Racial, Ethnic, and Gender Variations in Cancer Risk: Considerations for Future Epidemiologic Research

Shelia Hoar Zahm and Joseph F. Fraumeni, Jr.

Division of Cancer Etiology, National Cancer Institute, Rockville, Maryland

There is no question that the risk of many cancers varies substantially by race, ethnic group, and gender. Although important clues to cancer etiology may come from investigating the differences in risk across subgroups of the population, epidemiologic research has often focused on white men. More descriptive and analytic studies are needed to identify and explain variations in risk among population subgroups. Especially important are studies to clarify the role of differential exposures, susceptibility, and diagnostic factors in cancer incidence, although differences in treatment may contribute to variations in cancer mortality. Improvements in classification of ethnicity, assessment of carcinogenic exposures in various subpopulations, and measures of host susceptibility states should augment future epidemiologic research designed to better understand mechanisms underlying the racial, ethnic, and gender differences in cancer risk. — Environ Health Perspect 103(Suppl 8):283–286 (1995)

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Introduction

Striking variations have been observed in the incidence and mortality rates for cancer, according to such factors as age, gender, race, ethnicity, time, socioeconomic class, marital status, and geographic location. Descriptive studies, by uncovering the patterns of cancers in populations, have yielded many clues to cancer etiology. The patterns are useful in monitoring variations that might point to new environmental hazards; in evaluating the effects of cancer prevention, screening, and treatment activities; and in predicting future trends that may help set priorities in cancer research. This paper discusses issues relevant to future epidemiologic research to clarify the effects of race, ethnicity, and gender on the risk of cancer.

Are the Variations Real and Meaningful?

Variations in cancer risk often are remarkable, even allowing for the fluctuation that might be expected as a result of chance and differences in diagnostic or reporting practices. Especially striking are variations by racial group. Incidence rates among African Americans are approximately 3 times higher for esophageal cancer, 2 times higher for multiple myeloma, liver cancer, cervical cancer, and stomach cancer, and 50% higher for cancers of the larynx, prostate, oral cavity and pharynx, pancreas, and lung than rates prevailing among whites (*1*). However, compared with whites, African Americans have a lower incidence of melanoma and cancers of the endometrium, thyroid, breast, bladder, leukemia, lymphoma, ovary, testis, and brain (*1*). Also well documented are gender differences in cancer risks, with higher rates generally among males, with some exceptions such as the predominance of cancers of the thyroid and gallbladder among females (*1*). Ethnicity has been less well studied in the United States, but is also known to affect cancer risk. For example, Hispanic subgroups, such as Cuban Americans, Puerto Ricans, and Hispanics from Central and South America, have cancer patterns that differ measurably from those of the white non-Hispanic population and from one another (*2–7*). Some data suggest, however, that greater efforts are needed to ensure precise designation of race and ethnicity and thus enhance research on ethnic differences in cancer risk (*8,9*).

Racial, ethnic, and gender differences in cancer risk often vary by such factors as age, geographic location, and tumor histologic type. For example, white women are at greater risk than African American women for postmenopausal breast cancer, while premenopausal breast cancer is more common among African Americans than whites (*10*). Mortality rates for oral cancer among women (but not men) are excessive in southern rural parts of the country, primarily because of the longstanding practice of snuff dipping in this region (*11*). Esophageal cancers, the majority of which are squamous cell carcinomas, occur at much higher rates among African Americans than whites, whereas whites have a higher risk of adenocarcinomas of the esophagus (*12*). Thus, the influence of age, geography, and other demographic variables should be considered, as well as cell type, to fully evaluate variations in cancer risk among special populations.

For some exposures and cancers, extensive epidemiologic research has been conducted based on cancer variations according to race, ethnic background, and gender. Examples include research on lung cancer and tobacco use by race (*13*), the effects of hormonal medications used by women (*14*), and cancer and diet among different ethnic groups (*15*). For many exposures, however, research has focused primarily on white men. For example, although women and minorities account for 46 and 18%, respectively, of the U.S. work force, a survey of 1233 published epidemiologic studies on occupational cancer found only a few studies with in-depth analyses of risks among white women (7%), nonwhite women (1%), or nonwhite men (3%) (*16*). There were legitimate reasons for excluding women and minorities from some occupational studies,

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Address correspondence to Dr. Shelia Hoar Zahm, Epidemiology and Biostatistics Program, Division of Cancer Etiology, National Cancer Institute, EPN 418, Rockville, MD 20892-7364. Telephone: (301) 496-9093. Fax: (301) 402-1819. E-mail: zahms@epndce.nci.nih.gov

Abbreviations used: NAT2, *N*-acetyltransferase; SEER, Surveillance, Epidemiology and End Results; SMR, standardized mortality ratio.

Table 1. Cancer mortality among male coke plant workers in the steel industry.

