THE COLOR OF CANCER

Disease and the Measure of Race in the United States from the 1920s to the 1990s

by Leyla Mei

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Date	David Nasaw Chair of Examining Committee
Date	Joshua Freeman Executive Officer
	Gerald Markowitz
	Gerald Oppenheimer
	Mae M. Ngai
	Vijay Prashad
	Supervisory Committee

THE CITY UNIVERSITY OF NEW YORK

ABSTRACT

The Color of Cancer:
Disease and the Measure of Race in the United States from the 1920s to the 1990s

by Leyla Mei

Advisor: Professor David Nasaw

This dissertation analyzes the ways in which cancer researchers in the United States understood, measured, and defined race between 1920 and the turn of the twenty-first century. Shifting interpretations of its relationship to carcinogenesis forced doctors to confront multiple definitions of race as they struggled to untangle the medical significance of various racial traits and explain epidemiologic patterns. At different times, race stood for nationality, culture, skin tone, physicality, genetics, socioeconomics, and biochemistry.

The measurement of race moved from a bodily notion early in the century, to a postwar assessment which increasingly incorporated external characteristics, to an internal schema in the 1990s. In the 1920s, cancer's designation as a disease of civilization structured the search for etiology in ways that affected groupings of whites and nonwhites, as researchers compiling statistics on cancer rates in different populations rationalized and naturalized racial categories. Case studies of four cancers with racial associations examine how disease identities resulted from patterns of incidence, and in turn shaped research agendas and consolidated racial and ethnic borders. Skin cancer's stark racial disparities were poorly understood until the discovery of the carcinogenic nature of ultraviolet light, prompting researchers to classify subjects according to changing combinations of race, ethnicity, and skin color

in their search for its causes. Varying associations of risk, race, and behavior marked studies into the etiology of cervical cancer; because of the disease's links with economic status and the correlation between race and class, race became a risk factor in that it appeared to determine the sexual practices which could affect incidence. Nasopharyngeal carcinoma's characterization as a disease of ethnic Chinese led scientists to pinpoint the specific traits which defined an individual as such, a list guided by racial ideology, stereotypically Chinese habits such as opium smoking, and a disregard for regional variations in Chinese culture. Finally, an examination of how prostate cancer became a "black" disease in the postwar U.S. reveals how new diagnostic technologies promote the illusion that race has an inherent biological basis, unsettling the prevailing social constructivist framework of race in ways that will have profound effects during the twenty-first century.

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Prologue

Racial Science in History

Throughout American history, scientists have generated knowledge about race. Their ideas have been used by diverse groups of people to explain, justify, and rationalize differences between human populations. At the same time, popular understandings of race have affected conversations among scientists, influencing approaches to its scholarly study. The flow of ideas about race within American society has necessarily been complex; culture, science, and politics have been and remain interwoven. Yet scientific knowledge of race—and, in particular, the discourse of racial science—has had a major impact on the ways in which Americans have assigned importance to physical and physiological differences between groups. The success, or lack thereof, of scientists in addressing the sources of such differences has shaped the meanings that people assign to them, often with grave social and political consequences.

During the eighteenth century, British and American scientists generally agreed that all humans, despite numerous physical differences in appearance, belonged to the same species. This theory, called monogenism, derived from both Christian theology and Enlightenment thought. The Enlightenment's focus on natural rights, egalitarianism, and universal reason supplied an optimistic faith in the potential for achievement of nonwhite races, given suitable environmental conditions and educational opportunities. Religious teachings emphasized the descent of humankind from a single set of ancestors, Adam and Eve, and the subsequent equality

of men under God.[1] These beliefs, while obviously insufficient to bring an end to the practice of enslaving Africans and Native Americans, nonetheless indicated that racial thinkers at the time considered all humans, as members of the group *Homo sapiens*, to have a singular origin.

Between the turn of the nineteenth century and its midpoint, the idea of monogenesis gradually gave way to the theory of polygenesis, which held that members of different races constituted separate species. Polygenists believed that variations between groups of people were innate, and therefore, permanent and unchangeable. One's environment had little effect on one's development; an Englishman in the tropics could no sooner turn African in skin color, temperament, or aptitude than a dark-skinned, nonwhite individual could become a member of the white race. Inborn differences, moreover, could be measured and quantified, enabling polygenists to classify people into racial types. The size and shape of one's skull, in particular, appeared to be good markers of race, because they were considered not only distinctive and immutable, but also excellent indicators of intelligence.[2]

Polygenism became an accepted scientific viewpoint in the United States in the mid-1800s through the work of a number of men whose scholarly credentials lent

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[1] On Christian theology and monogenism, see Nancy Stepan, *The Idea of Race in Science: Great Britain, 1800-1960* (Hamden, CT: Archon Books, 1982), 1-2. On the influence of Enlightenment thought on the theory of monogenism, see Thomas F. Gossett, *Race: The History of an Idea in America* (New York: Oxford University Press, 1963, 1997), Chapter III.

[2] On the differences between monogenism and polygenism, see George M. Fredrickson, *The Black Image in the White Mind: The Debate on Afro-American Character and Destiny, 1817-1914* (Hanover, NH: Wesleyan University Press, 1971, 1987), 71-73; Stepan, 2-4. A useful textbook on the history of racial science is John P. Jackson, Jr., and Nadine M. Weidman, *Race, Racism, and Science: Social Impact and Interaction* (Santa Barbara, CA, Denver, CO, and Oxford, England: ABC-CLIO, 2004); Chapter 2 describes polygenism in the United States in the early- to mid-nineteenth century.

academic respectability to the field. The earliest of them, Samuel George Morton, was a well-known physician and student of natural history in Philadelphia, where he taught at the Pennsylvania Medical College. An early interest in paleontology and geology gave way to a fascination with craniology. Through his relationships with physicians and other scientists around the world, he amassed a collection of several hundred human skulls; nicknamed the "American Golgotha," it was the largest one in the world at the time.[3]

Morton's vast collection enabled him to compare cranial measurements among blacks, whites, and indigenous peoples from throughout the Americas. In *Crania Americana*, the 1839 volume detailing his research, he described racial differences in temperament and ability. American Indians were "averse to cultivation," "slow in acquiring knowledge," and "fond of war," while blacks were "joyous, flexible, and indolent," their small skulls marking them as the "lowest grade" among the races.[4] By the time of his sudden death in 1851, Morton, writes Thomas F. Gossett, "had convinced most of the scientific community…that the theory of separate species among human races was the most logical explanation for the differences between races."[5] The mantle of polygenesis subsequently passed to two of his followers: Josiah Clark Nott, a physician, surgeon, and native of South Carolina; and an English-born Egyptologist named George Robin Gliddon. While Gliddon contended that neither climate nor environment was sufficient to produce the

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[3] Jackson, Jr. and Weidman, 45; Gossett, 58.

[4] For descriptions of Morton's findings in *Crania Americana*, see Jackson, Jr. and Weidman, 47; Gossett, 59.

[5] Gossett, 63.

stark racial differences between blacks and whites, Nott seized upon the biological dictum that individuals of two separate species could not produce fertile offspring to argue that mulattoes were inherently weaker and less fertile than adults of either race. The pair collaborated on an eight-hundred page tome, entitled *Types of Mankind*, in 1854. The book, which brought polygenist ideas to a readership outside of academia, proved immensely popular. As Gossett notes, the first printing sold out immediately, "[e]ven at the price of $7.50," and went through at least nine editions before the turn of the twentieth century.[6]

In the 1840s and 1850s, educated Americans in both the South and the North were beginning to look to science for evidence of the biological status of the races, hoping that it could settle definitively the persistent issue of inferiority versus equality. Given its scientific and popular standing at the time, polygenism might well have provided proslavery forces with a powerful rationalization for the peculiar institution. Most slaveholders, however, were reluctant to embrace the theory, finding that it conflicted with the monogenist teachings of the Bible. No less a slavery apologist than George Fitzhugh accepted, at least in the 1850s, that blacks and whites constituted a single species.[7] Nevertheless, polygenist thinking did serve to buttress the proslavery position; Nott and Gliddon both supported slavery, the former a fervent believer in the inherent inadequacy of blacks. But disagreement over whether blacks and whites had separate origins or a single one was ultimately of less

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[6] Gossett, 64-65. See also Fredrickson, 77-82.

[7] Gossett, 66. Fredrickson writes that Fitzhugh eventually capitulated to a polygenist position, writing in an 1861 review of John H. Van Evrie's *Negroes and Negro "Slavery"* of "demonstrative reasoning, demonstrative proof, that the negro is of a different species, physically, from the white man." Fredrickson, 69.

consequence to justifying the institution of slavery than the primary, unifying notion that African Americans were inferior to whites.[8]

The turn from monogenism to polygenism in the first half of the nineteenth century marked a fundamental shift in how scientists thought about the place of humans in the world. Monogenism emphasized man as a social and spiritual being governed primarily by social laws; race, a product of environment and civilization, was changeable and independent from nature. Polygenism, in contrast, interpellated man as a biological entity guided by scientific principles, including those which established fixed biological types and essences. No longer did culture determine race; instead, race produced culture.[9] The shift from monogenism to polygenism occurred within the context of increasing European nationalism, colonialism, and industrialization, all of which magnified the geographic and cultural expanse between white and nonwhite peoples. At the same time, science itself was becoming more empirical and quantitative—indeed, more "scientific." New fields arose; these included comparative anatomy and physiology, which emphasized systematic analysis of racial data. The word "biology" was itself introduced into the scientific lexicon in 1802, suggesting a new way to approach the study of living organisms.[10] In the United States, the American school of anthropology, based on the polygenist

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[8] Gossett and Fredrickson differ on the extent to which Southerners embraced polygenism as a defense of slavery. Their disagreement is based on conflicting positions on William Stanton's *The Leopard's Spots: Scientific Attitudes Toward Race in America, 1815-1859* (Chicago: University of Chicago Press, 1960). While Gossett accepts Stanton's contention that the doctrine had little effect on the proslavery defense, Fredrickson argues that the polygenist position—particularly Nott's stance on racial intermixing and his anti-black views—were "basic" to it. See Gossett, 66; Fredrickson, 76-82.

[9] Stepan, 3-4.

[10] The argument about why the shift from monogenism to polygenism occurred when it did, as well as the descriptions of changes in scientific methodology and lexicon, is Nancy Stepan's. See Stepan, 4-5.

work of Morton, Nott, and Gliddon, became the site where scientists studied race; the discipline centered on physical anthropology, and especially racial typology.[11]

The debate over single versus multiple origins was finally settled with the 1859 publication of Charles Darwin's *On the Origin of Species*. Darwin showed that species were not fixed and static, created by God in a specific, unchanging form, but were dynamic and constantly evolving through a process of adaptation and natural selection. Organisms survived because they were better suited to their environment, and passed these adaptations on to their offspring. Species were interconnected, with more advanced ones developing from those less complicated. Although *On the Origin of Species* did not directly address human evolution, Darwin wrote that all earthly organisms, both living and extinct, had evolved from the same primordial life form. By the time he published *The Descent of Man* in 1871, which did explicitly examine humans and the traits that made them distinct from animals, his theory of evolution had become widely accepted among scientists.[12]

Although Darwin's work ostensibly supported monogenism, it was far from incompatible with polygenist thinking. In fact, scholars in both the natural and the social sciences drew on his ideas into the twentieth century to justify racial hierarchy and argue for the innate inferiority of nonwhites. The polygenist emphasis on racial typology persisted, as did the belief in biology as the determinant of culture.

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[11] George W. Stocking, Jr. refers to these scientists as founders of the "American School of Anthropology," while Fredrickson calls it the "American school of ethnology." George W. Stocking, Jr., *Race, Culture, and Evolution: Essays in the History of Anthropology* (Chicago: University of Chicago Press, 1968, 1982), 39; Fredrickson, 74. See also Stocking, Jr., Chapter 2, for a discussion of the shift in French anthropology from monogenism to polygenism in the first half of the nineteenth century.

[12] Jackson, Jr. and Weidman, 61-67; Stepan, 47-49.

Biologist Alfred Wallace, whose writings on human evolution predated Darwin's, contended that man's intellectual capabilities rendered natural selection irrelevant, for they enabled him to react to changing environmental conditions by utilizing technology, rather than through the forced adaptations which characterized the rest of the animal world. The differentiation of people into races with recognizable physical traits, therefore, must have occurred so far back in time that the races were, by the nineteenth century, largely distinct.[13] Other remnants of polygenism surfaced in the work of Frederick L. Hoffman, a statistician at the Prudential Life Insurance Company, who would become an influential writer on the racial distribution of cancer. In his 1896 *Race Traits and Tendencies of the American Negro*, he maintained that the biracial children of black-white unions were inferior to both parents, both physically and morally. He claimed that African Americans, who had an innate tendency toward poverty, immorality, and crime, were headed toward certain extinction unless they could pull themselves together as a race, without assistance from whites.[14]

As a social Darwinist, Hoffman applied Darwin's model of evolutionary change to human races. Social Darwinists, notably Herbert Spencer, an English-born sociologist, believed that the races were engaged in a struggle for survival; the fittest would flourish, while the weaker ones would remain mired in poverty and disease. It was a positive process in that it eliminated races which lacked adaptive abilities. To Spencer, evolution was linear and hierarchical. Highly evolved races with the most

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[13] Stepan, 85-87; Jackson, Jr. and Weidman, 67-68; Matthew Frye Jacobson, *Barbarian Virtues: The United States Encounters Foreign Peoples At Home and Abroad, 1876-1917* (New York: Hill and Wang, 2000), 144-145.

[14] Stocking, Jr., 52-53; Gossett, 281-282; Fredrickson, 249-251.

advanced civilizations could be found at the top, while those further down the hierarchy were less developed, both culturally and biologically. He believed that members of the higher races, whose ancestors had made extensive use of their mental faculties, inherited a larger brain capacity than lower races, according to a Lamarckian process through which acquired characteristics could be inherited by future generations. Spencer compared the intellectual abilities of primitive races with those of children, finding in them a "vanity about clothes and small achievements, but little sense of justice."[15]

Although social Darwinists such as Spencer, a sociologist, and Hoffman, a statistician, pioneered the application of the theory of evolution to the study of human populations, the domain of racial science at the end of the nineteenth century was occupied largely by anthropologists, many of whom continued to promote anthropometry as a way of measuring and codifying racial types. Indeed, the practice of physical anthropology reached its peak around this time.[16] Furthermore, the "American school" of Gliddon, Nott, and Morton had faded, superseded by a focus on ethnology, or the linguistic, cultural, and archaeological aspects of human societies. The field of anthropology was in a state of growth and professionalization, aided by such figures as Daniel G. Brinton, an ethnologist with academic posts at the Academy of Natural Sciences in Philadelphia and the University of Pennsylvania, who is credited with its transformation from "a romantic pastime to an academic

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[15] Gossett, 149. See also Gossett, Chapter VII, on race and social Darwinism, and Jackson, Jr. and Weidman, 76-84, on Spencer's theories.

[16] Elazar Barkan, *The Retreat of Scientific Racism: Changing Concepts of Race in Britain and the United States Between the World Wars* (Cambridge: Cambridge University Press, 1992), 4.

discipline."[17] Institutions including the University of California established departments of anthropology, while others, such as Harvard University, expanded their graduate divisions or established connections with museums. New York, California, Washington, DC, and Chicago became major regional centers of a brand of academic anthropology which favored associations with museums of natural history.[18]

The ideas about race which were circulating within anthropology and the natural sciences at the time reached American consumers through a number of outlets. World's fairs interpreted ethnography for an eager public. The 1893 World's Columbian Exposition in Chicago famously contrasted the White City, a celebration of Euro-American scientific, artistic, and technological achievement, with the ethnological exhibits of the Midway Plaisance, where races were displayed, in their native habitats, in a procession from least to most civilized.[19] Popular magazines, including *The Atlantic Monthly* and *Harper's Weekly*, as well as late nineteenth-century travelogues by writers such as Charles Dudley Warner and Richard Harding Davis, offered accounts of "primitive" and "backward" races which drew on notions

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[17] Lee D. Baker, *From Savage to Negro: Anthropology and the Construction of Race, 1896-1954* (Berkeley, CA: University of California Press, 1998), 33. Stocking, Jr. offers a somewhat different assessment of Brinton's role in the growth of the field of anthropology. "Brinton's prestige in his own lifetime," he writes, "was considerable, but his intellectual influence was personal rather than institutional." He "trained no professional anthropologists, and his organizational ties were to groups like the American Philosophical Society, in which anthropology was only one of a number of scholarly interests pursued by private individuals." Stocking, Jr., 277-278.

[18] For a discussion of the transformation and institutionalization of American anthropology at this time, see Stocking, Jr., 277-283.

[19] The 1893 World's Fair has been a favorite site of analysis of scholars examining attitudes toward race in the late nineteenth century. Robert Rydell's *All the World's a Fair: Visions of Empire at American International Expositions, 1876-1916* (Chicago: University of Chicago Press, 1987) remains the standard work on the subject. Baker's book also contains a description of the fair, focusing on uses of anthropology there. Baker, 55-61.

of post-Darwinian evolutionism. So persistent were the racial themes of savagery and civilization in popular writings that they eventually grew to influence the impressions of later travelers to such regions.[20]

By the beginning of the twentieth century, the idea of a hierarchy based on a foundation of skin color and civilization was firmly entrenched within American attitudes toward race. While anthropology and its subfield, ethnology, were the main academic disciplines shaping popular understandings of race, a new science of eugenics was emerging, the followers of which would promote its doctrines within law and public policy for the next half-century. The word "eugenics" was coined by Francis Galton, an English statistician, naturalist, and cousin of Charles Darwin. His discovery of the work of Gregor Mendel, an Austrian monk whose experiments with pea plants in the 1860s had uncovered the basic principles of heredity, prompted him to combine an interest in the mental abilities of different races with the study of human heredity. In *Hereditary Genius* and *Natural Inheritance*, his books published in 1869 and 1889, respectively, he attempted to outline the heritability of traits such as intellectual ability, artistic inclination, tendency toward disease, and alcoholism.[21] The importance of eugenics lay in the ways in which it connected race to hereditarianism and genetics.[22] Eugenicists believed that mental, moral, and physical differences between races were hereditary; hence, the human race could be improved through a program of selective breeding to concentrate positive traits while weeding

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[20] Jacobson, 141. See also Chapter 3 for a lengthy discussion of travelogues, "ghetto sketches," and "fictions of the foreigner."

[21] Daniel J. Kevles, *In the Name of Eugenics: Genetics and the Uses of Human Heredity* (Cambridge, MA: Harvard University Press, 1985, 1995), 3-19; Jacobson, 154-155.

[22] Stepan, 112.

out ones that were detrimental to society. This translated, predictably, to an emphasis on Anglo-Saxon superiority at the expense of other, less fortunate groups.

In the United States, influential figures in the eugenics movement included Madison Grant, Charles B. Davenport, and Harry Laughlin. A New York conservationist, Grant was involved in the creation of the Bronx Zoo and served as an officer of the American Museum of Natural History. In his 1916 volume, *The Passing of the Great Race*, he wrote of the threat to the old-stock "Nordic" races from inferior "Alpine" and "Mediterranean" peoples who were thronging American shores.[23] Davenport, a biologist by training, resigned from his post at the University of Chicago in 1904 to found an institute for genetic research at Cold Spring Harbor, Long Island. He thereafter established the Eugenics Record Office, which employed a team of field workers to crisscross the country and compile data from families with "hereditary" conditions such as feeblemindedness and criminality.[24]

American eugenicists were less concerned with African Americans, whose inferiority had long been established, than with the Eastern and Southern Europeans who had been arriving in the U.S. in record numbers since the late nineteenth century. These new immigrants brought with them undesirable moral and mental qualities, all the more dangerous because of what appeared to be their exceptionally high birthrates. Not only would they dilute the American germ plasm, but they threatened to replace superior genes with inferior ones. Examination of immigrants at Ellis Island and other ports of entry aimed to identify and exclude the physically and

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[23] Jacobson, 160-162; Jackson, Jr. and Weidman, 111.

[24] Jacobson, 157-160. See also John Higham, *Strangers in the Land: Patterns of American Nativism, 1860-1925* (New Brunswick, NJ: Rutgers University Press, 1955, 1988, 1992), 151.

mentally unfit, while tests of young men inducted into the army during World War I revealed high levels of feeblemindedness among recruits, particularly those who traced their ancestry to countries including Poland, Italy, Russia, Greece, and Turkey.[25]

During the first few decades of the twentieth century, the study of eugenics found supporters among a wide range of people, including academics, physicians, and legislators. When Congress began to investigate the issue of immigration restriction, Harry Laughlin, a eugenicist who directed Davenport's Eugenics Record Office and favored sterilization, testified before the House Committee on Immigration as a scientific expert, supplying lawmakers with statistics which supposedly verified the degeneracy, social inadequacy, and racial inferiority of the new immigrants. In 1924, Congress enacted the Johnson-Reed Immigration Act, designed to sharply restrict the influx of undesirable racial types, while increasing the percentage of newcomers from Northern and Western Europe. The legislation probably marked the peak of the political influence of eugenicists in the U.S.[26] In Europe, the American example provided a model, and the field of eugenics supplied a scientific rationalization, for the Nazis, who carried the practice to grotesque new heights during the 1930s and 1940s, resulting in forced sterilizations, mass displacements, and the deaths of millions.

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[25] Jacobson, 168-169. See also Kevles, 80-81. On medical examination of immigrants, see Amy L. Fairchild, *Science at the Borders: Immigrant Medical Inspection and the Shaping of the Modern Industrial Labor Force* (Baltimore: Johns Hopkins University Press, 2003); Nayan Shah, *Contagious Divides: Epidemics and Race in San Francisco's Chinatown* (Berkeley, CA: University of California Press, 2001).

[26] Higham, 314; Mae M. Ngai, *Impossible Subjects: Illegal Aliens and the Making of Modern America* (Princeton, NJ: Princeton University Press, 2004), 24. The first chapter of Ngai's book examines the Johnson-Reed Act of 1924.

While the Nazi project led to a thorough discrediting of eugenics by the end of World War II, the tenets of the field had been under attack for some time. Led by Franz Boas, a professor at Columbia University and curator at the American Museum of Natural History, cultural anthropologists began to dismantle scientific racism by separating biology from culture. They criticized the eugenicists' insistence on the hereditability of moral and intellectual traits, as well as the polygenic notion, strains of which persisted well into the twentieth century, tying race and civilization to evolutionism. In his 1911 book, *The Mind of Primitive Man*, Boas argued that race, culture, and language were distinct variables, rather than overlapping ones, and that many claims about Anglo-Saxon superiority could be explained by both environmental and cultural variables and by the observer's prior experiences with his or her subject. His emphasis on cultural relativism and resolve in arguing for the scientific instability of racial hierarchies made him a major figure in the struggle to establish a new framework for understanding race in the early- to mid-twentieth century.[27]

Boas was equally adamant about the need to refute Nazi scientific racism, unsuccessfully organizing an attempt in the 1930s to denounce it.[28] Although he died before the end of World War II, visions of Nazi atrocities, Japanese American internment, and discriminatory treatment of African American soldiers convinced a number of postwar scientists, including Boas's former student, Ashley Montagu, of

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[27] Barkan, 76-90; Stocking, Jr., 195-233. Stocking, Jr.'s book contains the most detailed discussion of Boas's work, in Chapters 7 ("From Physics to Ethnology"), 8 ("The Critique of Racial Formalism"), and 9 ("Franz Boas and the Culture Concept in Historical Perspective").

[28] Michelle Brattain, "Race, Racism, and Antiracism: UNESCO and the Politics of Presenting Science to the Postwar Public," *American Historical Review* 112, 5 (Dec. 2007), 1390-1391.

the need to craft a carefully worded statement to clarify to the public the meaning of race. In 1949, the United Nations Educational, Scientific and Cultural Organization (UNESCO) convened an international group of scholars to put together a rebuttal of Nazi ideology. The result was the 1950 UNESCO statement on race, which asserted a single origin for mankind, denounced the assumption that intelligence testing revealed hereditary differences in ability, and declared, finally, that "'race' is not so much a biological phenomenon as a social myth." While the statement generally met with approval from the national and international press, widespread scientific controversy over the section on race as a social myth—particularly among geneticists and physical anthropologists, who had not been included on the UNESCO committee—prompted the agency to assemble another group of scholars. A year later, in 1951, UNESCO released a second statement which backtracked substantially on the issue of social myth, suggesting instead a schema in which race could be considered a series of recognizable physical and physiological characteristics.[29]

The 1950 UNESCO statement revealed scientific acknowledgement that race and racial differences were not fixed, permanent phenomena, but depended on the beliefs of those who were embuing them with meaning.[30] It represented a formal end to the racial science of the past century and a half, abandoning racial typologies and hierarchies while recognizing the importance of social perceptions and context. Although it lasted scarcely a year before it was supplanted, the statement signaled that race might not be a valid biological category for analysis. In doing so, it laid the

[29] All material on the 1950 and 1951 UNESCO statements is from Brattain's article, 1386-1413.

[30] See Brattain, 1413.

foundation for the scientific turn to socioeconomic variables as measures of race in the 1960s and 1970s, a framework which persisted until the 1990s, when advancements in genetics and biochemistry would introduce new ways of understanding the science of race.

Introduction

Locating Race and Disease

In the summer of 2007, the journal *Cancer* published an article describing racial differences in the characteristics of breast carcinomas in newly diagnosed women. The authors found that African Americans in their study were more likely than Caucasians to have an aggressive form of cancer known as the "triple-negative" phenotype, so named for its negative tests for estrogen, progesterone, and HER-2 receptors. Black women, moreover, presented with higher grade tumors at a more advanced stage of the disease.[1] Other scientists had also found that tumor biology varied by race. An article in the December 2006 issue of the same journal suggested that larger tumors in African American females, combined with a higher rate of the estrogen receptor-negative form of breast cancer, contributed to increased mortality when compared with Hispanic or white women.[2] A separate, earlier investigation reported that uterine tumors in black women responded less favorably to drug therapies than similar tumors among whites.[3]

These studies marked the latest efforts in the ongoing task of assessing the impact of race on medicine and health. For centuries, observers have noted unequal rates of disease among racial groups, using them at different times as evidence of biological

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[1] Gloria J. Morris, Sashi Naidu, Allan K. Topham, Fran Guiles, Yihuan Xu, Peter McCue, Gordon F. Schwartz, Pauline K. Park, Anne L. Rosenberg, Kristin Brill, and Edith P. Mitchell, "Differences in breast carcinoma characteristics in newly diagnosed African-American and Caucasian patients," *Cancer* 110, 4 (Aug. 2007), 876-884.

[2] Wendy A. Woodward, Eugene H. Huang, Marsha D. McNeese, George H. Perkins, Susan L. Tucker, Eric A. Strom, Lavinia Middleton, Karin Hahn, Gabriel N. Hortobagyi, and Thomas A. Buchholz, "African-American race is associated with a poorer overall survival rate for breast cancer patients treated with mastectomy and doxorubicin-based chemotherapy," *Cancer* 107, 11 (Dec. 2006), 2662-2668.

[3] Nicholas Bakalar, "Despite Equal Cancer Care, a Racial Disparity Persists," *New York Times*, Sep. 26, 2006 (accessed online Jan. 7, 2008).

inferiority, divergent ancestry, or, in the case of communicable illnesses such as cholera, moral failure. More recently, doctors have attributed patterns of morbidity and mortality to environmental conditions, genetic inheritance, or a combination of the two. Within medical circles today, researchers and clinicians generally consider race to be of no more biological consequence than the length of one's arms or the size of one's feet. "[I]n medicine," read a 2001 editorial in the *New England Journal of Medicine*, "there is only one race—the human race."[4] But as contemporary studies clearly indicate, race continues to affect health by influencing not just one's response to treatment and short- and long-term survival rates, but the very illnesses to which one might be susceptible.

This dissertation historicizes the current debate over the significance of race to the landscape of disease by investigating notions of race in biomedical research on cancer in the United States between 1920 and the turn of the twenty-first century. Medicine has been both constitutive of and reflective of broader cultural trends surrounding race, and scientific work on different forms of cancer shaped and reshaped its meanings during this time. As doctors struggled to understand the pathways of cancer causation, their shifting interpretations of the association between race and carcinogenesis led to a constant revision of the ways in which they measured and classified race. Their work not only affected categorizations of race and ethnicity, but also played a crucial role in determining how race would get defined in the twentieth century.

Since the 1920s, skin cancer, nasopharyngeal carcinoma, prostate cancer, and cervical cancer have encompassed particular disease identities that have resulted, in part, from patterns of incidence. For a portion of the history of each of these forms of cancer,

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[4] Quoted in Denise Grady, "Imperfect, Imprecise but Useful: Your Race," *New York Times*, July 4, 2006 (accessed online Jan. 10, 2008).

the disease's expression has aligned with existing boundaries of race. Prostate cancer, the most common non-skin malignancy in American men today, carries a strong association with African Americans, whose rates of the disease are the highest in the world. Similarly, cervical cancer's historical connection to low-income women generated a research focus on black females, a population for whom race and class have been inextricably linked. Nasopharyngeal carcinoma, a type of cancer which involves the portion of the upper throat which is located behind the nose, disproportionately affected Chinese and Chinese Americans, while the etiology of skin cancers, particularly malignant melanomas, put those with the palest skin at the greatest risk.

The racially-inflected disease identities of these four types of cancer have, in turn, shaped research agendas. In providing scientists with a place to begin their work, epidemiologic configurations consolidated racial and ethnic borders. Existing racial ideologies determined who might be susceptible or resistant to a specific type of cancer, bringing a disease component to categorizations of both nonwhites and ethnic Caucasians. What scientists already understood about disease causation affected how they classified blacks, whites, and Asians, and what they subsequently discovered sometimes caused them to redraw racial boundaries. When studies showed, for instance, that blacks seemed curiously immune to skin cancer, doctors sketched a line around black Africans, African Americans, and Afro-Caribbeans, separating all blacks from all non-blacks and effectively dividing the world by level of pigmentation. Disease identities affected not only the groups into which people could be placed as subjects for research, but also functioned as a means of distinguishing members of one race or ethnicity from another.

Associations between different types of cancer and specific ethnic or racial groups both influenced where scientists looked for the causes of disease and impacted how they went about searching. Prevailing ideologies of race and what it represented shaped research methodology by pointing doctors to the places where it appeared to be located. In the late nineteenth century, race generally referred to one's country of origin, with groups distinguished by boundaries of nationhood. It comprised both a biological component and a hierarchical one, an internal as well as an external measurement. Social Darwinists promoted the idea of higher and lower races that possessed physical, moral, and mental attributes which had been acquired over time. By the 1920s and 1930s, scientists interpreted race as a combination of pigmentation and culture. Race indicated color and associated habits and practices; it encompassed both physicality and behavior.

Through mid-century, the notion of race continued to incorporate physiological criteria, such as the chemistry of the surface of the skin and the size and shape of one's organs. While most researchers believed that these were inherited traits, some continued to argue that they represented true "racial" characteristics, retaining the view that race encompassed a series of immutable, unchangeable physical and physiological features. In the postwar period, this perspective gradually faded, and the measurement of race began to move in two different directions. On the one hand, the field of genetics, a formerly peripheral specialty whose scientific standing skyrocketed with discoveries in the 1950s about the nature of DNA, brought a new, molecular component to the study of race. Researchers hoped to be able to determine the genetic markers which made populations look different and experience disparate health outcomes. Many anticipated, as well, that new work in genetics and its handmaiden, molecular biology, could uncover

disease pathways and isolate the mechanisms responsible for turning on and off chronic illnesses such as cancer.

On the other hand, understandings of race in the postwar period began to incorporate anew a collection of lifestyle factors that, as primarily social characteristics, lay outside the body. Unlike in the 1920s and 1930s, these habits and practices derived not from an idea of race informed by skin color and national boundaries, but from the socioeconomic criteria associated with one's level of income. Scientists aiming to quantify race and its effects turned to indicators such as diet, smoking status, and number of years of education, variables which epidemiologists and public health researchers believed could serve as markers of the conditions that led to race-based health disparities. This formulation of race relied on a strong correlation with class and its attendant social effects; it aimed to measure the medical significance of race by using socioeconomic variables to gauge its consequences. The idea of racial difference as biological reality had fallen out of favor, replaced by a belief that economic and social indicators, which were "real," might offer a reliable way to explain racial discrepancies in rates of disease. The use of socioeconomic factors to represent race in medical research continues to this day.

The story of race and cancer in the twentieth century has also been shaped by the changing relationship between technology and medical understandings of color, ethnicity, and race. The ways in which race functioned within the etiology of different forms of cancer were intertwined with technological developments, as advancements in diagnostic procedures altered associations between race and disease by locating race in different places at different times. The Pap smear, for instance, was in large part responsible for a

decades-long decrease in morbidity and mortality from cervical cancer, as it offered doctors a convenient way to screen for the disease at its early stages, when it was most treatable. As the test became less expensive and more widely used, it redefined risk in behavioral terms. Cervical cancer most often affected low-income women with a particular combination of sexual and social practices, including multiple sexual partners. Associations between class and race, moreover, meant that the disease was most prevalent among poor, non-white females. The discourse surrounding cervical cancer helped to cast race as risk, as the groups of women who were most likely to develop cervical cancer were also the ones with the greatest chance of practicing the hazardous behaviors that could lead to the disease. Work on the etiology of skin cancer similarly shaped perceptions of race as skin color. First blacks, and then ethnic whites, were classified according to skin tone as scientists slowly uncovered the link between ultraviolet light and dermal malignancies. The practice of using adjectival expressions of skin color, common among skin cancer researchers since the 1920s, faded with the introduction in the 1960s of the skin reflectometer, a device which assessed the amount of light refracting off a surface. Rather than describing someone as "pale" or "very dark brown" or "coal black," doctors were now able to reduce skin color to a precise measurement of the degree of the organ's absorption of light.

By the end of the twentieth century, race had returned inside the body. After decades of moving away from an emphasis on physicality, a new, bodily conception of race emerged, shaped in large part by technological advancements that introduced innovative ways of detecting and measuring biological and chemical markers. Increasing awareness of a racial divide in the incidence of and death rate from prostate cancer,

sharply higher among African American men, fueled an upsurge in research on the disease. Aided by new diagnostic technologies, such as the PSA test, which measured blood levels of the amount of a specific protein produced by the prostate gland, and by procedures that could assess levels of hormones and antibodies, doctors uncovered chemical evidence of racial differences between whites, blacks, and Asians. African Americans had the highest testosterone and PSA readings, while Asian Americans showed the lowest PSA levels. These, and similar tests, seemed to confirm that race was now detectable in the blood and scientifically quantifiable, lending credence to the idea that racial differences were discernable underneath the skin—an outdated notion of race made viable again by modern technologies.

As the history of cancer illustrates, the measurement of race moved from a bodily notion early in the century, to a postwar assessment which increasingly incorporated external characteristics, back to an internal schema in the 1990s. The potential consequences of this most recent shift in racial thinking are immense. It complicates the idea, which has become commonplace over the last few decades, of race as a social construction. According to this view, phenotypic differences in skin color, hair color and texture, and body type hold no inherent meaning, and are significant only in the cultural value which we have assigned to them. Race as a system of classification may correspond to physical discrepancies between groups of people, but the differences that define racial boundaries are only external. Disease acts upon bodies without regard for race, and any health disparities among races result from economic and social forces, not from innate biological distinctions. Moreover, geneticists tell us that we share over 90% of our DNA with one another, making us more similar than dissimilar, and most genetic

variation does not align with categories of race. Social constructionists disavow any inference of inherent intellectual ability or predisposition to a number of chronic ailments, including cancer, heart disease, and high blood pressure, believing instead that racial differences in health or mental aptitude result primarily from environmental conditions.

The new, bodily conception of race instead suggests that there is more to race than mere social construction; that it leaves an imprint upon our genes which can be measured through assays and blood tests and serological analyses. It is, in some ways, an extension of the idea that external similarities within races come from shared ancestry. If a particular population displays a distinctive hair texture or eye color, for instance, then it isn't really so much of a stretch to imagine that this group might also have developed differences in how the organs metabolize fat or react to particular substances. This notion of race, of course, has its critics. Notably, researchers working in the field of population genetics are focusing on ancestry outside of traditional racial categories, and a branch of public health examines the health consequences not of race, but of racism. But the new, bodily conception of race is uniquely dangerous in its alignment of biochemical differences with existing boundaries of race. When internal, genetic variations correspond to external, phenotypic ones, then racial categories appear to be natural and biologically valid. If African Americans, in other words, show the highest PSA readings among all groups in the United States regardless of income, access to health care, or level of education—all socioeconomic criteria—then why isn't this evidence of a biologically distinct race?

For most of American history, the great scourges of public health have been infectious and communicable illnesses such as smallpox, cholera, and tuberculosis, which can spread easily from one person to another under the right conditions. During the nineteenth century and early years of the twentieth, epidemics frequently ravaged urban areas where people lived in crowded, substandard housing without reliable access to clean water or adequate sanitary facilities. Both government officials and the popular press vilified residents of these neighborhoods—often non-whites, immigrants, and the poor—as disease-carrying threats to the nation's public health. At different times, Jews, Irish immigrants, and Chinese Americans, among others, shouldered the blame for outbreaks of typhus fever, cholera, and bubonic plague. But public health could also function as a vehicle for Americanization. Middle-class status offered protection from the diseases of the ethnic ghettoes to those who acquired wealth and relocated out of the old neighborhoods. By adhering to the healthy, hygienic lifestyle that was expected of proper Americans, they demonstrated their capacity for self-regulation, thus allowing access to the privileges and benefits of citizenship.

Historians have found a rich trove in the relationship between ethnicity and disease. Outbreaks and epidemics of infectious illnesses, in particular, offer a convenient lens through which to examine social and cultural attitudes toward contagion, cleanliness, and health. Among the best works on ethnicity and public health is Charles Rosenberg's *The Cholera Years: The United States in 1832, 1849, and 1866*. An elegant and persuasive writer, Rosenberg is particularly adept at illuminating the cultural dimensions of each successive epidemic. Howard Markel's *Quarantine! East European Jewish Immigrants and the New York City Epidemics of 1892* and Alan Kraut's *Silent Travelers:*

Germs, Genes, and the "Immigrant Menace" share a similar approach to the history of ethnicity and immigration, offering insights into how medicine has been used to designate certain groups as undesirable. *Science at the Borders: Immigrant Medical Inspection and the Shaping of the Modern Industrial Labor Force*, by Amy L. Fairchild, explores linkages among ethnicity, health, and citizenship through the medical examination to which all arriving immigrants were subjected. Fairchild demonstrates that public health practices at the beginning of the twentieth century excluded immigrants from Asia and the Middle East at much higher rates than newcomers from Europe, an important intervention in the historiography of immigration.

In contrast to the extensive literature on ethnicity and public health, just a handful of historians have explored the relationship between race and disease. In a recent book entitled *Fit to be Citizens? Public Health and Race in Los Angeles, 1879-1939*, Natalia Molina examines the effects of science and public health on the racialization of Mexicans, Chinese, and Japanese, employing a comparative approach to show how local officials used concerns about infectious disease to cast citizenship in racial terms. Keith Wailoo's *Drawing Blood: Technology and Disease Identity in Twentieth-Century America* and *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Health* contain excellent discussions of sickle cell anemia's status as a "Negro" disorder and what it reveals about the relationship between racial and disease identities. The best monograph on race and public health is Nayan Shah's *Contagious Divides: Epidemics and Race in San Francisco's Chinatown*, which traces the transformation of Chinese immigrants from hygienic pariahs to bourgeois citizens from the late nineteenth to the mid-twentieth centuries. Shah provides a sophisticated framework for

understanding public health, race, and immigration as contested sites, connecting public health as a coercive mechanism to the processes of racial formation.

By the 1920s, the bacteriological revolution of the late nineteenth century, combined with Progressive Era improvements in municipal services such as sanitation and the delivery of clean drinking water, had greatly reduced death rates from infectious and communicable ailments. Abandoning the moral dimensions of illness that scholars such as Rosenberg and Markel have described, scientists began to concentrate on the biological causes of disease. The discovery of penicillin and widespread use of antibiotics after World War II inspired many observers to put their faith in medical science, as it finally seemed capable of conquering the frontiers of disease.

In the midst of such progress against the illness-related suffering that had crippled previous generations, the Surgeon General's landmark 1964 report on smoking marked a crucial turning point. As historian Allan Brandt has argued, the report identified individual behaviors as risk factors for lung cancer, marking a shift from a strictly medical notion of disease to one which incorporated genetics, nutrition, and personal habits. As its name suggested, the "risk factor" approach sought to assess one's risk for contracting a particular illness, usually a chronic, non-communicable one. It not only suggested a return to a moral etiology, but also turned race into a risk factor in itself; now, race affected both the diseases with which one might be afflicted and one's probability of survival. However, unlike the associations of decades past between disease and ethnicity or race, class no longer provided a protective status. Instead, the genetic inescapability of race meant that certain ailments could affect anyone, regardless of place of residence, length of time in the U.S., or income bracket.

Historians have only recently begun to examine the dimensions of the chronic, degenerative illnesses that have supplanted communicable ones as the gravest threats to the nation's health.[5] In *Making Sense of Illness: Science, Society, and Disease*, Robert Aronowitz makes an important contribution to the sociology of medicine in his study of how social factors have influenced understandings of chronic fatigue syndrome, ulcerative colitis, Lyme disease, and coronary heart disease. Jeremy Greene's recent *Prescribing by Numbers: Drugs and Definitions of Disease* traces the ways in which hypertension, diabetes, and high cholesterol came to be measured as deviations from a numerical norm and the role of pharmaceutical companies in promoting medication to treat these new, symptomless diseases. Stephen Pemberton and Keith Wailoo's *The Troubled Dream of Genetic Medicine: Ethnicity and Innovation in Tay-Sachs, Cystic Fibrosis, and Sickle Cell Disease* brings race and ethnicity to the study of chronic disease, exploring their roles in shaping the histories and identities of each hereditary illness.

Within work on cancer, the standard social and cultural history of the disease is James T. Patterson's *The Dread Disease: Cancer and Modern American Culture*; published in 1987, it is now over two decades old. More recent books have focused on breast or lung cancer, the two most commonly-examined forms of the illness. Examples of the former include Barron Lerner's *The Breast Cancer Wars: Hope, Fear, and the Pursuit of a Cure in Twentieth-Century America* and Robert Aronowitz's new book,

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[5] While the historical study of chronic, degenerative disease is fairly new, that of chronic, communicable illnesses which feature acute episodes is not. The literature on tuberculosis is particularly extensive. See, for instance, Sheila Rothman, *Living in the Shadow of Death: Tuberculosis and Social Experience of Illness in American History* (New York: Basic Books, 1994); Georgina D. Feldberg, *Disease and Class: Tuberculosis and the Shaping of Modern North American Society* (New Brunswick, NJ: Rutgers University Press, 1995).

Unnatural History: Breast Cancer and American Society. Allan Brandt's epic *The Cigarette Century: The Rise, Fall, and Deadly Persistence of the Product that Defined America* offers a masterly account of not only the process by which smoking became entrenched in American society, but also the scientific battles over its relationship with lung cancer. It will likely stand as the definitive work on both tobacco companies in the twentieth century and the changes in epidemiologic methodology engendered by the study of chronic disease, particularly lung cancer. No historian has yet written a monograph on race and cancer.

I have tried to be as careful as possible with my use of terminology, and although I learned a great deal through this project about the fields of epidemiology, biochemistry, and genetics, my training is ultimately in history, and not in science. I hope those with the requisite backgrounds will not find that I have grossly misunderstood the terms and concepts I employ. Furthermore, as my dissertation investigates the discourse surrounding biomedical research on cancer, I have focused on the work of physicians and other investigators working in mainstream medical fields. I have not included the stories and experiences of patients with forms of the disease, nor do I examine alternative narratives of health and healing. Both are important elements of the social history of cancer, but ones which lie beyond the scope of this project.

Finally, a note on usage: as this dissertation analyzes the ways in which cancer researchers understood, measured, and defined race during the twentieth century, race is central as both a term and a concept. I consider race to be a social and political category with meanings that are not fixed and absolute, but which change over time. Race is a

historically produced system of classification that translates phenotype and identity—both self-identification and the ways in which one is perceived by others, including governmental entities—into political, legal, and cultural considerations. While I regard race as a social constructed category of difference, the ramifications of race and racism impact people's lives in numerous ways and affect outcomes of health and disease. Thus, in the chapters that follow I employ race as a shifting category of representation best understood within the historical processes that give it meaning.

My dissertation begins in the 1920s, by which time chronic, degenerative illnesses such as cancer and heart disease had displaced the infectious and communicable ailments that imperiled populations in earlier times. By 1920, most physicians had already abandoned the idea that cancer's origins lay in moral degeneracy, much like the roots of alcoholism or criminality. Instead, they increasingly saw it as a disease of the body, caused by a combination of cellular mechanisms and chronic irritation. In Chapter 1, I investigate theories of cancer etiology in the 1920s. The observation that the illness affected people from "primitive" cultures less often than white Europeans and Americans led to cancer's designation as a disease of civilization, an association which guided epidemiologic research,; it both influenced and was shaped by racial classifications. At the same time, researchers keen on establishing how quickly cancer rates were increasing, in which organs, and for whom, rationalized and naturalized racial categories in collecting statistics on various populations. Cancer's identity as a disease of civilization structured the search for etiology in ways which affected groupings of both whites and nonwhites.

