Socioeconomic Status and Multiple Myeloma Among US Blacks and Whites

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ABSTRACT

Objectives. This study examined the relation between socioeconomic status (SES) and risk of multiple myeloma among Blacks and Whites in the United States.

Methods. This population-based case–control study included 573 cases (206 Blacks and 367 Whites) with new diagnoses of multiple myeloma identified between August 1, 1986, and April 30, 1989, and 2131 controls (967 Blacks and 1164 Whites) from 3 US geographic areas. Information on occupation, income, and education was obtained by personal interview.

Results. Inverse gradients in risk were associated with occupation-based SES, income, and education. Risks were significantly elevated for subjects in the lowest categories of occupation-based SES (odds ratio [OR] = 1.71, 95% confidence interval [CI] = 1.16, 2.53), education (OR = 1.36, 95% CI = 1.06, 1.75), and income (OR = 1.43, 95% CI = 1.05, 1.93). Occupation-based low SES accounted for 37% of multiple myeloma in Blacks and 17% in Whites, as well as 49% of the excess incidence in Blacks. Low education and low income accounted for 17% and 28% of the excess incidence in Blacks, respectively.

Conclusions. Our results indicate that the measured SES-related factors account for a substantial amount of the Black–White differential in multiple myeloma incidence. (*Am J Public Health.* 2000;90:1277–1281)

In the United States, the annual age-adjusted incidence and mortality rates for multiple myeloma rose sharply from the 1950s to the 1980s and then leveled off, with rates 2-fold higher among Blacks than among Whites.[1,2] The causes of multiple myeloma, the reasons for the rise and stabilization of incidence rates, and the reasons for the racial disparity in incidence are unclear.[3] Socioeconomic status (SES) as an independent risk factor has been evaluated in several epidemiologic studies of multiple myeloma, with an increased risk associated with higher SES in some studies[4–6] and with lower SES in others.[7–9]

We examined the effect of occupation-based SES, income, and education on multiple myeloma in a multicenter population-based case–control study among US Blacks and Whites. Our purpose was to assess the relation between SES and risk of multiple myeloma and to evaluate the effects of SES on the disparity in incidence rates between Blacks and Whites.

Methods

This study was one component of a large population-based case–control study of multiple myeloma and cancers of the esophagus, pancreas, and prostate. Cases for the study consisted of Black and White residents of Atlanta, Ga (DeKalb and Fulton counties); Detroit, Mich (Macomb, Oakland, and Wayne counties); and New Jersey (10 counties) residing in areas covered by population-based cancer registries. Eligible cases—those aged 30 to 79 years with multiple myeloma newly diagnosed between August 1, 1986, and April 30, 1989—were identified from pathology, hematology, outpatient, and tumor registry records. Because of the poor prognosis of multiple myeloma, a rapid reporting system was developed to identify and interview cases. The average interval between diagnosis and interview was 128 days. Among both Black and White eligible cases, approximately 7% were too ill to be interviewed, and 21% died before they could be interviewed.

Population controls were selected from the same geographic areas as the cases, proportional to the expected race, sex, and age distribution of the cases for the 4 cancer sites combined, based on incidence data from the 3 study areas. Controls younger than 65 years were selected by random-digit dialing; we used a 2-step selection process that involved identification of eligible households followed by selection of eligible controls (i.e., controls in the designated race–sex–age stratum).[10] Controls aged 65 to 79 years were randomly selected

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from rosters of Medicare recipients for each study area provided by the Health Care Financing Administration stratified by race, sex, and age.

Cases and controls were interviewed in person by trained interviewers. Detailed information was obtained on sociodemographic factors, medical history, use of alcohol and tobacco, dietary factors, and lifetime occupational history. Subjects also were asked to report their total income (including money received by their spouse) before taxes for the past calendar year, the number of persons supported by this income, and the highest level of schooling completed.