			All cancers								Lung cancer							
	% Employed		White				Nonwhite				White				Nonwhite			
Work area	White	Nonwhite	Obs	Exp	SMR	(95% CI)	Obs	Exp	SMR	(95% CI)	Obs	Exp	SMR	(95% CI)	Obs	Exp	SMR	(95% CI)
Coke plant[a]	100	100	199	193.8	103	(89–118)	121	106.7	113	(94–136)	11	12.2	90	(45–161)	26	9.6	271	(177–397)
Coke oven	42	91	80	80.2	100	(79–124)	104	92.9	112	(91–136)	8	4.7	170	(73–335)	23	7.6	303	(192–454)
Side	26	71	45	47.2	95	(70–128)	61	67.3	91	(69–116)	5	2.7	185	(60–432)	5	5.4	93	(30–216)
Partial topside	13	1	22	25.1	88	(55–133)	2	1.2	–	–	2	1.6	–	–	0	0.1	–	–
Full topside	3	19	13	7.8	167	(89–285)	41	24.4	168	(121–228)	1	0.5	–	–	18	2.2	818	(485–1293)
Nonoven[a]	58	9	119	113.6	105	(87–125)	17	13.8	123	(72–197)	3	7.3	41	(8–120)	1	1.2	–	–

Abbrevations: Obs, observed; Exp, expected; SMR, standardized mortality ratio; 95% CI, 95% confidence interval. [a]Coke plant and nonoven data for lung cancer includes other respiratory system cancers. From Lloyd (*22*).

such as the small number of subjects or events, lower exposures, and difficulty in tracing. There were some studies, however, in which the data for women and minorities could have been given more consideration. Important clues to cancer etiology may have been missed because of lack of attention to groups other than white men. More descriptive and analytic studies are needed in epidemiologic research on cancer to generate and test etiologic hypotheses based on variations in cancer risk by racial, gender, and ethnic subgroups.

What Accounts for the Variations in Risk?

Racial, ethnic, and gender variations in cancer risk may reflect differences in environmental exposure or differences in susceptibility and biologic response, although some patterns may be due at least partly to differences in diagnostic, reporting, and (in terms of mortality) treatment practices. Many of the racial and ethnic patterns of cancer risk are due at least partly to socioeconomic differences (*17–20*).

Exposures

Some of the strongest evidence that environmental exposures affect cancer patterns across racial and ethnic groups comes from observations of large international variations in risk and substantial changes in risk among populations that have migrated from one area of the world to another. This was recently demonstrated in a study of Asian American migrants whose breast cancer risk increased over several generations to attain rates prevailing in the U.S. white population (*21*).

Differences in cancer risk among special populations may reflect variations in the intensity or duration of specific carcinogenic exposures. Among workers at a coke plant within the steel industry, there was almost a 3-fold increase in lung cancer mortality among nonwhites (mainly African Americans), whereas white workers had a slightly lower than expected rate (Table 1) (*22*). Examination of work areas within the coke plant revealed that 91% of nonwhites but only 42% of whites were employed near the coke oven, particularly at the top side of the ovens, where there was heavy exposure to the by-products of coal carbonization. The elevated risk of lung cancer among nonwhites in the coke plant thus simply reflects their work assignments and exposures rather than a racial differential in susceptibility. This study illustrates the importance of detailed job titles and exposure assessment in identifying racial differences in cancer risk as well as the risk factors involved. If only broad occupational categories were used, an important occupational carcinogen would have been missed.

Tobacco smoking explains some of the gender and racial differences in cancer risk. For example, a large case–control study of oral and pharyngeal cancer in the United States showed that variation in the prevalence of use and the risks associated with tobacco and alcohol accounts for much of the higher incidence among men than women and among African Americans than whites (*23*). Detailed information on the amounts of tobacco and alcohol consumed helped clarify their role in the etiology of these tumors. Similarly, the role of diet in the etiology of cancer may be better understood if differences in portion sizes consumed by men and women are considered along with qualitative patterns in dietary and nutritional habits. In general, refinements in the assessment of lifestyle and other environmental exposures will allow more accurate comparison of risks across subgroups of the population.

Biologic Response

On the other hand, there is evidence that subgroups of the population may experience different cancer risks, even when types and levels of various exposures are precisely defined. In a recent study of esophageal cancer, Brown et al. (*24*) found that after detailed controlling for amount of smoking and alcohol consumption, the odds ratios associated with these exposures were substantially greater among African Americans than whites (Table 2). At each exposure level, African Americans had estimated annual incidence rates about 3 to 9 times higher than those for whites. Although the racial differences in risk may be due to differences in the types of alcohol consumed or to dietary and other exposures that

Table 2. Risk of squamous cell esophageal cancer in white and African American men by cigarette smoking and alcohol consumption.[a]