Chapters 2 through 5, which proceed roughly chronologically, investigate the relationship between race and cancer etiology through case studies of four different cancers that have had racial associations in the twentieth century. In Chapter 2, I look at skin cancer, an affliction whose stark racial disparities were poorly understood until the discovery of the carcinogenic nature of ultraviolet light, prompting researchers to scrutinize first physical, then physiological, and finally environmental factors in their search for its causes. Shifting associations of risk, race, and behavior marked studies into the etiology of cervical cancer, the subject of Chapter 3. Due to the disease's links with economic status and the correlation between race and class, race became a risk factor because it appeared to determine the sexual practices which could affect incidence. Chapter 4 investigates the relationship between nasopharyngeal carcinoma (NPC) and Asians. NPC's characterization as a disease of ethnic Chinese led scientists to pinpoint the specific behaviors, traits, and characteristics which defined an individual as such, a list guided as much by pre-existing knowledge of stereotypically Chinese habits, such as opium smoking, as by a disregard for regional variations in Chinese culture. In Chapter 5, I analyze how prostate cancer became a "black" disease in the postwar U.S. and how new research has unsettled prevailing frameworks of race in ways that will have profound effects into the twenty-first century.

For American scientists studying skin, nasopharyngeal, cervical, and prostate cancers between 1920 and 1999, racial disparities guided work on the disease. Differences in rates of cancer incidence forced researchers to confront multiple definitions of race as they struggled to find one which could explain epidemiologic patterns. Doctors working to understand how race could act on the body sought to

quantify not only its effects, but its medical foundations, as well. In the process, they created knowledge about the meanings of race and the ways in which it could be measured. What they discovered would play a major role in transforming racial discourse in the twentieth-century United States.

Chapter 1

Cancer, Race and Statistics

"Cancer," wrote a prominent researcher on the illness in 1925, "is essentially a disease of civilized and highly urbanized populations."[1] The idea that cancer struck those with refined ways of living had emerged toward the end of the nineteenth century and beginning of the twentieth, when observers began to notice that "primitive" races seemed curiously immune to it. In the 1920s, American physicians viewed the racial distribution of cancer through the lens of civilization. For the next decade and a half, the discourse of civilization would play a major role in shaping scientific approaches to the relationship between cancer and race until the eve of World War II, by which time its importance as an etiologic theory had dwindled.

In the 1920s, the notion of race encompassed two aspects: a set of bodily markers, notably skin color, and a series of cultural ones, derived from one's nationality or country of ancestry; it indicated both physical attributes and behavioral ones. "Civilization" invoked the latter. Because it made reference to a society's intellectual and moral achievements, it was an explicitly cultural categorization. It suggested race only indirectly, in the sense that a particular population's degree of advancement was determined by skin color. Cancer, therefore, most affected those who had progressed the

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[1] Frederick L. Hoffman, *Some Cancer Facts and Fallacies; An Address Delivered, in substance, before the Mayo Foundation, Rochester, Minn.; the Medical Societies and the public at Sioux Falls, S.D., Fargo, N.D., Winnipeg, Man., Regina, Sask., Victoria, B.C., and San Francisco, Cal.—July-August, 1925, by Frederick L. Hoffman* (USA: Prudential Press, no date), in Frederick L. Hoffman Collected Papers, History of Medicine Division, National Library of Medicine, Bethesda, MD (hereafter FLH NLM), 1916-1931, v. 2.

furthest from backwardness. The disease's epidemiologic patterns followed racial lines, with incidence rates directly corresponding to skin tone.

While civilization was the organizing theme for work on the racial distribution of cancer in the 1920s and early 1930s, differences in morbidity and mortality were expressed and given meaning through the use of statistics. Statistical categories both naturalized racial classifications and provided a medical underpinning for them. Statistics measured rates of incidence in "whites," "coloreds," "Negroes," and other groups, categories for which the boundaries were inconsistent and shifting. Race, within this context, was less a biological designation than an ideological one, determined by a tenuous combination of skin color, physical appearance, and cultural attributes. By using race as the basis for measuring disease, statistics lent scientific legitimacy to a particular understanding of it by validating and medicalizing "racial" differences between groups.

Those who labored to uncover links between race and cancer etiology in the 1920s and 1930s may have done so within the discursive framework of civilization, but their work represented only one part of the ongoing research on the dread disease. Scientists struggled to understand cancer's cellular mechanisms, singling out heredity, viruses, and microorganisms as possible causes. At the same time, they aimed to improve methods of detection and treatment. In an era in which medical certainties regarding cancer were few, doctors and public health authorities urged prevention as the best way to avoid the disease's ravages and implicated a sense of individual responsibility in knowing the signs and symptoms of cancer.

The researcher who described cancer as a disease of civilization in 1925 was Frederick L. Hoffman, a statistician with the Prudential Life Insurance Company. He

emerged as a principal figure in early-twentieth century cancer research, eventually assuming a position of leadership in the American Society for the Control of Cancer. In his extensive writings on cancer in "uncivilized" and "native" groups, he sought both to explain the disease's rarity in certain populations and to quantify his findings through the use of numbers. Consequently, he played a key role in articulating the meanings of the term "civilization" and in naturalizing the boundaries that were based upon them. In Hoffman's time, race was an idea which invoked both cultural designations based on nationality, and physical or physiognomic ones. The two concepts were inseparably linked—so much so that each one failed to make sense without the other, both within the discourse of race and as explanations for cancer etiology.

Cancer and Medical Knowledge

As the decade of the 1920s opened, Americans had little reason to think that the rapid pace of recent scientific progress would come to an end. Since the turn of the twentieth century, public health campaigns had dramatically reduced mortality from communicable diseases such as diphtheria and typhus. While housing reform, improved sanitation, and other municipal upgrades meant fewer deaths, particularly among children and infants, the effects of such improvements reached across spectrums of age and class. Mortality rates for children under the age of one stood at 92.3 per 100,000 in 1920, down from 162.4 in 1900.[2] Rates of tuberculosis, the country's top killer for much of the nineteenth century, were falling, from over 400 deaths per 100,000 in the mid-nineteenth

[2] Gerald N. Grob, *The Deadly Truth: A History of Disease in America* (Cambridge, MA: Harvard University Press, 2002), 222.

century to 194 in 1900.[3] Most important, advancements in basic medicine and diagnostic technologies had altered the ways in which scientists thought about illness, transforming disease from a series of symptoms arising from one's circumstances and surroundings to a medical condition brought about by a specific microorganism.

The era of bacteriology had begun nearly forty years earlier, with Robert Koch's 1882 discovery of the bacillus responsible for causing tuberculosis. The German scientist demonstrated conclusively that the illness was produced by a single pathogen, and not by miasmas, the invisible clouds of gases stemming from environmental sources on which earlier observers had blamed outbreaks of disease. Researchers rushed to determine the microbes responsible for one ailment after another; by 1900, they had identified about thirty such causal organisms.[4] Such new etiologic understandings may not have brought about an immediate and corresponding creation in effective therapies—the development of sulfa drugs, penicillin, and other medications that could effectively cure specific illnesses was still years away—but they did streamline the process of diagnosis and remove the terror with which previous generations had associated infectious and communicable disease. The eradication of, or at least the ability to treat successfully, diseases that were once the nation's top killers, would come with its own consequences, however. Reducing the threats of tuberculosis and diphtheria did not automatically guarantee that an individual would enjoy years of good health. The longer one lived, the more time he or she had to fall prey to a chronic ailment or a disease of old age. As Americans would soon see, cancer qualified on both counts.

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[3] Grob, 210.

[4] Ibid.

By 1920, American physicians had become increasingly familiar with cancer, but it remained a mysterious disease of indeterminate origin. Amidst its many scientific uncertainties, perhaps the only thing of which doctors could be sure was that it had a long history. Ancient Egyptians texts made reference to the illness and described various treatments for it, including excision of the affected area or, less invasively, the application of arsenic-based ointments to it.[5] Hippocrates himself named the disease in the fifth century B.C.E., comparing the blood-rich veins that radiated from a tumor to the legs of a crab.[6] In the second century A.D., Galen theorized that cancer developed from a concentration of black bile, one of four fluids which, along with mucous, blood, and yellow bile, governed the processes of the body.[7] More recently, seventeenth-century healers applied various salves and poultices to tumors, including ones made with tobacco plants, tinctures of mercury, "rotten apples, frog spawn, fresh veal, or even pigeon, cut up while still alive."[8]

Medical knowledge about the disease had not progressed much in the intervening millennia. At the end of the nineteenth century, cancer's causes remained obscure and options for treatment, where they existed at all, were not only unreliable, but often resulted in the death of the patient. Both physicians and laypeople thought of cancer as an alien invader that took over an individual's body and slowly ate away at his or her insides. It "tore at the flesh," writes historian James T. Patterson, "often ravaging one's

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[5] James Ewing, *Neoplastic Diseases: A Text-Book on Tumors* (Philadelphia and London: W.B. Saunders Company, 1919), 17.

[6] James T. Patterson, *The Dread Disease: Cancer and Modern American Culture* (Cambridge, MA: Harvard University Press, 1987), 12.

[7] Ewing, 17.

[8] Adam Wishart, *One in Three: A Son's Journey into the History and Science of Cancer* (New York: Grove Press, 2007), 10.

private organs and leaving victims feeling dirty."[9] Cancer struck without warning, selecting its victims haphazardly. Death often followed on the heels of a diagnosis, when doctors could identify the disease at all, instilling fear in those who saw no way to control its erratic path. While surgery was the treatment preferred by most orthodox physicians, adherence to surgical procedures of anesthesia and antisepsis varied by practitioner. Since surgery was not routinely performed within a sterile environment, post-operative infections were common. The physicians, for instance, who treated President James Garfield after a shooting in 1881 "probed for the lodged bullet with their fingers, virtually guaranteeing the development of infection."[10]

For those living before the last few decades of the nineteenth century, a diagnosis of cholera, typhoid, or malaria might have spelled a death sentence. Disease, thought physicians and laypeople alike, resulted from some combination of social and moral factors. It could arise from behavioral quirks, such as consuming undercooked food or breathing fumes released by piles of rotting garbage. Or, as Charles Rosenberg has demonstrated, it might serve as God's punishment to the sinful. As observers noticed in the cholera epidemics of 1832 and 1849, the disease seemed disproportionately to affect alcoholics, immigrants, prostitutes, and the poverty-stricken.[11] Assigning a moral etiology to infectious ailments served as a way to reduce the random nature with which they seemed to strike; it represented an attempt to gain a measure of comprehension, and consequently, control, over diseases that held nearly insurmountable potential to inflict

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[9] Patterson, 30-31.

[10] Patterson, 18.

[11] Charles E. Rosenberg, *The Cholera Years: The United States in 1832, 1849, and 1866* (Chicago: University of Chicago Press, 1962, 1987).

pain and suffering. The more that people could isolate vectors of disease—be they race, ethnicity, or social class—the greater the likelihood of avoiding sickness altogether.

Cancer may not have incited panics in the same way that outbreaks of bubonic plague or yellow fever did, but its unpredictability and tendency to lie concealed within inaccessible parts of the body, combined with its often rapid course after diagnosis, gave the disease a unique capacity to incite dread and anxiety among Americans. Many late nineteenth-century observers considered cancer a symbol of degeneracy, like alcoholism, drug addiction, or criminality, that marked unfortunate families from generation to generation. The popular belief that it was contagious further served to isolate its victims, who were shunned and stigmatized. As Patterson notes, residents often gave "a wide berth" to the New York Cancer Hospital, an Upper West Side institution that was the first of its kind in the country, "as if the virulent germs of cancer were able to penetrate the walls and contaminate the surroundings."[12] Relatives of those who fell victim to the disease sometimes hid a diagnosis of cancer from friends and acquaintances. Even the prominence of such cancer patients as Grover Cleveland and novelist Alexandre Dumas could not shield them from contemporary prejudices. Under cover of darkness, Cleveland underwent secret treatments aboard a yacht in New York Harbor for cancer of the jaw during his presidency. He concealed news of his operation from both the press and the public, and the complete story about his bout with cancer did not emerge until after his death in 1917. Dumas died from the disease in the 1890s, yet, as with many of

[12] Patterson, 38.

his contemporaries, shame and stigma surrounding the mysterious ailment kept obituaries from listing it as the cause of death.[13]

The list of scientific unknowns about cancer contrasted sharply with recent advancements in other areas of medicine and public health. At the turn of the twentieth century, physicians were experiencing a rise in income and social status that brought new legitimacy and authority to the profession. In the eighteenth and early nineteenth centuries, as Paul Starr writes in *The Social Transformation of American Medicine*, limited transportation networks meant that regular medical care was impractical for the majority of Americans who lived in rural areas. The indirect expenses of travel, either for a doctor to see a patient or vice versa, made the cost of treatment prohibitively high; as a result, few physicians were able to support themselves solely through the practice of medicine.[14] Some could make ends meet only by simultaneously taking on other responsibilities, such as dentistry, veterinary medicine, or midwifery.[15] Although as professionals they enjoyed greater status than manual laborers, their salaries at the time placed them comfortably within the ranks of the middle class, and often at the low end.

During the nineteenth century, a career in medicine presented few barriers to entry, and guaranteed neither financial prosperity nor social status. There were, notes Starr, physicians at the top of the profession who had attended elite universities and medical schools, often in Europe, and secured an affluent living attending to wealthy, prominent patients. Many practitioners, however, took only a smattering of specialized courses, while others served in apprenticeships and received no formal medical education

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[13] Patterson, 36-37.

[14] Paul Starr, *The Social Transformation of American Medicine* (New York: Basic Books, 1982), Chapter 2.

[15] Starr, 81-85.

at all. As Starr reports, "the proportion of medical school graduates among practicing physicians" in five counties in New England from 1790 to 1840 "ranged from 20 to 35 percent."[16] Moreover, medical therapeutics at the time ran the gamut from ineffective to deadly, an assortment of tinctures, salves and patent medicines, as well as surgeries practiced in unsterile environments. The limits of medicine's ability to effectively treat various ailments probably contributed to social perceptions of doctors as inexpert laymen with little to offer by way of cure. As a result, medicine as a line of work garnered less prestige than other careers at the time. An 1851 study by the newly formed American Medical Association found that over three times as many men who graduated from eight prominent colleges between 1800 and 1850 became lawyers or clergymen as entered the field of medicine, with even lower rates among honors students. "As late as 1870," Starr writes, "a medical journal remarked that when a young man of merit and ability chose to become a doctor 'the feeling among the majority of his cultivated friends is that he has thrown himself away.'"[17]

Because the medical profession lacked standards for education and licensing, as well as a powerful organization which could lobby for its members' interests on a national level, physicians found that a medical degree often conferred only limited legitimacy. Toward the end of the nineteenth century and the beginning of the twentieth, a number of developments converged to strengthen the influence of physicians as a group, coming in the midst of a period of increased professionalization, when plumbers, butchers, and other skilled workers similarly established licensing requirements and

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[16] Starr, 63-64.

[17] Starr, 82-83. The colleges were Amherst, Brown, Dartmouth, Hamilton, Harvard, Princeton, Union, and Yale.

professional standards. First, the American Medical Association reorganized and expanded. In 1900, it had just 8,000 members out of a national population of about 100,000 physicians.[18] It subsequently brought into its allopathic ranks homeopaths and Eclectics, two groups with which it had previously disagreed on medical orthodoxy.[19] The AMA also changed its fee and membership structure, requiring physicians who wanted to belong to county medical associations or the national organization also to join their state medical associations. Local membership skyrocketed, which in turn increased the clout of physicians at the state level, where, as Starr notes, "many vital political decisions…were then being made."[20]

Second, the movement, beginning around 1870, to professionalize American universities and shift the intellectual focus of the faculty to research expanded to encompass medical education, as well. Schools including Harvard and the University of Pennsylvania imposed new curricular requirements, tightened standards for students, and lengthened the academic year.[21] Physicians began to receive comprehensive training according to academic standards that were now both rigorous and uniform. Third, the Pure Food and Drug Act of 1906 made illegal many of the patent medicines that had been advertised in previous decades, promising cures for ailments from headaches to kidney disease to tuberculosis; this did much to eliminate the medical "quacks" who undermined public confidence in the efficacy of medical science. Fourth, the bacteriological

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[18] Starr, 109.

[19] Eclectics were botanic doctors who believed in empiricism and reform of the regular profession, which they thought placed too much emphasis on bleeding and drugging of patients. Homeopaths considered disease a matter of spirit, and called for reduced dosages of drugs which would produce the patient's symptoms in a healthy individual. See Starr, 96-97.

[20] Starr, 109.

[21] Starr, 112-116.

revolution of the late nineteenth century created an increasing reliance on diagnostic technologies including the microscope and the spectrometer, instruments that could only be read and used by trained professionals. These developments, combined with a newfound political influence at the state level, transformed doctors from people whose jobs involved perhaps no more than advanced technical skills to experts who derived considerable authority from specialized knowledge that was largely inaccessible to the general public. Physicians, clinicians, and other members of the medical community began to enjoy increased prestige for their ability to marshal science to alleviate human suffering.

Cellular Etiology

For all the progress by 1920 in the fields of medicine and public health, the scientific community had yet to answer successfully the challenge of cancer. Numerous studies documented the alarming upsurge in rates of incidence and mortality, figures that were familiar to doctors and the public alike. Francis Carter Wood, a prominent physician, estimated that at least 88,400 Americans died of cancer in 1920, an increase of nearly ten percent from 1910.[22] The American Society for the Control of Cancer reported an official rate of 91.9 cases per 100,000 people in 1924, up from 63.0 in 1900.[23] By 1939, Thomas Parran, Jr., the Surgeon General of the United States, reported that the

[22] Francis Carter Wood, *Cancer: Nature, Diagnosis and Cure* (New York: Funk & Wagnalls Company, 1923), 1.

[23] American Society for the Control of Cancer, *Suggestions for Popular Talks on Cancer* (New York: American Society for the Control of Cancer, 1927), 12.

disease killed 140,000 Americans annually, or "more than twice as many as live in Atlantic City."[24] Only heart disease claimed more lives each year.

The terror and unease that cancer generated reflected medicine's lack of progress in understanding the disease, much less in finding a cure for it. At the time, there were about half a dozen major theories regarding its causation. The oldest hypothesis blamed cancer on parasites or some form of microbiotic organism able to jump from person to person, leaving a trail of carcinoma in its wake; studies of "cancer houses" and "cancer streets" seemed to confirm a relationship between geography and the disease. Two theories related cancer causation to abnormal cellular growth. The first held that tumors grew from masses of tissue that had been misplaced during embryonal development, while the second implicated a breakdown in "tissue tension," the complex group of forces believed to keep unrestrained cellular growth in check.[25] After Peyton Rous successfully induced tumors in healthy chickens around 1910 by injecting them with tiny particles from cancerous birds, he speculated that the disease might be linked to specific viruses. Although his ideas displayed initial promise, they failed to take hold until much later, when scientists in the 1970s revived his search for viruses that could lead to an increased risk of cancer.[26] Another theory with early potential was the notion of heredity. Perhaps the best-known scientist on this subject was Maud Slye, a researcher at the University of Chicago. Her selective breeding of mice to isolate specific cancer strains garnered critical attention in the 1920s and 1930s, when colleagues hoped that her work could help

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[24] Thomas Parran, Jr., "Cancer and the Public Health," *Science* 90, 2341 (Nov. 10, 1939), 428.

[25] See Ewing, 94-95, 98, 114-116.

[26] One such virus is the human papilloma virus, which causes some strains of cervical cancer in women.

to explain why an elevated incidence of the disease could sometimes be found in certain "cancer families."

The most widely accepted theory for the formation of cancer attributed the disease to trauma, a term that was somewhat loosely defined. Doctors believed that cancer could develop after an injury from "a single or repeated more or less contusing, crushing, or lacerating" blow.[27] A tumor could form, as well, through protracted, mild injury to an area. Called "chronic irritation," such trauma could be thermal, as in the heat from solar radiation or x-rays, or it could result from various chemical substances, including tar, mineral oil, and certain kinds of dyes that were just beginning to be recognized as carcinogens.[28] It might take a mechanical form, which, if prolonged, could transform an ordinary blemish into a cancerous lesion. Medical books for lay audiences advised readers not to let clothing rub against any moles or cysts, not to pick at any growths on their skin, and to avoid accessories with parts that fitted snugly or pinched any part of the body. Jagged teeth should be repaired immediately by a dentist, lest they lead to cancers of the mouth and lips. As James Ewing, a professor of pathology at Cornell University Medical College in New York City, wrote in his 1919 textbook on cancer, the disease "has been clearly traced to laceration by rough instruments, rusty nails and pins, thorn pricks, insect bites, surgical wounds, and blows without visible destruction of tissue."[29]

In addition to the theories that appeared in established medical sources, such as *Annals of Surgery*, *The American Journal of Cancer*, and *The New England Journal of*

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[27] Ewing, 110.

[28] See, for instance, Arthur Purdy Stout, *Human Cancer: Etiological Factors, Precancerous Lesions; Growth; Spread; Symptoms; Diagnosis; Prognosis; Principles of Treatment* (Philadelphia: Lea & Febiger, 1932).

[29] Ewing, 111.

Medicine, self-published books and pamphlets offered to curious readers a flurry of alternative hypotheses. Some of these authors were laypeople convinced that they had uncovered crucial evidence overlooked by experts, while others were physicians, chemists, and biologists who based their ideas on their own scientific training. One blamed cancer on dogs.[30] Another claimed that premature birth resulted in fetal imperfections, which increased the supply of oxygen to abnormal cells and produced cancer.[31] The abundance of plausible—or not so plausible—theories regarding the causation of the disease revealed the haphazard state of oncological research at the time and reflected the urgency with which Americans sought answers. The disease seemed to represent a detour along mankind's seemingly unstoppable march of scientific advancement; its grip on the national imagination laid bare the significance of the battle to untangle its mysteries.

A cure might be the ultimate goal, but one first had to understand the ways in which the disease formed and progressed throughout the body. If mainstream physicians, laboratory researchers, and other scientists couldn't quite agree on the precise etiology of cancer, they did generally acknowledge that it required two sets of conditions in order to take hold. First, an individual had to possess a "constitutional susceptibility," or some particular biological makeup that carried with it a tendency for abnormal cellular growth. As much as doctors insisted that cancer was not a directly inheritable disease—that is, a parent could not pass down breast or liver cancer to an offspring—they did believe that a propensity for tumor formation was transferable from one generation to the next. While

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[30] Samuel Walter Cort, *Cancer: Is the Dog the Cause?* (London: Bale & Danielsson, 1933).

[31] Caleb Wyand Geeting Rohrer, *Researches in Cancer: Part One* (Baltimore: The Brentwood Printing Company, 1934).

the exact nature of heredity in cancer was still under investigation, it seemed clear to contemporaries that one's ancestry played a critical role in its expression, and the work of Slye and her colleagues before World War II directly addressed this search to isolate predisposing factors. As Canadian researcher Madge Thurlow Macklin wrote,

> There is by no means unanimity of opinion as to the heredity of cancer. One argument which is brought forward is that if heredity is at the basis of cancer, it is not cancer itself which is inherited but only the predisposition to it, since cancer as such is not present at the time of birth….The important thing is that we inherit some factor, call it a predisposition, a potentiality, or just "cancer". In our constitutional makeup there is something which differentiates us from the person who cannot develop cancer, and that factor is bound up in what we receive from our parents and in what we are capable of passing on to our offspring.[32]

The second requirement for the development of a cancerous growth called for the presence of "exciting" causes. These included trauma, viruses, mental stress (which could affect one's nerve endings, thereby leading to uncontrolled cellular growth), and the commonly cited "chronic irritation." Many referred to this combination as "the seed and the soil," with one's genetic constitution supplying the latter, and external factors the former.

Etiologic investigations into the causes of cancer in the 1920s largely reflected the state of the field of epidemiology at the time. During the first half of the nineteenth century, epidemiologists looked closely at patterns of disease in populations to isolate their sources and vectors. John Snow's elucidation of the process of transmission of cholera in mid-nineteenth-century England famously illustrated the techniques of the discipline. By mapping the relationship between cases of the disease and subscribers to each of the two companies which supplied water to city residents, Snow demonstrated

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[32] Madge Thurlow Macklin, "Human Tumours and Their Inheritance," *Canadian Medical Association Journal* 27, 2 (Aug. 1932): 184, 187.

that Londoners who drew their water from the upper Thames, before it had been contaminated with sewage, had fewer instances of cholera than those who drank from the lower part of the river.[33]

In the United States, the methodology of Snow and his colleagues led to measurable improvements in health and wellness in the late nineteenth and early twentieth centuries. At the same time, the popularity of Robert Koch's germ theory began to supplant the field's earlier reliance on quantitative work. Koch's postulates outlined a path of causality based on the identification and isolation of a particular microorganism, which could then be made to induce disease. Each illness was associated with a singular, specific pathogen. The success of Koch's theories in illuminating modes of causation for infectious diseases led students of chronic ailments, including cancer and heart disease, to adopt them, as well. As a result, writes Allan M. Brandt, "[t]he tradition of environmental and behavioral investigation that had once characterized the search for 'cause' was now deemed by many researchers to be primitive and imprecise. The center of action had shifted to the laboratory. The 'field' was now beneath the lens of the microscope."[34] Practitioners all but abandoned the population as their unit of study, turning instead, as Mervyn Susser has argued, to the search for "specific agents [and] singular causes."[35]

Although rates of communicable illnesses declined in the early twentieth century, epidemiologists continued to apply the same methodologies which had produced fruitful

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[33] Rosenberg, 193-194.

[34] Allan M. Brandt, *The Cigarette Century: The Rise, Fall, and Deadly Persistence of the Product that Defined America* (New York: Basic Books, 2007), 119.

[35] Mervyn Susser, "Epidemiology in the United States After World War II: The Evolution of Technique," *Epidemiologic Reviews* 7 (1985), 149.

results for infectious diseases to the study of the chronic afflictions. The process worked better for some disorders than for others. For pellagra, for instance, which Joseph Goldberger determined in the 1920s was caused by a lack of niacin, the outcome was both unambiguous and successful.[36] But overreliance on infectious disease methodology led cancer researchers to favor disease trajectories that implicated microorganisms, cellular deficiencies, and other agents which could be classified as causes in the development of cancerous tumors. Even when scientists identified behavioral or environmental factors, their fetishization of the model of causation, to paraphrase Brandt, led them to concentrate on the mechanism of disease at the cellular level, equating exposure to a carcinogen with contact with an infectious agent.[37] This focus on a singular, rather than a multifactorial etiology for cancer, as well as the belief that cancer could be made to follow the paradigm of infectious diseases, would persist for decades.

Detection and Treatment

By the end of the 1920s, work on the cellular mechanisms of carcinogenesis, such as the theory of displaced embryonal tissue, appeared to be going nowhere. As a result, researchers largely abandoned their earlier hypotheses, save for two areas: heredity and chronic irritation. Studies of the former would continue in the coming years, most often in experiments to isolate particular chemical and physiological pathways in laboratory animals. But chronic irritation would become the major scientific theory regarding the

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[36] See, for instance, Jeremy A. Greene, *Prescribing by Numbers: Drugs and the Definition of Disease* (Baltimore: The Johns Hopkins University Press, 2007), 11. For a detailed discussion of Goldberger's investigation of pellagra, see Elizabeth W. Etheridge, *The Butterfly Caste: A Social History of Pellagra in the South* (Westport, CT: Greenwood Press, 1972).

[37] Brandt, 120.

causation of cancer until the 1950s, when the focus would shift to socioeconomic and other environmental and behavioral factors. Its appeal reflected just how little scientists understood the disease.

Researchers considering chronic irritation as an etiologic factor singled out the role of the individual, a move which dovetailed with public educational efforts in the late 1920s and early 1930s. Such campaigns were spearheaded primarily by the American Society for the Control of Cancer (ASCC). Founded in 1913 by a group of wealthy New York philanthropists, the ASCC in the 1920s and 1930s bore little resemblance to the powerful, well-funded group it would become after World War II. It was, instead, a small organization which depended on deep-pocketed donors, including John D. Rockefeller, Jr., to stay afloat. Although limited in influence, particularly outside the Northeast, the ASCC did its best to enact its official slogan to "Fight Cancer with Knowledge." By distributing pamphlets, publishing articles in magazines, and sending its medical experts to give public talks, it aimed to instruct citizens on the symptoms, treatment, and prevention of cancer.[38]

Despite its constraints of size and status, the ASCC was the most visible cancer organization on the scene, its existence an indication of a growing urgency within the medical community. Physicians, public health authorities, and the ASCC promoted the twin goals of early prevention and detection on the one hand, and personal responsibility for knowing the signs of the disease and seeking treatment on the other. They "encouraged people to inspect themselves for the early warning signs of cancer and to undergo regular medical checkups; warned of the dangers of quackery and folk remedies;

[38] Patterson, 71-78, 90-95.

and sought to undermine popular beliefs that might encourage delay."[39] As historian David Cantor writes, "[t]he message was that cancer was curable if caught early and treated by a recognized physician, and the public was thus urged to turn to their physicians at the first suspicion of cancer."[40] "Cancer," wrote Francis Carter Wood, "is not an epidemic disease which can be controlled by public health or police methods: it is rather a question of personal hygiene and personal understanding of the conditions under which cancer arises. The individual himself must take the necessary preliminary steps to place himself in proper hands in order that he may be cured of the disease when it is still in an early stage."[41]

Throughout the history of public health, the best "treatment" for incurable illness has often been prevention. Before antibiotics provided an effective therapy for tuberculosis in the period following World War II, public health authorities urged people to avert its spread by curtailing public spitting, ending the practice of sharing drinking cups, and covering their mouths when coughing. More recent assaults on heart disease have warned of the risks of smoking, high blood pressure, and obesity. In the 1920s and 1930s, physicians and public health advocates maintained that people put themselves at risk for cancer through entirely controllable measures. The key to avoiding cancer lay in steering clear of the behaviors that seemed to precipitate it, including chronic irritation of persistent cracks, sores, moles, and lacerations.

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[39] David Cantor, "Introduction: Cancer Control and Prevention in the Twentieth Century," *Bulletin of the History of Medicine* 81, 1 (Spring 2007), 5-6.

[40] Cantor, 5-6.

[41] Wood, 4.

To encourage watchfulness, cancer prevention messages often emphasized the element of fear, aiming to frighten people into obtaining the requisite checkups and exercising the proper amount of vigilance toward their bodies. J. Ellis Barker, the author of a volume for laypeople, claimed that cancer was "undoubtedly by far the worst" among "the important diseases which torment mankind." It "inflicts unspeakable agonies upon the poor sufferers, and frequently cancer patients become horrible to their own families by the intolerable smells which emanate from them. Consumptives are frequently cheerful to the end…[They] are apt to order new clothes and to make plans of travel during the last days of their lives, but most cancer sufferers are deeply depressed and despondent from the beginning of their disease."[42] "Popular pessimism," reported the ASCC, "is in part due to the fact that many cases are operated on too late, and…failures are widely advertised."[43] As Barker observed, cancer generally began as a painless growth, becoming dangerous only if allowed to progress unchecked. "Only when a large-sized tumour has developed which presses upon some tender spot," he wrote, "…does the afflicted person become aware that something is wrong. Then, as a rule, it is too late for a successful operation. The sufferer is doomed.[44]

Many doctors considered fear to be an unnecessary emotion, given the advances in knowledge about the illness. "[T]he fear of cancer," wrote one physician, "is inestimably worse than cancer itself and the more cancer is spoken of to the intelligent public the sooner it will be robbed of its unknown terrors. Most people believe that once

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[42] J. Ellis Barker, *Cancer: How it is Caused; How it can be Prevented* (New York: E.P. Dutton & Company, 1924, 1.

[43] American Society for the Control of Cancer, *Suggestions for Popular Talks on Cancer*, 15.

[44] Barker, 1, 2.

a diagnosis of cancer is established it means certain death. Nothing could be more erroneous."[45] Such rhetoric, however, departed wholly from the reality of technological knowledge at the time. Physicians could urge early diagnosis as much as they wanted, but without the tools to identify cancerous cells, such utterances were all but meaningless. George Papanicolaou's test for cervical cancer came into use within a few years of its development in the late 1930s and early 1940s, but even a decade later it remained the only reliable method to detect *any* kind of cancer at an early stage.[46] Clinicians could remove and microscopically examine cells from suspicious growths on the skin or other visible parts of the body, but such techniques were of no use if the cancer was growing internally, out of the visual range of eyes and instruments. The strikingly graphic slides of neoplastic cells that appeared in medical textbooks and journals were nearly always pathological specimens which had been obtained from bodies during autopsies, as no one, save for Papanicolaou, had yet figured out a way to look for the disease in inaccessible organs. Furthermore, fear, shame, and a refusal to contemplate what might amount to a judgment of certain death kept many cancer patients cloistered away at home. By the time they actually made the trip to the doctor's office, their tumors could have progressed to an extremely serious stage.

The medical community's emphasis on prevention and early detection stemmed in part from the scientifically proven conviction that patients in whom cancer did not spread

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[45] Albert Soiland, *Cancer: A Professional Responsibility and a Public Liability* (New York and London: D. Appleton and Company, 1928), 44.

[46] Patterson, 199. An ASCC pamphlet from the early 1930s mentioned new technologies that allowed for earlier diagnosis, including an x-ray study of the intestine with barium, cytoscopy of the urinary tract, proctoscopes for examining the rectal region, analysis of stomach contents, and transillumination of the breast. It is unclear, however, how effective and widely used these procedures were at the time. New York City Cancer Committee, American Society for the Control of Cancer, *Cancer: Then and Now* (New York: The Chemical Foundation, Inc., no date (probably 1932)), 22; in Frederick L. Hoffman Papers, Columbia University Rare Book and Manuscript Library (hereafter FLH Papers), Box 39, Scrapbook 2.

from a primary organ to other parts of the body had a better prognosis than those in whom the disease metastasized. It also, however, reflected the grim state of available treatment. Just as physicians, oncologists, and laypeople had promoted all manner of theories on the causes of cancer, so a similar assortment advocated a hodgepodge of cures. Early therapies ranged from jolts of electricity to the injection of minerals, ointments, and other compounds. At the least, they were ineffective against the disease; at the worst, they succeeded in killing the patient.[47] Other "quack" cures included ointments that supposedly lifted the cancer out by its roots, salves containing cod-liver oil, sarsaparilla bark, hemlock, sulphuric acid, zinc, camphor, or gold, and one method that involved "strangling the growth by tying a tight cord or wire about the base, using the chain saw, and freezing the cancer."[48] Sigismund Peller, who trained in Europe and settled in the United States, tried unsuccessfully for many years to get funding for what he called the "Principle of Inverse Association." According to his theory, which he devised in part from working with sailors in the U.S. Navy, those who survived skin cancer were rewarded with lifelong immunity from all forms of malignancies. He thus recommended inducing a mild form of skin cancer in young people to protect them from deadlier, more serious types of neoplasms later in life. Needless to say, his attempts to generate in healthy adolescents a disease to which was attached considerable social and medical stigma never caught on.[49]

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[47] Patterson, 63-65.

[48] Ira Kaplan, "Cancer Cures," *American Journal of Cancer* 16, 1 (Jan. 1932), 210-211.

[49] See Sigismund Peller, "Skin Irritation and Cancer in the U.S. Navy," *American Journal of the Medical Sciences* 194, 3 (Sep. 1937), 326-333; and *Not In My Time: The Story of a Doctor* (New York: Philosophical Library, Inc., 1979).

A significant number of researchers believed that surgery represented the only infallible cure for cancer. As one physician wrote, surgery "stands out as the one preeminent agent that has been of real service and which has stood the test of time in the battle against cancer."[50] Typically conducted with a scalpel, a radio-electric knife, or electrocautery, the latter two of which were heated up to destroy cancer cells, surgeons aimed to completely remove the cancerous growth, as well as the surrounding tissue. Other mainstream treatments involved some form of radium or x-ray therapy. X-ray cures emerged around the turn of the twentieth century, shortly following the discovery of the rays themselves in 1895. James Patterson notes that it quickly became clear to physicians that the technology was both difficult to manage and dangerous. By 1911, a number of cases had been documented of death or serious illness, including cancer, as a result of exposure to radiation. Many of those afflicted were x-ray technicians and physicians.[51] Nevertheless, members of the medical community continued to tout x-rays as therapy for the disease well into the 1940s. During the same time period, radium was a popular cure, particularly for forms of skin cancer. Faith in its possibilities remained high even after reports in 1925 of mouth cancer in New Jersey, where women applying the chemical to watch dials routinely dipped paintbrushes into their mouths to bring the bristles to a point. Because both radium and x-rays had worked in some cases on specific kinds of cancers—mostly external lesions on the skin—members of the medical community continued to rely on them, despite their dangers. They had few alternatives.

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[50] Soiland, 99.

[51] Patterson, 64.

Cancer and Civilization

The search for information about the nature, origins, and etiology of cancer led researchers in the 1920s and early 1930s to explore a range of epidemiologic variables. Because cancer rates had been increasing since at least the beginning of the century, a number of researchers began to focus on changes in the American lifestyle which were related to the nation's increased industrialization and could be contributing causes. Scientists, said J. Ellis Barker, the writer to whom cancer was unsurpassed in its ability to inflict agony upon the sufferer, "have looked for the cancer cause in the use of soap…, in piped water, in vaccination, in bad teeth…, in the use of sponges, in the use of enamelled [*sic*] saucepans, in emery employed for cleaning knives, in paper, etc."[52] Others singled out air pollution, overcrowding in urban areas, and the rise of the automobile.

Of all the transformations wrought by industrialization, observers spent by far the largest amount of time dissecting American patterns of diet and exercise. A number of writers, including Frederick L. Hoffman, the Prudential Insurance Company of America's chief statistician and a leading figure in the American Society for the Control of Cancer, noted the simultaneous expansion of leisure time and contraction of the number of hours spent in physical exertion. Americans, he claimed, were consuming greater amounts of food than at any time in history, much of it highly processed. "Habits of over-eating and of an excessive number of meals are the rule," he wrote, "with the result that the average adult bodyweight is easily 25% in excess of what it need be for a thoroughly healthy and vigorous existence."[53] The consumption of meat and sugar, too, had "increased

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[52] Barker, 17.

[53] Frederick L. Hoffman, *On the Causation of Cancer; An Address Delivered before the American Association for Cancer Research, Buffalo, NY, April 17, 1924* (USA: Prudential Press, no date), 3.

enormously" over the past fifty years. "It is not, of course, the meat or the sugar," Hoffman continued, "but the excess and the manipulated character of the food eaten, plus excessive frequency in eating, that causes the mischief and the disarrangement of the human machine."[54] In short, the combination of a sedentary way of life, an abundant food supply, and an overly processed diet was endangering the health of Americans by robbing their bodies of the optimal configurations of food and exercise which had sustained men and women throughout history.

The list of dietary restrictions varied by practitioner. Physicians warned against consuming too much salt, too much milk, too little milk, too many spicy foods, excessive amounts of meat, or fruits and vegetables of the wrong type. One took issue with the supposedly carcinogenic nature of the "succulent but harmless" tomato.[55] Some said to eat frequently, while others recommended against numerous meals. Certain combinations of food were to be avoided, but the specifics of such groupings inevitably varied. Extremes of temperature could be perilous; therefore, hot soups and stews spelled trouble. Medical pronouncements regarding the carcinogenic potential of the American diet may have been inconsistent, and even contradictory, but they highlighted the depths to which researchers delved in their attempts to isolate lifestyle factors in the etiology of cancer.

The changes in American ways of living to which Barker, Hoffman, and their colleagues alluded were commonly viewed as the byproducts of civilization. Beginning in the nineteenth century and accelerating after the Civil War, the U.S. economy had

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[54] Ibid., 5.

[55] Alex. Theodore Brand, *Cancer: Its Cause, Treatment and Prevention* (London: John Bale, Sons & Danielsson, Ltd., 1922), 3.

grown at an accelerated rate. Railroads, the country's first large corporations, pioneered new forms of organization and reduced space to time, enabling travelers to reach New York from Chicago in days rather than weeks and at a fraction of the cost. Meat-packing companies ballooned to the size of industrial behemoths. Relying on economies of scale to turn a profit, they refined techniques of mass production, butchering hogs and cattle by the thousands and transporting the packaged meat in refrigerated railroad cars to distant markets. Factories that produced everything from shoes to steel opened and expanded in cities and towns across the country. At the same time, thousands upon thousands of immigrants, mostly from European countries, entered the United States each year. Between 1880 and World War I, 25 million European immigrants arrived on American shores.[56] Many of them settled in urban areas and found work producing consumer goods for markets at home and abroad.

To many contemporaries, these were irrefutable signs of progress. Economic prowess set the United States apart as the most advanced nation on earth, the pinnacle of cultural and social development to which the rest of the world should aspire. But civilization had its seamy underbelly, too, which challenged the notion of unimpeded progress and unsettled those who bothered to look beneath its glossy exterior. For one, the influx of so many immigrants brought to light a host of urban problems. Marked by poverty and a tendency to cluster in ethnic neighborhoods, these newcomers found themselves prey to unscrupulous landlords and overcrowded housing which lacked adequate sanitary facilities. For another, immorality seemed to be rampant: drunk with power, politicians ran their offices on graft and corruption; employers unbound by health

[56] Mae M. Ngai, *Impossible Subjects: Illegal Aliens and the Making of Modern America* (Princeton, NJ: Princeton University Press, 2004), 18.

and safety regulations chased profits at the expense of their workers; and alcohol abuse, prostitution, and aberrant sexual practices flourished.

Observers noted with alarm what appeared to them to be the erosion of the very foundations of American culture. But the changes that were remaking society were influencing more than just demographics and American virtues; they were affecting the nation's health, as well. Diseases such as cholera and tuberculosis had the potential to cause serious problems in urban areas where poor immigrants lived cheek-by-jowl in unventilated, germ-infested tenements. For the middle and upper classes, the risks were less from acute, infectious illnesses than from longer-term conditions which affected one's nervous system or mental faculties. Beginning around 1870, physicians began to notice a new disease cropping up among their patients. A decade later, the illness, known as neurasthenia, had achieved near-epidemic status. As historian Gail Bederman has written, medical textbooks described the disorder as "'a lack of nerve force'…[that] resulted when a highly evolved person seriously overtaxed his body's finite supply."[57] More than just a mental illness, it manifested such somatic symptoms as headaches, muscle spasms, and a wide range of male sexual problems, including impotence and involuntary emissions. In the late 1800s, doctors also began to diagnose an increasing number of their female patients with chlorosis, an ailment marked by "poor appetite, gastric disturbance, and faint green pallor," as well as "intense lethargy," headache, and

[57] Gail Bederman, *Manliness & Civilization: A Cultural History of Gender and Race in the United States, 1880-1917* (Chicago: University of Chicago Press, 1995), 85.

dyspnoea, or labored respiration.[58] Although physicians had recognized the disease for centuries, rates of chlorosis in the U.S. peaked after about 1890.

The etiologic explanations for both chlorosis and neurasthenia illuminated the ways in which each disease served as a mirror for contemporary cultural concerns. Neurasthenia affected men and women who could not handle the changes associated with shifting gender roles. Many middle-class women moved from the home, where they had been safely ensconced for much of the nineteenth century, and into colleges, universities, and social and political work. Their exposure to "more demanding mental activity…drained their capacity to be healthy mothers," writes Bederman, and triggered the development of neurasthenia. For men, changes in the nature and organization of work provided new sources of stress. Frederick Jackson Turner's famous 1893 pronouncement of the end of the frontier echoed contemporary laments about the decline of the strenuous, rugged spirit associated with westward expansion. Whereas men had once tested their mettle against savage Indians, wild animals and an open, untamed landscape, now they passed their days in corporate jobs, a new class of middle managers who oversaw workers and business practices but who were not themselves directly involved in the production of goods of any kind. An earlier, more rugged masculinity was replaced by a system of Victorian ideals that stressed manly self-restraint and control. As a result, "the increased pace and technological advancement of modern civilization placed greater demands" on men "as businessmen and professionals," with devastating consequences for their health.[59]

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[58] Keith Wailoo, *Drawing Blood: Technology and Disease Identity in Twentieth-Century America* (Baltimore: Johns Hopkins University Press, 1997), 17.

[59] Bederman, 85-87.

As historian Keith Wailoo has written, a number of medical and social factors converged in the increased visibility of chlorosis at the end of the nineteenth century. First, advancements in diagnostic technology lent the disease a scientific legitimacy. The hemacytometer and heboglobinometer, new instruments that could be used to examine a patient's blood cells, medicalized chlorosis and conferred on it a changed identity that was a mark, in part, of one's hemoglobin level. It could now be diagnosed with a precise, scientific measurement rather than by relying on a physician's subjective observation of symptoms. Second, the same developments in the social status of women which put them at risk for neurasthenia also increased their susceptibility to chlorosis. To many physicians, the disease "merely highlighted the sad consequences of social change in women's lives, and most of this sad tale could be chronicled by examining the transformation of the blood." It revealed "the precise hematological dangers of freedom, capricious behavior, mutual female indulgence, and collective women's activities."[60] By 1920, the disease had all but disappeared, a victim of social reform, additional improvements in medical technology, the Progressive Era's focus on scientific, rather than moral management of disease, and the continued erosion of Victorian ideals of gender.