Information obtained on usual occupation title was coded according to the *Standard Occupational Classification Manual.*[11] A 3-level occupation-based SES score (high, medium, low) was assigned to each Standard Occupational Classification code. We decided to have 3 occupation-based SES levels to avoid a potential small numbers problem. The usual occupation was defined as the longest job held. The mean duration of the usual occupation was 24 years (25 years for Whites, 22 years for Blacks). This occupation-based SES indicator was created by one of us (M.D.) with information (average earnings and number of years of training required for that particular job) presented in the 1987 version of *CFKR Career Materials* (CFKR Career Materials, Meadow Vista, Calif). Our scoring system has not been validated directly, but SES-related associations that use this system have been observed in our previous studies.[12,13]

We used unconditional logistic regression to estimate odds ratios (ORs) and approximate 95% confidence intervals (CIs) for multiple myeloma, with adjustments for age (30–39, 40–49, 50–59, 60–69, ≥70); study area (Atlanta, Detroit, New Jersey); and, where appropriate, race and sex.[14] SES variables were entered in the models as dummy variables. Ordinal variables were used to test for trend. We used the EPICURE program for personal computers[15] to obtain odds ratios and 95% confidence intervals. To quantify the proportion of excess risk among Blacks that might be explained by low SES, we computed the population attributable risks and 2-sided 95% confidence intervals (adjusted for age, sex, and study area) by the methods of Bruzzi et al.[16] and Benichou and Gail,[17] respectively.

Of the 309 Black and 581 White cases identified for the study, interviews were successfully conducted with 206 (66.7%) Blacks (91 men and 115 women) and 367 (63.2%) Whites (193 men and 174 women). The response rate was 67% among both Blacks and Whites for the random-digit-dialed controls and 61% among Blacks and 57% among Whites for the Health Care Financing Administration controls. We conducted analyses with 967 Black controls (614 men and 353 women) and 1164 White controls (742 men and 422 women).

Results

As shown in Table 1, White subjects tended to have higher occupation-based SES, income, and education than did Blacks. In both races combined, the risks of multiple myeloma were associated with lower occupation-based SES (P_{trend}=.0005), income (P_{trend}=.009), and education (P_{trend}=.010) (Table 2). Risks were significantly elevated for subjects in the lowest categories of occupation-based SES (OR=1.71, 95% CI=1.16, 2.53), education (OR=1.36, 95% CI=1.06, 1.75), and income (OR=1.43, 95% CI=1.05, 1.93). The gradients in risk were similar for Blacks and Whites.

When we adjusted the occupation-based SES analysis by income and education, the association between low occupation-based SES

TABLE 1—Distribution of Cases and Controls by Sociodemographic Factors: Multicenter Population-Based Case–Control Study Among US Blacks and Whites, 1986–1989

	Blacks				Whites			
	Cases		Controls		Cases		Controls	
	n	%	n	%	n	%	n	%
Study site								
Atlanta	39	18.9	196	20.3	25	6.8	252	21.6
Detroit	89	43.2	420	43.4	167	45.5	443	38.1
New Jersey	78	37.9	351	36.3	175	47.7	469	40.3
Age, y								
30–39	9	4.4	26	2.7	3	0.8	27	2.3
40–49	22	10.7	101	10.4	19	5.2	150	12.9
50–59	41	19.9	242	25.0	74	20.2	332	28.5
60–69	79	38.3	309	32.0	136	37.1	344	29.6
≥70	55	26.7	289	29.9	135	36.8	311	26.7
Mean age, y	62.3		62.3		65.3		61.4	
Sex								
Male	91	44.2	614	63.5	193	52.6	742	63.7
Female	115	55.8	353	36.5	174	47.4	422	36.3
Occupation-based socioeconomic status								
High	4	1.9	41	4.2	34	9.3	188	16.2
Medium	53	25.7	314	32.5	158	43.1	569	48.9
Low	147	71.4	608	62.9	172	46.9	404	34.7
Missing	2	0.9	4	0.4	3	0.8	3	0.3
Education								
College	31	15.0	176	18.2	112	30.5	483	41.5
High school	52	25.2	247	25.5	116	31.6	370	31.8
0–11 y	123	59.7	544	56.3	137	37.3	302	25.9
Missing	0	...	0	...	2	0.5	9	0.8
Annual household income, $								
High (≥25 000)	34	16.5	220	22.8	122	33.2	556	47.8
Medium (10 000–24 999)	64	31.1	350	36.2	148	40.3	373	32.0
Low (<10 000)	91	44.2	330	34.1	53	14.4	119	10.2
Missing	17	8.2	67	6.9	44	12.1	116	10.0

TABLE 2—Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for Multiple Myeloma by Socioeconomic Status (SES) Indicators: Multicenter Population-Based Case–Control Study Among US Blacks and Whites, 1986–1989