		Odd ratios 95% Confidence intervals[a]		Incidence rates, per 100,000 person-years	
Cigarette smoking	Drinks per week	White	African American	White	African American
Light	0–7	1.0	1.0	0.5	1.4
Light	8–14	1.8 (0.5–6.1)	5.7 (2.0–15.8)	0.9	8.0
Light	15–35	4.6 (1.7–12.8)	10.6 (4.1–27.2)	2.3	14.8
Light	36–84	19.7 (7.2–53.4)	39.5 (14.5–107.8)	9.8	55.3
Light	≥85	29.0 (7.2–116.5)	31.0 (9.8–98.5)	14.5	43.4
Heavy	0–7	3.3 (1.0–10.8)	4.5 (1.4–14.6)	1.6	6.3
Heavy	8–14	8.7 (2.4–32.4)	14.2 (4.1–49.1)	4.4	19.9
Heavy	15–35	22.1 (7.8–62.3)	36.8 (13.9–97.2)	11.0	51.5
Heavy	36–84	28.5 (10.1–80.2)	42.1 (15.8–112.6)	14.2	58.9
Heavy	≥85	35.4 (10.0–125.5)	149.2 (39.2–567.4)	17.7	208.9

[a]Odds ratios (95% confidence intervals) are adjusted for age, geographic area, and income. From Brown (*24*).

interact with alcohol or tobacco, the findings raise the possibility of racial disparity in a susceptibility factor that remains to be identified. The esophageal cancer study is part of a large National Cancer Institute multicancer case–control study designed to shed light on the racial differences in rates of multiple myeloma and cancers of the prostate, esophagus, and pancreas, which occur at higher rates among African Americans than among whites.

There also is some suggestion that lung cancer risk may vary by gender (*25,26*) and ethnicity (*27*) even after controlling for level of smoking. Other risk factors, such as diet or inborn susceptibility, may be responsible. The high rates of thyroid and gallbladder cancers among women have led to studies suggesting the importance of reproductive and hormonal factors in the development of these tumors (*28,29*).

In a search for susceptibility states that may vary demographically, recent research has centered on pharmacogenetic studies; notably, the possible role of the P450 family of genes, whose polymorphic expression may affect the metabolism of carcinogens, either by activation or detoxification. Table 3 presents some genetic polymorphisms that vary by race and affect the metabolism of occupational carcinogens. For example, the *CYP1A1* gene that influences metabolism of polycyclic aromatic hydrocarbons shows polymorphic variation among whites, Asians, and African Americans (*30–33*). There is also a different pattern of mutations among Asians and Caucasians for *N*-acetyltransferase (NAT2), which plays a role in the metabolism of aromatic amines that are bladder carcinogens (*34,35*). Studies conducted in Japan, China, Italy, and Arkansas have shown racial differences in the activity of *CYP1A2*, which is involved in the metabolic activation of aromatic amines, heterocyclic amines, and other carcinogens (*36,37*). Future studies on head and neck cancers might focus on *CYP2E1*, since it is inducible by ethanol and also participates in the activation of certain nitrosamines.

Table 3. Examples of genetic polymorphisms of metabolic enzyme activity that vary by race.

Gene	Occupational carcinogen
CYP1A1	Polycyclic aromatic hydrocarbons
CYP2A1	Aromatic amines
CYP2D6	Polycyclic aromatic hydrocarbons
CYP2E1	Benzene, butadiene
NAT2	Aromatic amines

Diagnosis and Treatment

For cancer incidence and to a greater extent mortality, some differentials in risk may reflect access to health care. Cancer screening, stage at diagnosis, and treatment practices vary across subgroups of the population and affect rates of cancer detection and survival (*38–44*). For example, African American and Hispanic women are less likely to undergo mammography than non-Hispanic white women (*38,40,41*). Minorities are more likely than white women to be diagnosed with advanced stages of the disease (*41,44–46*). Accounting for socioeconomic factors lessens but does not eliminate the observed racial and ethnic differences in stage at diagnosis and survival (*45–47*). These factors should be kept in mind when drawing etiologic inferences from the demographic patterns in cancer incidence and mortality.

Recommendations

In future epidemiologic research on avoidable causes of cancer, it is important that we not overlook opportunities to evaluate cancer risk among women and minorities. Immediate attention should be given to data already collected but not yet analyzed. The National Cancer Institute's program to fund small grants for further analyses of existing data sets should help fill the gaps in our understanding of the distribution of cancer among various subgroups of the population and the causes of the differential risks. In addition, all new data resources should include, whenever possible, a diversity of population subgroups. At the National Cancer Institute, the Surveillance, Epidemiology and End Results (SEER) Program of population-based cancer registries recently expanded its coverage of minorities by adding registries from Los Angeles and northern California, where large populations of Hispanics and Asian Americans are located (*48*).

Suggestions are occasionally made to remove information on race from data systems to avoid discrimination. In fact, inclusion of race allows monitoring to detect disparities that point to avoidable hazards and medical inequities. In addition, we need to improve exposure assessment, with special attention to potential differences among women and minorities. Specialized methods may be needed to assess lifestyle and other environmental exposures across subgroups of the population. Finally, biological markers of exposure and susceptibility states should be incorporated into epidemiologic studies whenever possible, remembering that normal values for white men may not be applicable to other segments of the population. As has happened in the past, future research that encompasses women and minorities may yield new insights into carcinogenesis that should eventually enhance the prospects for cancer prevention in the entire population.

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