Cancer, like chlorosis and neurasthenia, exposed fears about the effects of refinement on the health of American men and women. In the 1920s and early 1930s, the discourse of civilization provided the primary framing mechanism for contemporary epidemiologic and etiologic research on the dread disease. Many noted that cancer rates appeared to correlate precisely with a particular society's level of intellectual,

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[60] Wailoo, 20, 26.

technological, and moral advancement. One observer, for instance, described a lower incidence in "wild people."[61] Frederick Hoffman, who was probably the most prolific writer on the subject of cancer in "uncivilized" peoples, stated that "native races…living under conditions of primitive life…are practically exempt from cancerous affections, though non-malignant tumors may be and often are of common occurrence."[62]

As historians including Bederman and Matthew Frye Jacobson have argued, the discourse of civilization in the late nineteenth and early twentieth centuries invoked a particular configuration of race, gender, and economics. By its very nature, civilization bore an imprint of race; at its core, it was an idea that could be used to divide the world by level of progress. It measured achievement along a continuum which placed people living in states of barbarism at one end and Europeans and Euro-Americans, who had reached the pinnacle of refinement, at the other. Civilization, writes Bederman, was an explicitly racial concept that "simultaneously denoted attributes of race and gender" and "conflated biological human evolutionary differences with moral and intellectual human progress."[63] Jacobson adds that the term had long been steeped in "a dense weave of ideas and assumptions regarding…proper comportment, manners, social bearing, and Judeo-Christian belief." It was, moreover, an economic concept, ranking "diverse peoples' ways of life according to a hierarchy of evolutionary economic stages," including "the fundamental social issues of property relations, the distribution of wealth,

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[61] Francis Carter Wood, *Cancer: Nature, Diagnosis, and Cure* (New York: Funk & Wagnalls Company, 1937), 17-18.

[62] Frederick L. Hoffman, *Cancer and Civilization; An Address by Frederick L. Hoffman, LL.D., Read Before The Belgian National Cancer Congress, Brussels, November 18-19, 1923* (USA: Prudential Press, no date), 5; in FLH NLM, 1916-31, v.2.

[63] Bederman, 23, 27.

modes of production, and patterns of consumption."[64] Thus, "the superiority of civilized society itself" could serve as a justification for the taking of lands occupied by "savages," transforming such populations not solely through contact with a more advanced nation, but also by creating a market for American and European consumer goods.[65]

By the 1920s, the link between skin color and civilization was beginning to fracture. "Civilization" had come to refer more generally to culture, invoking biological race only implicitly. Whereas at the end of the nineteenth century race could be expressed plainly through the use of the term, now "civilization" largely alluded to a group's cultural attributes. These societal features were, of course, determined by the physical attributes of race, but they also took precedence over them. The subtle shift in the meaning of "civilization" occurred in part because the notion of race had itself changed in the intervening years, from a view in the late nineteenth century "which centered on physiognomic difference and hierarchy," as historian Mae M. Ngai notes, "to twentieth-century racial ideas that linked race to both physiognomy and nationality."[66] Race, in other words, denoted not only a collection of physical, bodily markers, but also an associated series of habits and customs that could be traced to one's ancestry. It both retained its evocation of phenotypic distinctions and added a behavioral element that brought it closer to the modern notion of ethnicity. The emphasis on culture can also be attributed to the influence of Franz Boas, who had been advocating a cultural basis to racial difference since the early years of the twentieth century. His perspective on

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[64] Matthew Frye Jacobson, *Barbarian Virtues: The United States Encounters Foreign Peoples at Home and Abroad, 1876-1917* (New York: Hill and Wang, 2000), 50.

[65] Jacobson, 51.

[66] Ngai, 8.

"cultural environmentalism," as George Fredrickson called it, was attracting followers among anthropologists in the 1920s and had been adopted by the sociologists of the Chicago School, among others.[67] In the years ahead, the primacy of biological race would fade even further. Jacobson writes that between 1924 and the post-WWII period, "culture eclipsed biology as the prime determinant of the social behavior of races….Strictly biological understandings of race as the key to the diversity of humanity gave way to cultural and environmental explanations."[68] In the early 1920s, however, most scientists continued to post an association between color and culture.

As with other pronouncements about the nature of cancer, the alleged link between rates of incidence and degree of civilization sparked a vigorous debate. Experts could not even agree on whether morbidity was in fact lower in "primitive" races. For every observer who insisted that the uncivilized were less likely to fall victim to the disease, another one speculated, as H. Gideon Wells did in the *American Journal of Cancer*, that the "supposed increase in cancer among primitive peoples when they are in contact with modern civilization…may well be only a matter of civilization revealing the cancers, not causing them."[69] The author of an ASCC handbook for physicians declared that medical professionals had in fact detected the disease among "uncivilized" populations.[70] "Those who point to the low cancer rates existing among primitive

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[67] See Fredrickson, 315; Matthew Frye Jacobson, *Whiteness of a Different Color: European Immigrants and the Alchemy of Race* (Cambridge, MA: Harvard University Press, 1998), 98-99; Henry Yu, *Thinking Orientals: Migration, Contact, and Exoticism in Modern America* (New York: Oxford University Press, 2001), 45.

[68] Jacobson, *Whiteness of a Different Color*, 98-99.

[69] H. Gideon Wells, "The Nature and Etiology of Cancer," *American Journal of Cancer* 15, 3 (July 1931): 1949.

[70] Raymond Voorhees Brokaw, *Cancer Compend: A Handbook for Speakers* (New York: American Society for the Control of Cancer, 1932), 32.

peoples," wrote another, "neglect to call attention to the fact that preventive medicine is itself a triumph of modern civilization."[71] Each side claimed to have the weight of scientific evidence on its favor, and each side denounced the other for favoring anecdotal data over rigorous experimentation.

To test the theory that cancer was a disease of civilization, researchers scrutinized "primitive" populations, hoping that whatever they learned about racial resistance to cancer would translate into useful insights about preventing the disease in the bodies of the civilized. The first step lay in determining which populations lived cultivated lifestyles and which could be considered primitive. To most, it was a relatively easy matter; one needed to look no further than skin color to establish where a particular group fell along the hierarchy of achievement, as the idea that race determined culture still prevailed. In general, "civilization" signified white, Euro-American culture and values; the rest of the world fell into the realm of the uncivilized. Nearly all investigators placed Africans, Mexicans, and Native Americans, including "Eskimos," in the latter category. Some added to the mix Japanese, Chinese, and "Hindus," while others, citing each nation's long history and rich cultural legacy, objected to the inclusion "in this group [of] races of vastly older civilization than our own."[72] For the most part, researchers considered African Americans to be uncivilized, although many claimed that their longtime residence in the United States, a civilized country, had brought numerous benefits to the population's health and well-being, the accumulation of which had only accelerated after emancipation. Frederick Hoffman's *Cancer in Native Races* provided

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[71] Madge Thurlow Macklin, "Is The Increase of Cancer Real or Apparent?" *American Journal of Cancer* 16, 5 (Sep. 1932), 1204-05.

[72] H. Gideon Wells, "The Nature and Etiology of Cancer," *American Journal of Cancer* 15, 3 (July 1931), 1948.

the most comprehensive division of the world by level of civilization. He counted all "non-European races" among the uncivilized, including Maoris, native Hawaiians, Burmese, Algerians, Egyptians, Cubans, East and Southeast Asians, and residents of Greenland and Iceland.[73]

Despite minor squabbling over the precise boundaries of the terms "civilized" and "uncivilized," no one in the medical community seemed to question the ideological underpinnings of each category; that some nations had indeed progressed faster than others seemed to be an indisputable consequence of history that could be confirmed by no more than a cursory glance around. Those who saw civilization as an etiologic factor in carcinogenesis were essentially saying that the illness's reach was limited to Caucasians. If it were a disease of civilization, then white Americans and Europeans were the only ones who were civilized enough to have to worry about it. The various tribes and nonwhite populations living in undeveloped areas without government, art, or Christianity still trusted folk remedies and faith healing. Since they had not yet attained a sufficient level of social and cultural development to embrace industrialization and modern medicine, they could not possibly suffer from an ailment brought on by luxurious living.

Within the literature on cancer in the 1920s and 1930s, the use of the term "civilization" did not necessarily reflect a sense of biological advantage, with the exception of the apparent immunity of black Africans and African Americans to skin cancer, a relationship that was beginning to garner scientific attention at the time. When Hoffman, for one, wrote about the rarity of cancer among people living under the

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[73] Frederick L. Hoffman, *Cancer in Native Races; An address delivered in substance before the American Association for Cancer Research, Albany, N.Y., April 1, 1926* (USA: Prudential Press, no date).

"conditions of primitive life," he was not describing biological distinctions but ethnic practices that corresponded to a group's level of refinement. Classifying societies within a framework of civilization did treat whites differently from nonwhites, but it honored them for their achievements, and not simply because they were white.

Still, observers seeking to establish a connection between cancer and civilization assumed that a society's degree of intellectual and technological achievement was determined largely by skin color, in effect overlaying culture with race. This viewpoint, of course, was simply a continuation of earlier beliefs regarding race and refinement. The hierarchy of civilization had always broken down along racial lines, and many would undoubtedly have argued that skin color itself was what enabled Europeans and their descendants to build societies that contrasted so sharply with those of "primitive" populations around the globe. The idea that progress could be measured in racial terms was hardly noteworthy, as European and Euro-American critics had been evaluating nonwhites by Western standards for centuries. While Americans ate processed foods and rode on trains and in automobiles, various nonwhite peoples still grew their own vegetables, hunted their own game, and relied on non-motorized forms of transportation. What was new was that observers were now using the *absence* of a chronic disease in a particular group as an additional way to underscore that population's cultural and social failures.

As the meaning of the term "civilization" within cancer research continued to associate cultural practices with disease levels, physicians began to describe connections between specific races and types of cancer. Two examples appeared frequently in both medical literature and popular publications starting in the early 1930s. Doctors noticed

that men and women in China suffered from cancers of the digestive system at unequal rates, a disparity which they attributed to cultural norms dictating a specific household status for each gender. "Chinese men have cancer of the gullet because they eat their rice at the first table when it is very hot while Chinese women never have it because they do not get theirs until the second table when the rice is cold," proclaimed an article entitled "What You Should Know About Cancer," which appeared in *Hygeia*, the American Medical Association's general-interest magazine.[74] Chinese men, wrote William J. Mayo, had elevated instances of pharyngeal and esophageal cancers due to their habit of "throwing it [rice] with some force by means of chopsticks into the mouth and pharynx."[75] The same two articles were among many that detailed what was known as "Kangri-burn cancer," named after a portable firebasket used by people in Kashmir, India. The basket contained an earthenware pan filled with hot charcoal, which residents wore under their clothing to keep warm during the winter. The practice resulted in high rates of "cancer just above the pubis" and "cancer of the skin of the abdomen."[76]

The instances of cancer in Chinese and Kashmiris were frequently cited in American public health materials as warnings against chronic irritation from a source of heat—rice in the case of Chinese men and burning coals for those in Kashmir. Investigators noticed additional associations between race and cancer that seemed to result from cultural habits. Japanese women, who traditionally breast-fed their children longer than European or American women, experienced a lower incidence of breast

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[74] George Gray Ward, "What You Should Know About Cancer," *Hygeia* 14 (Sep. 1936): 823.

[75] William J. Mayo, "Susceptibility to Cancer," *Annals of Surgery* 93, 1 (Jan. 1931), 17.

[76] Mayo, 17; Ward, 823. For a more detailed description of Kangri-burn cancer, see Frederick L. Hoffman, *Cancer in Native Races*, 33.

cancer.[77] Penile cancer struck Jewish men less often than their non-Jewish counterparts, while Jewish women had a reduced prevalence of uterine cancer, both statistical anomalies that were attributed to the religious practice of circumcision. Whether most observers would have listed these groups alongside "the most effete residents of New York or Paris, London or Sauk Centre" or with "American Indians, African Zulus, and Australian bushmen" seemed to matter less than the discovery that cancer indeed afflicted nonwhites, and at rates that were sometimes lower, and sometimes higher, than in the general American population.[78] Within the framework of civilization, the primary category of analysis had shifted entirely to culture.

The discovery of relationships between certain forms of cancer and the racial "types" of Chinese, Indians, Jews, and Japanese brought to light flaws in earlier assumptions which directly correlated cancer rates with skin color and its proxy, civilization. It also served to mark these groups as civilized, as they had evidently reached a level of refinement sufficient to produce a disease brought on by a luxurious lifestyle. In the following years, articles in the medical literature continued to move away from the dichotomy of "civilized" and "uncivilized." By the mid- to late 1930s, researchers for the most part had abandoned the notion of civilization, recognizing the ubiquity of cancer even in groups once designated as "primitive." In the words of one set of authors writing in 1940, the issue had become a "mooted question."[79] But even though

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[77] See Madge Thurlow Macklin, "Human Tumours and Their Inheritance," *Canadian Medical Association Journal* 27, 2 (Aug. 1932), 183; M. Pinson Neal, "Cancer Viewed as a Preventable Disease," *Journal of the Missouri State Medical Association* 33, 11 (Nov. 1936), 411.

[78] Tobey, 19.

[79] Quinland and Cuff, "Primary Carcinoma in the Negro: Anatomic Distribution of Three Hundred Cases," *Archives of Pathology* 30, 1 (July 1940), 401-402.

clinicians confirmed that all people, regardless of race, could exhibit signs of cancer, they still hadn't solved the puzzle of the disease's racial distribution. Anecdotal evidence, which continued to suggest differential rates of the disease in discrete populations, led scientists to seek new answers to an old question: why did some individuals came down with cancer, while others of similar racial or ethnic background, under equivalent conditions, remained free of the disease? What kinds of variables could reasonably explain the uneven distribution of cancer among different groups, both white and nonwhite?

The statistics, meanwhile, were grim. Scientific progress in finding effective treatments could not keep pace with what appeared to be dramatically increasing rates of cancer among white Americans. Census reports and figures compiled by Frederick L. Hoffman estimated the death rate at 62.9 per 100,000 in 1900, about 100 per 100,000 in 1920, and 120 per 100,000 in 1940.[80] A failsafe cure could be years or even decades away. Alarmed, a number of observers suggested a program of eugenics among cancer-prone individuals to reduce the likelihood of the disease among future generations. One researcher declared that it was "rational and logical to apply preventive measures of eugenics to this problem."[81] Emil Holman, writing in 1938, agreed. "[T]he multiple occurrence of cancer in a family or in two succeeding generations is sufficient medical and legal evidence to warrant sterilization when desired, or abortions when necessary.

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[80] Patterson, 79, 95.

[81] Alfred Scott Warthin, "Heredity of Carcinoma in Man," *Annals of Internal Medicine* 4, 7 (Jan. 1931): 696.

The question arises also whether, ultimately, for the good of the race, compulsory sterilization, might in certain instances, be desirable."[82]

The calls for eugenic measures or reproductive caution among those from "cancer families" never reached the levels of decades past, when a loosely organized movement both inside and outside the scientific community encouraged social engineering through selective breeding. Drug addicts, criminals, the feeble-minded, the poverty-stricken, and numerous nonwhites, including Jews, Asians, and Eastern Europeans, could be controlled, eugenicists thought, by promoting policies of enforced sterilization. Although only a handful of scientists appeared to take seriously a policy of eugenics as a judicious approach to the problem of cancer, that they advocated it at all indicated the lengths to which they thought society should go in order to exercise some level of command over the seemingly uncontrollable affliction.

"Natural" Categories: Statistics and the Definition of Race

The discourse of civilization reflected primarily the cultural aspects of what race represented in the 1920s and early 1930s. Race, however, also denoted physical attributes, which provided scientists with another way to classify their subjects. The medical profession gradually shifted away from the use of the term "civilization" in the 1930s, but the underlying issues remained the same. Cancer was not the sole preserve of Caucasians; it could, and did, affect nonwhites. But were cancer rates for these groups higher or lower than those found among white Americans? Was the disease on the rise among blacks and other people of color? Which organs were the most affected? Overall,

[82] Emil Holman, "Concerning the Hereditary Nature of Cancer," *Surgery, Gynecology and Obstetrics* 66, 2 (Feb. 1, 1938): 248.

available evidence seemed to show that "coloreds"—a group that nearly always meant African Americans, but sometimes included Mexicans, Asians, and Native Americans, as well—had lower rates both of the disease itself and of mortality from cancer than whites did. One study found a rate of 83.09 cancer deaths per 100,000 for whites in 1928; the rate for American Indians in the same year was 30.52.[83] The contrast was especially striking for skin cancer, a disease that particularly affected fair-skinned blondes and redheads, but to which blacks seemed to be essentially immune, their highly pigmented skin apparently a racially-based protective mechanism.

Although some researchers managed to establish, within available parameters, the rough, relative rates of disease in whites and blacks, the task of coming up with precise data on how cancer affected other racial groups proved more challenging. The difficulty rested on two issues: the dearth of reliable, relevant statistics and inconsistencies in how the medical profession defined race. To concerned observers, access to dependable statistics was essential to understanding the epidemiology of cancer. Worthwhile figures on whether its rates were increasing, its relationship to other diseases, and its relative frequency in organs, tissues, and racial groups could only be collected through statistical studies. But these surveys suffered from their own problems of conception and design, ranging from confusion regarding the difference between crude and adjusted rates to small sample sizes, lack of standardization of racial categories, improper collection of data, and questions about the trustworthiness of cancer diagnoses. "The careless use or the incomplete collection of figures misleads to an extent as to seriously impair any investigation," wrote Harold B. Wood in 1932. In order to determine whether deaths

[83] E. Payne Palmer, "Cancer Among the Indians of the United States, with an Analysis of Cancer in Arizona," *Southwestern Medicine* 22, 12 (Dec. 1938): 483.

from cancer were increasing, he called for studies that isolated a single organ and adjusted for "the oft-termed cancer age." Hospital records, the mainstay of many articles, revealed nothing about rates of the disease in the general population. Instead, Wood encouraged a heightened level of precision. "Let us then be more careful of our statistics; compare comparable factors; give definitions which define; report figures which correctly confirm, deny or explain conditions."[84] The results could go a long way toward furnishing truthful and accurate reports that would prove useful to colleagues.

Wood's pronouncement, however, was more effective as rhetoric than as practice. The state of medical knowledge in the 1930s necessitated that most data on cancer incidence be culled from death certificates. While this seemed to be a useful way to determine whether mortality from cancer was on the rise, reliance on such statistics was in fact fraught with problems. Doctors could not always isolate cancer as the cause of death, especially since not everyone in the 1930s distinguished between benign and malignant tumors. An individual whose name appeared on the cancer death rolls might have died from a completely unrelated ailment, only to have his death attributed to the dread disease after a pathologist discovered a neoplasm during an autopsy. Furthermore, using death certificates as one's main data source meant that a person with cancer did not become statistically significant until he or she died. Awareness that more people were dying from cancer might bring a new urgency to educational and fundraising efforts, but it did little to help those who were actually living with the disease. As many physicians realized with alarm, each new victim, regardless of whether cancer had been diagnosed

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[84] Harold B. Wood, "Reliability of Cancer Statistics," *American Journal of Surgery* 18, 1 (Oct. 1932): 31-35.

during his or her lifetime or at death, represented someone who had been failed by medical science.

If the conclusions that could be drawn from epidemiologic investigations into cancer were limited by a heavy reliance on death records, they were also complicated by shifting racial categories. Some clinicians considered "Negroes" to be American blacks only, while others included blacks in Africa and the Caribbean, as well as mixed-race blacks. "Colored" usually just meant African Americans, but at other times it incorporated a hodgepodge of nonwhites. Of course, since blacks constituted nearly all of the nonwhite population in the United States at the time, using the category of "colored" as a shorthand way to represent African Americans would not, in most cases, lead to wildly inaccurate results. Nationwide, the numbers of Asian Americans, Latinos, and Native Americans were probably too small to merit their inclusion in general studies; researchers did, however, give these groups special attention if a statistical anomaly, such as an unusually high rate of a certain type of cancer, piqued their curiosity. But the lack of standardized racial categories made more difficult the task of amassing accurate information. If one physician's "Negro" patient was another one's "colored," while a third failed to organize his records by race at all, then any data gleaned from these doctors might be so inconsistent or even contradictory as to render it useless.

Although still in its infancy by the standards of a discipline such as mathematics, which had been around for thousands of years, by the 1930s the legitimacy of the field of statistics was well established. Historians and other scholars have written extensively on the linkages between early statisticians and the growing eugenics movement in Europe and the United States in the late nineteenth and early twentieth centuries. Indeed, Francis

Galton, a cousin of Charles Darwin and the founder of eugenics in the 1860s, came to statistics by way of his studies of heredity; he spent years, as Daniel J. Kevles notes, "trying to ferret out the laws of inheritance."[85] Galton was concerned, as were many eugenicists, with pinpointing the factors that led to alcoholism, criminality, sexual deviance, and other forms of degeneracy that seemed to be perilously on the rise. The nascent field of statistics offered a methodology that could be used to provide a scientific quantification of the surrounding world. Convinced that statistics must "incorporate the theory and methods of mathematical probability," Galton transformed the field from the "mere data gathering" of the mid-nineteenth century to a science that enabled practitioners to think in terms of both causation and correlation. As Karl Pearson, Galton's biographer and a eugenicist in his own right, wrote in 1930, "Thousands of correlation coefficients are now calculated annually….[T]hey form…the basis of investigations in medical statistics, in sociology and anthropology." Galton's work had "not only enormously widened the field to which quantitative and therefore mathematical methods can be applied, but it has at the same time modified our philosophy of science and even of life itself."[86]

The power of the field of statistics stemmed from its applicability in interpreting the natural and social worlds. It offered a scientific language through which to address important social issues and lent scientific authority to a process that practitioners could use to identify the weak and undesirable elements of society. Statisticians measured the circumference of human skulls, the length of limbs, and the height of men and women in

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[85] Daniel J. Kevles, *In the Name of Eugenics: Genetics and the Uses of Human Heredity* (Cambridge, MA: Harvard University Press, 1985, 1995), 13.

[86] Kevles, 17.

the hopes of discovering numerical correlations with various forms of degeneracy. Others made use of the I.Q. test, a new tool which emerged around World War I, to assess the intelligence of segments of the population. More often than not, their results broke down along lines of ethnicity, class, and race. Statisticians could, to be sure, put their skills to less controversial use by calculating, for instance, the probability of wrinkled seeds among generations of pea plants. But the concept of "standard deviation" measured just what its name suggested: the extent to which an individual deviated from a standard. With respect to calculating human intelligence or head size or one's likelihood of committing a crime, experts invariably used as their model someone who resembled themselves. Thus, white males of Western or Northern European heritage became the scientific norm for statistical studies of physical and mental attributes.

The influence of statistical thinking on social policy in the United States in the first decades of the twentieth century owed much to the trust in science and expertise that characterized the Progressive Era and years following. In the wake of World War I and the often extreme nationalist sentiment that it engendered, many were eager to reestablish an American identity that included components of character and race. The Reed-Johnson Immigration Act of 1924 offers an instructive look at the weight which policymakers gave statistical science at the time. The legislation created a system of quotas by using categories of the Census Bureau to first divide the world's populations by nationality or race, and then rank each group according to a hierarchy of desirability. In her discussion of the debate over the bill, Mae M. Ngai observes that Congress considered the Census Bureau's categories to be "objective divisions of an objective reality."[87] The

[87] Ngai, 29.

classifications of the census "created a vocabulary for public discourse on the great social changes taking place in America at the time—industrialization, urban growth, and, of course, immigration," and would shape the demographics of the U.S. for the next four decades.[88]

Despite the pitfalls for researchers seeking precise numbers on cancer morbidity and mortality, statistics did serve both to rationalize and to reinforce racial categories. Combining all blacks into one group, for instance, split the world by level of pigmentation, eliding the national, cultural, and social differences between African Americans and blacks from other countries. Such an approach assumed that skin color trumped environment, that similarities between blacks worldwide took precedence over any local variations in diet, housing, or health care. At the same time, the category of "colored," when it was used as a catchall term for nonwhites, created a divide between Caucasians on the one hand and everybody else on the other; it both reified skin color as a legitimate criterion by which to define groups and naturalized differences among populations that were based on it. Statistics also provided a medical foundation to "racial" distinctions that rested not just on skin color and other physical traits, but also on cultural ones. A study which showed that "whites," however the group was defined, were less likely to suffer from liver cancer than "blacks" validated the idea that races differed from one another in ways which were scientifically measurable. Uneven rates of cancer in whites and blacks offered proof that "white" and "black" were reasonable medical designations, and legitimized the categories as logical ones for medical research.

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[88] Ngai, 29-30. The point about census categories is from Margo Anderson's *The American Census: A Social History* (New Haven: Yale University Press, 1988), as referenced by Ngai.

Within the field of cancer research, the most prominent statistician working in the 1920s and early 1930s was Frederick L. Hoffman, who was by then in his final years of a long career with the Prudential Life Insurance Company. Born in Germany in 1865, he spent his young adulthood apprenticing, at the behest of his parents, at a number of European companies, including a mercantile firm. Intent on both pursuing his longstanding interest in science and traveling to America, he ignored his parents' pleas to become a businessman and departed for the United States in 1884. He gained his citizenship six years later. Hoffman held neither a medical degree nor a doctorate in a biomedical field, and, although in the 1880s he snuck into a handful of mathematics classes at Western Reserve University in Cleveland, Ohio before he was discovered and thrown out, he did not finish college. His knowledge of statistics was apparently self-taught.[89]

For insurance companies such as Prudential and the Metropolitan Life Insurance Company, the keeping of accurate statistics was central to survival. If their main purpose was to provide monetary compensation to surviving family members upon the death of a policyholder, then it was in their best interest to figure out which types of people constituted the gravest insurance risks. Theoretically, statistics on the health and mortality of various groups could be used to determine which populations should be excluded from coverage. If Chinese in San Francisco or blacks in the American South seemed to be particularly unhealthy, exhibiting high rates of tuberculosis or other diseases, including mental disorders, then a company could deny insurance to individuals

[89] For additional biographical details, see Ella Hoffman Rigney, *Frederick L. Hoffman*, no publisher, no date. This biography, written by Hoffman's daughter some time after 1971, fills four looseleaf binders in Box 31, FLH Papers. See also Frederick L. Hoffman, *The Life Story of a Statistician, 1865-1884*, no publisher, no date; Box 9, FLH Papers.

from these groups based on an evaluation of risk. Ascertaining the health of specific populations, then, could enable a life insurance company to protect its resources by pre-screening applicants based on a combination of ethnicity, class, race, and health.

The assessment of risk, in fact, was a major element of the new epidemiological model of disease. It marked a shift not only from the centuries-old configuration of infectious and communicable illnesses, but also from the perspective, common before the 1920s and 1930s, that chronic ailments were unavoidable degenerative diseases, the byproducts of aging and the signs of a body breaking down. Now, conditions such as cancer and heart disease were seen as afflictions which, with proper detection and treatment, could be managed as long-term illnesses. The insurance industry, as medical historian Jeremy A. Greene has argued, was instrumental in promoting the new model of risk assessment. "By the early twentieth century," he writes, "companies like Metropolitan Life Insurance were able to use their own claims records as large data sets to measure physiological variables against mortality. In doing so, the insurance industry recast physiology in terms of *risk*, a term with specific financial connotations that the insurance industry had developed to analyze broad population-based policies in quantifiable terms."[90] The resulting studies provided scientists with a substantial source of data from which conclusions could be drawn about the causation of and mortality from various diseases.

Frederick Hoffman, for his part, believed that statistics were of little use unless interpreted correctly, an occurrence which happened less often than he found

[90] Greene, 12. Emphasis in original.

acceptable.[91] "The most perilous though most common practice and habit of thought on the part of even men in very responsible positions," he wrote, "is to guess at the truth and give utterance to guesswork opinion. Yet guessing is at all times unjustifiable when the facts are available and possibly readily accessible to contradict a statement which should not be uttered as a matter of opinion."[92] To Hoffman, statistics represented "the facts, as they have been collected in an orderly fashion."[93] His concern was not that people would manipulate them to rationalize untruths, but that wrongful interpretations might produce misleading outcomes. He trusted that there was a single, correct way to interpret a set of statistics; this would be, presumably, his own.

When Hoffman arrived at Prudential in 1894, its rating system was modeled on those used by companies in England. Finding it unsuited to social conditions in the United States, he set about revising it. In the early 1900s, much of his work involved tuberculosis, a subject, he wrote, "of very considerable financial importance, on account of the heavy losses sustained from tuberculosis during early years of life."[94] Through studying mortality records and death certificates, Hoffman determined that people who died from tuberculosis were often of low body weight and concluded that an individual who carried too few pounds on his or her frame was predisposed to the disease. He found, moreover, that for insurance purposes the healthiest males were between 5'5" and 5'10", and weighed somewhere between 130 and 169 pounds. In light of his discoveries

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[91] Frederick L. Hoffman, *Utility and Limitations of Statistics; An address delivered before the student body of the Babson Institute, Babson Park, Mass.*, no publisher, no date, 1.

[92] Ibid.

[93] Rigney, iv.

[94] FLH to Dr. Leslie D. Ward, dated May 31, 1910, Box 1, FLH Papers.

regarding body weight and tuberculosis, he recommended "a more severe standard of minimum weight" to "successfully eliminate a fair proportion of applicants predisposed to consumption and perhaps also to respiratory diseases."[95]

Despite his unshakeable faith in statistical truth, as well as what seemed to be an ironclad finding that low body weight indicated a predisposition to tuberculosis, Hoffman's conclusions were questionable. In mining mortality records to create a list of characteristics of those who suffered from tuberculosis, he confused the causes of the disease with their consequences. Victims of tuberculosis may, in fact, have died at weights significantly lower than those found among healthy adults, but the disease was known as consumption for a reason. Medical texts from the nineteenth century described "'frequent and harassing'" coughs that later developed into "'hollow rattles' and 'graveyard coughs'" as "[a]n initial 'ruddiness' of the face gave way to a 'deathlike paleness.'"[96] Tuberculosis, writes historian Howard Markel, "consumes with a passionate and incisive energy, slowly, inexorably devouring the very structure of the lungs and other critical organs with the single goal of conquering its host."[97] Faced with such a collection of symptoms, maintaining an adequate caloric intake was probably not foremost in a patient's mind. Hoffman's discovery of a correlation between tuberculosis and low body weight may have been genuine, but his assignation of cause and effect was not. It was as though he had first announced that people who drank red wine were less

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[95] FLH to John F. Dryden, dated August 2, 1910, Box 1, FLH Papers; FLH to Dryden, dated May 31, 1910, Box 1, FLH Papers.

[96] Sheila M. Rothman, *Living in the Shadow of Death: Tuberculosis and the Social Experience of Illness in American History* (New York: BasicBooks, 1994), 4.

[97] Howard Markel, *When Germs Travel: Six Major Epidemics That Have Invaded America and the Fears They Have Unleashed* (New York: Vintage Books, 2005), 27-28.

likely to suffer from heart disease than teetotalers or consumers of other alcoholic beverages, and then ascribed the differential to an unknown quality in the wine itself, without taking into account the social and cultural factors that might make an individual more likely to imbibe red wine in the first place.

The methodology that Frederick Hoffman followed in his early work on tuberculosis established a pattern to which he would adhere for the rest of his career. Because he lacked biomedical training, he could not participate in clinical studies. He did not have access to the latest technologies by which to diagnose or treat cancer; even if he had been able to get his hands on a supply of radium or a Roentgen ray machine, he had no patients on whom he could try various therapies or from whom he could gather data for case reports. Rather, Hoffman amassed a broad range of anecdotal evidence by drawing on personal correspondence and interviews with physicians who were working with nonwhite populations. He went on several major research trips throughout North and South America under the auspices of the Prudential Life Insurance Company. Finally, mortality records, one of the few available sources of statistical information on cancer, provided him with a large body of data. Together with the material he collected on his travels, it provided the foundation for his numerous articles on cancer. He was easily the most prolific writer on race and cancer in the first third of the twentieth century, publishing articles on cancer in Hawaii, Mexico, British Malaya and the Philippines, and the American South, and delivering lengthy addresses on "Cancer and Civilization" and "Cancer in Native Races" before audiences of cancer researchers.[98]

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[98] See, for instance, Frederick L. Hoffman, *Cancer and Civilization*; Frederick L. Hoffman, *Cancer in Native Races*.

Hoffman's interest in "Negroes," Mexicans, and other nonwhites stemmed, in part, from what he identified as a dearth of "facts" and vital statistics about these populations. He capitalized on his immigrant status, believing that it conferred on him a sense of legitimacy; as an outsider, he considered himself less biased about questions of race than were American-born writers.[99] Hoffman first encountered African Americans during a steamer trip to New Orleans on the Mississippi River not long after he arrived in the United States; years later, he recalled his astonishment at how poorly they had been treated by the white passengers and crew. At the Life Insurance Company of Virginia and later at Prudential, he discovered that neither business offered insurance to blacks; because of their high mortality rates, they were considered too great a risk. At the bequest of both Prudential and the National Commissioner of Labor, he began to investigate the economic, "moral" and "sociological state of Negroes" in order to determine their suitability for insurance. Much of his source material came from the Hampton Institute in Virginia, where he worked briefly, and from statistics compiled from the U.S. Army and from state boards of health across the country.[100]

The resulting book, *Race Traits and Tendencies of the American Negro*, appeared in 1896, published by the American Economic Association under the auspices of the Macmillan Company. Hoffman's daughter described the work as "the first compilation of facts regarding the Negro to have been published."[101] *Race Traits and Tendencies* predicted the eventual demise of African Americans, based on their high rates of

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[99] See Frederick L. Hoffman, *Race Traits and Tendencies of the American Negro* (New York: The Macmillan Company for the American Economic Association, 1896), v.

[100] Rigney, 90-118A.

[101] Rigney, 119.

mortality and infectious disease. As in his work on tuberculosis, he obscured the causes and effects of the low social position of African Americans. He concluded, for instance, that his subjects were inherently less intelligent, less healthy, and more dependent than whites while failing to acknowledge how a lack of access to education, health care, and other resources might have contributed to, and even produced, their condition.

Reviewers at the time dubbed *Race Traits and Tendencies of the American Negro* the most important book about blacks since *Uncle Tom's Cabin*. It had, in the words of a legal historian writing a century later, a "significant impact on American life," justifying, "among other things[,] massive racial discrimination in the insurance industry."[102] George Frederickson, who called Hoffman an "extreme racist" and "articulate philosopher of racial Darwinism," wrote that the book "became a prized source of information and conclusions for anti-Negro writers for many years to come and also had the practical effect of helping to convince most white insurance companies that they should deny coverage to all Negroes on the grounds that membership in the race by itself constituted an unacceptable actuarial risk."[103] Although in later years Hoffman would backtrack from the racial proclamations of his earlier work, his views moderated by time and experience, *Race Traits and Tendencies* both established his authority on and piqued his interest in the subject of the health and mortality of nonwhites, a topic to which he would return again and again over the course of his long career.

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[102] See Paul Finkelman's introduction in Frederick L. Hoffman, *Race Traits and Tendencies of the American Negro* (American Economic Association, 1896; Clark, NJ: The Lawbook Exchange, Ltd., 2004), i.

[103] George M. Frederickson, *The Black Image in the White Mind: The Debate on Afro-American Character and Destiny, 1817-1914* (Hanover, NH: Wesleyan University Press, 1971, 1987), 314, 249-250.

By using his expertise to gather and interpret statistics about mortality, Hoffman aimed to provide a foundation by which others could work on lengthening human longevity. To him, insurance constituted a form of public service, and it was therefore "not only logical but important that an insurance company should aid in a crusade against disease."[104] In 1925, Dr. Rudolph Matas, perhaps the leading researcher on the health and physiology of African Americans at the end of the nineteenth century and early years of the twentieth, praised Hoffman's work on the subject, calling his statistical studies "of immense strategical importance in guiding us to the points of attack where the enemy seems to be most securely entrenched and where the conditions seemingly best suited to his existence are to be found." It is possible, Matas continued, "that by continued analysis and intensive study of the life habits of the cancer population, the true nature of these conditions may be revealed and in this way a more certain mode of prevention may be obtained than we now possess."[105]

Hoffman occupied a central position in the field of cancer research in the period before World War II. He was one of the founding members of the American Society for the Control of Cancer and served as the chair of its Committee on Statistics.[106] By his own account, he published 1,200 articles during his lifetime, about one hundred of which were on cancer, and amassed a library of over 100,000 items on the disease.[107] Hoffman's articles reached the desks of William Howard Taft and Irénée du Pont, a former head of the chemical company bearing his name. In 1950, researchers were still

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[104] Ibid., v-vi.

[105] Rudolph Matas to FLH, dated December 30, 1925, Box 26, FLH Papers.

[106] Rigney, 189-194.

[107] FLH to Mrs. Frances J. Rigney, dated February 5, 1942, Box 22, FLH Papers.

referencing *The Mortality from Cancer throughout the World*, Hoffman's monograph which had been published thirty-five years before, a testament to his continued stature within the field.[108]

The influence of Frederick Hoffman on the field of cancer research in the 1920s and following was a consequence of the convergence of a number of factors within the contemporary scientific struggle to understand the condition. First, his work took up urgent questions of etiology, including the roles of civilization, skin color, and behavior, categories which appeared to have an influence on disease patterns, although their definitions were fluid. Second, his association with the Prudential Life Insurance Company ensured him access to large data sets from the corporation's own records, and his background in statistics enabled him to make sense of these numbers. Finally, his position in the insurance industry placed him on the front lines of the ongoing recasting of physiology as risk, as Jeremy A. Greene puts it. If his work on rates of incidence and mortality of cancer in whites and nonwhites would play any role in the discovery of important details about cancer, researchers fervently hoped that it could both illuminate the causes of the disease and enhance public health efforts at preventing it.

Conclusion

The discussion of civilization as a carcinogenic agent would diminish even further after World War II, when researchers began to focus less on the etiologic effects of a group's behaviors and customs and more on the unchanging physical and physiological characteristics that served as identifying markers for blacks, whites, and Asians. It would

[108] See Herbert L. Lombard and Evelyn A. Potter, "Epidemiological Aspects of Cancer of the Cervix," *Cancer* 3, 6 (Nov. 1950), 960-968.

constitute a discursive shift from a search for cultural factors to an exploration of the roles of genetics in explaining varied susceptibility to cancer. Whereas earlier investigators had pointed to the wintertime practices of Kashmiris or the hygienic habits of Jews, they now looked for traits that differed by group—and which, they believed, were as inherent to one's chemical makeup as hair texture or eye color. In the process, they would ascribe a predetermined package of genetic material to a particular population, defined by either skin color, in the case of whites and blacks, or ethnicity, for Chinese.

For the time being, however, researchers continued to rely on statistics, as epidemiologic surveys were one of the few sources of available data on rates of cancer incidence. Myriad problems, including inconsistencies in the ways that subjects were grouped and multiple gaps in the collection of information, rendered the resulting numbers unreliable at best, and in many instances may have been explicitly inaccurate. Despite these difficulties, the principles that underlay such work served to both naturalize and rationalize the categories into which investigators organized their subjects. When doctors mapped percentages of whites and nonwhites with stomach cancer, for example, they upheld racial designations as legitimate medical distinctions. In doing so, they transformed social categories into biomedical ones, privileging distinctions between groups while obscuring those within them.

By the eve of World War II, the themes of cancer research and prevention for the next half-century were firmly in place. Doctors admitted that much remained unexplained about the disease at the same time that they highlighted the current state of knowledge and insisted that the medical profession was on the verge of a breakthrough.

Educational campaigns emphasized the role of personal responsibility, a familiar public health refrain, in identifying and responding to the early warning signs of cancer, and enlisted the individual in the battle to keep disease rates from rising. Finally, scientific awareness of racial variations in cancer incidence would lead researchers to seek understandings of race which could suitably explain the differentials. In the coming decades, the interplay of knowledge, responsibility, and patterns of incidence not only would shape the fields of cancer research and medicine, but also would influence racial discourse in the United States by aligning and realigning categories of race, biology, genetics, and culture in new and unexpected ways.

Chapter 2

Color, Complexion and Sunlight in the Development of Skin Cancer

Of all the organs that could be affected by the dread disease, cancer of the skin probably received the most early attention from the scientific community. For one thing, it tended to be visible on the body. A tumor on one's face or arms or abdomen might ooze pus or discolor the surrounding area, signs of good health gone awry that would be difficult to miss. A patient could ignore an external growth on his or her body—and plenty of people did, as medical journals were replete with photographs of neoplasms which had reached the size of golf balls or even softballs—but would have to do so willfully. In contrast, cancers of the lungs, stomach, and other internal organs were infinitely harder to detect, and cancer screenings were not a routine part of medical examinations. Doctors could utilize exploratory procedures like surgical biopsies for patients who displayed unusual bleeding or discharge, changes in bowel habits, or persistent indigestion, all of which were among the American Society for the Control of Cancer's seven early warning signs of cancer, but they generally did not think to perform such tests unless the disease had become symptomatic.

Skin, however, was easily accessible, allowing physicians in the 1930s to diagnose the illness through a routine examination. Moreover, the disease appeared to be both avoidable and highly treatable, characteristics that allowed it to mesh neatly with public health goals of prevention and early detection. In an era in which the etiology, diagnosis, and treatment of nearly every form of cancer remained murky and poorly understood, the prospect of meeting successfully the challenge of skin cancer provided an

opportunity by which medicine could set scientific progress back on track. The unique features of the disease, noted one physician, left "little excuse for failure."[1]

Skin cancer also displayed irregularities in its expression that provided researchers with a tremendous clue regarding its etiology: Caucasians appeared to be highly susceptible, while blacks rarely exhibited signs. Differential rates of racial incidence had first been observed at the end of the nineteenth century by Rudolph Matas, a physician whose work on the "surgical peculiarities of the Negro" influenced later cancer researchers, including Frederick L. Hoffman.[2] Investigators in the 1930s continued to make note of skin cancer's distinctive epidemiology. "Malignant melanoma," stated a physician from a cancer clinic in Atlanta, Georgia, "is an uncommon tumor in the negro, if the scarcity of reports on the subject is accepted as an indication."[3] "[S]ailors, farmers, and others whose countenances are more or less weather-beaten are prone to develop cancers of the face and lip," wrote another. "Negroes, with their pigmented skins, seldom have such cancers."[4] In published articles, skin cancer rates for whites generally exceeded those of blacks by about a factor of four, although rates sevenfold or more were not uncommon in some samples.[5] A study by the Metropolitan Life Insurance Company found that rates among its policyholders varied from less than

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[1] M.F. Engman, Jr., "Cancer of the Skin," *Weekly Bulletin of the St. Louis Medical Society*, 31 (1936), 103.

[2] Matas published an article entitled, "Surgical Peculiarities of the Negro," *Transactions of the American Surgical Association* 14 (1896).

[3] Everett L. Bishop, "Melanoma in the Negro," *American Journal of Cancer* 16, 3 (May 1932): 522.

[4] James A. Tobey, *Cancer: What Everyone Should Know About It* (New York: Alfred A. Knopf, 1932), 264.

[5] See, for example, Bishop, 522-526; W.A.D. Anderson, "Disease in the American Negro: I. Melanoma," *Surgery* 9 (Mar. 1941), 425-427.

one per 100,000 for "colored persons" to 2.4 per 100,000 for white men, a differential of about three and one-half times.[6]

The unique features of skin cancer—its accessibility, its epidemiology, and later, the ways in which it adhered to an established model of causation—gave the disease a visibility that would not be matched in other fields of oncology for years. In the 1930s, scientists investigating skin cancer were actively seeking to explain, rather than simply to observe, racial differentials in rates of incidence. It presented perhaps the clearest example of a racial disparity in a form of the dread disease. Consequently, researchers took a close look at the various physical, physiological, and environmental factors which they hoped could explain its causes. As research proceeded apace during the following decades, the ways in which doctors wrote about race would shift, and advancements in knowledge of the illness introduced new ways of defining and organizing racial groups.

The Disease in the 1930s

Physicians in the 1930s knew about two major types of cancerous skin tumors: epitheliomas and melanomas. Epitheliomas arose in the outermost, epidermal layer of skin and consisted of either a basal cell or a squamous cell growth. The former, which was usually found on the face "above the level of a line drawn through the upper lip to the lobe of the ear," began "as a small, shiny or scaly, gray, yellow or yellowish-red nodule." The area formed a scab that would fall off and reform; it bled on minimal impact, and looked to be of a firm consistence. These tumors grew slowly and were not

[6] *The Mortality from Cancer: A Study of the Experience Among the Industrial Policyholders of the Metropolitan Life Insurance Company, 1911 to 1930; Monograph 1 in a Twenty-Year Mortality Review* (New York: Metropolitan Life Insurance Company, 1935), 12, 75-76.

considered to be particularly dangerous. Squamous cell epitheliomas, which occurred either on the patient's face below the nose or somewhere on the body, began "as a warty patch or a crusted area" and carried more potential for harm.[7] In the disease's early stages, it presented similar to a basal cell carcinoma; later on, however, the edges of the growth would ulcerate and widen. Joseph Jordan Eller provided a description of the process in his 1939 textbook on skin tumors:

> …the central ulcer deepens and becomes excavated, forming a crater. The base of the ulcer is extremely irregular and may be covered with necrotic tissue. Pearl bodies may be expressed by manual pressure….The papillary type of squamous-cell carcinoma grows as a cauliflower-like tumor on a broad base…It may be covered with crusts and scales, which upon removal reveal a papillary surface which bleeds readily. The tumors are usually firm in consistence, often reaching the size of a man's fist.[8]

Melanomas, which developed in the skin's melanin-producing cells, sometimes formed from normal skin, appearing first as brownish spots, and then developing into darker nodules. At other times, melanomas arose from pre-existing nevi, or moles, which grew larger and darker and turned a blue-black color. These growths, according to Eller, had a tendency to bleed and form scales; ranging from pinhead- to apple-sized, they were usually found on a patient's face or lower extremities, particularly the toes, soles, and heels of the feet.[9] The primary characteristic which distinguished a melanoma from a basal-cell or squamous-cell epithelioma was, of course, its remarkable ability to metastasize. One physician dubbed malignant melanomas "one of the most dreaded and

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[7] Francis Carter Wood, *Cancer: Nature, Diagnosis, and Cure* (New York: Funk & Wagnalls Company, 1923), 27-29; Joseph Jordan Eller, *Tumors of the Skin: Benign and Malignant* (Philadelphia: Lea & Febiger, 1939), 250.