	Blacks[a]			Whites[a]			Total[b]		
	Cases	Controls	OR (95% CI)	Cases	Controls	OR (95% CI)	Cases	Controls	OR (95% CI)
Occupation-based SES									
High	4	41	1.00	34	188	1.00	38	229	1.00
Medium	53	314	1.33 (0.45, 3.92)	158	569	1.21 (0.79, 1.85)	211	883	1.22 (0.83, 1.80)
Low	147	608	2.02 (0.70, 5.81)	172	404	1.57 (1.01, 2.42)	319	1012	1.71 (1.16, 2.53)
Test for trend			$P = .01$			$P = .03$			$P = .0005$
Education									
High (college)	31	176	1.00	112	483	1.00	143	659	1.00
Medium (high school)	52	247	1.09 (0.66, 1.79)	116	370	1.06 (0.77, 1.43)	168	617	1.11 (0.86, 1.44)
Low (0–11 y)	123	544	1.32 (0.84, 2.08)	137	302	1.35 (0.99, 1.83)	260	846	1.36 (1.06, 1.75)
Test for trend			$P = .19$			$P = .06$			$P = .009$
Annual household income, $									
High (≥25 000)	34	220	1.00	122	156	1.00	156	776	1.00
Medium (10 000–24 999)	64	350	1.12 (0.70, 1.81)	148	373	1.19 (0.88, 1.61)	212	723	1.24 (0.96, 1.61)
Low (<10 000)	91	330	1.48 (0.90, 2.42)	53	119	1.21 (0.80, 1.84)	144	449	1.43 (1.05, 1.93)
Test for trend			$P = .04$			$P = .23$			$P = .010$

[a]ORs are adjusted for age, sex, and study area.
[b]ORs are adjusted for age, race, sex, and study area.

TABLE 3—Estimated Percentage Attributable Risk (AR)[a] in 3 Geographic Areas,[b] by Socioeconomic Status (SES) Indicators: Multicenter Population-Based Case–Control Study Among US Blacks and Whites, 1986–1989

	Controls (% in Medium-SES Indicators)	% AR for Medium-SES Indicators (95% CI)	Controls (% in Low-SES Indicators)	% AR for Low-SES Indicators (95% CI)	Annual Age-Adjusted Incidence Rates of Multiple Myeloma: In the 3 Geographic Areas	In the Absence of Each Low-SES Indicator[c]	Explained by Each Low-SES Indicator[d]
Occupation-based SES							
Blacks	32.5	6.9 (–14.5, 28.3)	62.9	36.8 (–1.8, 75.5)	13.40/100 000	8.47/100 000	4.93/100 000
Whites	48.9	7.2 (–8.6, 22.9)	34.7	17.1 (3.2, 31.1)	5.15/100 000	4.27/100 000	0.88/100 000
Education							
Blacks	25.5	2.2 (–9.6, 14.1)	56.3	14.2 (–7.4, 35.7)	13.40/100 000	11.50/100 000	1.90/100 000
Whites	31.8	1.6 (–7.8, 11.0)	25.9	9.6 (0.0, 19.1)	5.15/100 000	4.66/100 000	0.49/100 000
Annual household income							
Blacks	36.2	5.9 (–8.8, 20.6)	34.1	18.8 (2.5, 35.0)	13.40/100 000	10.88/100 000	2.52/100 000
Whites	32.0	7.9 (–4.4, 20.2)	10.2	3.2 (–2.8, 9.6)	5.15/100 000	4.97/100 000	0.18/100 000

Note. CI = confidence interval.
[a]All ARs were adjusted for age, sex, and study area of residence and were based on odds ratios calculated relative to high SES.
[b]Atlanta, Ga (DeKalb and Fulton counties); Detroit, Mich (Macomb, Oakland, and Wayne counties); and New Jersey (10 counties).
[c]Estimated incidence rates if cases due to the lowest level of each SES indicator were eliminated.
[d]Estimated incidence rates attributable to lowest level of each SES indicator.

and multiple myeloma remained. The results for income were not altered when the number of people supported by that income was added to the logistic models. We examined the interaction for combined risk factors, such as low occupation-based SES, low income, and low education. Combined effects of these variables were not indicated. The odds ratio for income varied by sex. The odds ratio for the lowest category of income was elevated in women (OR = 2.03, 95% CI = 1.30, 3.16) but not in men (OR = 0.96, 95% CI = 0.61, 1.53) (data not shown).

Among Blacks, 37% of multiple myeloma occurrence was related to low occupation-based SES, compared with 17% among Whites (Table 3). This difference is partly the result of the higher odds ratio associated with low SES among Blacks than Whites (2.02 vs 1.57) and the higher proportion of low-SES subjects among Black than White controls (62.9% vs 34.7%). The total average annual age-adjusted incidence rates of multiple myeloma in the 3 study areas combined during the study period were 13.40 per 100 000 among Blacks and 5.15 per 100 000 among Whites, yielding an excess among Blacks of 8.25 cases per 100 000 per year. The proportion of the disease not explained by low SES was applied to the total rates to estimate incidence rates in the absence of low SES (8.47 per 100 000 among Blacks and 4.27 per 100 000 among Whites). The average annual age-adjusted incidence rates associated with low occupation-based SES were estimated to be 4.93 per 100 000 among Blacks and 0.88 per 100 000 among Whites, yielding an excess among Blacks of 4.05 per 100 000 per year. Low occupation-based SES thus may account for 49% of the excess occurrence among Blacks (i.e., 4.05 of the 8.25 per 100 000 difference in average annual age-adjusted incidence rates between Blacks and Whites).

When attributable risk calculations were done for education and income, our findings were as follows: among Blacks, 14% of multiple myeloma occurrence was related to low education, compared with 10% among Whites (low education accounting for 17% of the excess occurrence in Blacks); among Blacks,

19% of multiple myeloma occurrence was related to low income, compared with 3% among Whites (low income accounting for 28% of the excess occurrence in Blacks) (Table 3).

Discussion

In this population-based case–control study, we found elevated risks of multiple myeloma associated with lower occupation-based SES, education, and income among both Blacks and Whites. The overall odds ratio of 1.71 (95% CI=1.16, 2.53) in the lowest occupation-based SES category is similar in magnitude to the odds ratio of 1.63 (95% CI= 1.21, 2.19) reported in a recent US population-based case–control study[8] and the odds ratio of 2.8 (95% CI=1.6, 3.0) observed in an Italian hospital-based case–control study.[9] Among subjects with less than a high school education, the odds ratio of 1.36 (95% CI=1.06, 1.75) in our study is consistent with the risk (OR=1.5, 95% CI=1.0, 2.0) reported in a nested case–control study within the American Cancer Society cohort.[7] In contrast, earlier mortality-based studies of multiple myeloma found positive associations with social class, possibly because persons of higher social class had better access to diagnostic facilities,[4–6] whereas other studies have indicated no clear relation to social class.[18–22]

In our study, low occupation-based SES accounted for 37% of multiple myeloma in Blacks but only 17% in Whites because of the much higher percentage of Black (62.9%) than White (34.7%) controls in the low-SES category. In addition, the risk associated with low SES was somewhat greater among Blacks than Whites. If low SES, as a proxy for true risk factors, is causally related to the risk of multiple myeloma, then it may account for 49% of the excess incidence observed among Blacks. Attributable risk calculations, however, are affected by how SES is measured (e.g., income, education, occupation-based index) and what criteria are used to categorize the data for the selected SES measure.

Low social class may be a surrogate for a set of negative environmental characteristics, such as poor housing, dangerous jobs that may result in differential exposure to occupational carcinogens, unemployment, lack of access to medical care, stressful home or work environments, poor nutrition, and exposure to infectious agents.[23] The specific SES-related exposures that contribute to the higher incidence of multiple myeloma among US Blacks are unclear, but suspicion has centered on possible infectious agents and immunologic mechanisms.

Studies have suggested that Kaposi sarcoma–associated herpesvirus, also known as human herpesvirus 8, may be involved,[24] perhaps through infection of bone marrow dendritic cells and production of interleukin 6, a powerful stimulator of plasma cells and promoter of myeloma cell growth.[25,26] However, the association between multiple myeloma and Kaposi sarcoma–associated herpesvirus infection has not been confirmed.[27,28]

Monoclonal gammopathy of undetermined significance is a common precursor to multiple myeloma[29,30]; an infectious agent may promote the development of this monoclonal gammopathy or its progression to myeloma. In parallel with the racial differences in multiple myeloma, monoclonal gammopathy of undetermined significance appears to be more common in Blacks than in Whites[31,32] and less common in Japanese than in Whites,[33] suggesting that the origins of multiple myeloma could be clarified by studies of the etiology and natural history of monoclonal gammopathy of undetermined significance.