[8] Eller, 263-264.

[9] Eller, 417-421.

vicious of all tumors," feared for the way in which it metastasized at an early stage and spread rapidly through the body to both lymph nodes and vital organs.[10]

The medical community at the time recognized the theoretical distinction between benign and malignant tumors of the skin. Researchers understood that each type of cancer cell exhibited different types of behavior; whereas malignant growths were more likely to metastasize, benign tumors were generally harmless. In reality, distinguishing between malignant and benign cells under the microscope was not always a straightforward task. As Eller's textbook read, "benign tumor cells may show the same irregularities as are found in malignant new growths." Moreover, malignancies did not always metastasize, and at times even spontaneously healed, further confounding scientists who were already bewildered by the intricacies of the dread disease.[11]

Still, knowledge about cancer seemed to be proceeding briskly, leading many to believe that its moment of vanquishment might be right around the corner. In the 1930s, research on the etiology of skin cancer focused on the role of chronic irritation "as a frequent, if not entirely essential, cause" of all types of cancer.[12] The concept of chronic irritation had been introduced in the 1860s by Rudolf Virchow, an influential German pathologist. Virchow, who had coined the term "leukemia" to describe cancer of the blood, pioneered the idea that cancer cells arose from other cells, and not from foreign matter in the blood, bones, or any other part of the body.[13]

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[10] Wesley W. Wilson, "Cancer of the Skin in Florida," *Journal of the Florida State Medical Association* 36, 10 (Apr. 1950), 620-621.

[11] Eller, 13-14.

[12] Ira I. Kaplan, "New York City and the Cancer Problem," *Radiology* 17, 2 (Aug. 1931), 112.

[13] Adam Wishart, *One in Three: A Son's Journey into the History and Science of Cancer* (New York: Grove Press, 2007), 29-44.

With skin cancer, the role of chronic irritation appeared to be unambiguous. As Francis Carter Wood proclaimed in 1923 in a widely-referenced textbook entitled *Cancer: Nature, Diagnosis, and Cure*, "There is no type of growth which gives clearer evidence of the relationship between irritation and tumor production than does cancer of the skin."[14] Researchers agreed that mechanical irritation, or the abrading of a part of the body from constant friction, could produce the disease. Patients were advised against wearing tight clothing which might unduly rub against their body; they were told not to pick at any growths on their skin and to avoid accessories or garments that pinched at a body part. One had to exercise vigilance even in selecting one's eyeglasses, as an improperly fitting pair might lead to cancer. As James A. Tobey warned in a 1932 volume written for the general public, "[p]ersons who wear glasses should endeavor to secure frames for them which do not irritate the bridge of the nose, and tight glasses should never be worn regularly."[15] People had to especially vigilant about moles and raised bumps on the skin, as these presented a ripe opportunity for cancer to develop. "Moles may be irritated and become cancerous during the use of electric or caustic treatment by those who pose as beauty specialists," wrote Wood. "Have the tumor cleanly cut or leave it alone."[16] An educational pamphlet produced by the Connecticut State Board of Health recommended careful monitoring of the skin to avoid the precursors to cancer:

> The prevention of skin cancer lies in taking care of minor skin blemishes….Pigmented moles should be carefully watched and brought to the attention of a physician when they appear to grow or change their

[14] Wood, 29.

[15] James A. Tobey, *Cancer: What Everyone Should Know About It* (New York: Alfred A. Knopf, 1932), 65.

[16] Wood, 29.

> appearance. Any mole on the foot is particularly dangerous. Those existing on parts of the body which are irritated by the friction of clothing, or even by shaving, should be especially watched and a physician consulted as to the advisability of removal as a means of preventing the possible development of cancer.[17]

Convinced that skin cancer could develop at sites of previous trauma, physicians urged their patients to attend to any burns, scars, and lesions to ensure proper healing. Left untreated, these areas could grow rough and create optimal conditions for the formation of tumors. "The thick scaly areas often noticed on the hands and faces of older people sometimes develop into cancer," claimed the pamphlet from the Connecticut Board of Health. "The early symptom of cancer of the skin," it continued, "is a small painless lump which generally develops in some old scar or blemish, and which continues to grow and may ulcerate causing a sore."[18] Other precancerous abnormalities included Paget's disease and senile keratosis, the latter of which was a common condition in the elderly and in farmers, sailors, and others who had spent a lifetime working outdoors. Old x-ray burns, "cutaneous horns," cysts, and even, in some cases, mechanical pressure on the scarred stump of an amputated leg all carried with them the potential to form skin cancers.

Doctors had long surmised that chronic irritation from ultraviolet rays might play a role in the production of skin cancer. The evidence in the 1930s, however, was mostly speculative, based more on clinical observation than on experimental research. As a result, members of the medical community remained divided on the issue. For each physician like M.F. Engman, Jr., who described sunlight as "the commonest type of

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[17] Connecticut State Board of Health, *Cancer Prevention and Cure*, no publisher, 1937, 3-4.

[18] Connecticut State Board of Health, 3-4.

trauma to the skin" and a major factor in the production of skin cancer, a more cautious colleague decried the lack of experimental confirmation and admonished the medical profession for elevating anecdotal data to the level of scientific proof.[19] Furthermore, medicine's grasp of the disease was such that doctors often offered advice that subsequent research would directly contradict. An editorial in a 1936 issue of the *Bulletin* of the American Society for the Control of Cancer, for example, confidently denied any relationship between cancer production and exposure of "otherwise protected areas of…skin to direct rays of sunlight." "If such were the case," the article read, "it would be noticed at once and the news would spread like wild fire." The article did, however, note that the long-term effect of cumulative exposure was a topic well-suited to laboratory inquiry.[20]

Public health materials on skin cancer, such as Tobey's volume and the numerous educational pamphlets from organizations including the ASCC and the Connecticut State Board of Health, shared a common theme which would constitute the foundation of the cancer prevention message for decades to come: the notion of individual responsibility. Information on cancer that was intended for general audiences invariably put the onus on each person to monitor his or her body carefully and note any changes that might be considered early warning signs of cancer. If doctors were still struggling to improve methods of finding internal cancers that were not only reliable, but also allowed for detection of the disease in its initial stages, then at least progress could be made against skin cancer, which presented few such challenges. Skin tumors were both visible to

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[19] See Engman, Jr., "Cancer of the Skin," 103-104.

[20] "Sunlight, Ultra-Violet and Skin Cancer," *Bulletin of The American Society for the Control of Cancer* 18, 9 (Sep. 1936), 9-10.

patients and accessible to physicians. Successful treatments, even cures, were no futuristic fantasy; they were already available in the form of surgery and x-rays. Physicians, however, could do nothing for cancer patients who had waited too long before seeking medical help. While lack of money, denial, or a sense of shame about the disease might keep individuals out of doctors' offices, educational campaigns aimed to eliminate ignorance of symptoms as a variable. But successful abolition of skin cancer in the United States would require the public to partner with the medical community. As informational materials emphasized, it was the duty of each individual to educate himself or herself about the signs of cancer and take the necessary measures to prevent the disease.

Skin Color and Skin Cancer

Researchers investigating skin cancer in the 1930s noticed that the disease displayed specific racial patterns. Whites, especially pale, freckled types who sunburned easily, seemed especially susceptible. In contrast, nonwhites, particularly Africans and their descendants, tended to exhibit low rates of lesions. Cancer of the skin, wrote one physician, "is frequent in the white and rare in the brown or negro races."[21] Another cited lower rates of skin cancer in "[d]ark-complexioned races" than among Caucasians

[21] Francis Carter Wood, *Cancer: Nature, Diagnosis, and Cure* (New York: Funk & Wagnalls Company, 1937), 31.

and a practical immunity in "Negroes."[22] "It is of interest to note," read a textbook on cancer, "that malignant melanomas are exceedingly rare in the negro."[23]

Even though the medical community had essentially reached consensus on the starkly contrasting rates of skin cancer among whites and nonwhites, it had yet to understand precisely how to account for the disease's racial differential. Some simply conferred a racial immunity upon all nonwhites. But others went to great lengths to distinguish both between and among various "colored" populations, hoping to isolate patterns of incidence that might help them to understand better the ways in which skin cancer developed and spread.

Disease identity provided the logic by which researchers classified their subjects. Since skin cancer exhibited a racial differential, scientists organized groups along racial lines, which, for the most part, translated to skin color. Overall, blacks, or, more commonly, "Negroes," dominated the contemporary discussion of race and skin cancer. The term served as a catchall for Africans, African Americans, and Afro-Caribbeans, among whom the majority of researchers made no attempt to distinguish. Members of the Negro race traced a common ancestry to the African continent; they shared a number of physiological characteristics with one another, the most important of which was skin tone. In this formulation, race became a measure of one's pigmentation, effectively reducing it to color. Skin color was assumed to be racially predetermined, and as such, it constituted a true racial trait. Furthermore, those who saw all blacks as Negroes supposed that the skin tone of their subjects did not vary. Africans and their diasporic

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[22] William J. Hoffman, "Industrial and Occupational Cancer," *Bulletin of the American Society for the Control of Cancer* 19, 2 (Feb. 1937), 1.

[23] Arthur Purdy Stout, *Human Cancer: Etiological Factors, Precancerous Lesions; Growth; Spread; Symptoms; Diagnosis; Prognosis; Principles of Treatment* (Philadelphia: Lea & Febiger, 1932), 574.

counterparts shared with one another the racial characteristic of pigmented skin, which physicians generally presumed was dark in color, and certainly always darker than the skin of Caucasians. Regardless of whether they lived in England, Trinidad, Nigeria, or the United States, blacks retained their skin color over generations because it was an essential, immutable part of their anatomy; one could no sooner change the length of one's arms than alter the shade of one's epidermis. According to this perspective, skin color existed in a dichotomy of white and black. If an individual's tone could not be described as the former, then it was the latter.

The vast majority of skin cancer researchers in the 1930s adhered to this framework. They neither recognized the diversity of colors among blacks nor attempted to map the range of human skin tones along a spectrum of light to dark. Most identified just two races: black and white. But others added a third group, which they dubbed "brown." Distinguished by both nationality and skin color, it included not just Arabs and South American Indians, but sometimes Chinese, Japanese, and Southeast Asians, as well, the latter three of which were commonly referred to as "Orientals."[24] Researchers discussed brown populations much less frequently than they did whites or "Negroes," and those who mentioned them nearly always did so within the wider context of "colored" races. One pair of scientists, for instance, wrote of "10,000 deeply pigmented oriental people and negroes."[25] Physicians did not attempt to determine how South Indians differed in skin tone from Chinese, or Malays from Mexicans. Instead, they adopted

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[24] The designation of "Oriental" would become more widespread in the 1940s. See, for instance, W.C. Hueper, *Occupational Tumors and Allied Diseases* (Springfield, IL: Charles C. Thomas, 1942), 224; Lauren V. Ackerman and Juan A. del Regato, *Cancer: Diagnosis, Treatment, and Prognosis* (St. Louis: The C.V. Mosby Company, 1947), 127-128.

[25] Ross E. Herold, "Case Report—Malignant Melanotic Tumors in the Negro," *New York State Journal of Medicine* 36, 19 (Oct. 1, 1936), 1418.

categories of "white" and "colored" because of their social and political utility, and then naturalized differences between groups by pointing to the epidemiology of skin cancer. Blacks occupied one end of the spectrum, whites the other. Populations with various shades of non-white and non-black skin took up the space in the middle.

A handful of scientists attempted to get at the etiology of skin cancer by isolating the characteristics which distinguished not just Africans from African Americans, but among black Africans from various countries, as well. Although it was an implicit acknowledgement that blacks were more than a monolithic mass, the researchers' motivation was likely the immense dearth of knowledge about the disease, rather than a greater sensitivity to ethnic and cultural distinctions in the African diaspora. Historian James T. Patterson has written that the proliferation of a large number of theories about a disease usually indicates that no one has yet grasped its causes.[26] A similar lack of understanding of the particularities of skin cancer led scientists in the 1930s to examine a wide range of variables in their quest to comprehend the disease. Because no one knew for certain what caused these tumors, physicians could not be sure which factors might affect an individual's susceptibility. A decade later, after the etiological effect of sunlight was well established, research on blacks would all but cease, to be replaced by an increasing number of studies that investigated shades of Caucasian complexions. But for now, all doctors had to go on was the curious fact that "Negroes" appeared to be nearly immune to tumors of the skin.

Investigators who subdivided their black subjects into regional categories immediately found notable differences between Africans and African Americans.

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[26] Patterson, 56.

American blacks rarely exhibited signs of melanoma; many practitioners, in fact, claimed never to have seen a case of skin cancer within the population. Melanomas which did occur in African Americans often appeared on the soles of the feet—an area with relatively light pigmentation—or developed from bluish-black moles on the face and upper body. Despite acknowledging that blacks and Europeans had racially intermixed for centuries, resulting in "mulattoes" and "those of mixed blood," doctors for the most part did not identify differences in skin tone among African Americans.[27] There were, of course, exceptions, and a few physicians wrote case reports describing patients who ranged in color from light brown to "very dark-skinned."[28] But in general, discussion among cancer researchers of skin tones among African Americans remained uncommon in the 1930s.

Black Africans, in contrast, did not display the same level of racial resistance as their American counterparts. The discovery by Sequeira and Vint, an influential pair of researchers, of 482 malignancies among 2,228 specimens from patients at the Native Hospital in Nairobi, Kenya, demonstrated that blacks were not immune to skin tumors.[29] T.F. Hewer found malignant melanoma "of very frequent occurrence in natives of the Anglo-Egyptian Sudan."[30] Dialogue about the range of skin tones among black Africans, moreover, appeared regularly in this segment of the medical literature, with physicians describing some Africans as "light and some darker brown" and others as "almost 'coal

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[27] Bishop, 523.

[28] See Bishop, 523.

[29] Herold, 1418; Anderson, 425.

[30] T.F. Hewer, "Malignant Melanoma in Coloured Races: The Role of Trauma in Its Causation," *Journal of Pathology and Bacteriology* 41 (1935), 473.

black."[31] Variations in color generally correlated with country of origin, or with ethnic group within a particular country. As Hewer discovered in the Sudan, "there are many distinct races, with different skin colours. These differences are racial and not due to interbreeding with Europeans."[32]

Researchers who studied the incidence of skin cancer among black Africans noticed that many such tumors occurred on the legs and feet. While some drew a connection to the decreased amount of pigmentation on the lower extremities, others focused on the possibility of trauma. Physicians had long speculated that a blunt injury could prompt carcinogenesis in the affected area. A blow to the chest might result in breast cancer, while a punch in the gut could lead to stomach or liver cancer. Even decades later, Americans who had digested a steady stream of public health pronouncements regarding the disease continued to believe in trauma as an etiologic factor. The noted cancer researcher Wilhelm C. Hueper, for instance, testified as an expert witness in a 1960s civil suit against the employer of a man who had died of pancreatic cancer—the consequence, his widow claimed, of falling off a building at work.[33]

Many believed that melanomas in black Africans resulted directly from trauma. As one physician claimed in 1936, "trauma has a definite bearing on the development and spread of the tumor" in cases of skin cancer in blacks.[34] Physicians described tumors on

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[31] Bishop, 526-538.

[32] Hewer, 474.

[33] See Wilhelm C. Hueper Papers [MS C 341], History of Medicine Division, National Library of Medicine, Bethesda, MD, Box 3, Vol. 1-12.

[34] Herold, 1421.

the soles of the feet following injury from nails, thorns, and pressure. Hewer, who found that 59.5% of his subjects had melanomas on their feet and another 15% on their legs, traced the etiology of skin cancer among Sudanese to their practice of going shoeless.[35] "[T]he high incidence of malignant melanomata on the lower extremity and especially on the sole of the foot in people who walk bare-foot in a country abounding with thorns and sharp stones is fairly strong evidence for the significance of trauma in their causation," he wrote. In contrast, "[t]he rarity of melanomata in the American negro may well be due to the fact that he wears shoes and that his feet are not subjected to the frequent punctures from thorns that are the lot of his African relation." Trauma, Hewer believed, was the crucial variable affecting the development of skin cancer in his subjects, "a more important predisposing factor than skin colour in the aetiology of malignant melanoma" among Sudanese.[36]

Although Hewer and his like-minded colleagues recognized differences in skin tone among African blacks, the suspected etiology of skin cancer in the population rendered such distinctions irrelevant. Skin color was not the primary factor in whether Africans developed cancerous skin lesions; rather, scientists believed that the disease resulted from physical trauma. Furthermore, the source of the trauma was significant, less blows and blunt force than habits and behaviors. The discovery of trauma-induced melanomas in Africans illuminated a potential reason for resistance to the disease among black Americans, suggesting that it was a consequence not of skin color, but of culture. Adopting the customs of the United States conferred a protective status on African

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[35] Hewer, 476.

[36] Hewer, 474-476.

Americans, making them less susceptible to skin cancer than black Africans whose bare feet suffered regular injury from sharp objects.

The ways in which scientists interpreted the relevance of behavior and skin tone to the development of skin cancer revealed differences in perceptions of black Africans and African Americans. Black Africans, as Hewer found in his work in the Sudan, possessed gradations in skin color and cultural characteristics which were a result of national origin. They had, in other words, both ethnicities and ethnically-derived skin tones. African Americans, in contrast, comprised a *racial* group, and as such lacked a distinctive culture or a range of skin tones. As "Negroes," they had a continent of origin—Africa—but no particular country of origin. Even though their skin color marked them as non-European Americans, their cultural heritage was varied, linked to both a number of African countries and to white America, which had given them the custom of wearing shoes. Whereas black Africans were members of specific ethnic groups which varied by country of origin, African Americans were a race, and one in which skin color constituted an unchangeable racial trait.

At the same time, investigators in the 1930s were making more of an effort to differentiate ethnic whites from one another. It was no longer sufficient to classify Caucasians as Northern or Southern European; instead, they were further subdivided into categories such as North Germans, Scottish, Irish, and Basques, the latter two of which were "distinguished by their whiteness."[37] Each group exhibited a characteristic coloring, from the fair-complexioned Scandinavians to the swarthier Italians, French, and

[37] Angel H. Roffo, "Cutaneous Cancer and the Sun: A Clinical and Experimental Study," *Urologic and Cutaneous Review* 43, 6 (June 1939), 414.

Syrians.[38] Whites, like black Africans, had countries of origin; therefore, they possessed both distinctive ethnicities and skin tones.

The attention to white ethnicity in the 1930s came on the heels of a period of race awareness that gathered momentum in the late nineteenth and early twentieth centuries. Scholars including Matthew Frye Jacobson, Noel Ignatiev, and David Roediger have explored the historical concept of whiteness and its subsequent effects on culture, politics, and citizenship.[39] As Jacobson has argued, the nineteenth century's monolithic conception of whiteness fractured in the early twentieth century, a consequence of changing demographic patterns that lent new visibility to "undesirable" groups such as Irish, Italians, and Jews. The nativist movement that culminated with the passage of the Johnson-Reed Immigration Act in 1924 represented a shift to a new form of "bedrock racism," the central principle of which was the "political 'fitness' among a now fragmented, hierarchically arranged series of distinct 'white races.'"[40]

The influence on the 1924 legislation of eugenists such as Charles Davenport and Harry Laughlin has been well documented by scholars such as John Higham, who cites the bill as evidence of discriminatory attitudes toward certain European ethnicities at the end of the nineteenth and beginning of the twentieth centuries. Higham argues that old-stock Americans—those of Northern and Western European origin and their descendants—were growing increasingly alarmed over the rising influx of "swarthy"

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[38] See, for instance, Hueper, *Occupational Tumors and Allied Diseases*, 225; Joseph Taussig and George D. Williams, "Skin Color and Skin Cancer," *Archives of Pathology* 30 (Sep. 1940), 721-730.

[39] Matthew Frye Jacobson, *Whiteness of a Different Color: European Immigrants and the Alchemy of Race* (Cambridge, MA: Harvard University Press, 1998), Noel Ignatiev, *How the Irish Became White* (New York: Routledge, 1995), David R. Roediger, *The Wages of Whiteness: Race and the Making of the American Working Class* (London: Verso, 1991).

[40] Jacobson, 42-43.

immigrants hailing from Eastern and Southern Europe. The Johnson-Reed Act, therefore, represented the culmination of a decades-long agitation for a more selective border policy.[41] More recent scholarship by Amy L. Fairchild and Mae M. Ngai contends that although eugenicists dominated Congressional discussion on the legislation, the 1924 law effectively drew a line around, rather than through Europe, thereby consolidating the legal boundaries of the white race.[42]

While the mechanism of the law produced specific racial formations of whites and non-whites after 1924, medical literature on skin cancer revealed a process of racialization in which pigmentation became a proxy for underlying ideological and political concerns. The discourse on skin cancer in the 1930s continued to disrupt a monolithic conception of whiteness. The white "race" consisted of Europeans and their descendants; they possessed national origins, ethnicities, and ethnic-specific skin tones. Paying attention to ethnic differences in skin color both allowed whites to retain their ethnicities and gave researchers a way by which to distinguish between, for instance, the pale, ruddy-faced Irishman and the "swarthier" individual of French ancestry. At the same time, blacks, Asians, and other non-Europeans became a single category of

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[41] See, for instance, John Higham, *Strangers in the Land: Patterns of American Nativism, 1860-1925* (New Brunswick, NJ: Rutgers University Press, 1992 [1955].

[42]Amy L. Fairchild, *Science at the Borders: Immigrant Medical Inspection and the Shaping of the Modern Industrial Labor Force* (Baltimore: Johns Hopkins University Press, 2003), Mae M. Ngai, *Impossible Subjects: Illegal Aliens and the Making of Modern America* (Princeton, NJ: Princeton University Press, 2004). Ngai has also examined the influence of immigration law, particularly the 1924 Act, on the racial formation of Europeans, Asians, and Mexicans. She finds that the quota system produced by the Johnson-Reed Act reconfigured the world into categories that distinguished "persons of the 'colored races' from 'white' persons from 'white' countries." The law racialized Chinese, Indians, and Japanese into a category of "Asian," defined both by country of origin and by their legal inability to obtain American citizenship. Mexicans, who by the late 1920s comprised the country's largest population of undocumented immigrants, became the objects of what Ngai calls "an emergent Mexican 'race problem'" that rendered them inassimilable and permanently foreign. Thus, while the 1924 law uncoupled ethnicity and race for Europeans, subsuming them under an umbrella of "white," racial and ethnic identities remained intertwined for Mexican and Asian immigrants. Euro-Americans possessed both a race and an ethnicity derived from nation of origin, while non-whites were simply members of "colored" races who were perceived as having no country of origin. See Ngai, 27, 7-8.

"colored," distinguished only by skin tone. The term "colored" underscored the ways in which all nonwhites could be classified regardless of differences of history, culture, ethnicity, and country of origin. Race, ethnicity, and national origin were reduced to skin color, which itself was broadly defined in opposition to whiteness. The naming of the category of "colored" used the pretext of disease to privilege disparities in skin color between races while failing to acknowledge those within them. The fact that dark-skinned individuals did seem to exhibit lower rates of skin cancer both justified such an approach and appeared to confirm the medical underpinnings of race.

Although researchers concurred on the infrequency of skin tumors in nonwhites, they were hard-pressed to pinpoint the responsible factors. At the time, two leading theories filled the pages of medical journals. Since no one was sure precisely what caused the disease, both theories seemed equally plausible and did not appear to spawn sharp debates. The first one was advanced by Rudolph Matas, the prominent late-nineteenth century physician, who sought to explain why malignant tumors and benign nevi, or pigmented moles, were so much rarer in tissue that contained relatively large amounts of pigment. Matas attributed the varying rates of incidence in whites and blacks to differences in the physiological systems which produced pigment. He argued that blacks, with their high degree of pigmentation, were accustomed to creating coloration. Therefore, because it was a normal function of their skin, the mechanism responsible for the process was not likely to make errors. The high numbers of tumors on the hands and feet of Africans and African Americans resulted not from traumatic injury, but from the lesser degree of pigmentation—and the greater potential for inaccuracies—in those areas.

In contrast, whites produced very little pigment, which rendered their own physiological system prone to mistakes and resulted in various forms of skin cancer.[43]

The second theory, advanced by James Ewing in the early 1930s, had nothing to do with physiological differences between whites and blacks. Rather, Ewing implicated the relationship between skin tone and visual observation. He suggested that the darker color of Africans and African Americans made malignant melanomas and other external lesions more difficult to detect. A tumor which might be apparent on the face or body of a Caucasian patient might be rendered nearly invisible by "Negro" pigmentation. As a result, physicians overlooked many cases that would have been evident in lighter-skinned individuals.[44] The reason that black patients displayed so many neoplasms on the palms of their hands and soles of their feet was simply because the paler pigmentation in those areas enabled doctors to see lesions more easily. Ewing did not argue, as Matas did, that blacks had empirically lower rates of the disease than whites. Instead, he attributed the reduced number of cases of skin cancer among them to the ways in which an individual's coloring could affect the thoroughness of a visual examination by a physician.

Unlike their colleagues who were investigating internal cancers such as liver or stomach, few physicians argued that black resistance to skin cancer was a result of the population's lack of civilization. Instead, the explanations for black immunity to skin cancer which were put forward by the medical community in the 1930s ranged from the biological to the physiological to the cultural. Scientists may have disagreed on whether the production of pigment, the shade of one's skin, or the wearing of shoes was the

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[43] See Herold, 1418.

[44] For a summary of this argument, see Everett L. Bishop, "Melanoma in the Negro," *American Journal of Cancer* 16, 3 (May 1932), 522-539.

crucial factor in accounting for Africans' and African Americans' low rates of the disease. They were, however, united in viewing blacks as a group that was distinguished, first and foremost, by skin color. Race equaled pigmentation, and the apparent lack of susceptibility of blacks to skin cancer could be traced directly to its effects. Matas blamed the system by which such pigment was produced, while Ewing suggested that dark skin could hide melanomas. A racial logic underlay the observations of even those who pointed to the habits of barefoot Sudanese; by comparing the group with African Americans, these researchers outlined a black diaspora that was defined by skin color. Some physicians acknowledged variations in skin tone among Africans and their descendants, while others failed to recognize them. But the medical literature was clear on one crucial point: blacks had color, while whites did not.

Sunlight, Race and Skin Cancer

By the early 1940s, some textbooks and medical sources had begun to cite chronic irritation from cumulative exposure to solar radiation as a factor in the development of skin cancer. It was not a new theory; as one researcher noted in a 1940 article in the *Journal of the National Cancer Institute*, scientists had been considering it for nearly fifty years.[45] The prominent cancer researcher C.P. Rhoads pointed to studies "which prove that mariners and farmers, exposed for years to bright sunlight, are much more liable to growths of the exposed areas than are those individuals who lead more sheltered lives."[46] Furthermore, the disease displayed a distinct geographic variance. It is "well known,"

[45] Harold F. Blum, "Sunlight and Cancer of the Skin," *Journal of the National Cancer Institute* 1, 3 (Dec. 1940), 397.

[46] C.P. Rhoads, "The Inciting Causes of Cancer," *National Bulletin* [of the ASCC] 22, 6 (June 1940), 6.

stated a 1945 article in the *Journal of the Medical Association of Georgia*, that the incidence of skin cancer increased the further one traveled south.[47]

Despite numerous articles touting growing evidence of an association between ultraviolet light and skin cancer, causality had not yet been conclusively demonstrated. At the time, scientists had managed to identify a mere handful of cancer-causing agents. In the 1760s, Percival Pott discovered high rates of scrotal cancer among chimney sweeps in Britain, subsequently linking the disease to the soot with which workers came into regular contact. Japanese researchers Katsusaburo Yamagiwa and Koichi Ichikawa produced cancer in rabbits in 1915 by painting the animals' ears repeatedly with a compound containing tar. In the 1930s, researchers outlined the cancer-causing properties of dozens of chemicals, including arsenic, petroleum, mineral oils, and aniline dye. Wilhelm C. Hueper, a German-born scientist, spent the bulk of his career working to uncover carcinogens that could lead to environmental and occupational cancers. In the 1930s, he lost his job as a scientist for the DuPont Corporation after the company blocked him from publishing data linking the compound beta-naphthylamine with bladder cancer.[48] Hueper eventually got a job with the National Cancer Institute, and in 1942 published the results of his work in a 900-page tome entitled *Occupational Tumors and Allied Diseases*, which offered chapter after chapter of analysis of various carcinogens in the workplace.

Exploration of the connection between sunlight and skin cancer, moreover, came at a time when scientists were struggling to understand the basic science of the human

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[47] Wm. L. Dobes and Lt. Philip H. Nippert, "Clinical Aspects and Treatment of Cutaneous Cancer," *Journal of the Medical Association of Georgia* 24, 2 (Feb. 1945), 25.

[48] Patterson, 187.

body, let alone the ways in which malignant growths formed and spread. Notable medical milestones were still years in the future. Antibiotics would not come into general use until after World War II, and Jonas Salk's development of a vaccine for polio lay more than a decade away. Some cancer researchers, meanwhile, were developing a vague awareness that hormones had something to do with breast and prostate cancer, and had noticed a correlation between cervical cancer and a high number of sexual partners, but they were nowhere near establishing public health recommendations for these forms of the illness. Even into the 1950s, notes James T. Patterson, "researchers were still groping to comprehend the basic biological processes of the disease. As one investigator admitted in 1949, 'looking for the cause of cancer is like looking in a dark room for a black cat that isn't there.'"[49]

But the main reason why the link between ultraviolet light and the development of skin cancer remained no more than an association, rather than a relationship of cause and effect, had to do with the procedures of biomedical research. In the 1940s, work on disease etiology followed one of two models: either the controlled experiment or the inferential method. The first approach advocated experimental laboratory science, which researchers utilized to test a hypothesis, often by experimenting on animals, or to assess the effectiveness of new vaccines. It relied on controlled experimentation of the sort that had elucidated the biological mechanisms involved in the etiology of many infectious diseases. Epidemiologists who employed the second approach relied on statistical methodology to analyze a large amount of data gathered either from the field or from multiple scientific studies to look for correlations between disease and particular

[49] Patterson, 188.

etiological factors. They would examine population demographics and social and economic data to establish, for example, that heavy users of alcohol had higher rates of lung cancer than non-drinkers.[50]

For many scientists, a correlation between skin cancer and sunlight was not enough to amount to a disease etiology. The success of Robert Koch's postulates in facilitating the identification of microbes responsible for infectious illnesses such as tuberculosis had led scores of researchers to distrust statistical, population-based studies, believing instead that the laboratory represented the only failsafe location to ascertain the source of disease. To them, a "strong association" did not constitute scientific proof. Furthermore, those who favored laboratory research often had misgivings about the reliability of clinicians to offer generalizations based on their experience, leading them to dismiss such data as anecdotal and unreliable.[51]

Despite disagreements over what could be considered adequate knowledge in the etiology of skin cancer, evidence was beginning to accumulate in favor of a causal role for ultraviolet light. In 1940, Harold F. Blum of the National Cancer Institute noted that available information lent "strong support" to the relationship between sunlight and skin cancer. Although he cautioned that it had not yet been accepted as a scientific certainty, his remarks revealed the growing importance of sunlight in the search for causality.[52] Scientists began to accept its probable role in the development of skin cancer.

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[50] For a discussion of the two models of research, see Mark Parascandola, "Two approaches to etiology: the debate over smoking and lung cancer in the 1950s," *Endeavor* 28, 2 (June 2004), 81-86; and Mark Parascandola, "Skepticism, Statistical Methods, and the Cigarette: A Historical Analysis of a Methodological Debate," *Perspectives in Biology and Medicine* 47, 42 (Spring 2004), 244-261.

[51] See, for instance, Allan M. Brandt, *The Cigarette Century: The Rise, Fall, and Deadly Persistence of the Product that Defined America* (New York: Basic Books, 2007), Chapter 5.

[52] Blum, "Sunlight and Cancer of the Skin," 397.

As medical research gradually grew to implicate the etiological function of ultraviolet rays, the ways in which physicians and their colleagues wrote about race shifted. Ten years earlier, scientists who had yet to fully grasp the significance of sunlight attempted to isolate the ways in which groups with low rates of the disease, notably Africans and African Americans, differed not simply from whites, but also from one another; their work centered on uncovering the unique characteristics of blacks that they hoped could have explanatory potential. A decade later, researchers all but abandoned the discussion of degrees of color among blacks. There were, of course, exceptions, including a Philadelphia physician named Meyer L. Niedelman who published a study in 1945 describing the variations in color among children with one white and one black parent. But his awareness of skin tone among mulattoes came within the context of physical differences between the two races, as he aimed to determine the degree to which pigment from each parent could be measured in the skin.

The medical community's enhanced comprehension of the etiology of the disease meant that blacks no longer held the key to the puzzle after 1940. Hence, Africans, Afro-Caribbeans, and African Americans became an indistinguishable mass, undifferentiated by nationality, cultural habits, or skin color. The ensuing discussions of race generally fell within one of two frameworks. The first focused on the physiological makeup of blacks, as medical researchers investigated both the chemistry of black skin and the group's internal anatomy. The second framework of race and skin color added white populations to the fray and examined the roles of skin tone and complexion among Caucasians, particularly with respect to cancer prevention.

As those seeking to explain the incidence of skin cancer among blacks stopped casting a wide net over myriad variables in an attempt to discover discrete causes for the disease, they began to focus more narrowly on specific aspects of black physiology. This new formulation of skin cancer and race identified bodily differences between racial groups, including changes in hair color and texture, skin tone, and the appearance of numerous facial features. It encompassed, as well, variations in the size of internal organs and the chemical processes of the body. All of these were then classified as racial traits, markers of physical disparities between whites and blacks.

Among those who addressed the issue of racial differences among groups was Wilhelm C. Hueper, the National Cancer Institute scientist whose work involved environmental and occupational tumors. In his textbook, he described "marked differences" in the incidence of particular diseases in various populations, including sickle-cell anemia among blacks; thalassemia in Italian, Greek, Armenian, and Syrian populations; hemophila among those of "Teutonic" descent and "Jewish racial mixture"; and familial night blindness in the Japanese. Certain groups displayed anomalous rates of cancers of specific organs, such as liver cancer among "African Negroes, and Asiatic Javanese and Chinese" and the "complete absence" of penile cancer among Jews. He reiterated, too, the higher rates of melanoma among African blacks and "members of fair-skinned races" than among "the colored races," as well as lower rates of skin lesions in general among African Americans. "The susceptibility to skin cancer caused by solar irradiation," he wrote, "is in general inversely proportional to the amount of skin pigment present, as it is the function of the melanin pigment to act as a screen against the

ultraviolet rays." In white skin, he continued, pigment occupied only the superficial layers of the epidermis, while in "Negroes" it penetrated into the deeper strata, as well.[53]

In addition to differences in pigmentation, Hueper found disparities in the surface properties of white and black skin. Not only did the oily epidermis of blacks reflect solar rays at a higher rate than "a dry skin which is more often found in white people," but increased vascularization caused abundant perspiration, resulting in "an effective cooling of the skin and, thereby, in reducing the burning effect of solar rays." The physiological characteristics of black skin, Hueper believed, both shielded African Americans from tumors caused by ultraviolet light and protected them from the carcinogenic effects of pitch, tar, and asphalt. This, he thought, rendered them particularly well-suited to employment in tar distilleries.[54] But racial discrepancies between whites and African Americans went beyond the color and chemistry of each's skin and the distribution of pigment therein. The groups differed from one another "in many more minute anatomic details, such as, the size the heart, liver and spleen…, the secretion of the apocrine and sebaceous glands, the type and color of hairs, the number and distribution of sweat glands, and the distribution of blood groups."[55]

Contemporaries of the German scientist agreed that African Americans possessed both anatomical and dermatologic peculiarities which rendered them less susceptible to skin cancer. "Some of the tropical races may even have internal mechanisms which protect them against the deeper effects of actinic exposure which the white races apparently do not possess," claimed two researchers, listing blood formation, utilization

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[53] See Hueper, *Occupational Tumors and Allied Diseases*, 762-768.

[54] Hueper, *Occupational Tumors and Allied Diseases*, 224, 765.

[55] Hueper, *Occupational Tumors and Allied Diseases*, 762.

of vitamins, and liver function among the racial differences.[56] Several saw physical abnormalities in the pigment of blacks, maintaining that its distribution throughout the epidermis protected against sunburn, whereas in whites it occupied only the superficial, basal-cell layer.[57] "In the Negro skin," read one article, "the pigment formed in the basal layer progresses toward the surface and is found in large amounts diffused throughout the squamous cell layer and stratum corneum, where it is in a perfect position to block the carcinogenic rays before they reach the deeper layers of the epidermis." White skin, in contrast, contained too little pigment near the surface of the skin to offer any protection from actinic rays.[58] Meyer L. Niedelman, the Philadelphia dermatologist who studied pigmentation in African Americans, wrote, "The skin of persons of the Negro race differs from that of members of the white race not only in structure and physiology but in its reaction to trauma and infection….In comparison with the skin of persons of the white race, that of Negroes has a thicker epidermis, a more abundant blood supply in the corium and a larger number of sudoriferous and sebaceous glands."[59]

The perspective displayed by these scientists of race as a confluence of particular traits was a fairly common one at the time. By the early 1940s, members of the medical community generally described race as a series of physical and physiological characteristics—such as hair color and texture, eye color, and properties of the skin—that were affected both by genetic history and by the social circumstances of one's daily life.

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[56] Richard S. Weiss and Adolph H. Conrad, Sr., "Light as a Factor in Skin Cancer," *Surgical Clinics of North America* 24 (Oct. 1944), 1028.

[57] Blum, "Sunlight and Cancer of the Skin," 410.

[58] Hall, 592.

[59] Meyer L. Niedelman, "Abnormalities of Pigmentation in the Negro," *Archives of Dermatology and Syphilology* 51, 1 (Jan. 1945), 1, 3.

People of similar ancestry might look alike because of the combined influence of shared genes, diet, customs, and climate. Scientists recognized that Africans in the United States had intermarried with both Europeans and Americans Indians since the seventeenth century; hence, any notion of a "pure" black race was long obsolete. If race seemed to have a bodily expression, it was only because people who shared a common descent displayed similar physical characteristics. The new awareness of social factors, however, did not preclude individual physicians from continuing to subscribe to theories of cultural or intellectual inferiority that were grounded in racial differences. Indeed, W. Montague Cobb, a physical anthropologist who insisted on the social foundations of the category of "American Negro," went on to discuss the size of the genitalia of African American men and women and whether the "more marked" webbing of the tendons of their hands had affected artistic achievement.[60] But it did suggest that many American researchers in the 1940s looked at race not as a series of immutable, physical characteristics, as people had understood it in the nineteenth century, or in terms of culture and nation, a concept prevalent earlier in the twentieth century. Rather, race appeared to be a product of both genetic heritage and social circumstances. As Cobb himself remarked, "Social and not scientific dictum defines an American Negro."[61]

At the same time, however, a degree of uncertainty remained over the distinction between an inherited or genetic trait and a truly "racial" one which would remain unchanged from one generation to the next. As much as researchers recognized the impact of social and environmental factors in explaining distinctions between races, they

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[60] W. Montague Cobb, "Physical Anthropology of the American Negro," *American Journal of Physical Anthropology* 29, 2 (June 1942), 158-159, 163.

[61] Cobb, 144.

appeared reluctant to abandon completely the notion of biological underpinnings. To Hueper, for instance, race was governed by a confluence of variables that determined not only external, phenotypic markings, but also internal ones such as the size and shape of particular organs. These traits might be due to the concentration of "special constitutional weaknesses or sensitivities" in populations defined by internal breeding. Such groups, furthermore, could experience differences of diet, climate, and habits that might account for regional fluctuations in the rates of incidence of certain diseases. But Hueper also speculated that the epidemiological patterns which he observed might be "attributable to a racial factor," which he distinguished from a crowding of traits due to reproductive isolation.[62] The characteristics that an individual inherited from generations of ancestors played out in ways that were generally predictable and understandable. But the possibility persisted of traits which might be present in diasporic populations. In other words, the physical and physiological differences between races could be due not only to genetics, or one's inherited characteristics, but also to biology, or the persistent expression of particular traits across time, space, and reproductive partner. These ambiguities regarding the concept of race—and the muddled distinction between biology and genetics—would become less important in the field of skin cancer in subsequent years as researchers turned their attention to white populations.

Complexion

By the end of the 1940s, the role of sunlight as an etiologic factor in the development of skin cancer had gained broad acceptance. Writing in 1949, Harold F.

[62] Hueper, *Occupational Tumors and Allied Diseases*, 762.

Blum, the National Cancer Institute researcher, described the process by which scientists had come to recognize causality. At the beginning of the decade, "the then existing evidence strongly supported the idea that sunlight is a major cause of cancer of the skin in man, there were numerous places where the picture was not very clear." Seven years' worth of additional experiments, however, "have amplified and rendered more interpretable the earlier data. The newer studies strengthen the general concept at several points and at no place conflict with it, so it seems possible, today, to take a much more definite position….We now have a number of lines of evidence, all of which converge to indict sunlight as the major cause of cancer of the skin in man."[63]

Whereas research ten years earlier had examined the physiology of black skin, mounting evidence in favor of the etiological role of ultraviolet light now prompted physicians to shift focus to their white patients. At the time, a common way for skin cancer researchers to categorize patients was by hair color, whether blonde, brunette, or redhead.[64] Even by 1940, the notion that blondes were more susceptible to skin cancer than brunettes was widespread enough that one set of authors referred to it as "[a] commonplace in dermatologic lore."[65] That same year, the ASCC's *Bulletin* addressed the questions of science writers regarding the varied susceptibility to sunlight of blondes and brunettes. "Negroes represent a strain of humans with an hereditary degree of protective pigmentation," read the article. "Brunettes are people with potentiality for temporary pigmentation when exposed to an irritant. Blondes lack this capacity to

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[63] Harold F. Blum, "Review: Sunlight as a Causal Factor of Cancer of the Skin in Man," *Journal of the National Cancer Institute* 9, 3 (December 1948), 247, 257.

[64] Frequently, "Negroes" comprised a special subset of the category of brunettes.

[65] Joseph Taussig and George D. Williams, "Skin Color and Skin Cancer," *Archives of Pathology* 30 (Sep. 1940), 721.

different degrees." The article went on to compare fair-haired people to albino mice, and brunettes to pigmented ones.[66] A handbook published several years later by the Michigan State Medical Society and state Department of Health and entitled *Cancer: A Manual for Practitioners* advised "[b]londes and other 'thin-skinned' persons" to "reduce their sunbathing to an even shorter period [than one hour per day]," as such individuals "may belong to a recognizable constitutional type in which solar irradiation is peculiarly carcinogenic."[67]

The ways in which whites were represented within the medical literature on skin cancer contrasted sharply with portrayals of blacks. By the 1940s, "Negroes" constituted a discrete race with specific, recognizable internal and external traits. Whites, however, lacked race, and were rarely, if ever, referred to as such. Instead, researchers after World War II focused on hair color and the ways in which it correlated with complexion and eye color. One study by A. Fletcher Hall, a physician in Santa Monica, California, examined the relationships of sunlight, complexion, and heredity to skin cancer among one hundred subjects. He first divided his sample by eye color into two categories; "'the brown-eyed group'" included individuals with eyes of dark or light brown, while "'the blue-eyed group,'" contained those whose eyes were light blue, dark blue, hazel, gray, or green. Hall then attempted to correlate eye color with each person's self-reported "original" hair color, which fell somewhere along a spectrum of black to light blond to "sandy or red." His results challenged the prevailing wisdom that brunettes had lower rates of skin cancer; of his patients with carcinoma, 47% had dark hair, while 28% were light-haired,

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[66] American Society for the Control of Cancer, *National Bulletin* 22, 4 (Apr. 1940), 7.

[67] Michigan State Medical Society and Michigan Department of Health, *Cancer: A Manual for Practitioners* (1944), 114.

prompting him to conclude that "it seems unjustifiable to continue to propagate the statement that 'blonds are especially susceptible to cancer of the skin.'"[68]

Hall's study was notable less for his results than for the ways in which he interpreted the category of "blond." The *New Century Dictionary*'s definition, which he included in his article, consisted of a tripartite description of a hair color, a complexion, and a hair color with an associated complexion: "'light colored, as golden or light brown hair; fair haired and fair complexioned, as a person; light in hue, as the complexion.'"[69] As Hall's study and the dictionary entry indicated, an individual's hair color suggested much more than merely the shade of his or her hair. It could encompass a range of physical characteristics, from eye color to complexion to physiological features of the skin. Redheads and blondes tended to have thin, fair skin. One team of researchers, whose work Hall cited, described "'a certain type of skin…[as] more fertile soil for the development of epithelioma; namely, that of the person with blond, sandy or ruddy complexion, which freckles readily but does not tan, usually associated with light hair and eyes.'"[70] Another physician noted that the type of skin most at risk for developing cancer was represented, in his opinion, by a "red-headed, blonde person who freckles easily."[71] The skin of blondes and redheads also tended to be dry and rough, further rendering such individuals prone to developing cancerous lesions. Hall reported that

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[68] A. Fletcher Hall, "Relationship of Sunlight, Complexion and Heredity to Skin Carcinogenesis," *Archives of Dermatology and Syphilology* 61 (Apr. 1950), 597.

[69] Hall, 597.

[70] Hall, 595.

[71] Charles Phillips, "Observation Based Upon the Study of 1,434 Skin Cancers," *Virginia Medical Monthly* 67, 7 (Jul 1940), 405-406. The ambiguity of the category of "blonde"—and whether it denotes hair color, complexion, or some combination of the two—is apparent here, as Phillips refers to a person who is both a blonde and a redhead.

"[b]lue-eyed children of blue-eyed parents" carried the greatest risk due to their "almost complete inability to acquire and retain a thick enough stratum corneum to protect them from rays of carcinogenic wavelengths in the amounts encountered in such regions as Texas, Arizona, Southern California and Australia."[72]

If people with blonde or red hair had light eyes, fair complexions, and thin skins, then the physiological attributes of brunettes offered them some protection. Brunettes tended to have brown eyes and more deeply pigmented skin. Hall identified "certain racial stocks and hereditary complexion patterns in which sunlight is not an important, if any, factor in skin carcinogenesis; these include certainly the Negro and Oriental races, probably the Mexican and Mediterranean and possibly all homozygous brown-eyed persons."[73] An Australian researcher who studied a type of skin cancer called "rodent ulcers" found that "[p]eople with brown eyes and olive skin rarely come into the rodent disease list. I have never seen an Italian with rodent disease…. No instance of a Chinaman or colored patient suffering from rodent ulcer could be found, although many of such people pursue outdoor occupations, generally as market gardeners."[74]

Categorizing an individual on the basis of the shade of his or her hair, whether blonde, brunette, or redhead, was intended to signify disparities in complexion that might affect one's propensity for developing skin cancer. Redheads, researchers agreed, tended to possess the thinnest, most susceptible skin, followed by blondes. Brunettes, a group which included not just Caucasians with brown hair but also members of the African

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[72] Hall, 606.

[73] Hall, 606. A "homozygous brown-eyed person" is someone who has two identical alleles for the trait on both members of a pair of homologous chromosomes. Since brown eye color is a dominant trait, an individual who is homozygous for the characteristic has two alleles for brown eyes, expressed as AA.

[74] E.H. Molesworth, "Rodent Ulcer," *M.J. Australia* 1, 878, 1927, Hall, 594.

diaspora, as well as a mishmash of other nonwhites such as "Orientals" and North and South American Indians, were thought to have a more resistant type of skin. But just as the terms "colored" and "white" elided differences within nonwhite groups while preserving the features that separated them from Caucasians, dividing populations by hair color minimized variations of race, color, and culture among "brunettes," while maintaining such distinctions among whites. Writers took care to identify the most vulnerable types, including "[b]lue-eyed descendants of blue-eyed parents," blondes with gray eyes, and redheads with green eyes.[75] Nonwhites, in contrast, invariably had brown hair and brown eyes, and few researchers took pains to distinguish among their spectrum of skin tones.

As these articles indicated, researchers in the early 1950s were beginning to pay more attention to nonwhite—and nonblack—groups, both inside and outside the United States. Their reasons were probably etiological. Although most scientists accepted the causative role of ultraviolet light in producing cancers of the skin, certainly not all of them did. Research on populations with phenotypic differences, therefore, could provide a way by which to test the theory. Perhaps the most significant writer at the time to address issues of race, ethnicity, and cancer was Paul E. Steiner, a professor of pathology at the University of Chicago. His monograph, *Cancer: Race and Geography*, appeared in 1954 and examined various types of cancer among "Caucasoids," "Negroids," and "Mongoloids." Most of his data were drawn from necropsy reports of the Los Angeles County Hospital; he also referenced published articles on the three groups in other parts of the world. Steiner aimed to use statistical methods to illuminate the etiology of human

[75] See, for instance, Morris Waisman, "The Dermatologic Dangers of Sunlight," *Journal of the Florida Medical Association* 39, 4 (Oct. 1952), 249-258.

cancers, and by examining populations of similar ancestry in different countries he hoped to isolate the effects of environment and heredity on the development of disease.

While Steiner did not study malignant melanomas, he conducted substantial research on carcinomas of the skin. From his data he identified 122 cases in whites, thirteen in Mexicans, and one in an African American. The thirteen Mexican cases he called "unexpected" because the subjects, like Chinese, Filipinos, and Japanese, were "moderately pigmented peoples." In comparing rates of skin cancer in several groups of immigrants versus those in their country of origin, Steiner found no statistical differences between Japanese and Chinese in the U.S. and Asia. Among African Americans, he noted, as had previous colleagues, lower rates of cancers of the feet and legs than among black Africans. Looking carefully at rates of skin cancer in groups of migrants could be particularly significant, for skin color was not likely to change substantially when populations moved around the globe. One might grow a few shades darker or lighter according to altitude or latitude, but only within the range determined by one's genes. Race, as Steiner found, was a factor in skin cancer only to the extent that it determined special customs or one's degree of pigmentation; he did not believe, as others had earlier, that certain races inherently possessed attributes which shielded them from the disease.

Steiner saw racial expression as the totality of an individual's physical, physiological, and cultural traits, which depended on both environment and genetic inheritance. "Observed racial differences," he wrote, "may be caused by genetical, cultural, or environmental factors, or by combinations."[76] Racial characteristics included

[76] Paul E. Steiner, *Cancer: Race and Geography; Some Etiological, Environmental, Ethnological, Epidemiological, and Statistical Aspects in Caucasoids, Mongoloids, Negroids, and Mexicans* (Baltimore: Williams & Wilkins Co., 1954), 5.

susceptibility to particular diseases, as well as the physical markers of race, including skin and hair color. He referred to a trait which was determined by inheritance, such as the tendency of a normal cell to mutate into a malignancy, as an "inherent constitutional factor." An "extrinsic factor," in contrast, was influenced by custom or culture and included "climate, altitude, air pollution, humidity, temperature, amount of solar and other types of irradiation…choice of food and culinary practice, occupational exposures, sanitary habits, [and] economic level."[77] To Steiner, race was determined by the ways in which the environment, and the environments of one's ancestors, had acted upon one's genes; the challenge in cancer research lay in figuring out the relative importance of geography versus inheritance.

In the mid-1950s, the association between skin cancer and sunlight was about as close as biomedical researchers had gotten to determining a path of causality for any form of the dread disease. In the field of lung cancer, British epidemiologists Richard Doll and A. Bradford Hill were working to establish cigarette smoking as a causative factor in the development of lung carcinomas, rather than simply a behavior with a statistical association with the disease; the relationship between smoking and lung cancer would be generally accepted by clinicians and researchers by 1955.[78] Their efforts, which introduced an experimental element to what historian Allan M. Brandt calls "the methodological rigors of randomization," would pioneer a sophisticated epidemiologic framework for illuminating the pathways of causation for chronic, noninfectious

[77] Steiner, 3.

[78] Brandt, *The Cigarette Century*, 153.

diseases.[79] But many physicians remained skeptical about the ability of epidemiologic methodology to establish causation in the absence of laboratory experiments that had been so successful with infectious illnesses.

The lack of conclusive proof led many physicians to continue to err on the side of caution. Among those who declined to throw their weight behind the finding that ultraviolet light caused skin cancer, a number of them probably did so not because they disagreed with it, but because they questioned the methodology of their colleagues who were promoting it. Cancer was, after all, an enormously complex disease with a multitude of unpredictable variables governing who would get it and when. For every heavy smoker who succumbed to lung cancer, for instance, there was another who appeared to be the picture of health. As three Colorado doctors wrote in 1954, the purpose of their jointly authored article was "to point out that excessive sunlight *may* produce skin cancer."[80] Even as late as 1962, a physician in Dallas, Texas published an article reviewing the "[c]onverging evidence" that implicated sunlight as a major cause of skin cancer, indicating that he still felt some of his colleagues needed to be convinced.[81] Such wariness may also have stemmed from an unwillingness to appear overly sanguine regarding the country's growing cancer problem. Decades of dead ends and false leads had taught medical professionals to exercise a degree of prudence in their dealings with patients. From the snake oil salesmen of the 1800s to the patent medicine peddlers of the

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[79] Brandt, *The Cigarette Century*, 136, 141. See Chapter 5 for a lengthy discussion of Hill and Doll's work in the 1950s as well as the scientific controversy regarding their methodology for determining a causal relationship between cigarette smoking and lung cancer.

[80] O.S. Philpott, A.R. Woodburne, and J.A. Phipott, Jr., "Skin Cancer and Sunlight," *Rocky Mountain Medical Journal* 51, 7 (July 1954): 610-611. Emphasis added.

[81] J.B. Howell, "Skin Cancer and Sunlight," *Skin* 1, 6 (July 1962), 197-199.

early twentieth century to the optimism of scientists after World War II, the medical community had been promising a cure for cancer for a long time. As of yet, however, none had emerged. Those who aimed to temper the exuberance of their boosterish postwar colleagues by urging restraint in interpreting news about scientific discoveries—or worse, accepting conclusions without clinical evidence—hoped to avoid a cycle of raised expectations followed by dashed hopes.

Epilogue: Post-1955

The discourse on race and skin cancer that was in place by 1955 or so would change in two significant ways in the next decade and a half. First, as researchers continued to expand their pool of subjects to groups of newly visible nonwhites, they began to include Asian ethnic groups, particularly those in Hawaii. The addition of Asians not just in the United States, but in other parts of the world, as well, ensured that the medical literature on skin cancer would continue to grow more complex and progress beyond its earlier dichotomy of black and white. Second, the increasing use of reflectometry and reflectance spectrophotometry to measure levels of pigmentation in the skin obviated the need for adjectival descriptions of skin color. Instead of relying on subjective portrayals of the lightness or darkness of a patient's skin, doctors could use an instrument to record both the degree to which light reflected off it and the amount of melanin present. The resulting numbers could then be used to evaluate an individual's susceptibility to cancers of the skin.

Aside from the inclusion of Asians and the development of new technologies for quantifying variations in skin tone, however, in many respects the language in which

physicians and other medical professionals wrote about skin cancer remained virtually unchanged after the 1940s. As the 1960s drew to a close, doctors were still describing susceptibility to the disease in terms of a confluence of eye color, hair color, and complexion, as "blonde, blue-eyed, fair-complexioned individuals" and races "with dark skin [which] are less susceptible than those with white skin."[82] The pattern by then was well established.

Conclusion

The story of skin cancer highlights several themes at the intersection of race and medicine. Between 1930 and 1955, skin cancer offered a clear example of the ways in which epidemiology could serve a scientific mission to understand a disease's etiology. People of color gained visibility within medical circles because they held unique, though at the time unknown, properties which increased their resistance to skin neoplasms. At the same time, differential rates of incidence among white and black populations appeared to support arguments that the two races were physiologically and chemically dissimilar. Whether these disparities were genuine and widespread matters less than the role that epidemiological patterns played in articulating them.

As a generation of scientists struggled to chip away at pieces of the puzzle, the ways in which nonwhites appeared in medical journals and textbooks underwent numerous transformations. Scientists focused on black skin tone and cultural explanations for the racial incidence of skin cancer in the 1930s, turning to the study of

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[82] Robert G. Freeman and John M. Knox, *Treatment of Skin Cancer* (New York: Springer-Verlag, 1967), 3; E.V. Cowdry and Warren H. Cole, *Etiology and Prevention of Cancer in Man* (New York: Appleton-Century-Crofts, 1968), 251.

the physiology of "Negro" skin in the subsequent decade, and later to an examination of complexion in ethnic whites. After the carcinogenic effects of sunlight were established, Caucasians assumed the spotlight as Africans, Afro-Caribbeans, African Americans, and other "colored" populations were relegated to the sidelines, a group distinguished most often by the fact that its members were not white. Each shift indicated a different way of understanding what race stood for and how it affected disease etiology. Racial disparities in rates of skin cancer among various white and nonwhite populations remained a constant throughout the decades, but the ways in which researchers looked at race changed to accommodate new discoveries about the causes of the illness.

Every scientist who studies disease in human populations must decide on a method by which to categorize his or her subjects. Sometimes groups are divided according to the absence or presence of an illness; at other times a biological or behavioral risk factor for a particular ailment provides the line of demarcation. Research on skin cancer revealed how shifting understandings of the causation of a disease could affect both racial categorizations and perspectives on what race itself meant. Because rates of incidence followed racial patterns, investigators chose to arrange subjects by race. But whether they classified groups by nationality and skin tone or combined them into an undifferentiated mass—in other words, how they viewed the boundaries of the categories of race—depended on whatever etiologic theory held sway at the time. Each time they changed focus, the position of people of color within the medical literature on skin cancer underwent a concurrent alteration, aligning in ways that were determined by each group's usefulness to answering the question at hand. The racial discourse of skin

cancer broke down and recategorized not just nonwhites, but Caucasians, as well, in ways that exposed the interconnectedness of medical science with ideologies of race.

Chapter 3

Race, Risk and Cervical Cancer

Since the early twentieth century, women's cancers have been a particular concern of cancer advocacy groups. In the 1910s, breast, cervical, and uterine cancers killed about one-eighth of all women over the age of forty-five, and by 1930, uterine cancer had become the deadliest form of the dread disease for females.[1] Cancers of the reproductive organs carried a unique stigma in a society characterized by sexual reserve. Discomfort with and social awkwardness around both diagnosis and treatment complicated campaigns on prevention and education, thereby hindering efforts at control. As a result, cervical and uterine cancers would remain a particular focus of researchers for much of the century.

The search for the etiology of cervical carcinoma followed the model of other forms of cancer that were also poorly understood at the time. Researchers who aimed to understand epidemiologic patterns of disease organized their subjects into distinct sets of people with rates that were anomalously high or low, and then extrapolated from the characteristics of each group to isolate possible causes. The extent to which such an approach would in time lead to new understandings of the disease's origins depended on the ways in which each population was defined. If researchers prioritized racial boundaries, their results emphasized chemical and physiological disparities between groups. When they focused on distinctive religious practices that might affect cancer

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[1] James T. Patterson, *The Dread Disease: Cancer and Modern American Culture* (Cambridge, MA: Harvard University Press, 1987), 74; P. Brooke Bland, "Remarks on the Prevention of Uterine Cancer," *New England Journal of Medicine* 202, 25 (June 19, 1930), 1195.

risk, they effectively reduced religion to behavior. Categorizing populations by race, religion, or class—rather than, for instance, by body mass or diet—accentuated differences between races, religions, and economic classes while eliding distinctions within them, making such discrepancies central to the task of determining cancer etiology.

From the 1930s to the close of the 1960s, inquiries into the causes of cervical cancer were characterized by shifting associations of race, risk, and behavior. Both the ways in which biomedical researchers took characteristics of an individual's lifestyle into account and the meanings they assigned to them underwent significant transformations. What made investigations of cervical cancer different from work on skin cancer, prostate cancer, and nasopharyngeal carcinoma were the ways in which early scientific recognition of a link between the disease and a patient's economic status shaped subsequent perspectives on race. Transposed onto social factors, race became a risk factor for cervical cancer because of the ways in which it determined behavior, particularly sexual habits that carried with them a stigma of immorality.

Despite the centrality to medicine of rigorous experimentation and principles of scientific analysis, it remains a human practice, and as such is not free from social and cultural bias. As the study of cervical cancer reveals, assumptions about a population can not just alter the course of research; they can lead to new understandings of the relationships among class, race, and disease, as well.

Cervical Cancer Etiology in the 1930s

Although today cervical and uterine cancers are considered separate diseases, doctors in the early 1930s did not always distinguish between the two. The cervix, also known as the cervix uteri, is the lower, narrow portion of the uterus that connects with the vagina. Cellular changes on its surface are the distinguishing mark of cervical carcinoma. The uterus, or womb, connects at the top on both sides to the fallopian tubes, and on the bottom to the cervix. Uterine cancer—cancer of corpus uteri, or body of the uterus—most often involves a growth on the lining of the uterus, known as the endometrium, and thus is commonly referred to as endometrial cancer. As cancer diagnosis became more sophisticated in the coming decades, most physicians would adopt precise terms which differentiated between endometrial and cervical cancers. At the time, however, those in the profession generally combined both illnesses into the broader category of uterine cancer.[2]

Even in the 1930s, the medical community was certain that cervical cancer did not have to be a fatal affliction if caught at an early stage. Like other forms of the dread disease, it could be treated—and even cured, by the standards of the time—if medical intervention came swiftly. Consequently, it was a focal point of public health messages which aimed to disseminate information about cancer's early warning signs. The American Society for the Control of Cancer (ASCC), the nation's preeminent anti-cancer organization in the 1930s, urged the nation to "Fight Cancer With Knowledge." The

[2] Although physicians in 1930 used the term "uterine cancer" to encompass cancers of both the cervix and the corpus uteri, I prefer instead to use the more precise notation of "cervical cancer" throughout this chapter. Since I am examining discourse on the disease rather than assessing medical literature for statistical or scientific accuracy, I do not believe that this choice has affected my analysis significantly. By about the mid-1930s, moreover, it appears that most physicians did in fact distinguish between cancers of the cervix and the uterus, and used the appropriate terminology to refer to them as such.

campaign aimed to instruct ordinary men and women about symptoms that should prompt a trip to the doctor, making it their obligation to seek medical attention. After World War II, the ASCC would reemerge as the American Cancer Society and engage in extensive fundraising to find a cure for the disease, but in the 1930s the organization was still small, meagerly funded, and focused on prevention and education.[3] As a result, its approach centered not on pressuring government or private agencies to increase funding for research, but on the responsibility of each individual to keep cancer at bay. If early intervention was crucial to avoiding death from cancer, then it was up to each person to be accountable for his or her health. Accordingly, its educational posters and pamphlets urged Americans to be on the lookout for indications of the disease: "Any lump, especially in the breast. Any sore that does not heal particularly about the tongue, mouth, or lips. Any irregular bleeding or discharge. Persistent indigestion with loss of weight."[4]

The ASCC's campaign came at a time when the seriousness of the threat posed by cancers of the female reproductive organs seemed to be on the rise. During the first few decades of the twentieth century, the number of women who died each year from uterine cancer nearly doubled, from 8,500 in 1900 to 14,871 in 1932.[5] It appeared to be among the most widespread types of cancer in women, and by many accounts was the commonest. "It is generally conceded," wrote one physician, "that cancer of the uterus is

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[3] Around this time, revenue from dues and annual donations totaled around $15,000. Patterson, 95.

[4] From a poster by New York City Cancer Committee, American Society for the Control of Cancer, reproduced in, *Cancer: Then and Now* (New York: The Chemical Foundation, Inc., no date (probably 1932)); FLH Papers, Box 39, Scrapbook 2.

[5] John A. McGlinn, "Can the Mortality and Incidence of Cancer of the Uterus Be Reduced?" *American Journal of Obstetrics and Gynecology* 29, 5 (May 1935), 621. McGlinn noted that "only 14,871" women died of the disease in 1932 as evidence that the frequency, which did not take into account population increase, had been greatly exaggerated; nonetheless, the number was still nearly double that of 1900. The incidence rate, as a measure of cases per 100,000 people, was not given.

the most frequent clinical form of malignant disease arising in women and as a lethal malady in the female it stands in the foremost rank. It constitutes," he continued, "…about one-third of all cases of cancer." For the sake of comparison, he noted that tuberculosis, which claimed around 28,000 female lives annually, remained the top killer in 1930 in the United States.[6]

But the gulf of medical knowledge between the two illnesses remained vast. Rates of tuberculosis were on the decline, the consequence of a comprehensive public health campaign in the early part of the century that both targeted unhygienic behaviors, such as spitting, and expanded funding for a network of sanatoriums where sufferers could avail themselves in isolation of the latest treatments, without having to worry about infecting healthy individuals. Scientists, moreover, were certain of its cause, the offending microorganism having been discovered by German bacteriologist Robert Koch nearly fifty years before. In contrast, cancer was just beginning to come into focus among medical researchers, who had much to learn about the disease. Lack of familiarity among physicians with the symptoms of cancer posed an additional obstacle. As Joseph Colt Bloodgood, a prominent surgeon at John Hopkins, observed, improper training of physicians meant that for most forms of the disease "inoperability or metastasis has doomed more than 50 per cent of the patients, while the probability of a cure in the remainder has been reduced to 10 per cent."[7] With regard to cervical cancer, the outlook was even more grim. "Apparently both surgery and radiation have reached their limits"

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[6] Bland, 1195.

[7] Joseph Colt Bloodgood, "Responsibility of the Medical Profession for Cancer Education, with Special Reference to Cancer of the Cervix," *American Journal of Cancer* 15, 3 (July 1931), 1577, 1579.

in the treatment of the illness, he wrote, declaring that it "is today predominantly a hopeless disease."[8]

Bloodgood's pronouncement notwithstanding, doctors had in their arsenal an array of tools for diagnosing the disease and treatments that had proven effective in some situations. Cancerous cells on a woman's cervix might have been less visible than malignant lesions on the surface of her skin, but the location of the organ still made it fairly accessible to physicians. Initial symptoms included irregular vaginal bleeding and discharge, or, as one observer described, "[s]potting, abnormal intermenstrual or postmenopausal bleeding, [and] watery—at times foul—discharge."[9] If the disease continued to progress, a "cauliflower growth" on the cervix unmistakably signaled the presence of cervical cancer.[10] Pelvic pain and discomfort were generally among the last symptoms to appear. Diagnosis could be confirmed by digital examination, supplemented by the use of a colposcope, an instrument which allowed direct magnification of the cells of the cervix. As with other forms of cancer, the principal types of treatment at the time consisted of surgery to remove cancerous cells, radiation therapy by way of either x-rays or beads of radium inserted into the cervix, or a combination of the two.

Many women with symptoms of the disease, however, probably did delay visiting their physicians for a number of reasons. First, irregular discharge and vaginal bleeding were often the markers of benign illnesses such as bacterial infections; hence, a

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[8] Bloodgood, 1577, 1579.

[9] Thomas Peightal, "Cancer of the Cervix: Symptoms and Diagnosis," *Medical Record* 132, 12 (Dec. 18, 1935), 550.

[10] Franklin I. Shroyer, "Prevention and Treatment in Cervical Uterine Cancer," *Radiology* 20, 2 (Feb. 1933), 137.

combination of denial and wishful thinking may have kept them out of the doctor's office. One observer lamented the nonchalance with which many females confronted these initial symptoms:

> The difficulty is that it is so hard to impress upon the average lay woman the importance of a blood-tinged discharge. If pain were only the first symptom, we would have a much better opportunity of seeing our cases early, but it is usually the terminal symptom. Leucorrhea [viscous white, yellowish, or greenish white discharge] does not impress upon women as being very serious; even if it is pink or blood-stained, they do not think much about it. Even if she has spotting or hemorrhage [*sic*], she is accustomed to having a menstrual flow every month and bleeding does not frighten her. If it were a man, he would run to the doctor right away, but not the woman.[11]

Second, Americans in the 1930s had good reason to fear cancer. Treatment with surgery and radiation worked in some cases, but for many patients, a diagnosis of cancer meant that death lurked nearby, waiting in the shadows for the right moment to strike. As historian James Patterson notes, "the large majority (probably four-fifths) of cancer patients did die from the disease, sometimes slowly, miserably, and at enormous emotional and economic cost to their families."[12] Biomedical knowledge had not yet progressed to the point where researchers could make any consistent statements about the disease. While boosters remained optimistic about the ability of scientists to find a cure for cancer, and soon, their assurance of imminent medical progress offered cold comfort to those who were living with, or, more commonly, dying from the disease. "Many people are afraid to even mention the name," said one physician. "They still endow the

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[11] George Gray Ward, "Cancer of the Cervix: Etiology and Prevention," *Medical Record* 132, 12 (Dec. 18, 1935), 548.

[12] Patterson, 113.

condition with an aura of mysticism and hopelessness and throw in the sponge at the mere mention of the dread word."[13]

The culture of the medical profession created additional hurdles for women. Although views of female sexuality were less restrictive than they had been prior to the 1920s, a decade in which sexual activity increased, particularly for young people, the new openness remained tempered by what historian Allan M. Brandt describes as "a strong crosscurrent of demands for moral rectitude and gentility."[14] More women may have been using birth control and engaging in premarital intercourse than before, but they most likely remained unnerved by the prospect of a gynecological exam. If, as Barron H. Lerner has written, women were uneasy with the mechanics of an examination for breast cancer, then inspections for cervical cancer must have caused at least as much as, if not more, distress.[15]

The discomfort of having one's sexual organs examined by a physician of the opposite gender might have been alleviated if women had the option of frequenting female doctors. Nearly all physicians in the 1930s, however, were male; women in the medical profession were most often employed as nurses—an essential, though subordinate role. The rate of medical school admissions for women had been falling since the late nineteenth century, a time when Victorian concerns about the appropriateness of male doctors examining female bodies prompted an upsurge in women

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[13] Patterson, 112.

[14] Allan M. Brandt, *No Magic Bullet: A Social History of Venereal Disease in the United States Since 1880* (New York: Oxford University Press, 1985, 1987), 122. See also John D'Emilio and Estelle B. Freedman, *Intimate Matters: A History of Sexuality in America* (Chicago: The University of Chicago Press, 1988, 1997), Chapter 11.

[15] Lerner reports that even in 1966, 19% of women in an American Cancer Society survey said that they felt embarrassed during cancer exams. Barron H. Lerner, *The Breast Cancer Wars: Hope, Fear, and the Pursuit of a Cure in Twentieth-Century America* (New York: Oxford University Press, 2001), 56.

joining the profession.[16] By 1909, however, the number of women enrolled in American medical schools had dropped by 35% over the previous fifteen years. For the next half-century, excluding times of war, medical schools would reserve 95% of their slots for men. Some administrators attributed the shift to changing attitudes among women, fewer of whom now harbored the desire to become doctors, while others kept admission rates low by claiming that women would abandon their careers after marriage.[17]

By 1930, doctors already knew that the prevalence of cervical cancer differed among various groups. As early as 1902, a researcher named W.J. Sinclair had reported in the *British Medical Journal* that the disease occurred "almost exclusively among the poor, the chronically overworked and underfed,… prolific, harassed, worried, drained by lactation, reposeless."[18] "Many writers," a physician observed over three decades later, "have noted cancer of the cervix to be a disease of the poor and ill-nourished classes."[19] Rates peaked among married women, and the disease was rare among Jewish females, an anomaly thought to be a function of the relationship between early circumcision and the near absence of penile cancer among Jewish men. Poverty, marital status, and religion appeared to be the three pertinent variables, and each would guide future etiologic investigations. None was a somatic category, however, the boundaries of class, marriage, and religion marked more by behavior than by physical or biochemical indicators. As a

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[16] Paul Starr, *The Social Transformation of American Medicine* (New York: Basic Books, 1982), 124.

[17] Starr, 124.

[18] W.J. Sinclair, "Carcinoma in Women, Chiefly in Its Clinical Aspects," *British Medical Journal* 2 (Aug. 1902): 321-327, in Douglas H. Sprunt and William M. Berton, "The Relationship of Socio-Economic and Racial Factors to Carcinoma of the Cervix as Indicated By a Mass Screening Program," *Acta Unio Internationalis Contra Cancrum* 16, 7 (1960), 1768.

[19] Frank R. Smith, "Etiologic Factors in Carcinoma of the Cervix," *American Journal of Obstetrics and Gynecology* 21, 1 (Jan. 1935), 18.

result, the social factors that contributed to the development of cervical cancer would find their way into the research spotlight, but scientists would not always identify them as such.

At the time, the most popular theory for carcinogenesis was chronic irritation, which could function as an explanation for everything from Kangri-burn cancer, caused by holding a basket of hot coals close to one's stomach, to oral cancers that resulted from constant abrasion from jagged teeth. Its application to cervical cancer etiology came from the disease's link to marital status. As a category, "marital status" functioned primarily to separate women who had given birth from those who had not; it did not imply, as it does today, corresponding social circumstances such as financial or emotional stability.[20] Researchers believed that the birthing process caused trauma to the cervix, resulting in irritation and inflammation that could lead to cancer. "Parturition," or childbirth, wrote Frank R. Smith, a physician practicing in New York City, "with its resulting damage to the cervix is a generally accepted etiologic factor in cancer of the cervix." Each successive live birth multiplied the damage. Those who had children in rapid succession, moreover, never gave their organs a chance to heal properly. "With the majority of cancer patients proving to be multiparous," he continued, "this factor increases in importance with the number of pregnancies."[21] As a physician named H.S.

[20] I realize that there are those who will argue that marriage today indicates precisely the opposite, or financial and emotional instability. Nevertheless, contemporary studies generally confirm that married men and women live longer and have higher incomes than their single counterparts.

[21] Smith, 24.

Crossen declared in 1933, it was "well established" that "cancer of the cervix comes from long-continued irritation in the form of chronic cervicitis."[22]

Identifying chronic irritation as an important factor in cervical cancer and centering research efforts on it enabled scientists to exert a measure of control over the disease. If cervical cancer formed when lacerations in the birth canal went untreated, then doctors had only to encourage women to exercise greater vigilance after childbirth to stem rising rates of incidence. Physician George Gray Ward recommended a seven-step preventive regimen, which included public education, twice-yearly gynecological examinations, prompt repair of all cervical lacerations after delivery, and the use of surgery, including by "radio-knife," to cure cases of chronic cervicitis.[23] Crossen advocated "simple excision of the affected area of the cervix." "It is clear," he continued, "…that an important step in preventing deaths from cancer of the cervix is the systematic and early removal of those chronic irritative lesions of the cervix which precede cancer."[24] A doctor based in Dayton, Ohio agreed that infections and irritations should be addressed, but proposed doing so with "a detoxicating diet, with colonic irrigations of two quarts of hot sodium bicarbonate solution morning and evening, until there is a noticeable softening of the mass and a reduction of the surrounding inflammation. About two weeks of such treatment will give the desired result."[25] Although his prescription may have been unorthodox, he supported the basic premise that the trauma of childbirth

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[22] H.S. Crossen, "Prevention of Cancer of the Cervix Uteri," *American Journal of Obstetrics and Gynecology* 26, 5 (Nov. 1933), 691.

[23] Ward, 548-549.

[24] H.S. Crossen, "Prevention of Cancer of the Cervix Uteri," *American Journal of Obstetrics and Gynecology* 26, 5 (Nov. 1933), 691.

[25] Shroyer, 136.

was the source of numerous forms of irritation that could become precancerous. Removal of the suspicious tissues constituted a crucial step in fighting cervical cancer.

Although treating cervical lesions before they had they chance to turn into cancer appeared to be a straightforward mission, a number of obstacles lay in its path. The central impediment was poverty, which affected people in myriad ways, not the least of which was in influencing their access to medical services. But observers also believed that the behavior of low-income patients contributed to their higher rates of cervical cancer. As Frank R. Smith revealed in his article in the *American Journal of Obstetrics and Gynecology*, many of his colleagues thought that "birth control [was] not practiced among the poor." He noted, moreover, that the patients in his study with cervical cancer suffered less than those in the control group from leucorrhea, an unusual whitish discharge that often signaled the presence of an infection, indicating that those in "the cancer group were too careless of body hygiene to recognize" it.[26] This combination of condescension and moralism toward the poor was not new within medicine; it echoed previous attitudes that blamed impoverished Americans for their own unhealthiness. In the mid-nineteenth century, for instance, contemporaries widely perceived cholera to be God's punishment upon those who were too ignorant or lazy to be bothered by living in squalor. Public health authorities and everyday citizens alike held immigrants, the poor, and racial and ethnic minorities responsible for outbreaks of bubonic plague, typhus, and trachoma at the end of the nineteenth and beginning of the twentieth centuries. Smith's observations, too, foreshadowed the argument, which would surface in the 1960s, that a "culture of poverty" kept poor people mired in their unfortunate situations and unable to

[26] Smith, 22, 23.

internalize the behaviors that might enable them to progress into the middle class. While the boundaries of such attitudes toward low-income women would shift in the coming decades, their outlines were firmly in place by the 1930s.

The 1940s: Funding and Diagnostics

Research on cancer slowed considerably from the end of the 1930s to the mid-1940s, a consequence of the drain on financial and intellectual resources brought about by World War II. The National Cancer Institute (NCI), founded by an act of Congress in 1937, spent just $2 million on research during the three and a half years between the attack on Pearl Harbor and the surrender of Japan; in the same period, the government expended $317 *billion* on the war effort.[27] In June of 1945, the NCI's budget for the previous fiscal year stood at $500,000, nearly unchanged since 1937-1938, the first year of its existence. Similarly, the budget of the American Society for the Control of Cancer, at $102,000, paled in comparison to the $15 million raised by the March of Dimes to fight polio. Many NCI staffers had jumped ship during the war to work in the field of radiation, and those who remained could not even produce enough work to fill the pages of the agency's journal. As James Patterson writes, "Kenneth Endicott, director of the NCI in the 1960s, recalled that 'the cancer research program was pretty dead all during the war years,' the NIH 'a nice quiet place out here in the country.'"[28]

Still, at the close of World War II, cancer researchers in the U.S. had reason for optimism. American scientific ingenuity, after all, had won the war. Improvements in

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[27] Patterson, 140.

[28] Patterson, 171.

radar, sonar, and anti-aircraft technology had given the Allies an edge in aviation, while the enhanced accuracy of bombers and submarines allowed more weapons to reach their targets. The atomic blasts over Nagasaki and Hiroshima, moreover, showcased American technological prowess to the world. Faith in science and technology, along with a confidence in the limitlessness of American progress, imbued the postwar research climate with a sense of hope. In addition, a booming economy after World War II played a large role in shaping the contours of cancer research in the ensuing decades. Increasing prosperity meant that ordinary Americans had more money to donate to charitable causes, making it possible for the newly reorganized American Cancer Society to raise $14 million for research by 1948.[29] At the same time, the country's growing affluence influenced cultural proscriptions regarding health and wellness. One's right to physical wellbeing moved from the exclusive domain of the upper class into the mainstream of American society.[30]

Medical investigation of cervical cancer, virtually imperceptible during World War II, began to pick up again, albeit slowly, near the end of the 1940s. While the handful of articles on the disease that appeared in medical journals continued to emphasize disparate rates among different populations, the groups into which authors divided their samples changed slightly. Still prevalent were distinctions of religion, as doctors continued to note the low incidence of the disease among Jewish women. But unlike a decade earlier, racial and ethnic disparities were beginning to attract attention, and not just between African Americans and Caucasians, but among whites, as well.

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[29] Patterson, 171.

[30] For a related discussion, see Patterson, 138-139.

Julian H. Lewis, a pathologist at the University of Chicago, described "the high incidence of uterine cancer" among "Negroes," while another observer pointed to the differential as evidence of the "racial proclivities" of cervical cancer.[31] Writing in the *British Journal of Cancer*, a researcher by the name of E.L. Kennaway examined the incidence of uterine cancer among a group of African Americans and ethnic whites, including Germans, Italians, Greeks, Polish, Irish, Austrian, and English and Scotch.[32]

The focus on ethnic whites stemmed directly from the connection between cervical cancer and economic status. For physicians who were looking for a pool of low-income white patients to study, New York City's Memorial Hospital provided a ready source. As Kennaway noted in his study of cervical cancer patients at the institution between 1916 and 1937, the disease in New York City was found almost exclusively "in the ward or clinic type of patient, rarely being seen in the type of patient who can afford private care."[33] Frank R. Smith's earlier study on "nationality" and cervical carcinoma similarly drew its sample from Memorial Hospital, where he held a position in the department of gynecology; in his work, he identified a high incidence among Italian and Irish women.[34] The ethnic white populations upon which these researchers drew for the most part consisted of people who had entered the United States in large numbers in the late nineteenth and early twentieth centuries, only to be targeted by proponents of

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[31] Julian H. Lewis, "Race and Disease," *The Proceedings of The Institute of Medicine of Chicago* 17, 5 (May 15, 1948), 116; Herbert F. Traut, "Cancer of the Cervix," *California Medicine* 70, 4 (Apr. 1949), 297.

[32] E.L. Kennaway, "The Racial and Social Incidence of Cancer of the Uterus," *British Journal of Cancer* 2, 3 (Sep. 1948), 186. Kennaway found the highest rates among Greeks, English and Scotch, Blacks, Italians, and Polish, and the lowest rates among Jewish women.

[33] Kennaway, 197.

[34] Frank R. Smith, "Nationality and Carcinoma of the Cervix," *American Journal of Obstetrics and Gynecology* 41, 3 (Mar. 1941), 424-430.

immigration reform in the 1910s and 1920s. Restrictionists argued that these newcomers were not only poor and uneducated, but also prone to various mental and physical afflictions, such as insanity and bodily deformities. Even if a majority of these populations no longer lived in poverty in New York City in the 1930s and 1940s, the correlation between the two persisted, as a number of physicians clearly continued to perceive them as indigent.

Although the association between black women and cervical cancer was just starting to surface in the 1940s, researchers did not assign an exclusively racial causality to the disease. Even Lewis, whose earlier monograph, *The Biology of the Negro*, had argued that blacks and whites responded to disease differently because of innate biochemical and physiological discrepancies between them, declined to cite immutable biological traits as the sole reason for the disparity, instead blaming "poor obstetrical care." He did not, however, abandon his previous viewpoint entirely; he thought that such an explanation could account for some, but not all of the excess, thereby leaving open the possibility that biology might play a role.[35] That the notion of a racial causation for cervical cancer failed to win a clear convert in someone like Julian H. Lewis, whose views on race were unambiguous, indicated the extent to which the disease's etiology at the time involved factors other than racial biology. Black women were poor, of course, but so were many whites, as evidenced by their emergence in medical literature on cervical cancer in the 1940s.

Along with the recognition of a racial divide in cervical cancer incidence, the expansion of the use of the Pap smear as a diagnostic tool marked the other major

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[35] Lewis, 116.

development of the decade. George Papanicolaou, a Greek immigrant who held both medical and doctoral degrees, had been working on the technology for a number of years, publishing a paper in 1917 in which he described the use of vaginal smears to determine the stages of the estrus cycle in guinea pigs. Several years later, he discovered that the technique could be applied to humans, in whom it could detect cancerous cells in the uterine cervix. Papanicolaou presented his findings at a conference in 1928, but his lecture, as well as the paper subsequently published in the meeting's proceedings, suffered a mediocre reception. His paper contained "such serious typographical errors as printing 'conscious cells' rather than 'cancerous cells' throughout," and was replete with "'poorly-reproduced photographs.'"[36] He felt, moreover, that insular thinking among pathologists and gynecologists had further prejudiced his audience against him. After spending roughly another decade working on his project, Papanicolaou in 1939 teamed up with Herbert Traut, a Cornell gynecologist whose association with New York Hospital facilitated access to samples of cells from female patients. Within four years, the two doctors published an article and a book detailing their findings, which were quickly confirmed by colleagues at other institutions.[37]

The Pap smear allowed physicians to screen for cellular changes in a woman's cervix, thereby enabling them to treat the area before it turned cancerous. By the end of the 1940s, it was the only preventive procedure for cancer that had an established record

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[36] Monica J. Casper and Adele E. Clarke, "Making the Pap Smear into the 'Right Tool' for the Job: Cervical Cancer Screening in the USA, circa 1940-95," *Social Studies of Science* 28, 2 (Apr. 1998), 259. See also Leopold Koss, "The Papnicolaou Test for Cervical Cancer Detection: A Triumph and a Tragedy," *Journal of the American Medical Association* 261, 5 (Feb. 3, 1989), 737-743.

[37] Casper and Clarke, 259-260; Koss, 737.

of effectiveness.[38] Its success as a diagnostic method can be attributed to a number of factors. First, at a time when the etiology of cancer continued to mystify the scientific community and treatment had yet to progress beyond surgery and radiation therapy, Papanicolaou's technique provided a way for physicians not simply to take command of the disease, but to do so at an early stage when medical intervention actually had something beneficial to offer. Second, the Pap smear as an inexpensive, simple technology got a major boost from its promotion by the American Cancer Society. As medical sociologists Monica J. Casper and Adele E. Clarke have written, the ACS, reorganized after World War II and newly flush with cash, emphasized early intervention at the same time that it poured funds into research to find a cure for the disease. After the organization endorsed the Pap smear in 1948, "pathologists and others, hoping to share in the newfound largesse of the ACS and its close ally the NCI," fell in line behind it.[39] As scientists refined the technique over the coming decades, the Pap smear would bring about an astounding reduction in American mortality from cervical cancer, from roughly 44 per 100,000 in 1947 to 8 per 100,000 in 1984, a nearly sixfold decrease in less than forty years.[40]

"Lifestyle" Factors

As the 1950s began, the association between cervical cancer and African American women garnered increased attention within the medical community. On the one hand, the disease's prevalence among African Americans was a familiar refrain. "It

[38] Patterson, 198-199.

[39] Casper and Clarke, 261,

[40] Casper and Clarke, 257.

is well known," wrote one physician, "that carcinoma of the cervix has a high incidence among colored women in the South."[41] Estimates of the disease's rank among all cases of cancer in black women ranged from 46% (compared to a corresponding figure of 25% for white women) to 63% or as high as 78%.[42] Data from the National Cancer Institute's surveys of cancer morbidity in ten metropolitan areas indicated that cervical cancer occurred twice as frequently in "Negro" women."[43]

On the other hand, the reasons behind the epidemiologic curiosity were still a matter of some debate. Scientists were relatively sure that the disease's expression was not a function of inherent biological traits. Unlike the case of skin cancer, for which African Americans' protective pigmentation was seen as a "true" racial characteristic, few at the time attributed their increased susceptibility to cervical cancer to a similar kind of racial factor.[44] "Many doubt," wrote Raymond F. Kaiser and Alexander G. Gilliam of the National Cancer Institute, "that the clearly excessive risk among the nonwhite women in the United States is due to actual racial susceptibility. Most workers contend that it is due instead to a number of factors which have been loosely classed as social or environmental."[45] Another physician agreed. Using similar language, he blamed "the

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[41] Manuel Garcia, "The Curability of Carcinoma of the Cervix in the Negro," *Southern Medical Journal* 45, 2 (Feb. 1952), 145.

[42] Garcia, 146-147. See also Bernard W. Robinson, "Malignancy in the Negro: A Statistical Review," *American Journal of Roentgenology and Radium Therapy* 66, 5 (Nov. 1951), 783-790.

[43] John R. Heller, Sidney J. Cutler, and William M. Haenszel, "Some Observations on the Epidemiology of Cancer in the United States," *Journal of the American Medical Association* 159, 17 (Dec. 24, 1955), 1629.

[44] See Heller et al., 1631.

[45] Raymond F. Kaiser and Alexander G. Gilliam, "Some Epidemiological Aspects of Cervical Cancer," *Public Health Reports* 73, 4 (Apr. 1958), 363.

influence of their economic and educational background, rather than…inherent biologic traits."[46]

The dismissal of immutable racial features as a factor in cervical cancer carcinogenesis probably came as a result of the progression of medical knowledge surrounding the illness. Since doctors from early in the twentieth century knew of the links between cervical cancer and poverty, their ensuing investigations focused on behavioral variables as keys to etiology. The increasing recognition of a racial divide in cervical cancer incidence in the 1950s could have functioned to debunk further the myth of differential biology by underscoring the etiologic importance of a series of lifestyle choices more common to those with lower incomes; in other words, it might have highlighted the relationship between race and class. Instead, however, the growing association between black women and cervical cancer added a racial component to the behaviors that were believed to play a role in carcinogenesis. As a result, race came to signify a set of characteristics that increased one's risk of developing the disease.

As scientists set about investigating the epidemiologic factors that stemmed from the circumstances of an individual's lifestyle, they discovered myriad variables from which to select. Each one shaped a patient's living situation and behavioral choices in innumerable, perhaps unquantifiable ways. One's health could be influenced by everything from access to medical services to tendency to seek treatment to likelihood of following through with prescribed therapies. Given the disease's disproportionate effect on poor women, doctors reasoned that an inadequate diet might be a contributing factor. One set of researchers crafted a study in which they questioned subjects on their

[46] Garcia, 147.

consumption of meat, beans, sweet potatoes, cereals, bread, green vegetables, organ meats, eggs, milk, and yellow cheese, hoping to isolate possible deficiencies of thiamine and riboflavin.[47] Others theorized that poor nutrition might affect the ability of one's tissues to resist disease.[48] Studies on diet and the etiology of cervical cancer generally either searched for a nutritional imbalance that might lead directly to cancerous growth, or tried to determine the overall connection between diet and carcinogenesis. Some would build on and expand the exploration of these relationships in the 1960s; however, dietary factors appeared to play a much smaller role to those interested in cervical cancer than, for instance, the eating habits of Chinese and Chinese Americans did to investigators of nasopharyngeal carcinoma.

If lifestyle factors comprised the largest group of epidemiologic variables in cervical cancer etiology, then sexual health and sexual practices represented the most substantial subcategory. Doctors knew that the frequency of the disease varied with a woman's marital status; single women exhibited low rates, while married women were more often afflicted. At one extreme were nuns, in whom the disease appeared to be exceptionally rare. One study of 3,280 nuns in four Canadian convents over a twenty-year period failed to find a single case of cervical cancer among 130 malignant tumors.[49] At the other extreme, the highest rates of cervical cancer could be found among poor

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[47] Edward Gomer Jones, Ian Macdonald, and Lester Breslow, "A Study of Epidemiologic Factors in Carcinoma of the Uterine Cervix," *American Journal of Obstetrics and Gynecology* 76, 1 (July 1958), 1-10. The authors did not explain the significance to cancer etiology of these two substances.