Another immunologic clue is provided by some studies indicating an excess risk of multiple myeloma among patients with autoimmune diseases and with certain conditions associated with chronic antigen stimulation, perhaps mediated by interleukin 6 production and stimulation of B-cell differentiation.[3] In our study population, an excess risk was observed among Blacks with a history of autoimmune disease but not among Whites.[34] An excess risk of multiple myeloma also has been suggested among patients with AIDS, although further work is needed to clarify this association.[35]

The role of lifestyle and other environmental factors associated with SES warrants further study, although no relation was found with tobacco or alcohol use in our study population.[36] Dietary and nutritional characteristics rarely have been studied as potential risk factors for multiple myeloma, although a relation to obesity has been suggested.[37] Occupational exposure to certain solvents and pesticides may play a role,[38–42] but the evidence is inconclusive.

Genetic determinants also may be involved, particularly in explaining the portion of the excess risk among Blacks that is unrelated to social factors. In both races combined, we found a significant 4-fold increased risk in subjects reporting a first-degree relative with multiple myeloma, in line with clinical surveys suggesting a familial tendency.[43] Although risks associated with a family history of hematolymphoproliferative (HLA) cancer were higher in Blacks than in Whites, the difference in odds ratios was not significant. Furthermore, in our study population, the risk of multiple myeloma was associated with the HLA-Cw2 antigen, which was related to 18% of the cases among Blacks and 11% of the cases among Whites but did not fully account for the higher incidence among Blacks.[44] Another genetic marker is suggested by a polymorphism of the poly(adenosine diphosphate [ADP]-ribose) polymerase gene, with an increased frequency of the B allele particularly evident among Blacks with monoclonal gammopathy of undetermined significance and with multiple myeloma.[45] The higher frequency of this B allele among Blacks (35%) than among Whites (14%) in the general population[46] suggests that it may contribute to the higher incidence of monoclonal gammopathy of undetermined significance and multiple myeloma among Blacks.

The strengths of our study include its population-based methodology, use of incident cases, relatively large numbers of Black and White subjects, in-person interviews of study subjects, and an occupation-based measure of SES supplemented by data on income and education. The main limitations are the relatively low response rates and our inability to ascertain and control for unknown confounding factors.

Of the 3 SES indicators, the occupation-based SES showed the strongest relation to multiple myeloma in both races, raising the possibility that work exposures may contribute to the risk associated with low SES. In our study, the low-SES jobs varied widely and included clerks, household workers, janitors, painters, waiters, nurses' aides, construction workers, machine operators, metal workers, production workers, assemblers, and truck drivers. Our preliminary analyses showed significantly increased risk of multiple myeloma for 2 of these low-SES jobs: nurses' aides (OR=2.62, 95% CI=1.36, 5.03) and metal and plastic processing machine operators (OR=3.82, 95% CI= 1.12, 12.99). When subjects with these occupations were removed from analysis (20 cases and 27 controls), the elevated risk of multiple myeloma for occupation-based low SES was not substantially altered.

We also considered potential biases in our study. The relation between low SES and poor prognosis[47] may have affected the distribution of interviewed vs eligible cases. However, any loss of low-SES cases would have underestimated the association of multiple myeloma risk with low SES. If this effect were differential by race, the estimates of attributable risk might have been influenced, but such an effect seems unlikely because the nonresponse rates due to death or serious illness were similar among Blacks (26.8%) and Whites (28.1%).

In summary, this case–control study indicated that the risk of multiple myeloma increases with decreasing SES, whether measured by occupation-based SES, income, or education, and it quantified for the first time the amount of the excess incidence in Blacks that may be attributable to SES. Further research, particularly in the area of molecular epidemiology, is needed to uncover the environmental and genetic determinants of multiple myeloma

and the reasons for the racial and socioeconomic differentials. □

Contributors

D. Baris analyzed the data and wrote the paper. This multicenter study was led by National Cancer Institute investigators L.M. Brown, D.T. Silverman, R. Hayes, R.N. Hoover, L.M. Pottern, and J.F. Fraumeni in collaboration with principal investigators from each center (Detroit: G.M. Swanson, A.G. Schwartz; Atlanta: J.M. Liff, R.S. Greenberg; New Jersey: J.B. Schoenberg). M. Dosemeci provided occupation-based socioeconomic status levels, and J. Lubin assisted with statistical analysis. All authors contributed significantly to the analysis and interpretation of the findings.

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