[48] Ruth S. Taylor, Leonid S. Snegireff, and John E. Gordon, "Cervical Cancer as a Mass Disease," *American Journal of the Medical Sciences* 229, 3 (Mar. 1955), 339.

[49] See Lauren V. Ackerman and Juan A. del Regato, *Cancer: Diagnosis, Prognosis, and Treatment* (St. Louis: C.V. Mosby Company, 1954), 894; Taylor et al., 341.

women, particularly African Americans, who married early and had multiple pregnancies and deliveries.

What, then, were the precise implications of marital status? To some observers, the significance of marriage lay in the extent to which it could predict sexual habits. Married women engaged in sexual activity, got pregnant, and had children, while single women did not. Although the premise, commonplace in the 1930s, that cancer developed from untreated lacerations in the cervix was losing ground as a theory of carcinogenesis, it continued to influence a number of researchers. If childbirth caused cervical trauma, they reasoned, then women with the most children should have the greatest propensity for the disease. Data from the studies of nuns appeared to support their contention. Others, however, deemed it an oversimplification. "Many other differences exist between the life of women of the outside world and those in a convent," wrote a team of epidemiologists. "Perhaps the issue is summarized as well as possible by Novak who recognizes 'considerable differences of opinion among gynecologists as to the importance or unimportance of chronic irritative lesions as predisposers to cancer.'"[50]

Aside from ongoing speculation regarding the carcinogenic capacity of chronic cervicitis, the medical community had few additional theories which could link marriage to cervical cancer. In a search for answers, they returned to populations with high rates of the disease in the hopes of isolating causative factors. Edward Jones, Ian Macdonald, and Lester Breslow, the physicians who tried to pinpoint deficiencies of riboflavin and thiamine, hypothesized that patterns of sexual activity might play a role. For a study of 858 cervical cancer patients and matched controls from the Los Angeles County Hospital

[50] Taylor et al., 338-339.

and private physicians' practices, they employed "skilled interviewers" to question subjects about their sexual behavior, noting that patients "had been told [at the beginning of the interview] that information of a very personal nature would be needed."[51] In collecting data on "frequency of coitus, duration of total marital state, years with first husband, total number of permanent partners, and total number of casual partners," they discovered that cancer patients married earlier and got pregnant at a younger age than those in the control group.[52] Another researcher had similar findings; in his study, women with cervical cancer "tended to marry relatively often, at a younger age, to have more pregnancies and deliveries, to be less often nulliparous, and to develop their disease if married, even though nulliparous."[53] Twenty appeared to be the all-important number. Women who married before this age had the highest rates of the disease: 44.6%, compared with 29.7% of controls in a study by H.L. Lombard and E.A. Potter.[54]

While some believed that early marriage affected the development of cervical cancer by increasing the duration of a woman's reproductive years, and hence her exposure to precancerous lesions and lacerations, others saw it as a shorthand way to identify a collection of socioeconomic criteria that played a role in cancer etiology. The social circumstances of poor African American women, the group at highest risk for the disease, brought about somatic consequences, of which cervical cancer was one. Low-income black women were more likely to marry early and to lack adequate access to

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[51] Jones et al., 5.

[52] Jones et al., 5-7.

[53] Damon, 507. A nulliparous woman is one who has never given birth to an offspring.

[54] H.L. Lombard and E.A., Potter, *Acta Unio Internationalis Cancer Cancrum* 6 (1950), 1325, in Taylor et al., 341.

obstetrical care. They had higher rates of venereal diseases such as syphilis, a consequence of poor "sex hygiene" which, according to one set of researchers from the National Cancer Institute, also led to high rates of cancer of the prostate and penis among "Negroes."[55] A number of scientists, including Edward Jones and his coauthors, identified instability of marriage, with its attendant corollaries of "rapid maturation sexually and a haste to begin early, and early to terminate, the reproductive phase of biologic destiny."[56] A different group of NCI researchers echoed the argument, naming "illegitimate births, syphilis, early sexual relations, multiple sexual partners, and prostitution" as outcomes of an unstable marriage.[57] Others attempted to locate biochemical evidence for the constellation of factors that led to early marriage and increased cervical cancer risk. Previous work had found unusually high estrogen levels in women with the disease, prompting Jones, whose study also measured urinary excretion of estrogen, to speculate that hormones might induce "a greater desire for early marriage and childbearing.[58] Lombard and Potter agreed, blaming "excessive hormonal stimulation" which led to "excessive sexual urge."[59]

The correlation that medical professionals drew between high rates of cervical cancer and a configuration of sexuality that included early sexual relations, insufficient sexual hygiene, venereal diseases, and "excessive sexual urges" revealed, for one, the explanatory burden placed on marriage. Researchers endowed the institution with the

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[55] Heller et al., 1631.

[56] Jones et al., 10.

[57] Kaiser and Gilliam, 364.

[58] Jones et al., 1.

[59] See Taylor et al., 341.

ability to predict both patterns of disease and social outcomes. It was an indicator of particular situations, both biochemical and non-medical. Someone who wanted to wed early was apparently so invigorated by hormonal urges that she could not control her desire to engage in sexual intercourse early, often, and with multiple partners. Sexually transmitted diseases, prostitution, and cervical carcinoma were the inevitable consequences of such an unfortunate lifestyle. It showed, as well, the extent of the association between poverty and sexual immorality. On the one hand, nuns represented paragons of virtue, rewarded for their celibacy and piety with a notable absence of the disease. On the other hand, the association between cervical cancer and sexual behavior seemed to explain its prevalence in poor people. The lack of sexual decency among low-income African American women, in particular, meant that they could ill escape cancer.

Medical historian Allan M. Brandt has illuminated the ways in which disease, the dimensions of which are often influenced by the intersections of gender, class, ethnicity, and race, can reveal a society's values. Medicine, he writes, "is not just affected by social, economic, and political variables—it is embedded in them." Venereal disease, in particular, offers a way to understand conflicts of sexuality and morality; since the end of the nineteenth century, it has been used as "a symbol for a society characterized by a corrupt sexuality." It has represented "pollution and contamination, and [been] cited as a sign of deep-seated sexual disorder, a literalization of what was perceived to be a decaying social order." During the twentieth century, it "came to be seen as an affliction of those who willfully violated the moral code, a punishment for sexual irresponsibility."[60] The relationship between cervical cancer etiology and sexual

[60] Brandt, 5.

behavior imbued the discourse surrounding the illness in the 1950s with a sense of moralism, drawing attention to the habits of a group of people who departed from a normative version of sexuality. That the highest rates of cervical cancer could be found among black women cast a racial tinge onto much of the discussion of sexual behavior.

The elevated frequency of cervical cancer among African American women contrasted dramatically with the near absence of the illness in Jewish communities. Researchers had known for decades that Jews had extremely low rates of the disease. One study reported rates seven and one-half times higher among non-Jews, while another found the incidence among non-Jewish women at Mount Sinai between 1893 and 1906 to be twenty times greater than for their Jewish counterparts.[61] But patterns among Jewish women posed a challenge for researchers, as they appeared to contradict the theory that the disease resulted from trauma during labor and delivery. Many Jews married young and had large families; if childbirth and its associated injuries were in fact the most important causative factors, particularly in women who were still maturing, then Jewish females should have rates that approached or exceeded those of other productive populations. Instead, they were significantly less likely than non-Jews to have the disease.

Doctors set about identifying alternate ways to explain the epidemiologic data from their Jewish patients. Some returned to circumcision, a practice that had been examined in the 1930s, in the hopes that it could explain the low incidence of both cervical and penile cancers within the community, but the results were inconclusive. The authors of one article, for instance, encountered an unexpected obstacle when they

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[61] Max Sugar and Walter E. Levy, "The Incidence of Carcinoma of the Cervix in Jewish Women," *New Orleans Medical and Surgical Journal* 103, 10 (Apr. 1951), 424.

realized that many of their male subjects did not know their circumcision status.[62] A second religious practice that could be relevant to the formation of the disease, and which set Jews apart from other groups, was a religious proscription against sexual activity during menstruation and after childbirth. Jewish law prohibited intercourse from the onset of menstruation to twelve days henceforth, for forty days after the birth of a boy and eighty days following the birth of a girl.[63] Perhaps, doctors reasoned, these abstentions played some role in curtailing the development of cervical cancer. The problems with such a premise were twofold. First, short of stationing an observer in the bedroom of each of their subjects, researchers had no way of ascertaining whether people actually adhered to religious recommendations, regardless of how firmly they were stipulated. Second, there was no obvious reason why abstaining from intercourse at these times would affect rates of cervical cancer; hypotheses mostly involved the protection of vulnerable tissue in the cervix from unnecessary irritation.

The status of Jewish women within the work of cervical cancer affected the direction of investigations into etiology. In earlier decades, and particularly in the late nineteenth and early part of the twentieth centuries, Jews were considered a racial group in the same way that blacks, Mexicans, Germans, and Irish were. By the 1950s, however, researchers generally agreed that "Jew" was a religious designation, rather than one which connoted a particular set of physical, physiological, and biochemical

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[62] John E. Dunn, Jr. and Philip Buell, "Association of Cervical Cancer With Circumcision of Sexual Partner," *Journal of the National Cancer Institute* 22, 4 (Apr. 1959), 749-764.

[63] Taylor et al., 343-344. See also Ernest L. Wynder, Jerome Cornfield, P.D. Schroff and K.R. Doraiswami, "A Study of Environmental Factors in Carcinoma of the Cervix," *American Journal of Obstetrics and Gynecology* 68, 4 (Oct. 1954), 1018-1020; Kennaway, 197.

characteristics.[64] Most scientists—at least among those interested in cervical cancer—grouped Jews with Hindus and Muslims, a designation underscored by their interest in religious differences in sexual practices. Research on Jewish women, then, served to reinforce the cultural origins of cervical cancer, as doctors clearly realized that one's religious orientation influenced one's habits, but not one's biochemical composition.

The relationship between cervical cancer and the Jewish religion also shaped the ways in which race functioned within the epidemiologic debate. Although the link between black women and the illness emerged slowly in the 1930s and 1940s, by the postwar period it was a well-recognized pattern. Cervical cancer had not become a "black" disease in the way that prostate cancer would later carry a heavy association with African American men, but its connection to behavior meant that race and culture would be inextricably linked. If Jews were able to avoid the disease by acting in particular ways, then the conduct of poor black women similarly put them at risk. Race had come to represent a variety of social and cultural factors that made women more vulnerable to cervical cancer: early onset of sexual activity, multiple sexual partners, instability of marriage, and poverty.

Race, Behavior, and Risk

The connection between race and cervical cancer, an association that had solidifed during the 1950s, became even more entrenched in the following decade. The disease's racial differential appeared to be increasing, at least in some locales. Whereas the

[64] The physician who suggested that to more convincingly resolve the issue of "racial" resistance his colleagues should examine Arab women, who, like Jews, were of "Semitic" origin, was in the minority. See the discussion section following Wynder et al., 1050.

National Cancer Institute in 1955 had reported that rates in black women were twice as high as for whites, a 1961 study of African Americans in New York City found rates in the two populations differing by a factor of three.[65] Earlier theories on carcinogenesis fell further out of favor, including the focus on untreated cervical lacerations, which had achieved a reasonable degree of acceptance among colleagues; this enabled new work, particularly in the burgeoning field of mental health, to enter the spotlight. As a result, the ways in which scientists described race as a risk factor for cervical cancer would shift, influenced not only by a growing emphasis on the materiality of risk itself, but also by an increasing assurance that race acted as a determinant of culture.

The reasons for the racial divide in the incidence of cervical cancer were to some extent a consequence of the disease's etiology, which scientists were still working to unlock. But the methodological approaches of a number of researchers offered a partial explanation as to why studies continued to confirm the differential. A pair of physicians who examined 350,000 vaginal smears of white and black women in Memphis and Shelby County, Tennessee over a five-year period discovered a rate of cervical carcinoma that was twice as high among African Americans in one screening, and three times as high in a subsequent screening. Their analysis, however, suffered from extreme economic bias, as they drew their "Non-White Group," a population "practically 100 percent Negro," from a charity clinic and the white group from private physician's offices. "[E]conomics," they admitted, "plays a part."[66]

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[65] Lucia J. Dunham, "Cancer of the Uterine Cervix in Negro Women in New York City," *Acta Unio Internationalis Contra Cancrum* 17, 7 (1961), 910.

[66] Sprunt, 1768-1769.

Other surveys which failed to uphold a link between race and the disease suffered from similar problems of design. As a doctor named Armand J. Pereyra reported in 1961, "[o]ur studies do not support the purported ethnic relationship in cervical cancer. On the contrary, they reveal a higher prevalence of cervical cancer among the whites than among the nonwhites." But his study, which examined whites, blacks, "Mexican-Indians," and a "scattering" of other nonwhites, drew its subjects from the population of inmates at the California Institution for Women in Corona, California, a group with little variation in economic and social factors.[67] Such work, which ostensibly aimed to investigate racial differences in cervical carcinoma, ended up instead providing evidence for the disease's link to class. So ingrained was the association with race, however, that many researchers proved unable to abandon it. Race itself—or, more specifically, being black—increased the probability that one would fall victim to the illness.

The transformation of race into a risk factor for cervical cancer came within a shift in biomedical approaches to chronic, degenerative disease. As a number of scholars have described, by the 1960s medical professionals increasingly saw ailments including heart disease and cancer in terms of individual risk.[68] Isolating characteristics such as smoking or high cholesterol allowed physicians to identify patients who were more likely to develop certain chronic illnesses. At the same time, high cholesterol and high blood pressure, conditions which increased one's chances of developing chronic ailments, themselves became diseases which were defined by a numerical cutoff and needed to be

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[67] Armand J. Pereyra, "The Relationship of Sexual Activity to Cervical Cancer: Cancer of the Cervix in a Prison Population," *Obstetrics and Gynecology* 17, 2 (Feb. 1961), 154-159.

[68] Robert Aronowitz, *Making Sense of Illness: Science, Society and Disease* (New York: Cambridge University Press, 1993); Allan M. Brandt, "'Just Say No': Risk, Behavior, and Disease in Twentieth-Century America," in *Scientific Authority and Twentieth-Century America*, Ronald G. Walters, ed. (Baltimore: Johns Hopkins University Press, 1997), 82-98.

treated. Pharmaceutical companies played a leading role in the transformation by promoting medications for these new conditions.[69]

The idea that each individual had the responsibility to take the precautions necessary to avoid disease had a long history in public health discourse, from prescriptions for women to shorten their hemlines to avoid sweeping up tuberculosis germs early in the twentieth century to more recent admonitions against houseflies, which were thought to spread polio. But unlike those models, which aimed to identify people who either did not know they were sick or who refused to seek medical attention, the new approach focused on distinguishing those individuals who were at risk for certain illnesses and providing clinical intervention, either through drug regimens or by encouraging behavioral modification. It reflected the shift from infectious and communicable afflictions, which could be contracted through an unlucky event, such as drinking contaminated water, to chronic ones that developed from a lifetime of bad habits. Everyone was now considered to have some level of risk, a formulation with the potential to shape conduct based on the latest public health pronouncements. Those who were at the highest risk were expected to change their habits to reduce the likelihood of getting sick or be held accountable for any ensuing illness. The focus on individual behaviors, such as smoking or consuming an unhealthy diet, brought a renewed emphasis on morality to the practice of medicine and marked a change from the more medical notion of disease causation that had gained sway with the advent of the era of bacteriology and reigned during much of the twentieth century.

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[69] Jeremy A. Greene discusses the central role played by drug companies in defining hypertension, diabetes, and high cholesterol as symptomless diseases that needed to be treated with new medications in *Prescribing by Numbers: Drugs and the Definition of Disease* (Baltimore: Johns Hopkins University Press, 2007).

In cervical cancer etiology, the range of individual behaviors seen as increasing one's risk was circumscribed by race. Studies of sexual activity, in particular, which researchers knew related in some way to the incidence of the disease, attributed social habits to specific races and ethnicities. One investigator, for instance, described marriage as "a relatively unstable institution among the Negroes," while another cited "poor penile hygiene" among "Negroes, Puerto Ricans, Hindus, and Chinese," even as he referred to race as "so-called race."[70] Conduct that undoubtedly could be found among different groups, although not necessarily ones defined by ethnicity, race, or even class, now occupied the intersection of race and risk. But unlike quitting smoking or watching one's diet, both of which lay within the purview of habits under the control of an individual, behaviors bounded by race were unchangeable. If race affected culture, which in turn influenced behavior, then the ways in which one conducted oneself were determined largely by circumstance and, therefore, unalterable.

Sexual behavior, a mainstay of earlier work on cervical cancer, continued to occupy those in the field in the 1960s, although the focus of their inquiries shifted slightly. Whereas earlier discussions of female sexuality had centered on marital status, a shorthand way of identifying those women who were likely to reproduce, and were thus at risk for the disease, attention now turned to details such as monogamy, number of sexual partners, and frequency of sexual intercourse. Economic and social circumstances, not marital status, became the primary determinants of sexual activity.

Perhaps the group of subjects who showed the most evident convergence of race with social and economic disadvantages was, as Armand J. Pereyra discovered, female

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[70] Dunham, 911; Lionel Sandler Auster, "Genital Cancer in Jews," *New York State Journal of Medicine* 65, 2 (Jan. 15, 1965), 273.

prisoners. He reported elevated levels of sexual activity among his population, demonstrated by "multiple marriages, common-law husbands…and incidental high venereal disease rate."[71] Prostitution was widespread, as well, reaching thirty percent of subjects in a separate study.[72] What these women had in common, he noted, was a "general promiscuity," of which sexual dysfunction was merely the symptom. His articles lacked the principled tone of, for instance, World War II campaigns against venereal disease, in which "promiscuous girls," who carried the potential to infect soldiers with dangerous ailments, represented the breakdown of traditional social boundaries.[73] Researchers, too, had implicated sexual immorality in the spread of cervical cancer a decade earlier. Unlike in the 1950s, however, when they in part blamed hormonal urges in poverty-stricken women for sexual patterns which could lead to the disease, their current work contained a critical undercurrent toward the low-income female prisoners whose "promiscuity" had saddled them with sexual illness and neoplastic growth. If these women could not restrain themselves from making poor choices, then perhaps cervical cancer was the inevitable result. The centrality of sexual behavior to investigations into cervical cancer etiology ensured that the discussion would retain a perceptible element of moral rectitude.

The most prolific writer in the 1960s on the subject of sexual behavior and the incidence of cervical cancer was Ira D. Rotkin, the director of the Cancer Research Project at the Kaiser Foundation Research Institute in California. At the time, a number

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[71] Pereyra, 157.

[72] K.S. Moghissi, H.C. Mack, and J.P. Porzak, "Epidemiology of Cervical Cancer: Study of a Prison Population," *American Journal of Obstetrics and Gynecology* 100, 5 (Mar. 1, 1968), 607-614.

[73] See Brandt, Chapter 5.

of scientists were investigating the psychological aspects of carcinogenesis. Studies in previous decades had examined the mental states of cancer patients after diagnosis, attempting to trace the effects on one's psyche as the illness progressed. But Rotkin and his colleagues were more interested in the ways in which emotional factors might influence the etiology of cancer. Did social maladjustment or neurosis, for instance, make someone more likely to develop neoplastic disease? How did attitudes toward sexuality and sexual behavior affect the formation of disease in one's reproductive organs?

Rotkin published some of his findings in a 1965 issue of the *Archives of General Psychiatry*. Entitled "Psychosexual Factors and Cervical Cancer," the article described the relationship between psychological and sexual variables in both cervical cancer patients and a group of controls. Writing with two colleagues, he discovered that women with the disease were less social than the control group, "less emotionally responsive," and suffered from fewer "clinical neuroses."[74] They found few differences between the two groups in "general affect toward coitus," measured by responses to the question of what subjects thought about during sexual intercourse. For instance, statements such as "'Wish it were over'" and "'I don't enjoy sex'" indicated negative affect; "'With my husband I didn't like anything, with my lover I thought sex'" signified ambivalence; and answers of "'Don't think, DO'" and " 'I'm thinking about what I'm doing, nothing else'" meant that the subject had a positive attitude toward sex.[75] Their data, the authors suggested, provided a "solid basis" for further investigation into "psychological variables,

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[74] I.D. Rotkin, N.L. Quenk, and M. Couchman, "Psychosexual Factors and Cervical Cancer," *Archives of General Psychiatry* 13, 6 (Dec. 1965), 535, 536.

[75] Rotkin et al., 534.

both, social and sexual, which may possibly influence neoplastic change in the human cervix."[76]

Other pursued a psychological basis to the growth of cervical cancer, as well. The authors of a textbook on the disease cited "[s]adness and despondency" as "fertile ground for the development of malignancy," finding "a high degree of personal unhappiness and adversity" in a group of forty-nine cancer patients and a poor response to medical intervention in "those with a poor background and hopeless future."[77] What was notable about studies such as this was not their inclusion of psychiatric and psychological principles, as doctors had been noting for years that cancer patients often appeared to be more depressed than those without the disease; it was a predictable reaction to a diagnosis that many considered akin to a death sentence. Rather, the ways in which the authors drew connections among sexuality, emotional health, and the development of neoplastic disease marked a new understanding of the relationship of bodily to psychological health, with sexuality as the mediator between the two. Whether one appreciated sex too much or not enough, evidence seemed to show that emotional well-being directly affected the body's ability to resist disease.

The incorporation into studies on cervical cancer of concepts from psychology and psychiatry reflected recent growth in the two fields. Before World War II, psychiatry was a minor specialty within medicine, plagued by a lack of precision in diagnosis, poor understanding of the etiology of various mental illnesses, and a shortage of effective therapies. The postwar period provided both a new model for patient care and expanded

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[76] Rotkin et al., 532-536.

[77] Graham et al., 32-33.

opportunities for doctors in the field. Care of the mentally ill moved from the asylum to the community, spurred by physicians who had succeeded in treating patients under a similar model during the war. Funds from the federal government to support research into mental illness and to provide training through fellowships and state and institutional grants increased the money available for professionals. The war, moreover, ushered in a period of increased acceptance for the principles of psychotherapy, which had proven to be an effective treatment for combat-related stress. As historian Gerald N. Grob has written, its popularity in the 1950s was enhanced by a number of factors which included the expansion of community practices in psychiatry, a "general receptivity toward psychological explanations," and "an economic prosperity that created a middle-class clientele able to pay for and eager to use psychological services."[78] A decade later, the movement for community psychiatry in the early and mid-1960s combined "[f]aith in the redemptive qualities of modern psychiatry" with a desire to end poverty and discrimination by dismantling the structural barriers that prevented individuals from reaching their full potential.[79]

Ira D. Rotkin, the researcher who identified "psychosexual" characteristics of cervical cancer patients, went even further than his peers in examining the sexual habits of women with and without cervical cancer. In one study, he looked at douching practices in cancer patients and a group of controls, including the substance used (among them Lysol, vinegar, saline, soap, and boric acid), as well as the effects of abortion,

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[78] Gerald N. Grob, *The Mad Among Us: A History of the Care of America's Mentally Ill* (New York: The Free Press, 1994), 224. See also Chapters 8 and 9.

[79] Grob, 250.

frequency of intercourse, type of contraception, and duration of menstruation.[80] In other work, he delved into the role of sexual positions, which he divided into seven combinations of sitting, standing, and lying down; it was, he noted, the first such work to do so. Rotkin also scrutinized the "masturbatory practices" of his group, quantifying his results into a chart listing the numbers of patients and controls who used various implements, such as rocks, streams of water, candles, and vegetables.[81]

Regardless of whether Rotkin was actually able to obtain reliable data on intimate sexual behavior through the use of questionnaires and interviews, his work, along with that of his colleagues, provided a catalog of the types of behaviors that were circulating in the sexual vocabulary at the time. Studies of sexual positions, masturbatory objects, and douching defined appropriate sexualities by outlining the practices which diverged from them. At the same time, as historians John D'Emilio and Estelle B. Freedman have written, sex by the 1960s had become an integral part of American culture. The "sexual liberalism" of the era, to use their term, allowed a greater degree of permissiveness regarding premarital and extramarital intercourse, masturbation, and sexual imagery.[82] The wide range of behaviors that Rotkin and other scientists examined in their studies simply reflected changing sexual values and mores.

It is one thing, however, to identify practices that are out of the ordinary and another to implicate them in the etiology of a chronic, potentially deadly illness. In their assertions that "promiscuous" women and the sexually deviant were far more susceptible

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[80] I.D. Rotkin and R.W. King, "Environmental Variables Related to Cervical Cancer," *American Journal of Obstetrics and Gynecology* 83, 6 (Mar. 15, 1962), 720-728.

[81] I.D. Rotkin, "Sexual Characteristics of a Cervical Cancer Population," *American Journal of Public Health* 57, 5 (May 1967), 815-829.

[82] See D'Emilio and Freedman, Chapter 12.

to the disease than the less sexually active, cervical cancer researchers in the 1960s chose the latter. Rotkin's approach assumed that the disease was somehow related to non-normative sexual practices, which then became a way by which to identify those who might develop it. Cervical cancer was, in many ways, an inescapable hazard for impoverished women of color. On the one hand, these women got cancer because their abnormal sexual habits put them at risk for the disease. On the other hand, sexual immorality was part of the culture of being poor and nonwhite.

The notion that low-income women of color were at greater risk for cervical cancer because of race-related social factors resonated with the broader idea that poverty itself was a kind of pathology with detrimental moral and social effects. Government officials believed that a "culture of poverty" kept the poor trapped in their circumstances. Unable to break free of the traditions which had been handed down from generation to generation, they failed to adapt to changing conditions and take advantage of new opportunities. The national War on Poverty, announced by President Johnson in 1964, sought to ameliorate impoverishment by instituting programs to ease the transition of poor people into the middle class. Head Start, community action programs, and a job corps aimed to give poor people "a hand up, not a handout" by equipping them with the educational and job skills necessary for productive employment.[83]

In 1965, around the time that Rotkin was studying the sexual behavior of cancer patients, Daniel Patrick Moynihan, a political science Ph.D. and assistant secretary of labor in the Johnson administration, published a report examining the status of African Americans. Entitled *The Negro Family: The Case for National Action*, it sought to

[83] Nicolas Lemann, *The Promised Land: The Great Black Migration and How It Changed America* (New York: Alfred A. Knopf, 1991), 144-153.

explain why African Americans continued to lag behind whites in educational and social achievement after ten years of civil rights victories, including the Supreme Court decision in *Brown v. Board of Education*, which struck down segregation in public schools in 1954, and the 1964 Civil Rights Act that outlawed, among other things, discrimination in employment. Moynihan found Negro social structure, particularly the Negro family, in deep disarray, "highly unstable, and in many urban centers…approaching complete breakdown." It is, he wrote, "the fundamental source of the weakness of the Negro community at the present time."[84] While the problem had its roots in slavery, urbanization, unemployment, poverty, and lack of education, Moynihan identified as the most important factor the disappearance of black men, who, humiliated by their inability to provide a stable income for their families, abandoned them. As a result, traditional male and female roles were reversed, forcing women to head families at a rate more than double that for whites.[85] "Broken homes," welfare dependency, juvenile delinquency, and lack of scholastic achievement followed.

The "culture of poverty" thesis and related belief that being poor was a pathological state meant that socioeconomics had the power to dictate behavior. Poverty could lead young people to crime, tear families apart, and encourage social breakdown. It might also produce disease, but not for medical reasons, such as lack of access to hospital care. Instead, poverty as pathology implicated social factors in disease etiology,

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[84] Daniel Patrick Moynihan, *The Negro Family: The Case for National Action* (Washington, DC: United States Department of Labor, Office of Policy Planning and Research, 1965), 5. See also Allen J. Matusow, *The Unraveling of America: A History of Liberalism in the 1960s* (New York: Harper & Row, 1984), 194-196; Frances Fox Piven and Richard A. Cloward, *Regulating the Poor: The Functions of Public Welfare* (New York: Pantheon Books, 1971), 192-195; Lee Rainwater and William L. Yancey, *The Moynihan Report and the Politics of Controversy* (Cambridge, MA: The M.I.T. Press, 1967).

[85] Moynihan, 15-27, 30-34, 9.

using a cultural argument to translate race into physical illness. Sexual deviance and abnormal sexual practices could increase a woman's chances of developing cervical cancer, but such behaviors inescapably belonged to the "culture" of being poor.

If the work of cervical cancer researchers was remarkable in the detail with which it catalogued sexual behavior, it was also significant both in the perspective that it offered on the sexual practices of a number of women in the 1960s and in its "kitchen sink" approach to the study of the disease. Examinations of such wide-ranging variables suggested that no one in the scientific community really understood what caused the illness. The recognition that cervical cancer tended to follow patterns of venereal disease would prove to be essential, as would early speculation that the carcinogenic agent might be a virus transmitted through sexual contact and more likely to be harbored in uncircumcised males.[86] But the breakthroughs that these discoveries would engender lay years in the future, and biomedical researchers still had a long way to go in determining the etiology of the disease.

By the end of the 1960s, the scientific community had for the most part reached agreement on a multifactorial etiology for cervical cancer. The habits and lifestyle choices common to those of low socioeconomic status appeared to create conditions that increased one's risk for the disease, although doctors were still not sure precisely what the variables were. As one group of investigators put it, "[n]o such agreement exists as to those specific factors which may identify the low socioeconomic groups and which may directly relate to…prevalence."[87] Race, by acting as a mediator of behavior, remained an

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[86] See, for instance, Pereyra, 157.

[87] David C. Figge and James L. Bennington, "A Study of the Epidemiology of Cervical Cancer," *American Journal of Obstetrics and Gynecology* 98, 4 (June 15, 1967), 524.

essential feature. Unlike prostate cancer, for which socioeconomic status could not explain racial differentials, cervical cancer incidence did appear to vary along economic and social lines. However, the insistence on a racial divide for the disease kept the focus away from class, in effect racializing a series of behaviors that researchers considered income-related. As the decade drew to a close, one physician's impertinent description of a composite high-risk candidate for cervical cancer succinctly summarized the often contradictory findings of the previous forty years of epidemiologic work:

> Patient is a 50.5 year old Negress immigrant who is tall, underweight, and unhappy; currently incarcerated at an urban prison for prostitutes located in Mexico City. She is separated or divorced (?) (patient unsure….I.Q. less than 100) from her uncircumcised, penniless, untouchable, Hindu husband, whom she married before the age of 20, after having been sexually promiscuous from the age of 12, which resulted in less than 3 full-term pregnancies complicated by syphilis and uterine prolapse. In addition to the preceding the examiner feels that her poor female hygiene is compounded by the fact that she prefers coitus during her menses as does her twin sister, who is presently being treated for cervical cancer in Tokyo, Japan.[88]

Conclusion

Between 1930 and 1970, biomedical researchers were remarkably consistent in their search for the etiology of cervical cancer. Early recognition of links to economics and religion suggested that development of the disease was influenced largely by behavioral factors. The precise variables that different generations of investigators scrutinized over the years may have changed, but they nearly always fell under the rubric of behavior—and in particular, because of the cervix's location and function, sexual behavior.

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[88] Richard T. Rappolt, Sr., "The Composite High Risk Cervical Cancer Candidate," *American Journal of Obstetrics and Gynecology* 95, 7 (Aug. 1, 1966), 1009.

Since sexual habits always remained central to studies of etiology, cervical cancer provides a way by which to chart changing perspectives on sexual behavior. Sexuality has always been a convenient focal point for fears of social and moral disorder, with venereal disease the inevitable result of poor decision-making. Researchers defined and redefined the boundaries of normative sexuality over four decades, considering any deviation to be a possible risk factor for the disease. The study of cervical cancer reveals, moreover, how the notion of individual accountability has shaped cancer discourse, as the link between sexual practices and cervical cancer ensured that the illness would retain a punitive element. It was each woman's responsibility to obtain necessary medical checkups, to get proper treatment for cervical lacerations, and to engage in normal sexual relations. Anything less raised the risk of developing a disease which, if not fatal, certainly might have been curtailed by practicing socially appropriate behavior.

The emerging importance of race to the epidemiology of cervical cancer, however, introduced an additional dimension to investigations into etiology. The ways in which race functioned within biomedical surveys diverged from other areas of cancer research. Racial differentials in skin cancer incidence, for example, led scientists to explore the idea of biological susceptibility, particularly in the 1930s. Despite early scientific identification of the relationship between behavior and cervical cancer, however, cervical cancer did not escape a racial etiology. The result was not a shift in etiologic work toward physiology or genetics. Instead, behavior became subsumed within the bounds of race. If behavior had a major influence on one's likelihood of developing the disease, then race circumscribed an individual's range of acceptable conduct. Furthermore, cervical cancer's established link with class meant that both race

and economics came to be seen as determinants of the personal habits that affected carcinogenesis. Low-income nonwhite women, especially African Americans, shouldered an increasing share of blame for their condition in the postwar years, yet seemed unable to break out of cultural prescriptions regarding behavior. Race became a risk factor for cervical cancer precisely because of the ways in which it shaped habits and determined conduct.

The study of cervical cancer, finally, offers an illustration of the ways in which epidemiology shapes examinations of etiology, and how race either complicates or confirms the adequacy of the categories into which investigators have organized their subjects. Epidemiologic methodology calls for populations to be divided along lines of demographic and behavioral variables for the purposes of scientific research, although such methods, with the notable exception of lung cancer, have largely proven incapable of determining the causation of chronic conditions. Since epidemiologic methodology excels at identifying associations between illnesses and the lifestyle or other factors that affect risk, subjects will always be grouped according to external categories in order to more easily isolate influential variables. As a result, race will continue to play a crucial role in the epidemiology of chronic disease, even if the mechanisms of its interactions within the body remain a mystery.

Chapter 4

Culture or Genetics?
Nasopharyngeal Carcinoma in Chinese and Chinese Americans

Articles on nasopharyngeal carcinoma appeared infrequently in the medical literature in the early twentieth century. Within the hierarchy of known human cancers, it occupied a lowly position, too rare to merit extended scientific studies. Unlike skin cancer, it was not easy to detect in its early stages. Even in advanced cases, cancer of the nasopharynx could encompass such a wide range of symptoms involving nearly every section of the head and neck that physicians often had a hard time recognizing it.

One of the few details of which medical doctors were aware was the disease's apparent propensity for Chinese victims. The earliest reference to their susceptibility was probably an article by Oscar Thomson which appeared in a 1923 issue of the *Chinese Medical Journal*.[1] Nasopharyngeal carcinoma's identity as a disease of Chinese populations would guide research for the rest of the twentieth century. As scientists in the postwar United States searched for ways to explain anomalous rates of the illness among Chinese immigrants and American-born Chinese, the ensuing discussion marked a moment in the classic, ongoing debate over the relative roles of genetics versus environment in the expression of disease. Such a conversation hinged not only on scientific advances in the fields of human genetics and molecular biology, but also on the specific groups which researchers chose to study. By narrowing the range of variables, the ways in which one selected his or her subjects not only affected the results, and hence

[1] See J. Oscar Thomson, "Cervical Lympho-sarcomas, with an Analysis of Ninety Cases," *Chinese Medical Journal* 37 (1923), 1001; cited in K.H. Digby, Wm. Lai Fook, and Yeung Tsaw Che, "Nasopharyngeal Carcinoma," *British Journal of Surgery* 28, 112 (Apr. 1941).

the conclusions which he or she might draw, but also illustrated which aspects of being Chinese were thought to be medically significant.

Throughout the decades of research on nasopharyngeal carcinoma, its association with the Chinese diaspora remained central. In pursuing the etiology of the disease, medical researchers could not separate the objectivity of science from the subjectivity of racial meanings. Rather, they demonstrated its fallacy by revealing the extent to which science was not only informed by, but also played a role in shaping racial ideologies. Doctors classified and reclassified the meanings of "Chinese," focusing on various arrangements of behavior, customs, and genes. Whether they emphasized environment or genetics depended on current scientific thinking. Being Chinese may have been a risk factor for cancer of the nasopharynx, but it was a fluid category, signifying different things at different times, all of which shared the possibility of contributing to neoplastic processes of disease. While nasopharyngeal carcinoma's racial association remained constant, definitions of what it meant to be Chinese changed over the course of the twentieth century to accommodate new etiologic knowledge and scientific discoveries, in the continual pursuit of an understanding of race which could sufficiently account for the disease's epidemiology.

Early Knowledge and Etiological Theories

Before World War II, nasopharyngeal carcinoma, or NPC, was neither a common nor a well-researched disease. One set of authors reported that it comprised just 2% of malignant growths seen at the Head and Neck Clinic of the Memorial Hospital in New

York City.[2] "Primary malignant disease of the nasopharynx," wrote another group led by Kenelm H. Digby, a professor of surgery at Hong Kong University, "cannot be a common disease in Great Britain. It receives scant attention in text-books on surgery and rarely appears in British statistics of malignant disease treated by radiotherapy."[3] Not only was the disease infrequently seen, but when a case did turn up in a physician's office, it was easily mistaken for other ailments, usually syphilis or tuberculosis. Digby's group described a patient who ingested cod-liver oil and undertook therapies of fresh air and sunlight in the hopes of ridding himself of a disease which he suspected was tuberculosis. When doctors removed and dissected his enlarged cervical glands, they discovered that he had not been afflicted with the infectious ailment after all, but with cancer of the nasopharynx. "The poor fellow eventually died," they reported, "possibly from an overdose of opium taken to end his sufferings."[4]

The telltale signs of nasopharyngeal carcinoma constituted a long list that could involve the ears, nose, eyes, scalp, mouth, throat, and neck. The earliest symptom was often a headache, sometimes confused for a brain tumor by physicians and patients alike. Tinnitus and deafness were common; many patients also gave off a "disagreeable odour" upon their "expired breath." Polyps in the nose caused sufferers to experience "nasal obstruction, unilateral to begin with, but eventually becoming bilateral," along with discharge of blood and excessive mucous from the organ. The disease could affect one or both of a patient's eyes; Digby and his researchers described "one case of a woman who

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[2] Charles M. Thompson and Elmer M. Grimes, "Carcinoma of the Nasopharynx," *American Journal of the Medical Sciences* 207, 3 (Mar. 1944), 342.

[3] Digby et al., 1941, 517.

[4] Digby et al., 1941, 521.

was very miserable and often wept." "[I]t was noticed," they wrote, "that the tears only flowed from one eye, the secretion of the opposite lacrimal gland apparently no longer being affected by emotional stimulus."[5] Paralysis of a nerve in the neck, moreover, rendered some patients unable to fully rotate their heads or shrug their shoulders. The chief symptom in the neck, however, was enlarged cervical glands. Photographs of unfortunate victims with growths on their necks filled the pages of medical journals. Some patients had slight enlargements visible only because they caused the individual's neck to appear asymmetrical. In extreme cases, though, the growths could cause a person to appear as though he had swallowed something about the size of a basketball, and it was now lodged between his chin and his shoulders.[6]

Treatment for the disease, Digby and his colleagues wrote in 1941, was "depressing." At the time, mainstream physicians held in their arsenal three major ways of treating cancer: surgery, x-rays, and radium. Surgery worked well with diseases such as skin cancer where the growths were visible, or in cases of internal cancer in which the tumors were well-defined and had not yet metastasized. Radium beads could be placed into a needle which was then inserted directly into the tumor; during the 1930s, it was a common treatment for breast cancer. Physicians had been tweaking x-ray therapy since the end of the nineteenth century, when they discovered that the waves could kill cancer cells; in the intervening decades, they continued to refine the treatment to minimize the loss of healthy, non-cancerous cells. But nasopharyngeal carcinoma did not respond well to any of the three available treatments. Because it spread rapidly to surrounding areas,

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[5] Digby et al., 1941, 523.

[6] See Digby et al., 1941, 521-533; Fred Z. Havens, "Carcinoma of the Nose, Nasopharynx and Paranasal Sinuses," *Surgical Clinics of North America* 21, 4 (Aug. 1941), 1009-1015.

surgery was often impossible. In cases in which doctors attempted to remove the growth, the disease returned quickly. Radium could effectively shrink enlarged glands, but carcinoma likewise returned, often within six months. Deep x-ray therapy held more promise, but for the treatment of nasopharyngeal cancer it was still largely untested.[7]

Around the time of World War II, then, nasopharyngeal carcinoma was rarely seen, not easily treated, and poorly understood. Most physicians probably had never encountered a case in their practices. Among those who conducted research on the disease, however, one feature stood out: rates among Chinese patients were markedly higher than for any other group. Digby observed a "great number of sufferers from this condition in Hong Kong and South China" and during the 1930s wrote several articles on it, many of which were based on observations of patients in his practice in Asia.[8] According to his data, between 1930 and 1938 cancer of the nasopharynx constituted 18% of all malignant tumors seen at the Hong Kong University Hospital.[9] A physician in Shanghai reported that while Chinese made up just 40% of his patients, they accounted for 100% of his sixteen cases of nasopharyngeal carcinoma.[10] By 1940 or so, the association between Chinese and NPC seemed to be well established, with different writers noting the disease's "great frequency in China" and "the interesting incidence of Chinese in these cases."[11] Moreover, the disease increased in frequency as one traveled

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[7] Digby et al., 1941, 536-537.

[8] Digby et al., 1941, 517.

[9] Hayes Martin and Stuart Quan, "The Racial Incidence (Chinese) of Nasopharyngeal Cancer," *Annals of Otology, Rhinology & Laryngology* 60 (Mar. 1951), 168-169.

[10] Ibid., 170.

[11] A.M. Dunlap, "Malignancy of the Nasopharynx and Eustachian Tube," *Chinese Medical Journal* 53, 1 (Jan. 1938), 68; Charles M. Thompson and Elmer M. Grimes, "Carcinoma of the Nasopharynx," *American Journal of the Medical Sciences* 207, 3 (Mar. 1944), 343.

southward, with Cantonese, including those in Hong Kong, exhibiting the highest rates. It tended to strike males rather than females, and displayed a relatively young age distribution compared with other types of cancer. Digby and his colleagues found the highest incidence of nasopharyngeal carcinoma in men between the ages of thirty-six and forty, perhaps fifteen or twenty years earlier than peak rates for lung or prostate or stomach cancer.[12]

Medical scientists initially suspected that exceptional rates of NPC among Chinese might be an irregularity related to lifestyle. "Chinese," in this formulation, designated a discrete group of people with distinctive cultural practices. Consequently, researchers scoured the habits of the population for behaviors which could account for the disease's epidemiologic patterns. The most prominent theory at play in the 1930s had appeared in a 1924 article by a researcher named Dobson, who attributed the susceptibility of southern Chinese to the characteristics of the houses in which they lived. Typical dwellings lacked sufficient means to ventilate soot and smoke from whatever substances were burned inside. The resulting pollutants, he speculated, irritated nasopharyngeal lymphoid tissues and predisposed occupants to the disease. "Native houses in this region are not ventilated," wrote Dobson. "A small hole in the roof or a window a foot square are about the only openings except the door, which is generally closed. The fireplace has no flue and is fed with dry grass or a poor quality of wood. Tobacco smoke is universally present, while guttering candles light the ancestral table

[12] Digby et al., 1941, 519.

and incense adds its quota. It is rare that one sees a kerosene lamp that does not smoke; open kerosene or clay lamps burning pea-nut [*sic*] oil are very general among the poor."[13]

Against the backdrop of contemporary cancer research, Dobson's inhalant theory seemed extremely promising. It paralleled the connections that scientists in other areas were just beginning to draw between lung cancer and tobacco smoke; by suggesting the carcinogenic potential of types of smoke peculiar to the southern Chinese way of life, it had the potential to explain NPC's unique patterns of incidence. Researchers soon discovered, however, that Dobson's assumptions failed to hold for other populations, notably women and American-born Chinese. When they began to examine these groups a decade or so later, they found rates of the disease contrary to what they expected. If Dobson had been right, then women, who spent far more time indoors and exposed to the suspected etiologic culprits of wood and kerosene smoke, should show a greater incidence of nasopharyngeal carcinoma than men. American-born Chinese adhered to a more Westernized way of life than their counterparts in Asia by eating an American diet and living in Western-style dwellings which, presumably, were well-ventilated. But rates among Chinese women were lower than those of the male members of their households, and Chinese Americans still developed NPC at much higher rates than non-Chinese, despite Americanized cultural practices.[14]

In light of new evidence of disease rates among women and Chinese Americans, Dobson's theory appeared inadequate to explain the susceptibility of Chinese to nasopharyngeal carcinoma. However, the detection of elevated rates among Chinese

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[13] Dobson, *Chinese Medical Journal* 38 (1924), 786, in Digby et al., 1941, 519.

[14] See Digby et al., 1941, 520; Lauren V. Ackerman and Juan A. del Regato, *Cancer: Diagnosis, Treatment, and Prognosis* (St. Louis: The C.V. Mosby Company, 1947), 331.

Americans represented an important etiological discovery. If a segment of a population relocated from its native environment to another part of the world, where migrants grew up in a foreign culture with separate customs, and still developed a particular disease at anomalous rates, then any hypothesis which focused exclusively on lifestyle factors could not sufficiently account for differences in disease incidence. Based in part on the deficiencies of Dobson's ideas, scientists began to look beyond cultural explanations. Over the next fifteen years or so, their search for similarities within the Chinese disapora would lead them to a genetic foundation for nasoharyngeal carcinoma. But Dobson's model, which offered an early argument for the importance of environmental factors in carcinogenesis, did not disappear completely. Despite its shortcomings, his inhalant theory enjoyed enormous longevity over the following decades. Nearly seventy years later, researchers in the 1990s were still testing it, their interest a measure of the continued uncertainty regarding the origins of the disease.

A Genetic Etiology

The turn from environmental to genetic factors in postwar research on the association between Chinese and nasopharyngeal cancer occurred in part because no reliable environmental carcinogens had been identified for the illness; NPC appeared to favor Chinese regardless of their place of birth or country of residence. But before investigators could assess the risk of developing the disease for various populations of Chinese, they first had to clarify what they meant by racial susceptibility, and indeed, what they meant by "Chinese."

Defining "Chinese" for the purposes of oncological research into the etiology of nasopharyngeal cancer did not at the time arouse a debate among scientists. For those working within the mainstream medical establishment, the answer to the question was straightforward: a Chinese person was someone whose ancestry could be traced to China. For African Americans, racial discourse emphasized percentages of blackness and whiteness, as well as variations in skin color, distinctions which could have severe legal and social consequences. Physicians investigating cancer etiology in the early to mid-twentieth century—particularly skin and cervical cancers, both of which had differential rates for whites and blacks—paid close attention to the boundaries of racial categories, as one's racial background was thought to influence the types of ailments to which one might fall prey. In contrast, the medical literature on NPC contained no discussions about the nature of race or genetics. Those writing about Chinese appeared to give little thought, moreover, to issues of miscegenation. Someone was either Chinese, or wasn't. The concept of multiracialism among Chinese lacked resonance to such a degree that, unlike with African Americans, it did not even represent a potentially volatile issue.

Once researchers accepted the unexamined assumption that Chinese people were those who either had been born in China themselves or whose forebears were, they set about locating the population in various parts of the world. The geographic sites in which Chinese emerged as research subjects offered a lesson in the reach of the Chinese diaspora. After China and Hong Kong, Singapore and Taiwan were the most frequent sites of study in Asia. In North America, Hawaii and New York topped the list, although other regions also appeared. One researcher examined sixty-four Chinese cancer patients

in Cuba, of whom twenty, or 31%, had nasopharyngeal cancer.[15] Another wrote in 1922 of seventy-nine cases of the disease at the Mayo Clinic in Rochester, Minnesota; he did not mention racial incidence, leaving colleagues to assume that the proportion of Chinese seeking treatment at the clinic was not high.[16]

At the time, the Chinese population of the United States was around 78,000.[17] Chinese began coming to the country in significant numbers in the 1840s, drawn by the search for gold in western states and by the promise of work on the railroads that were snaking across the expanse of the U.S. from coast to coast. From the mid-nineteenth century until the 1950s, most Chinese immigrants to the U.S. were from Guangdong, a province in southern China bordered by Hong Kong and Macau. Since the Tang dynasty (618-907), Guangdong province had been a hub of international trade and commerce. Its principal city, Canton, was a thriving, cosmopolitan metropolis, the population of which included ethnic Chinese, Jews, Arabs, Persians, and Indonesians who traded with British and other Europeans for silk, porcelain, gems, woods, and incense. By the 1840s, Canton was China's point of entry both for Westerners and for foreign influence, and place of exit for exported goods such as tea.[18] Within Guangdong province, up to half the immigrants came from Taishan County, one of ninety-eight counties in the province. Just half the size of Rhode Island, Taishan County supplied about 45% of immigrants in 1876

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[15] E. Martinez, "El Cancer de la Naso-faringe en los Chinos," *Boletín de la Liga Contra el Cáncer* 15 (1940): 276, in Martin and Quan, 170.

[16] G. New, "Syndrome of Malignant Tumors of the Nasopharynx," *Journal of the American Medical Association* 79 (July 1922), 10, in Martin and Quan, 170.

[17] The Chinese population of the U.S. in 1940 was 77,504. Ronald Takaki, *Strangers From a Different Shore: A History of Asian Americans* (Boston: Little, Brown and Company, 1989), 239.

[18] Peter Kwong and Dušanka Miščević, *Chinese America: The Untold Story of America's Oldest New Community* (New York: New Press, 2005), 19-21; Sucheng Chan, *Asian Americans: An Interpretive History* (Boston, MA: Twayne Publishers, 1991), 5-7.

and 50% after 1882.[19] Up to the 1960s, more Chinese immigrants in the U.S. could trace their ancestry to this small area of China than to any other county.[20]

While most Chinese—about 77% by 1870—settled in California, smaller numbers made their way to Boston, New York, and points in the Midwest, such as Chicago.[21] After 1865, they also settled in southern states such as Louisiana, South Carolina, and Mississippi, where they served as field hands and plantation workers, replacing slaves who had been emancipated during the Civil War.[22] Hostility toward the newcomers surfaced nearly with the first immigrants, whom adversaries labeled as dirty, diseased, immoral, and opium-addicted. Restrictive legislation soon followed. The 1875 Page Law curbed the entry of Chinese women, who were assumed to be coming to the U.S. for purposes of prostitution. Economic competition between Chinese and working-class whites prompted the passage of the 1882 Chinese Exclusion Act, which barred most classes of Chinese workers from entering the country for a period of ten years. (The legislation was renewed in 1892 and then indefinitely until its repeal during World War II.) As a result, the populations of various Chinese communities across the United States began to contract as some men returned to China while others, faced with a shortage of Chinese women, remained lifelong bachelors. Some practiced homosexuality or married and had children with non-Chinese women, but the sight of Chinatowns filled with single, aging men remained the norm until well into the twentieth century.

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[19] Kwong and Miščević, 19.

[20] Kwong and Miščević, 20.

[21] Takaki, 79.

[22] For an excellent discussion of Chinese in the South after the Civil War, see Moon-Ho Jung, *Coolies and Cane: Race, Labor, and Sugar in the Age of Emancipation* (Baltimore: Johns Hopkins University Press, 2006).

Despite their numbers, both as a percentage of the overall population and in raw terms, Chinese in California did not attract much attention from investigators of nasopharyngeal cancer until the 1960s. The reasons for this omission are unclear. It is possible that those who were likely to undertake such research simply settled in other parts of the country, where they drew on local residents for their clinical data. Perhaps, also, Chinese NPC sufferers in California were less likely than those in other parts of the country to seek help for medical problems, possibly for financial or cultural reasons. The appearance of Chinese in New York in the medical literature on cancer of the nasopharynx is less surprising than the neglect of California Chinese, for the city boasted the second-largest Chinatown in the United States, after San Francisco; in 1940, its population stood at 12,753.[23] Unremarkably, Hawaii found its way into research on cancer of the nasopharynx. Although Chinese in Hawaii constituted a minority, even among Asians, the archipelago had long been a destination for Chinese immigrants, who made the journey across the Pacific to work on the territory's sugar plantations and constituted about 9% of the population of the islands in the early 1920s.[24] Thus, with the exception of California, the geographical locations of American research on nasopharyngeal cancer predictably followed the patterns carved by the entry and settlement of Chinese immigrants.

If the first step in determining the link between race and nasopharyngeal cancer lay in finding populations of Chinese to study, then the next was outlining what one meant by racial susceptibility. In 1951, Hayes Martin and Stuart Quan, two physicians

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[23] Takaki, 250.

[24] Takaki, 132. During the second half of the nineteenth century, 46,000 Chinese landed in Hawaii. Takaki, 31.

practicing in the Head and Neck Service of New York City's Memorial Hospital, published an article on the subject in the *Annals of Otology, Rhinology and Laryngology*. Entitled "The Racial Incidence (Chinese) of Nasopharyngeal Cancer," the piece, which would soon assume a position of influence within the field, reviewed 358 cases of the disease observed and treated at their hospital between 1935 and 1950. Statistical evidence demonstrated, they wrote, that the Chinese were "more susceptible to nasopharyngeal cancer than other races." "[S]o far as we know," they continued, "this is the only example of racial susceptibility (excluding environmental factors) to a specific anatomic form of cancer so far established."[25]

By identifying Chinese "racial susceptibility" as a major reason for elevated rates of cancer of the nasopharynx, Martin and Quan made it clear that they favored a genetic basis for the ailment. Although others had reached the same conclusion—the article included a literature review of work by like-minded colleagues—their own experiences furnished the most convincing data. All thirty-seven of their cases of nasopharyngeal carcinoma were in Chinese immigrants. Some of the patients, moreover, had resided in the U.S. for decades, absorbing and practicing an American way of life. "At Memorial Hospital," they reported, "we have observed several cases in Chinese professional men—physicians, merchants, and educators who had come to this country as children and had lived for many years under American standards of diet, hygiene, and sanitation." To these authors, the fact that NPC continued to occur in Chinese migrants who grew up under significantly better living conditions than those in China demonstrated the importance of heredity over environment. Although they had yet to see a case of the

[25] Martin and Quan, 168-169.

disease in an American-born Chinese "so far," they did not rule out the possibility. "For the time being, at least," they asserted, "it seems safer to hold that the tendency is racial and/or inherited and that it is not determined by the immediate environment."[26]

The Memorial Hospital physicians were careful to distinguish between a "racial susceptibility independent of any environmental influence" and one "due to some local or geographic environmental factor."[27] A risk factor related to the local environment could be something like Dobson's inhalant theory or a regional difference in air quality, whereas a predilection that was unaffected by such variations would constitute an inherited tendency toward the development of the disease. Quan and Martin's preference for genetic over environmental factors as the primary cause of nasopharyngeal cancer prompted a response from others who were eager to test a particular component of their hypothesis. These scientists tended to focus on one of two issues, each of which could either complicate or lend support to a genetic heritage for the disease. First, the team from Memorial Hospital speculated that other "Oriental" races, which "in general have at least a distant common origin," might share a predisposition to NPC.[28] Second, their assertion that nasopharyngeal cancer continued to occur at high rates among Chinese immigrants in the United States inspired studies of data gathered in different parts of the country.

In 1960, the *Hawaii Medical Journal and Inter-Island Nurses' Bulletin* invited a Honolulu physician named L.Q. Pang to submit an article based on a talk he had given the previous year at the annual meeting of the American Laryngological Association.

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[26] Martin and Quan, 171.

[27] Martin and Quan, 168-169.

[28] Martin and Quan, 171.

Pang analyzed nearly three dozen cases of nasopharyngeal cancer seen in his practice, alluding to the "well known fact that the Chinese are the most susceptible to this disease."[29] Of his thirty-four cases, twenty-seven, or 80%, occurred in Chinese; four Filipinos, two Japanese, one Korean, and one Caucasian accounted for the remaining 20%.[30] Pang took issue with Quan and Martin's claim that they had never seen an instance of nasopharyngeal carcinoma in an American-born Chinese. The statement, which he called "amazing" and "certainly contradictory" to his experience, "was made to refute the environmental factors as a causative factor and to hold that the tendency is racial and inherited." In contrast to the results from Memorial Hospital, a full 60%, or sixteen individuals, of Chinese patients with NPC in Pang's study had been born in the U.S.[31] While he declined to contest the idea of a genetic basis for the disease and remained neutral on the issue of etiology, Pang's work provided clinical proof of cases of the disease among Chinese Americans and filled in some of the evidentiary gaps in the work of his colleagues.

In response to the first of Martin and Quan's issues, the susceptibility of other "Oriental" races, a number of scientists took up the challenge to test rates of NPC among an expanded pool of subjects. The Memorial Hospital physicians had listed "Filipino, Japanese, Korean, Indo-Chinese, etc." among the "Oriental" groups which ought to be investigated.[32] But "Oriental" was a flexible category, and the groups which constituted

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[29] L.Q. Pang, "Carcinoma of the Nasopharynx," *Hawaii Medical Journal and Inter-Island Nurses' Bulletin* 19, 5 (May-June 1960), 357.

[30] These figures are taken directly from Pang's article. His arithmetic, however, appears to be faulty, as 27 plus 8 is 35, not 34. Pang, 358.

[31] Pang, 358.

[32] Martin and Quan, 171.

it varied. One team consisting of a Taiwanese physician and a prominent American cancer researcher named E.V. Cowdry, who worked at Washington University in St. Louis, looked at rates of NPC among Japanese, Thais, Indochinese, South Vietnamese, and Indonesians. They found that, for the most part, other "Orientals" did not develop the disease at rates similar to Chinese.[33] In Punjab, India, a second group of researchers set about examining figures for cancer cases at the Victoria Jubilee Hospital, part of the Medical College in Amritsar. Their aim, noted the authors, was "to find out if it [nasopharyngeal cancer] has any racial predilection for all orientals or if there are any special factors operating in China to account for this heavy incidence, i.e. 18 per cent of all cancer cases." Data revealed rates of the disease in India that corresponded to the figures for American hospitals: in both countries, cancer of the nasopharynx accounted for around 2.5% of the overall cancer caseload. This, wrote the scientists, was "clear proof against any racial predilection of this disease for all orientals."[34]

The work of the Indian researchers, Cowdry's team, Martin and Quan, and others who probed the issue of "Oriental" susceptibility exposed the workings of a discourse rooted in ideologies of colonialism and power. As scholar Edward Said has argued, Orientalism served as a "corporate institution for dealing with the Orient," a system of knowledge by which the West could imagine, understand, and classify the East.[35] It provided an idea against which Europe and America could define themselves, not just

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[33] See Shu Yeh and E.V. Cowdry, "Incidence of Malignant Tumors in Chinese, Especially in Formosa," *Cancer* 7, 3 (May 1954), 425-436.

[34] Tulsi Das, G.M. Taneja, M.R. Chaddah, and D.B. Minocha, "Nasopharyngeal Carcinoma," *Annals of Otology, Rhinology, and Laryngology* 64, 4 (Dec. 1954), 890.

[35] Edward W. Said, *Orientalism* (New York: Vintage Books, 1978), 3.

culturally and ideologically, but also materially.[36] If Asia and its residents were effeminate, steeped in luxury, and weak in military prowess, then the image of Westerners stood in opposition to such qualities. Said writes that after the Enlightenment European culture not only produced the Orient "politically, sociologically, militarily, ideologically, scientifically, and imaginatively," but Orientalism also enabled the West to know and represent the Orient in ways that reverberated throughout the twentieth century.[37]

The uses of race and ethnicity within medicine, of course, have long been a source of tension for physicians and patients alike, and scientific definitions of each have sparked controversy since the nineteenth century. Moreover, although medicine may be based on tenets of the physical world, it nonetheless remains a creation of humankind, and as such is not immune to shifting social currents. The use of the term "Oriental" did not necessarily imply that one subscribed to its underlying ideology; its appearance in the literature in articles on nasopharyngeal cancer may simply have indicated its ubiquity in fields as diverse as literature, politics, and medicine. But when doctors in the 1950s employed "Oriental" to refer to ethnic groups linked more by the ways in which Europeans and Americans viewed them historically than by—to the extent which they could be applied—considerations such as a shared genetic heritage, it illustrated the continued convergence of and inextricable linkages between contemporary scientific and racial ideologies.

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[36] For a discussion of the role of Orientalism in the making of Chinatowns in the U.S. both as physical spaces and as imaginary ones, see John Kuo Wei Tchen, *New York Before Chinatown: Orientalism and the Shaping of American Culture, 1776-1882* (Baltimore: Johns Hopkins University Press, 1999).

[37] Said, 3.

Because "Oriental" represented an ideological category, rather than one centered on biomedical criteria such as age, sex, or body weight, the ethnic groups which constituted it depended on researchers' interpretations of the term. Subsuming Indians, for instance, under the umbrella of Orientalism revealed the ways in which the boundaries of the category rested upon political and ideological considerations, rather than on narrowly defined methodological ones. In the U.S., Indians had legally been grouped with East Asians for much of the 20th century, suffering alongside Koreans, Chinese, and Japanese as targets of anti-miscegenation laws, immigration exclusion, and the denial of landownership under the California Alien Land Law.[38] However, physical anthropologists in the 1950s—even those who insisted on the increasingly outdated practice of dividing humans into major taxonomic units—did not classify Indians and other South Asians as "Mongoloids."[39] At the same time, Chinese, Koreans, Japanese, Filipinos, Indonesians, and Vietnamese, who were at one time considered "Mongoloids," were linked by an assumption of a common genetic heritage based on physical similarities. As a category for medical research, "Oriental" suffered from outlines too broad and boundaries too porous to furnish consistent results.

Although the search for a genetic basis for cancer of the nasopharynx made its way to the forefront of medical research on the disease in the early- to mid-1950s, a handful of scientists continued to develop theories implicating environmental factors in its etiology. The newest one came from a researcher by the name of Balasingham, who

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[38] Takaki, 300.

[39] See, for instance, Stanley M. Garn and Carleton S. Coon, "On the Number of Races of Mankind," *American Anthropologist*, New Series, 57, 5 (Oct. 1955), 996-1001; Carleton S. Coon, *The Origin of Races* (New York: Knopf, 1962). The other four taxonomic units were Caucasoid, Congoid, Australoid, and Capoid. Coon argued that the five races of mankind evolved separately into *Homo sapiens*, with Caucasoids achieving *sapiens* status first.

worked at the Singapore University Medical School. The scientist took as a starting point Chinese people's reputed addiction to opium, speculating that the burning substance might produce some kind of carcinogenic material which could irritate the nasal passages of exhaling addicts. Others found the idea promising but expressed reservations regarding some of its precepts. Kenelm H. Digby wrote, "[o]pium smokers do certainly inhale and expel the smoke through their nostrils, and the theory would largely interpret the geographical and racial distribution of the complaint….But many of the cases in the wide experience of my friend, Dr. Shi Man Wai, and in my own observation, have not appeared to be opium smokers."[40] The researchers investigating nasopharyngeal cancer in Amritsar, India offered another word of caution, calling the hypothesis "unlikely" since "opium contains no carcinogenic substance."[41] Digby outlined additional theories which his colleagues continued to advance despite their "fantastic" nature. These included the habit of Chinese to "take their food very hot," which Digby dismissed as irrelevant since people ate through their mouths, not their noses, and the "excessive hawking of phlegm from the back of the throat habitual among the coolie class" which some thought predisposed Chinese to nasopharyngeal catarrh, or inflammation of the mucous membranes.[42]

At the end of the 1950s, researchers were not much closer to determining the causes of nasopharyngeal cancer than they had been in 1930. They knew that the disease favored Chinese people, that rates were higher among southern Chinese than their

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[40] The reference to Balasingham is in Kenelm H. Digby, "Nasopharyngeal Carcinoma," *Annals of the Royal College of Surgeons of England* 9, 4 (Oct. 1951), 255.

[41] Das et al., 892.

[42] Digby, 1951, 254-255.

neighbors to the north, that other "Oriental" groups did not appear to share a predisposition, and that even those who had lived for years in the United States continued to develop it at high rates. But studies probing genetic links to the disease yielded conflicting data, and continued attempts to isolate environmental carcinogens peculiar to Chinese habits and ways of life, which had looked so promising just a few years earlier, seemed not to be generating satisfying results. In fact, scientists had achieved greater success in determining what did not cause cancer of the nasopharynx than what did. But medical research proceeds in fits and starts, and what appear to some to be dead ends can spur the imagination of others. The difficulties of confirming a genetic etiology for nasopharyngeal carcinoma would play a role in leading researchers back to an exploration of environmental factors in the coming years.

Race and Environment

As the decade of the 1960s began, scientists interested in cancer of the nasopharynx for the most part fell into one of two camps: those who supported a genetic basis for the disease and those who favored an environmental etiology. The genetic explanation seemed to be based upon two pieces of evidence. The first drew upon the observations of physicians who saw Chinese patients in their practices. These doctors collected data which demonstrated clearly that Chinese men, in particular, could develop the disease at a rate up to fifty times that of Caucasians. The second was Hayes Martin and Stuart Quan's argument for an inherited tendency for NPC, an assertion based on the fact that they had seen cases of the disease among Chinese immigrant "professionals" who enjoyed an American standard of living. At the same time, researchers including

Paul Steiner and L.Q. Pang downplayed the possible genetic connection. Whereas Steiner emphasized environmental factors, Pang put forth data from his own practice, countering Martin and Quan's less than comprehensive evidence with his own observations confirming that American-born Chinese could, in fact, suffer from nasopharyngeal cancer.

The first major challenge to the prevailing framework came in 1962 in the pages of the *Journal of the National Cancer Institute*. Written by Calvin Zippin and colleagues at the University of California at San Francisco and the Tumor Registry of the California State Department of Health, the article, entitled "Studies on Heredity and Environment in Cancer of the Nasopharynx," attempted to determine whether predisposition to the disease was a result of heredity, environment, or some combination of the two. The authors first categorized 361 instances of nasopharyngeal cancer from the California Tumor Registry by the patient's race, age, sex, and place of birth. Next, they gathered data on annual rates of cancer incidence among residents of New York State, which they selected because the state Department of Health, unlike its counterpart in California, required physicians to report all cases of the disease.[43] By drawing on census information on the age distributions of U.S.- and foreign-born Chinese males in California, Zippin and his co-authors were able to use incidence rates from New York to extrapolate the number of expected cases of nasopharyngeal cancer for the entire state of California. In other words, if the rate of the disease among fifty- to fifty-five-year old males in New York State was 2.0 cases per 100,000, then the team would use that figure

[43] For some reason, physicians in New York City were not required to report cases of cancer; thus, the data for New York State were exclusive of New York City.

to calculate a raw number of cases that could be expected among American-born Chinese of the same age living in California.[44]

The researchers discovered that the ratios of observed to expected numbers for Caucasians ranged from 0.4 to 0.8, which were within the anticipated range for males both over and under the age of fifty-five. (A ratio of 1.0 would have indicated a perfect correlation between observed and expected numbers.) For Chinese, in contrast, the team found ratios from 3.3 to 5.0 for both American-born Chinese men of all ages and foreign-born Chinese males under fifty-five, meaning that rates of nasopharyngeal cancer among these subsets were higher than what the scientists predicted based on incidence data from native- and foreign-born New Yorkers. For immigrant Chinese men over fifty-five, the ratio was 28.8, a figure which they dubbed "extreme…not only dramatically higher than that for Caucasians, but…also more than 8 times as high as that for native-born Chinese under age 55."[45] In other words, immigrant Chinese had the greatest rates of nasopharyngeal carcinoma and white Americans the lowest, while the rates for Chinese born in the United States fell somewhere in between.

In discussing their results, Zippin and his co-authors gave equal weight to environmental and genetic hypotheses. Although the uneven rates for American-born and immigrant Chinese under fifty-five seemed to support the former, they cautioned that "[t]he sorting of environmental influences is an extremely difficult task." As earlier scientists had discovered, it was a virtual impossibility to first isolate and then assign causality to particular customs and habits thought to increase one's risk for the disease.

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[44] Calvin Zippin, Irene S. Tekawa, Kay U. Bragg, Dorothy A. Watson, and George Linden, "Studies on Heredity and Environment in Cancer of the Nasopharynx," *Journal of the National Cancer Institute* 29, 3 (Sep. 1962), 483-490.

[45] Zippin et al., 488.

At the same time, the team noted that non-immigrant Chinese still had a greater risk for nasopharyngeal cancer than Caucasians born in the U.S. "If it were possible to assume that the environment of native-born Chinese were the same as that of native-born Caucasians," they wrote, "and also that the completeness of reporting were the same, the difference between these two groups would suggest the operation of genetic factors as well." As a way to examine further the effects of environment and genetics, the researchers suggested comparing second-generation Chinese Americans who lived apart from their parents with those who remained in households that retained a strong immigrant influence:

> It would be of interest to follow an American-born Chinese group who may have left their home environment as a result of military service, college studies, or some geographically specialized field of work. A comparison of the frequency of occurrence of cancer of the nasopharynx among such individuals with the rate for others born in this country, but not displaced from their home environment, might provide more refined observations by which to evaluate further the importance of environment in this disease."[46]

Zippin and his colleagues were the first to publish an article in a major journal offering a framework which departed from the previous model of either a genetic or an environmental etiology. Their work was also the earliest large-scale investigation of nasopharyngeal cancer among Chinese in California. It was their decision, however, to include American-born Chinese into their study in a comprehensive manner which underscored the necessity of a new theory which incorporated both an environmental and inherited basis for the disease. If one noticed high rates among Chinese in China, one might suspect an environmental origin. A similar incidence among migrant Chinese who adopted non-Chinese diets and customs could suggest a racial predisposition. But a

[46] Zippin et al., 489-490.

researcher who found among Chinese Americans born in the United States a rate lower than that of their immigrant peers but higher than for American-born Caucasians had to acknowledge the possible roles of both genes and environment. While it might not have been feasible at this stage to isolate the relative importance of each, it certainly was impossible to deny that each probably had an effect on the development of the disease.

Other cancer researchers in the 1960s also began to devote increased attention to the importance of genetics. In "Nasopharynx Cancer in Chinese of California," a 1965 article in the *British Journal of Cancer*, Philip Buell argued that neither genetics nor environment alone could adequately explain differential rates of the disease among native- and foreign-born Chinese. "While the reduced incidence among the U.S. born Chinese would support an environmental hypothesis," he observed, "it does not in itself rule out genetic etiology. This is true not only because…one can postulate genetic-environmental interaction. One can also postulate selection against a genotype as a cause of reduced incidence in the filial generation."[47] Buell hypothesized that susceptibility to nasopharyngeal carcinoma was a trait with a genetic heritage; alleles inherited from one's parents turned the genes for the disease on or off. Alternatively, low rates of the disease among locally born Chinese could be a result of selection through failure to reproduce. Because records showed parental ages that were markedly higher for men than for women, he speculated that postponed fertility, combined with a relatively early expression of the disease in males, might have caused men to die from cancer of the nasopharynx before they had a chance to reproduce.

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[47] P. Buell, "Nasopharynx Cancer in Chinese of California," *British Journal of Cancer* 19, 3 (Sep. 1965), 467.

Buell's article was notable less for what it revealed about current knowledge about genetics than for what it left out. Since the rediscovery of the work of Gregor Mendel at the turn of the twentieth century, scientists had known the basic laws of inheritance. In experiments with pea plants in the 1860s, Mendel demonstrated that the frequency of occurrence of traits in hybridized plants could be predicted with some degree of certainty. If, for instance, two parent organisms each contained elements for a dominant characteristic such as tallness (*A*) and its recessive counterpart, shortness (*a*)—known as a "simple" trait because its expression was determined by a single gene—then approximately one-quarter of the offspring would be homozygous for *A* (that is, they would contain two identical copies of the same allele, or *AA*), another one-quarter for *a* (*aa*), and one-half would be heterozygous, or contain one copy each of *A* and *a* (*Aa*). Since tallness was dominant over shortness, then three-quarters of offspring—those displaying either *AA* or *Aa*—would be tall.[48]

At the end of the nineteenth century and the first few decades of the twentieth, the field of genetics was influenced by eugenists who aimed to improve human populations by encouraging selective breeding of those from desirable "stock" and limiting reproduction, through involuntary sterilizations, if necessary, of those deemed socially unfit. The list of undesirables included, at various times, alcoholics, paupers, criminals, the insane, the feeble-minded, African Americans, Jews, southern and eastern Europeans, and those with chronic diseases such as tuberculosis. Proponents of social reform through eugenics embraced Mendel's work, using his principles to calculate the likelihood that children would inherit objectionable traits of their parents. As scholar

[48] See, for example, Daniel J. Kevles, *In the Name of Eugenics: Genetics and the Uses of Human Heredity* (Cambridge, Mass.: Harvard University Press, 1985, 1995), 41-42, 194.

Daniel J. Kevles has written, "[t]he enthusiasts of eugenics were unquestionably stimulated by the advent of Mendelian genetics in 1900 and its application to human heredity."[49]

The study and practice of genetics before World War II continued to rely on the principles of Mendelian inheritance, as well as on mathematical and statistical methodologies. In its "early years" from 1930 to 1945, as Kevles writes, the field of human genetics was a fringe specialty "populated by a small band of pioneering enthusiasts, entrepreneurs, and evangels….The community was small in size—fewer than two hundred people published any research in the field at all, while fewer than fifty published more than once."[50] In the 1950s, a number of developments attracted new researchers and boosted the profile of the field. First, the establishment of the American Society of Human Genetics and its associated journal, the *American Journal of Human Genetics*, lent credibility to the specialty while at the same time providing scholars with a place to publish their work. Second, concern over the effects of atomic radiation on humans resulted in an influx of federal research money. Finally, two crucial new discoveries raised scientific hopes of determining the processes of hereditability for various human traits as well as diseases such as sickle cell anemia: James D. Watson and Francis Crick's 1953 identification of the double-helix pattern of DNA, and the determination in 1955 by two researchers that normal human cells contained forty-six, not forty-eight chromosomes.[51]

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[49] Kevles, 70.

[50] Kevles, 205.

[51] For a discussion of the growth of the field of human genetics in the 1950s, see Kevles, 223-238. James D. Watson gives a slightly different date, 1956, for the correct determination of the number of human chromosomes. James D. Watson, *DNA: The Secret of Life* (New York: Alfred A. Knopf, 2004), 321.

By the mid-1960s, the field of human genetics had been transformed, and researchers hoped to utilize biochemical methods in their search for information about heredity. But much remained unknown. Work in molecular biology, which focused on the interactions between proteins and nucleic acids, had produced a theory of how DNA regulated protein synthesis, but such progress had come primarily through the study of microorganisms, such as *E. coli*, and not in complex, multicellular life forms.[52] In humans, scientists had managed to map the genetic profiles for only a handful of diseases that were known to be inherited, including Down's Syndrome and some blood disorders, and certainly no one yet understood how the pathways of genes and heredity worked for different types of cancer.[53]

Buell's attempt to formulate a generalized genetic hypothesis for nasopharyngeal carcinoma before he had the ability to identify individual chromosomes or the locations of specific genes revealed an awareness of the significance of genes to disease. Others similarly invoked the field of genetics in theorizing about the etiology of NPC. One colleague, for instance, described the environmental and racial arguments as "not mutually exclusive." "[I]t is reasonable," he continued, "to postulate that some environmental factor or factors may exert a triggering effect in genetically susceptible persons."[54] The willingness of these researchers to incorporate the premises of a new field, no matter how preliminary and incomplete its theories, showed confidence in continued scientific progress and its applications to medicine. Researchers were aware,

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[52] Kevles, 265.

[53] Kevles, 253-254.

[54] K. Shanmugaratnam, "Nasopharyngeal Carcinoma in Asia," in *Racial and Geographical Factors in Tumour Incidence*, A.A. Shivas, ed. (Edinburgh: Edinburgh University Press, 1967), 176.

of course, of the enormous gaps in their knowledge. While they understood, Kevles writes, that "the human genetic code was carried in the coils of DNA compacted in the chromosomes of the cell nucleus, it was unknown where each gene was situated in the tangle, and what function each fulfilled. Human geneticists, and even most molecular biologists, recognized these difficulties, but in the opinion of the latter group especially they were difficulties that molecular genetics would eventually overcome."[55]

Several years after the publication of Buell's article, a number of leading researchers on nasopharyngeal cancer gathered in Singapore for a conference on the disease. Their papers were collected into a volume which subsequently appeared in 1967. Entitled *Cancer of the Nasopharynx*, it summarized a number of trends gaining prominence not only in work on nasopharyngeal carcinoma, but also in the wider fields of medical research and public health. First, contributors to the volume recognized, as Buell and Zippin had, the inadequacy of an etiologic model that failed to incorporate both environmental and genetic influences. Second, researchers began to explore a far greater range of environmental factors than they had previously; articles investigating dental hygiene, alcohol consumption, Chinese tea, and the concentrations of arsenic and nickel in Taiwanese cigarettes filled the pages of the book. Third, it was becoming more difficult to take definitions of race and ethnicity for granted. A number of authors now drew on the fields of sociology and anthropology to offer explanations of these terms.

The attempts of researchers to define race and ethnicity marked an acknowledgement that because these were indistinct concepts, their application within medicine required a clarification of precisely what each one meant. They came, as well,

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[55] Kevles, 266.

within a climate in which scientists were working to dismantle the notions of scientific racism that had persisted for well over a century, reaching a grotesque apogee in the Nazi project during World War II. The 1950 UNESCO statement on race, drafted by an international committee of scholars, aimed to clarify common understandings of race. It referred to race as less biological phenomenon than social myth, signaling the moral pitfalls of continuing to view race as a collection of immutable physical, moral, and intellectual traits, and marking a crucial moment in the separation of biology from culture.

Whether or not the contributors to *Cancer of the Nasopharynx* read the 1950 UNESCO statement, they would certainly have been aware of a political climate which sharply disapproved of the view that the physical and mental characteristics of race were rooted in biology and, therefore, unchangeable. Among the contributors to *Cancer of the Nasopharynx* to foreground their papers with a discussion of terminology was I. Polunin, who wrote that race referred to "a biological group within a species," used "to describe a group of individuals who interbreed and show certain genetically-determined characteristics which distinguish them from other such groups." An ethnic group, in contrast, "could be described as a group of individuals with cultural characteristics in common, and an awareness of a common identity which tends to maintain these characteristics." Polunin provided these descriptions as a way of rebutting the argument that differences in disease profiles among various populations could be attributed to biological disparities between races. "This may not be true for nasopharyngeal cancer," he admitted, "but it does show the importance of careful investigations to determine whether persons in high-risk populations have ways of living significantly different from

those of low-risk populations."[56] John C. Bailar III, a researcher at the National Cancer Institute, gave a similar meaning to race, describing it as "a group of persons each having some or all of certain distinct and characteristic inherited traits. Some of these traits may be related to the incidence of various diseases, including cancer."[57] Another writer, R.J. Walsh, approached race from the perspective of physical anthropology, examining blood groups, plasma protein groups, skin pigmentation, hair texture, and measurements of height and weight for populations with a propensity for cancer of the nasopharynx.

The efforts of Bailar, Walsh, and Polunin to sort out the meaning of race before using it in a conceptual way departed from the approach, common in preceding years, of letting race rest on the reader's understanding of it. Instead, researchers now seemed much more aware not only of the ways in which they used race as a term, but also the extent to which they could—or could not—define it scientifically. The problem with the definitions of race put forth by contributors to *Cancer of the Nasopharynx* was their vagueness. For the most part, authors were content to describe the physical component of race simply as a collection of "genetically-determined characteristics," as Polunin put it, or "certain distinct and characteristic inherited traits," in the words of Bailar. With the exception of R.J. Walsh, however, no one attempted to identify any of these genetic markers. Instead, in discussing their Chinese subjects, scientists fell back on ascribing to race a series of behaviors that could readily be identified and classified. In essence, they reduced race to ethnicity.

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[56] I. Polunin, "The Ways of Life of Peoples With High Rates of Nasopharyngeal Carcinoma," in *Cancer of the Nasopharynx*, C.S. Muir and K. Shanmugaratnam, eds. (Copenhagen, Denmark and Flushing, NY: Munksgaard / Medical Examination Publishing Company, Inc., 1967), 106-107. I am assuming, perhaps incorrectly, that the author was male.

[57] John C. Bailar III, "Race, Environment and Family in the Epidemiology of Cancer of the Nasopharynx," in Muir and Shanmugaratnam, eds., *Cancer of the Nasopharynx*, 101.

Among epidemiologists, of course, it was a common practice to approach disease etiology by first identifying a group of people with the ailment and then drawing generalizations about them. If the causation of a disease was not well understood, then epidemiologic methodology could allow investigators to pinpoint characteristics common to the afflicted. This style of research was not new in the 1960s, and it did not end with the decade. But what was notable about nasopharyngeal carcinoma was the way in which researchers, because of the disease's unique patterns of incidence, targeted their work by pinpointing behaviors common to Chinese. In doing so, they not only helped to define what was Chinese by isolating a series of ethnic practices, particularly those common to the Cantonese populations under study, but also inferred processes of cultural reproduction by noting particular behaviors that they believed identified an individual or a family as Chinese, regardless of place of domicile or immigrant status.

The behaviors identified by researchers as characteristically "Chinese" generally pertained to diet and living conditions. In an effort to explain "the Chinese way of life," I. Polunin reported that Chinese tended to take meals which included "rice, small quantities of meat, fish and other sea-foods [*sic*], green leafy vegetables, and soya bean products." They drank soups and teas at very hot temperatures, which, he noted, "might cause mild inflammation of the pharynx." Polunin examined typical houses and systems of ventilation, as well as the kinds of smoke that could be produced, the most notable of which were from incense burned for religious purposes and from opium.[58] Bailar, the National Cancer Institute scientist, suggesting studying migrant Chinese populations to test theories of the disease's genetic versus environmental origins. "It might be worth

[58] Polunin, 106-109.

studying such persons," he wrote in reference to the first generation of offspring from one Chinese and one non-Chinese parent, "to determine the level of risk for nasopharyngeal cancer. Specifically," he asked, "would it be possible to compare the frequency in persons of mixed ancestry living in a Chinese environment with the frequency in persons of similar ancestry living in a non-Chinese environment?"[59] Bailar, however, did not specify precisely what made an environment Chinese. Was it the food consumed, the style of dwelling, or the type of fuel used for cooking? How much of a factor was location?

An article by John Higginson and K. Shanmugaratnam offered perhaps the clearest illustration of the range of behaviors that researchers considered notable in the search for the causes of nasopharyngeal carcinoma. The authors first reviewed the etiologic factors which had previously been discussed and dismissed in the medical literature: internal pollution, as from smoke from "burning grass, poor quality wood, tobacco, candles, incense, kerosene lamps and lamps burning peanut oil in the poorly-ventilated houses in China," infections, excessive hawking of phlegm, the taking of hot food and drink, alcohol, tobacco smoking, opium smoking, and viruses.[60] The authors went on to present the results of their own survey of nasopharyngeal cancer patients and a control group in Singapore. They found that "[m]arital status, religion, and bowel habits were essentially similar in both groups." Although those with cancer had slightly lower levels of education, the groups were similar in nearly every other respect. Both ate "rice, curries, fried food, noodles, and a variety of other foodstuffs, viz. vegetables, eggs, pork,

[59] Bailar, 102.

[60] K. Shanmugaratnam and J. Higginson, "Aetiology of Nasopharyngeal Carcinoma: Report on a Retrospective Survey in Singapore," in Muir and Shanmugaratnam, eds., *Cancer of the Nasopharynx*, 130-134.

meat, sea-foods, salted and dried foods, preserved meat, etc." at approximately the same rates, and used the same quantity and quality of fats in cooking. They showed similar ingestion of milk, "'cooling drinks,'" tea, other non-alcoholic beverages, spices, and soy sauce. The groups used Chinese medicines similarly, including digestive powders, "tonic wines, strengthening foods, strengthening pills and tablets, sore throat cures, cough drops and lozenges, [and] herb drinks." Finally, both the cancer cases and the control group lacked "a positive relationship with the state of dental health," as many patients were missing teeth or reliant upon dentures.[61]

The lists of customs and habits that researchers investigated reinforced ideas about what it meant to be Chinese. On the one hand, they took as a starting point behaviors which had already been identified as stereotypically Chinese. Thus, they looked at opium smoking, even though Polunin, among others, recognized that the practice "is nowadays only common among oldfashioned [*sic*] Chinese men of the labouring classes and the opium-growing tribes in South-West China and bordering territories."[62] Similarly, they focused attention on the tendency of Chinese to ingest hot foods and beverages for links to cancer of the nasopharynx, as it had been reported decades earlier as a possible risk factor for stomach cancer. In the 1960s, as in the 1930s, however, researchers could not establish any relationship among temperature, eating habits, and disease. On the other hand, these scientists played a role in the continual defining and redefining of what was Chinese. Chinese people had bad teeth, ate rice, took Chinese medicines, and lived in poorly ventilated houses in which they burned

[61] Ibid., 132-133.

[62] Polunin, 109.

wood, tobacco, and incense. They lived, as Bailar noted, in "Chinese environments," except when they lived in "non-Chinese environments." But researchers also normalized as Chinese the way of life of the Cantonese population, as they not only had higher rates of nasopharyngeal cancer, but because southern Chinese—and, in particular, those from a small corner of Guangdong province—represented the greatest percentage of Chinese immigrants in the United States. The customs and living conditions reported as Chinese in studies of the disease were not standard throughout China, a vast country populated by a number of ethnic subgroups, each of which had its own particular ways of life.

The papers in *Cancer of the Nasopharynx* exposed how much about the disease remained unknown, even at the end of the 1960s. Despite advances in genetics and molecular biology, the basic etiology of nasopharyngeal carcinoma continued to baffle researchers. As John Higginson wrote in the introduction to the book, "[a]lthough there was initial wide acceptance of the view that environmental carcinogens inhaled through the nose were probably the aetiological factors in this cancer, it soon became clear that there was little supporting evidence, and the view that genetic susceptibility might be of primary significance definitely merits more consideration."[63] The evidence, however, did not clearly implicate one or the other, or even give many clues as to the relative significance of each. In response, medical researchers often cast a wide net, exploring variables that seemed irrelevant to the etiology of a disease that affected one's nasal passages. It could appear at times as though they were playing a game of roulette, spinning a wheel of local and behavioral factors and hoping to get lucky by chancing upon the correct one. Meanwhile, physicians were at a loss when it came to dispensing

[63] C.S. Muir and K. Shanmugaratnam, eds., *Cancer of the Nasopharynx* (Copenhagen, Denmark and Flushing, NY: Munksgaard / Medical Examination Publishing Company, Inc., 1967), 11.

advice to their patients on how to reduce their risk of nasopharyngeal cancer. A 1968 article by E.V. Cowdry put the matter succinctly: "Chinese, particularly, should exercise special precautions, but it is not known what these should be, except to avoid dietary deficiencies and prolonged irradiation."[64]

Epilogue: Cultural Carcinogens

In the following years, scientists would identify a number of agents that appeared to help explain the etiology of nasopharyngeal carcinoma. First, they noticed that patients with the disease had an unusual frequency of antibody titers to the Epstein-Barr virus, although they were unsure if the virus preceded the development of cancer or resulted from it. Second, a scientist named J.H.C. Ho, who worked at the Institute of Radiology at Queen Elizabeth Hospital in Kowloon, Hong Kong, observed that boat-dwelling Tanka people, a subgroup of the Cantonese, had higher rates of NPC than Cantonese who lived on land. On their boats, the Tanka cooked food in the open air, casting further doubt upon Dobson's inhalant theory. Furthermore, Ho ascertained that southern Chinese, and especially fishermen who spent long periods of time at sea, ate a diet which included a large quantity of salted fish. Certain Chinese populations salted the fish, using substances containing nitrates, as a method of preservation, and it was "for many years a common and favorite item of food among most Chinese, rich or poor, in or outside China in Southeast Asia or the United States."[65] Ho made two additional observations that later researchers deemed "crucial" to his salted fish theory. Since the

[64] E.V. Cowdry and Warren H. Cole, *Etiology and Prevention of Cancer in Man* (New York: Appleton-Century-Crofts, 1968), 34.

[65] J.H.C. Ho, "Nasopharyngeal Carcinoma (NPC)," in *Advances in Cancer Research* (New York: Academic Press, 1972), 81.

process of salting the fish was incomplete, "the product, aged several days to weeks, became partially putrefied, liberating a pungent odor offensive to those who were not raised in the southern Chinese culture, including most Chinese from the northern provinces." He realized, too, that parents in southern China often fed their babies salted fish mixed with soft rice during the process of weaning.[66]

Subsequent studies confirmed Ho's suggestion of a link between Chinese salted fish and nasopharyngeal carcinoma: from a young age, Chinese were exposed to cancer-causing chemicals that resulted from the incomplete salting and drying of the fish. It explained both patterns of early expression of the disease among Chinese and why immigrants to the West continued to show anomalously high rates even after they transitioned to a higher standard of living. Ho's theory, one should note, was made possible not by advancements in the fields of biochemistry or genetics, but by the same methodology which had earlier identified other characteristics of a Chinese way of life. It was, finally, the principles of epidemiology, not anthropological definitions of ethnicity or race or the pathways of genetic inheritance, that enabled the identification of a major factor in the etiology of nasopharyngeal carcinoma. By paying careful attention to differences between southern Chinese and other populations, including northerners and Southeast Asians, some of whom also had an increased risk for the disease, Ho was able to identify patterns that others had missed. Like previous work, his theory depended on the ways in which he classified Chinese. Unlike his colleagues, however, he realized that the categories of "Chinese" and even "Cantonese" were too imprecise to take into

[66] Mimi C. Yu, Brian E. Henderson, "Nasopharyngeal Cancer," in David Schottenfeld and Joseph F. Fraumeni, Jr., eds., *Cancer Epidemiology and Prevention* (New York: Oxford University Press, 1996), 607.

account important regional distinctions that were, ultimately, an essential component in carcinogenesis among Chinese and Chinese Americans.

Conclusion

The search for the etiology of nasopharyngeal carcinoma reveals the extent to which the processes of scientific inquiry are interlaid within a web of social and cultural meaning. The association between the disease and Chinese and other "Oriental" groups not only determined which populations were considered susceptible to it, but guided scientists in their quest for answers, as well. Researchers employed various combinations of behavioral, cultural, genetic, and environmental definitions of what it meant to be Chinese, utilizing and discarding each one as knowledge about disease etiology advanced. Initially, drawing the outlines of a Chinese population was as simple as identifying those who traced their origin to China. Emigration and resettlement would later complicate the task, prompting doctors to define "Chinese" by pinpointing a series of behaviors and customs which marked adherents as Chinese. Advancements in genetics and molecular biology introduced new ways of classifying human populations in the 1950s and 1960s, even as disease pathways remained unknown.

For all the efforts of researchers in the 1960s to clarify their uses of race and ethnicity with anthropological and sociological descriptions, the categories mattered only in the extent to which scientists questioned their meanings. Ultimately, the correlation between early, repeated consumption of Chinese salted fish and the later development of nasopharyngeal cancer made apparent the irrelevance of standardized definitions of ethnicity and race to the search for etiological agents. But the descriptions scientists

utilized over the course of the twentieth century alternately illuminated and obscured what it meant to be Chinese, as each successive characterization proved insufficient for explaining nasopharyngeal cancer's unique epidemiology. It is perhaps too simple to state that "Chinese" meant whatever scientists said it did. But whether the category bore the boundaries of genetics, ethnic practices, or both, in the search for the causation of nasopharyngeal carcinoma medical researchers were the ones who continued to define, redefine, and normalize the meanings of "Chinese."

Chapter 5

Race and Prostate Cancer: The Making of a "Black" Disease

Prostate cancer ranks as the most commonly diagnosed non-skin malignancy in American men today and is the second leading cause of cancer deaths among them. For those who are born in and live their entire lives within the United States, the risk of developing the disease is between 9 and 11%. It will represent the cause of death for between one-quarter and one-third of those diagnosed with it.[1] By age eighty, up to 80% of men may exhibit evidence of a latent form of the disease which is detectable at autopsy but never advances to the clinical stage. The American Cancer Society estimated that in 2007 approximately 218,890 men in the U.S. would receive a diagnosis of the disease, and some 27,000 would die from it.[2]

The etiology of prostate cancer remains largely unknown. Scientists have linked it with a high dietary intake of saturated fat and for some time have suspected that sex hormones, such as testosterone, play a role in its development. Measures that may reduce one's risk include consumption of selenium and vitamin E, while smoking, height, weight, body size, lycopene intake, and cardiorespiratory fitness appear to have no effect on carcinogenesis. The latest research suggests the possible importance of vitamin D: the lower one's blood levels, the greater one's risk.

Worldwide, the highest rates occur in North America, Europe, and Australia, and the lowest in East and South Asia. Within the United States, the disease shows distinct

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[1] Gary D. Grossfeld and Peter R. Carroll, "Natural History," in Peter R. Carroll and Gary D. Grossfeld, eds., *Prostate Cancer* (Hamilton, Ontario: BC Decker, Inc., 2002), 149.

[2] http://www.cancer.org/docroot/CRI/content/CRI_2_2_1X_How_many_men_get_prostate_cancer_36.asp?sitearea= (accessed July 11, 2007).

racial patterns, preferring white men to Asian Americans, whose rates of incidence are 38% lower than among Caucasians.[3] But it is the relationship between prostate cancer and African Americans which has provoked the most comment. Black men in the U.S. have the highest rates of the disease in the world, nearly 63% higher than for white American males.[4] Not only do they suffer greater mortality at all stages of diagnosis, but their death rate from the disease has been increasing more rapidly, as well. One study found that prostate cancer mortality among black men in the U.S. increased by 136% between 1950 and 1992, while the rate for whites rose only 21%.[5] Other data indicate that African Americans between the ages of forty and seventy are two to three times more likely to die from the disease as whites.[6]

This was not always so. Until around 1950, prostate cancer in the United States disproportionately affected Caucasians. Studies categorizing clinical cases or patterns of incidence by race sought not to explain elevated rates among blacks, but rather to demonstrate that African Americans suffered from the illness, too. Over the second half of the twentieth century, incidence among African Americans began to surpass that for whites, and prostate cancer gradually became a "black" disease. The postwar search to find an explanation for prostate cancer's racial configurations led to sharp debates over

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[3] Ahmedin Jemal, Taylor Murray, Alicia Samuels, Asma Ghafoor, Elizabeth Ward, and Michael J. Thun, "Cancer Statistics, 2003," *CA: A Cancer Journal for Clinicians* 53, 1 (Jan./Feb. 2003), 22.

[4] Jemal et al., 22.

[5] T.A. Pifath, M.K. Whitman, J.A. Flaws, A.D. Fix, and T.L Bush, "Ethnic Differences in Cancer Mortality Trends in the U.S., 1950-1992," *Ethnicity and Health* 6, 2 (2001), 105, in Keren Patricia Dimah and Agber Dimah, "Prostate Cancer Among African American Men: A Review of Empirical Literature," *Journal of African American Studies* 7, 1 (Summer 2003), 27.

[6] L.A.G. Ries, B.F. Hankey, and B.K. Edwards, *Cancer Statistics Review 1973-1987* (Bethesda, MD: US Public Health Service, 1990; NIH Publication 90-2799), in Isaac Powell, "Keynote Address: Prostate Cancer Among African Americans Men—From the Bench to the Community," *Journal of the National Medical Association* 90, 11 (Suppl., Nov. 1998), S705.

the very nature of race, as well as its relationships to class, ethnicity, genetics, behavior, and the environment. The social and economic conditions of African Americans in the U.S. were central to the discussion, as some researchers struggled to disaggregate race from class, while others argued for the inseparability of the two. Advances in epidemiologic methodology, biomedical technology, and diagnosis changed the ways in which scientists discovered and treated the disease, with results that both underscored and challenged prevailing frameworks of race and disease.

At the twilight of the twentieth century, prostate cancer's racial associations were not only well known, but had played a major role in reintroducing a bodily underpinning to the notion of race. For the previous twenty years or so, the idea of race as a social construction had been gaining sway, particularly within academic circles, its significance rooted in culture and no more, and no less, than what we assigned to it. While the new, bodily conception of race echoed the nineteenth and early twentieth century's focus on physicality, it differed in its abandonment of the view that members of various races displayed biological characteristics which remained unchanged from one generation to the next; that immutable physical differences in skin color, hair texture, and the shape of one's facial features could serve as racial markers. Instead, it rested on the principles of genetics. Genes, not biology, determined phenotypic distinctions between races, a consequence of a group's shared ancestry. Native Americans, or Asians, or African Americans displayed similar physiological characteristics as a result of reproductive choices that concentrated specific traits within a population. The new conception of race, likes its historical predecessor, retained a grounding within the body, a scheme which contemporary cancer research only strengthened. If medical technologies, including a

recently developed diagnostic test that measured levels of a protein called prostate-specific antigen, appeared to confirm biochemical differences between groups, then perhaps race was more than a social construction. Twentieth-century medical science had apparently returned us to a notion of race that, far from disregarding the body, instead remained embedded within it.

1930s and 1940s: The Disease that Wasn't

Compared with cancers of the skin, cervix, and nasopharynx, prostatic carcinoma generated little interest in the 1930s and 1940s. The male prostate, a walnut-sized gland found at the base of the penis and in front of the rectum, surrounds the top part of the urethra, the tube that drains urine from the bladder. It consists of glandular and fibrous tissue surrounded by a capsule of connective tissue, and its main role within the body, as we know now, is to produce seminal fluids.[7] But for doctors at the time, the organ's inaccessibility and the paucity of medical knowledge regarding its function meant that a list of the illness's symptoms would be unavoidably imprecise. At least skin cancer, nasopharyngeal carcinoma, and cervical cancer produced somatic markers that were recognizable as signs of disease; prostate cancer, in contrast, generated no unique indications of its own. The few symptoms which doctors warned patients to be aware of, including frequent or difficult urination, could well be the result of a non-cancerous ailment or even a psychological issue.

Because physicians lacked a reliable screening method for detecting the disease, most cases were found at autopsy, which may have aided statistical record-keeping but

[7] National Cancer Institute, *Cancer of the Prostate: Research Report* (NCI / NIH Publication No. 91-528, 1990), 1-2.

was of no consequence to the men who would eventually die from the illness. Digital rectal examination represented the only non-invasive diagnostic weapon in the medical arsenal, and the inconsistent results it yielded made it of questionable value. Worse yet, since researchers knew virtually nothing about the disease's etiology and development, they could not construct a cancer prevention message. Chronic irritation, which was being explored as a possible cause of other types of cancer, seemed irrelevant to carcinogenesis in the inaccessible prostate. Options for treatment were limited, as well. Surgeons could perform a simple prostatectomy, in which they removed diseased tissue from the gland, or a radical one, which excised the entire prostate as well as some of the surrounding tissue. Or physicians might treat the cancer with radium, using fine-gauge needles to implant seeds of radioactive material into the prostate and nearby vesicles.[8]

Just a handful of articles on prostate cancer from the 1930s and 1940s mentioned race, indicating that the disease was visible at the time almost exclusively in Caucasians. Non-white groups generally become notable within medical research only when scientists discover an epidemiologic peculiarity, such as extremely high or low rates of a disease within a particular population. Many hoped, for instance, that an examination of the rare cases of skin cancer among blacks, another preoccupation of cancer researchers at the time, would point them toward the disease's origins. But unlike with skin cancer, doctors who were concerned with race and prostatic carcinoma aimed to document not the immunity of African Americans, but their vulnerability. One study that investigated data from white and black men admitted to the Charity Hospital in New Orleans with symptoms of prostate cancer found rates to be slightly higher among the latter group.

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[8] John R. Caulk and S.B. Boon-Itt, "Carcinoma of the Prostate," *American Journal of Cancer* 16, 5 (Sep. 1932), 1038-1039.

"The values," the authors wrote, "indicate that the negro is undoubtedly as susceptible to this condition as the white man."[9]

Much of the literature on race and prostatic disease made note of the correlation between rates of benign prostatic hypertrophy or hyperplasia (BPH) and prostate cancer. A common disorder, BPH could cause an enlarged prostate gland to block the urethra and lead to urinary problems, symptoms which mimicked those of prostate cancer. Typically, the data on racial incidence of diseases of the prostate were inconsistent, with a number of studies reporting that both BPH and prostate cancer were rare in "Mongolians" and "Negroes," while others found the exact opposite—that African Americans were somewhat more likely than whites to suffer from prostatic enlargement.[10] BPH was among the non-cancerous conditions which affected the proper functioning of the prostate and compounded the difficulties of obtaining a definitive diagnosis of cancer. At issue for scientists was whether prostatic enlargement could lead to prostate cancer, a question which would not be resolved until much later in the century.

That American scientists concerned with prostate cancer before World War II did not focus on race was a consequence of both the disease's patterns of incidence and the state of medical knowledge at the time. Caucasians appeared to suffer the highest rates; therefore, they were the group which garnered the most attention. The dearth of research on race was less a willful neglect of the issue than a reflection of a more general lack of interest in the disease as a whole. Physicians knew little about prostate cancer, and had no ready way of learning more. As life expectancy expanded, prostate cancer was

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[9] Vincent J. Derbes and Stella M. Leche, "The Occurrence of Carcinoma of the Prostate in Whites and Negroes," *Urologic and Cutaneous Review* 41 (Oct. 1937), 701-702.

[10] Edgar Burns, "Prostatic Obstruction in Negroes," *Journal of Urology* 44, 2 (Aug. 1940), 177-182.

becoming an increasingly important cause of cancer deaths in men. Without a reliable diagnostic test, however, medicine had little to offer. As one doctor wrote in 1948, "[t]o date our answer has been primarily one of palliation."[11]

The Increasing Visibility of Race in the 1950s and 1960s

By the mid-1950s, researchers no longer had to struggle to include African Americans in discussions of prostate cancer. Instead, they were beginning to notice that members of the racial group presented more cases of the disease than their numbers indicated they should. The prostate was becoming a high-risk site for cancer in African Americans, much as the nasopharynx had proven to be in Asians and Asian Americans. As one physician wrote in 1956 in his state medical journal, clinicians doing cancer exams had to be sure to examine the prostates of their black patients, just as they should inspect the skin of head, face, and neck for their white patients, the "sites of predilection in the two races."[12] That same year, a set of researchers from the National Cancer Institute stated that the incidence of prostate and penile cancer was "definitely higher for Negroes."[13] As statistics now indicated, rates of incidence among African Americans were on the rise, whether due to greater diagnosis or increased life expectancy, as prostate cancer, more than any other form of the dread disease, was an illness of old age. Researchers, however, were reluctant to venture theories that might explain prostate

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[11] Joseph Memmelaar, "Carcinoma of the Prostate Today," *Journal of the Maine Medical Association* 39, 12 (Dec. 1948), 361.

[12] Alvin S. Crawford, "Clinically Important Facts About Cancer Morbidity," *Journal of the Indiana State Medical Association* 49, 4 (Apr. 1956), 388.

[13] John R. Heller, Sidney J. Cutler, and William M. Haenszel, "Some Observations on the Epidemiology of Cancer in the United States," *Journal of the American Medical Association* 159, 17 (Dec. 24, 1955), 1629.

cancer's racial differential; one of the few groups to do so were the NCI scientists, who implicated entirely controllable factors, including "sex habits and sex hygiene, the prevalence of venreal [*sic*] disease…and medical care."[14] That they focused on environmental reasons probably had as much to do with their methodological biases as epidemiologists as with the continued lack of progress in uncovering the histopathology of the disease.

The NCI investigators' emphasis on behavior and environment over other factors foreshadowed a trend within prostate cancer research that would continue into the 1990s. The 1953 discovery by Francis Crick and James Watson of the double-helix structure of DNA heralded a new era of molecular biology, and scientists in many areas of cancer studies, such as nasopharyngeal carcinoma, had begun to investigate genetic links between race and disease. Prostate cancer researchers, however, continued to focus on epidemiology, addressing their questions toward external causes rather than the cellular foundations of the disorder. This produced a twofold effect. First, it suggested that cancer could be prevented if one adopted the proper behavioral and lifestyle modifications. Some combination of diet, religion, marital status, and access to health care might stave off the disease; the problem was that no one knew precisely which arrangements would work. Second, the emphasis on external variables kept race within the realm of the social, rather than the genetic or biological. If racial differentials could be explained by social factors, then it would lend credence to the view that the physical markers which distinguished members of one racial group from another were no more than phenotypic window dressing to essentially identical internal mechanisms.

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[14] Ibid., 1631.

There were signs, as well, that the concept of race as a convergence of environmental and hereditary factors was gaining broader acceptance. Paul E. Steiner, the University of Chicago pathologist whose 1954 monograph, *Cancer: Race and Geography*, addressed a number of sites of cancer in blacks, Mexicans, Asian Americans, and whites, attributed racial differences in cancer incidence to "environmental or hereditary factors." The category of hereditary differences could be further broken down into environmental causes, which came about due to shared customs or the influence of particular elements on those living in the same geographical area, or "genetical" sources, such as the predisposition in some families toward certain types of retinoblastoma or neurofibromatosis.[15]

Although Steiner argued against the existence of genetically "pure" races, he persisted in dividing his subjects into "Caucasoids," "Mongoloids," and "Negroids," the three "major ethnic subdivisions of mankind."[16] He saw race as a way to signify distinctions in culture, skin color, and geography that affected disease rates by variably involving diverse environmental and "genetical" variables. Steiner's interest was less in charting frequency rates for different forms of cancer than in using his data to illuminate the causes of disease. "[B]y far the most important reason for interest in the ethnic and geographical aspects of cancer is their revelation of clues to etiology," he wrote.[17] If each form of cancer could be found all over the world, then its causes must be "ubiquitous." Documented racial and geographical variations in cancer incidence,

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[15] Paul E. Steiner, "Etiological Factors in Cancer Revealed by Ethnic and Geographical Studies," *Proceedings of the Institute of Medicine of Chicago* 20, 1 (Jan. 15, 1954), 7-8.

[16] Ibid., 10.

[17] Steiner, "Etiological Factors," 6.

however, offered an opportunity to sort out the relative importance of "extrinsic" and "intrinsic" factors.[18]

Steiner failed to find a higher rate of prostate cancer among "Negroids" in Los Angeles, but did determine that African Americans with cancer were more likely to have prostatic carcinoma than members of other racial groups. In other words, black and Caucasian males had similar chances of developing prostate cancer, but African Americans who were already afflicted with some kind of malignancy were more likely to have prostate cancer than any other form of the disease.[19] Lower rates in Mexican and Japanese immigrants were inconclusive based on small sample sizes and the need for comparative data from the subjects' countries of origin. "Apart from frequency," he concluded, "no differences associated with race have been recorded in cancer of the prostate."[20] His results disagreed with the those of other researchers, who were increasingly discovering evidence of differential racial incidence. At a loss to explain his contradictory findings, Steiner refused to implicate either an environmental or a genetic etiology, instead urging his colleagues to initiate studies comparing migrants to those who remained in their home countries, work which "might lead to dissociation of these factors and contribute to the solution of this important problem."[21]

By 1965, prostate cancer had become the most common form of the dread disease in American men over the age of fifty, with an estimated 33,000 new cases in the United

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[18] Ibid., 6.

[19] Paul E. Steiner, *Cancer: Race and Geography* (Baltimore: Williams & Wilkins Co., 1954), Chapter 10, 144-154.

[20] Ibid., 151.

[21] Ibid., 154.

States that year. Death rates were on the rise, from 14.5 per 100,000 in 1958-1959 to 15.7 for whites and 19.4 for nonwhites in 1961. Approximately 14,000 men died from the disease in the U.S. in 1962, making it the leading cause of mortality for men over seventy-five.[22] Prostate cancer's growing incidence resulted both from enhancements in life expectancy, as the illness was very much one of old age, and from wider access to improved medical care, which increased the likelihood that a patient would see a practitioner who had some familiarity with the disease. At the same time, however, researchers warned of the potential for inaccuracies in statistics on the morbidity and mortality of prostate cancer, as the organ's inaccessibility made an infallible diagnosis difficult, if not impossible, in a majority of cases. Still, scientists appeared to be making progress in puzzling out the disease's etiology, now theorizing that it might be caused by age-related hormonal imbalances, and possibly an excess of androgen.[23]

While some researchers were focused on the laboratory-based investigations that they hoped would unlock the mysteries of the common disease, epidemiologists turned their attention to more sophisticated studies that took into account a wider range of variables than before. This was probably a function, in part, of a more comprehensive record-keeping by the U.S. Census Bureau and other agencies, which enabled comparisons across a broader assortment of categories. It also likely resulted from the ongoing uncertainty surrounding the disease's origins. Scientists were moving ahead in their understanding of etiology, but a complete biomedical command of prostate cancer did not seem imminent. Consequently, as with early work on skin and cervical cancers,

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[22] Harry Grabstald, "The incidence, clinical and pathological classification of cancer of the prostate," *CA: A Cancer Journal for Clinicians* 15, 1 (Jan.-Feb. 1965), 31.

[23] Clyde E. Blackard, "Carcinoma of the Prostate: A Review," *The Journal—Lancet* 87, 12 (Dec. 1967), 477.

researchers examined an extensive network of factors in an attempt to isolate the ones that might relate to carcinogenesis.

Although doctors knew that nonwhites, and especially African Americans, were more likely to both develop and die from prostate cancer—one study found rates that were 44% and 22% higher, respectively, for black men—race was just one of a number of variables on which epidemiologists focused.[24] It diminished in importance next to age, which was the primary predictor of incidence of prostatic carcinoma; rare in those under forty, disease rates increased sharply after the age of sixty. Geographic area mattered, as well. Age-adjusted rates ranked highest in the Northeast and on the West Coast, and lowest in the South; and were slightly higher in urban than in rural areas. Researchers found a lower frequency among foreign-born over American-born whites, and immigrants tended to show higher rates than those in their country of origin. Family history played a role, as close relatives of men who had died of prostate cancer were more likely to develop the disease.[25]

Given the prostate's function within the male reproductive system, a number of studies considered the impact of marital status. "There is little need," wrote one team of epidemiologists, "to rationalize the desirability of studying the relationship of marital status to cancer of the male genital organs such as the prostate."[26] Men who were married, widowed, or divorced (the "ever marrieds") were more likely to develop cancer

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[24] Harold F. Dorn and Sidney J. Cutler, *Morbidity From Cancer In the United States* (Public Health Monographs No. 56, 1959).

[25] For a summary of the epidemiological literature on prostate cancer to 1963, see Haitung King, Earl Diamond, and A.M. Lilienfeld, "Some Epidemiological Aspects of Cancer of the Prostate," *Journal of Chronic Diseases* 16, 2 (Feb. 1963) 117-153.

[26] King et al., 137.

of the male genital organs than lifelong bachelors, and married men had higher rates of mortality than their single counterparts. Marital status served as a way to identify a subject's sexual habits and behaviors, much as cervical cancer researchers considered the category a proxy for regularity of intercourse and frequency of pregnancy and childbirth, both of which were thought to affect the conditions for carcinogenesis. In the midst of the uncertainty surrounding the disease, doctors speculated further that cultural variations in sexual conduct could partially explain racial differentials. Walter B. Quisenberry, for instance, who wrote extensively on cancer among ethnic groups in Hawaii, hypothesized that a greater emphasis on intercourse, rather than on foreplay, among Japanese might be a contributing factor to low rates of prostatic carcinoma within that population.[27]

While it was the conjectural similarities between the etiologies of prostatic and cervical carcinomas which initially led students of the former to examine male sexual practices, the income distribution of each disease forced a divergence in the direction of scientific research. The epidemiology of cervical cancer, an illness that disproportionately affected low-income women, induced a twofold moralistic rejoinder in the 1960s. On the one hand were those who blamed cancer sufferers for making poor choices, such as aggravating cervical inflammation by giving birth multiple times, the theoretical precursor to cancer. On the other hand, investigators implicated the culture of poor people for promoting sexual promiscuity and its risky, cancer-causing behaviors. Prostate cancer, in contrast, seemed to strike higher-income groups, such as managers

[27] W.B. Quisenberry, "Sociocultural Factors in Cancer in Hawaii," *Annals of the New York Academy of Science* 84, 17 (Dec. 1960), 795-806; in King et al., 149.

and pharmacists, as often as it affected farm laborers and dockhands.[28] That work on the disease escaped the moralism of the literature on cervical cancer was most likely a direct result of these class-based patterns.

Such discussions on the relationship between income and behavior underscored the changing discourse of class within biomedicine and marked the beginning of the use of the term "socioeconomic status." Researchers had previously examined income and social class, often together, but socioeconomic status combined the two into a single category of analysis, the boundaries of which were rarely defined. Later, socioeconomic status, or SES, would become a shorthand way to identify nonwhites, an implicit acknowledgment of the correlation between race and income in the U.S. But for now, occupation stood as the measure of socioeconomic status without bearing a direct association with race. Consequently, race became a factor unto itself within prostate cancer epidemiology in the 1960s, equivalent to age or nativity or religion. At the time, it was not seen as an indicator of economic and social circumstances, or of environmental conditions which could themselves affect carcinogenesis. Scientists recognized the importance of genes and their potential to revolutionize medicine, but knew little about how they worked. Race, then, represented just one of a number of epidemiological variables, and it stood primarily for phenotype.

[28] While most studies reported roughly equivalent rates of morbidity and mortality across income group, some reported higher incidence among wealthier men. See King et al., 141-146 for a summary of the literature.

The Social Variables of Race from 1970 to the Mid-1980s

After its introduction in the 1960s, socioeconomic status as an epidemiologic category of analysis escalated in usage throughout the next decade and a half. As before, writers rarely attempted to define the criteria by which they assigned subjects to a particular socioeconomic group; they generally used occupation as a proxy for SES, while a handful chose to measure educational level. For the most part, they compared socioeconomic status among white ethnicities, and tended to find that it had little effect on mortality rates, despite the presumption that lower-income men would have reduced access to quality medical care, thus delaying diagnosis and complicating treatment.

In 1978, Virginia L. Ernster and a group of colleagues from the University of California at Berkeley reported the results of a study which they described as the first contemporary attempt to explain differences in incidence and mortality rates along axes of both race and socioeconomic status. They suspected the influence of social factors in the rapidly rising levels of disease among African Americans, arguing that "the sizeable black excess in prostatic cancer rates has appeared only within the last several decades, too short a period for genetic mechanisms to be entirely responsible and a time during which the social experience of blacks has altered considerably."[29] The study differed from recent work in its limitation to whites and blacks—excluding Asians, Latinos, and Native Americans—and in its incorporation of recent data showing dramatically higher rates among nonwhites. In the San Francisco-Oakland metropolitan area, for instance, during the four-year period from 1969 to 1973 the annual age-adjusted incidence of

[29] V.L. Ernster, S. Selvin, S.T. Sacks, D.F. Austin, S.M. Brown, and W. Winkelstein, Jr., "Prostatic Cancer: Mortality and Incidence Rates by Race and Social Class," *American Journal of Epidemiology* 107, 4 (Apr. 1978), 312.

prostate cancer in blacks was 89.9 per 100,000, nearly 60% higher than the white rate of 51.6, and over four times higher than the figures of 20.5 and 20.7 for Chinese and Japanese residents.[30] Between 1947 and 1969, the incidence rate for whites rose 22.8%, but for blacks the increase was 55%.[31]

Ernster and her co-authors determined that socioeconomic status had no effect on either mortality from or incidence of prostate cancer among whites and blacks in Alameda County. African Americans showed higher rates across every socioeconomic level. The research team reported that excess risk for blacks diminished somewhat with rising age; once subjects reached the age of eighty, morbidity and death rates leveled out. Racial differences were most striking in the younger age categories, with young black men over four times as likely as whites to die from the disease.[32]

For the most part, other researchers similarly failed to establish an indisputable link between socioeconomic status and incidence of prostate cancer. One article reported "no clear relationship" between the two, while another found "no consistent correlation."[33] As with any type of medical research on a complex disease, however, unambiguous proof was nearly impossible to establish, and contradictory findings soon surfaced. In two studies written in the first half of the 1980s, an investigator named Hari H. Dayal, from the Fox Chase Cancer Center in Philadelphia, examined the links among

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[30] John E. Dunn, Jr., "Cancer Epidemiology in Populations of the United States—With Emphasis on Hawaii and California—and Japan," *Cancer Research* 35, 11 (Nov. 1975), 3241.

[31] Hari H. Dayal and C. Chiu, "Factors Associated with Racial Differences in Survival for Prostatic Carcinoma," *Journal of Chronic Diseases* 35, 7 (1982), 553.

[32] See Ernster et al., 314-318.

[33] Willis L. Owen, "Cancer of the Prostate: A Literature Review," *Journal of Chronic Diseases* 29, 2 (Feb. 1976), 95; Ernest L. Wynder, Kiyohiko Mabuchi, and Willet F. Whitmore, Jr., "Epidemiology of Cancer of the Prostate," *Cancer* 28, 2 (Aug. 1971), 346.

SES, race, and survival from prostate cancer. He argued that race was not a factor by itself, and racial differences in survival disappeared after adjusting for socioeconomic status. SES accounted for all of the racial differences in prognosis; it mitigated the effects of race by determining the length of time a patient would live after a disease diagnosis, with those in the high-income group displaying the most favorable results. "[R]ace," he and his co-authors wrote, "does not have a statistically significant influence on survival after adjustment for SES is made."[34]

Dayal, et al.'s finding that socioeconomic status could fully explain racial disparities in certain aspects of prostate cancer epidemiology directly challenged the work of Ernster and others. Their disagreements may have stemmed from differences in the ways in which each team designed its study and defined its terms. Ernster's mortality data, for instance, covered the five-year period from 1968 to 1972 and included just four hundred cases. In contrast, Dayal calculated survival rates for 2,831 patients between 1977 and 1981, but made no distinction between deaths from prostate cancer and those due to other causes. Moreover, the researchers diverged in their descriptions of socioeconomic status. In one study, Dayal assigned an SES score calculated from the median rent, income, years of schooling, and percentage of residents living below the poverty level in each subject's census tract; in the other, he used zip code information that identified the percentage of high school graduates. While Ernster also relied on census data, she limited her criteria to the percentage of individuals aged twenty-five and older with some college education.

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[34] Hari H. Dayal, Lincoln Polissar, and Steven Dahlberg, "Race, Socioeconomic Status, and Other Prognostic Factors for Survival From Prostate Cancer," *Journal of the National Cancer Institute* 74, 5 (May 1985), 1005. See also Hari H. Dayal and C. Chiu, "Factors Associated with Racial Differences in Survival for Prostatic Carcinoma," *Journal of Chronic Diseases* 35, 7 (1982), 553-560.

Despite their conflicting conclusions, Ernster and Dayal agreed on the necessity of isolating the effects of epidemiologic variables. By removing socioeconomic status as a factor, they hoped to determine the effects of race as race, rather than race as a marker of social and economic conditions. The problem, however, was that the basis for their objective in studying race and socioeconomic status simultaneously was the very reason why separating the two would not work. If, as Dayal wrote, "[r]ace and socio-economic status are highly associated in most populations," then one could not toss out the second without affecting the first.[35] He believed that racial differences in prostate cancer survival were "not due to biologic differences in the disease between the two races" but to "differences in the distribution of socioeconomic variables."[36] But if racial disparities could be attributed to socioeconomic factors, then race failed to have any explanatory power of its own, and had meaning only when it served as a means to distinguish the latter. Meanwhile, Ernster implicated race over SES in incidence and mortality from prostatic cancer, but urged that future studies concentrate "on more specific etiologic factors which might differentiate blacks and whites, such as dietary or sexual practices, [or] occupational exposures," all of which were purely social variables.[37] She did mention the importance of examining genetic characteristics, but only as a factor equivalent to other cultural and social measures. Once Dayal and Ernster removed SES from the equation of disease, the question that remained was whether race was a function of genetics, the environment, or a combination thereof. But biomedical researchers had

[35] Dayal et al., "Factors Associated," 559.

[36] Dayal et al., "Race, Socioeconomic Status," 1006.

[37] Ernster et al., 318.

no way to further isolate each variable, as socioeconomic status would always continue to shape gene-environment interactions in ways that were not easily quantifiable.

Some of the increased visibility of African Americans within work on prostate cancer probably resulted from heightened attention to the issues of race and disease brought about by the civil rights and black nationalist movements of the 1960s and 1970s, as well as by increased funding for research on cancer. Illnesses with a disproportionate impact on African Americans acquired symbolic significance as they came to represent black suffering, racial inequality, and the unequal distribution of health care resources. In cities across the country, the Black Panther Party took up the cause of sickle cell anemia, holding workshops in community health centers to both promote awareness of the disease and adopt it as a means to engender empowerment, self-sufficiency, and social change.[38] Prostate cancer researchers benefited, moreover, from a shift in governmental funding priorities during the 1970s. In 1971, President Nixon declared war on the dread disease and signed the National Cancer Act into law, promising to increase appropriations for the National Cancer Institute from $400 million in fiscal year 1973 to nearly $1 billion by the end of the decade. Twice as much federal money went toward fighting cancer as any other disease.[39]

With more funding available for their work, researchers could expand the range of their investigations. As part of the project to sort out the relative influence of heredity and environment, a number of scientists undertook comparative studies of African Americans and black Africans. Those in other fields of cancer research, notably

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[38] Keith Wailoo, *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Health* (Chapel Hill, NC: University of North Carolina Press, 2001), 182-183.

[39] Patterson, 251, 256.

nasopharyngeal carcinoma and skin cancer, had utilized this approach to varying effects, evaluating a high-risk group of subjects alongside one which was ostensibly genetically similar, but that lived under different environmental conditions. Within the field of prostate cancer, reports on the disease among blacks in the West Indies and different parts of Africa dated back several decades. Although the studies lacked a comparative approach, they did provide data on incidence among black populations worldwide. Whereas the age-adjusted rate among African Americans in Alameda County, California was around 65 per 100,000, the same studies reported rates of 29.2 among Rhodesians, 10.2 in Nigerians, 9.4 in black South Africans, and 4.4 among Ugandans.[40] Migrants who moved from low to high-incidence regions eventually assumed an increased risk for the disease. More recent work confirmed the differential incidence between American and African blacks and revealed significant physiological disparities between the groups. One study, for instance, reported notably higher levels of plasma testosterone and estrone (a form of estrogen) among U.S. versus Nigerian subjects.[41] Overall, comparative analyses of prostate cancer in black men revealed that if one could assume genetic correspondence among black populations, then the disease was either triggered or in large part caused by environmental factors.

Work continued, as well, on other possible etiologic agents. "Ever-married" men with children were at greater risk than both single and childless men, as were those who had a "greater sexual drive as indicated by multiple sexual partners, extra-marital activity

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[40] Wynder, 344; L.M. Franks, "Etiology, Epidemiology, and Pathology of Prostatic Cancer," *Cancer* 32, 5 (Nov. 1973), 1093.

[41] Marvin A. Jackson, Joseph Kovi, Martin Y. Heshmat, T.A. Ogunmuyiwa, George W. Jones, A.O. Williams, E.C. Christian, E.O. Nkposong, M.S. Rao, Aaron G. Jackson, and B.S. Ahluwalia, "Characterization of Prostatic Carcinoma Among Blacks: A Comparison Between a Low-Incidence Area, Ibadan, Nigeria, and a High-Incidence Area, Washington, DC," *The Prostate* 1, 1 (1980), 185-205.

and frequency of venereal infection."[42] Benign prostatic hypertrophy probably did not lead to prostatic carcinoma, but the relationship between the two conditions continued to generate controversy. Jews generally had lower morbidity and mortality rates than Protestants and Catholics. Smoking, distribution of body hair, baldness, and height and weight appeared to have no effect. Men who were occupationally exposed to cadmium had an increased risk for the disease, perhaps because the element restricted the body's ability to absorb zinc; those with cancer were found to have significantly lower levels of zinc in the prostate, the male repository for the mineral.[43] Some researchers suggested that diet might play a role, as evidence showed a positive relationship between prostate cancer and high intake of dietary fat. One study found elevated consumption of buttermilk and Tabasco sauce among cancer patients.[44] Others investigated hormonal factors, including levels of androgen, testosterone, and estrogen, but, writing in 1984, found the etiologic role of endocrine function to be "largely unclear at present."[45]

By the mid-1980s, the uses of race within prostate cancer research had changed little over the preceding decades. Medically, race had a primarily social meaning, measurable in one's place of residence, years of schooling, income, occupation, and diet; physically, it was indicated by external appearance. Even studies designed to make the

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[42] Haroutune K. Armenian, Abraham M. Lilienfeld, Earl L. Diamond, and Irwin D. Bross, "Epimiologic Characteristics of Patients with Prostatic Neoplasms," *American Journal of Epidemiology* 102, 1 (July 1975), 47-54; Editorial, "The Epidemiology of Cancer of the Prostate," *Journal of Chronic Diseases* 28, 7/8 (Aug. 1975), 344.

[43] Mandel and Schuman, 42-44; Ronald K. Ross, Annlia Paganini-Hill, and Brian E. Henderson, "The Etiology of Prostate Cancer: What Does the Epidemiology Suggest?" *The Prostate* 4, 4 (1983), 338.

[44] Jack S. Mandel and Leonard M. Schuman, "Epidemiology of Cancer of the Prostate," in *Reviews in Cancer Epidemiology, Volume 1*, Abraham M. Lilienfeld, ed. (New York: Elsevier/North-Holland, 1980), 2-83; George B. Hutchison, "Incidence and Etiology of Prostate Cancer," *Urology* (Supplement) 17, 3 (Mar. 1981), 4-10.

[45] W. Dana Flanders, "Review: Prostate Cancer Epidemiology," *The Prostate* 5, 6 (1984), 625.

most of the correlation between race and socioeconomic status by isolating the effects of the latter ultimately reinforced the idea of race as a social category, as those who concluded that the disease's racial patterns could not be explained by socioeconomic status refused to attribute them instead to biology. Studies of migrants helped to confirm the role of environmental factors, but accounting for elevated incidence rates in African Americans still presented a challenge. If socioeconomic status could account for racial differentials, then race had little significance outside of it. But if epidemiologic patterns were unrelated to socioeconomic status, then additional elements were at work. This would prove to be epidemiologists' central conundrum over the next ten to fifteen years, even as new diagnostic procedures gave them additional evidentiary tools.

Detecting Disease: Diagnosis and Etiology from the Mid-1980s to the 1990s

With scientists determined to ascertain the etiology of prostate cancer, rates continued to climb. The American Cancer Society estimated that 73,000 American men would develop the disease in 1982 and 23,300 would die from it.[46] By the end of the decade, these numbers reached 99,000 and 28,000, respectively.[47] Early diagnosis made a tremendous difference in survival rate. For the 25% to 30% of patients who showed clinical evidence of distant metastases at the time of diagnosis, the death rate from the disease approached 80%.[48] In contrast, those with localized occurrences were twice as

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[46] Joseph Kovi, "Carcinoma of the Prostate: A Disease of Black Men?" *Journal of the National Medical Association* 75, 2 (Feb. 1983), 121.

[47] Robert P. Huben, "Early Detection of Prostate Cancer," *Seminars in Surgical Oncology* 5, 3 (1989), 202.

[48] Peter T. Scardino, "Early Detection of Prostate Cancer," *Urologic Clinics of North America* 16, 4 (Nov. 1989), 635; A. Wayne Meikle and Joseph A. Smith, Jr., "Epidemiology of Prostate Cancer," *Urologic Clinics of North America* 17, 4 (Nov. 1990), 709.

likely to be alive five years hence than men whose cancer had spread throughout the body.[49] Doctors, however, lacked a reliable screening test for detecting the disease in its initial stages. Digital rectal examination, the most common method thus far, had serious limitations. Tumors had to be large enough to be palpated, and false negatives were extremely common. In a review article, Peter T. Scardino, a professor of urology at Baylor College of Medicine in Houston, Texas, reported the results of a large survey in which the technique identified prostate cancer in just 45% of cancer patients; another study reported an even higher false-negative rate of 55%.[50] False positives were a problem, as well, as up to one-half of prostate nodules found by palpation turned out to be benign prostatic hypertrophy, a common, non-malignant condition that could cause inflammation of the prostate gland and urinary disturbances, but was generally not fatal.[51] Still, digital rectal examination was widely used, as it was inexpensive and did not require any elaborate equipment. Its lack of sensitivity may have restricted its effectiveness, but in the hands of a skilled practitioner, and with a little bit of luck, it could find tumors.

Transrectal ultrasonography presented another way for physicians to detect early-stage prostate cancer. A relatively new technique, it could identify tumors too small to be palpated; the authors of one large study from Japan found transrectal ultrasound to be twice as sensitive as digital rectal examination when cancer diameters were less than 1.5

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[49] Yoshihito Ban, Ming C. Wang, and T. Ming Chu, "Immunologic Markers and the Diagnosis of Prostatic Cancer," *Urologic Clinics of North America* 11, 2 (May 1984), 269.

[50] Scardino, 646-647.

[51] Huben, 203.

centimeters.[52] But what the procedure offered in sensitivity it lacked in specificity, as non-malignant conditions such as benign inflammation frequently produced inaccurate results. It presented no greater positive predictive value than digital rectal examination and needed to be performed with specialized medical equipment, adding to its cost and diminishing its availability. Because of its shortcomings, the Diagnostic and Therapeutic Technology Assessment panel of the American Medical Association in 1988 dubbed it an "investigational" rather than an established technique for effectively detecting the initial stages of prostatic carcinoma.[53]

While digital rectal examination and transrectal ultrasonography provided two methods of screening for cancer through the physical detection of tumors, scientists hoped to identify a prostate cancer-specific immunologic marker that would improve the reliability of early diagnosis. In 1936, a team of researchers had discovered elevated levels of an enzyme called acid phosphatase in patients with metastasized forms of the disease. In 1964, prostatic acid phophatase, or PAP, was reported as the type found in the human prostate.[54] The first serum tumor marker to be discovered, PAP was still the basis of assays being performed in the late 1980s.[55] Initially, the test detected the disease's earliest stages, A and B, at 0%, stage C at 30%, and stage D at 70%; later, these numbers improved to 20% for stages A, B, and C, and 80% for stage D.[56] Since early diagnosis was crucial for long-term survival, a test that was most effective at revealing prostatic

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[52] See Huben, 203.

[53] Huben, 203-204; Scardino, 647.

[54] Ban et al., 269-270.

[55] Huben, 202.

[56] P. Guinan, R. Bhatti, and P. Ray, "An Evaluation of Prostate Specific Antigen in Prostatic Cancer," *The Journal of Urology* 137, 4 (Apr. 1987), 686-687.

carcinoma at its latest stage, only after it had already spread throughout the body and rendered a complete cure nearly impossible, held little promise for fighting the second-leading cause of cancer deaths in American men.

In 1979, M.C. Wang and associates identified a new immunologic marker for prostate cancer: prostate-specific antigen, or PSA. It appeared to be more sensitive than prosatic acid phosphatase; one study reported that PSA tests detected a greater number of cases of early disease than PAP, and levels correlated more closely with tumor volume.[57] But prostate-specific antigen displayed less specificity than PAP, for conditions which caused simple prostatic inflammation routinely produced elevated readings. False negatives were not unusual, either, particularly at lower PSA levels. Furthermore, the test provided no information about the location of the tumor, if one in fact was present. Despite its shortcomings, however, the PSA test showed a positive predictive value between one-and-a-half and three times higher than for either digital rectal examination or transrectal ultrasonography.[58] While flawed, it also represented the best way to date of finding prostate cancer at an early stage, when the odds of survival were most favorable.

Although both the American Cancer Society and the American Urological Association now recommend an annual prostate-specific antigen test (as well as a yearly digital rectal examination) for all men over the age of fifty, the diagnostic procedure faced an uncertain future in the mid-1980s. It met with heavy early resistance from members of the medical profession, who objected to the test's lack of precision. As one team of urologists noted, "[a]n ideal marker for prostatic cancer should be 100 per cent

[57] Huben, 202.

[58] The highest reported positive predictive value for the PSA test was 52%. The lowest and highest values for digital rectal examination were 22% and 34%; and 17% and 35% for transrectal sonography. See Scardino, 647-650.

sensitive and 100 per cent specific."[59] Prostate-specific antigen failed on both counts. The controversy revolved around two characteristics of prostatic disease. First, the PSA test was mediocre—and significantly less successful than the prostatic acid phosphatase assay—at distinguishing between neoplasms and benign prostatic hyperplasia. In one study, twelve of twenty-nine patients with reported hyperplasia had elevated PSA levels; in another, the corresponding number was 21%.[60]

The second difficulty with the PSA test, and the more significant one from a medical standpoint, had to do with the unique features of prostate cancer. The disease progressed slowly (a characteristic which enables today's accepted approach of "watchful waiting"); one study determined that small, clinically detectable tumors took about two years to double in size.[61] Moreover, prostate cancer was extremely widespread within the American population. Autopsies of men who had died of other causes revealed evidence of tumors at rates of 15-25% in men between the ages of fifty and fifty-nine, 20-30% for sixty to sixty-nine year olds, 30-45% for seventy to seventy-nine year olds, and over 50% in those older than eighty.[62] These men harbored a form of the disease, termed "latent" or "histologic" prostate cancer, which had failed to develop to a clinically significant stage during their lifetime. Such unsuspected, latent cancers were not believed to adversely affect the health of those who had them, and did not appear to influence patterns of mortality.

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[59] Guinan et al., 687.

[60] Guinan et al., 688; Scardino, 649.

[61] Scardino, 641.

[62] Scardino, 642.

Therein lay the dilemma of prostate cancer. As Peter T. Scardino, the urology professor, wryly observed, "clinically important cancers are rarely detected when they are curable, and clinically unimportant cancers do not need to be treated."[63] The disease's prevalence meant that a majority of men would statistically be likely to have some form of it, with the oldest ones carrying the greatest odds. But how could doctors identify clinically significant tumors which would be worthwhile to treat at an early stage, while at the same time differentiating them from benign conditions or latent cancers? One researcher estimated that if physicians could find and minister to every case of prostate cancer in American males for 1989, a year in which about 30,000 were expected to die from the disease, the treatment itself would kill 75,000.[64] Many doubted that the PSA test would offer much of a solution, given its problems with both sensitivity and specificity. "The ultimate role of prostate specific antigen," wrote the authors of one article, "remains to be determined."[65] "At present," another urologist agreed, "the search must continue for an accurate and cost-effective screening method for the diagnosis of early prostate cancer, for without such a method our efforts to increase the curability of prostate cancer are critically limited."[66]

In the continuing search for the causes of the disease, additional research produced conflicting data on several theories that had seemed promising over the past several decades. Benign prostatic hyperplasia did not appear to be either a risk factor for or a precursor to prostate cancer. Studies on occupational exposure to cadmium failed to

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[63] Scardino, 643.

[64] Scardino, 651.

[65] Guinan et al., 688.

[66] Huben, 204.

generate conclusive results, as did work on the relationship of levels of zinc to rates of morbidity and mortality. Sexual history, including marital status as a marker of sexual activity and number of children as a measure of fertility, correlated with increased risk of prostatic carcinoma in some surveys but not in others. Data positing a potential link with venereal diseases, particularly those transmitted in viral form, were unconvincing.

New work, however, was beginning to confirm the etiologic significance of dietary factors. Until the late 1970s, the role of nutrition in the development of prostate cancer had not garnered much research attention, particularly as an explanation for racial differentials. As one physician wrote in 1971, "[t]he high rate among Negroes in America is not likely to be related to overnutrition, since Negroes are known to not eat as well overall."[67] In the 1980s, the possible significance of cadmium led to studies of oysters, which are rich in the element. Scientists also reported a correlation between the death rate from the disease and consumption of fats, meats, milk, sugar, and coffee.[68] The increasing interest in the relationship between diet and prostate cancer was related to new discoveries of the possible effects of nutrition on cancers of organs which were related to endocrine function, including the breasts, ovaries, and endometrium. Although the precise biochemical mechanism had yet to be determined, researchers thought that food additives could be acting as carcinogens, that dietary deficiencies might be stimulating neoplastic growth through cellular malfunctions, or that dietary excesses could be fueling particular metabolic processes.[69]

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[67] Wynder et al., 354.

[68] Kovi, 122.

[69] Mandel and Schuman, 58-59.

Researchers also posited a strong role for androgens, or male hormones, in the development of the disease, since the prostate gland relied upon androgen stimulation for normal growth and functioning and cancer rarely developed in castrated men. Furthermore, androgen deprivation for those with metastatic disease was a "well recognized" form of therapy which produced positive results.[70] Some theorized that prostate cancer cells were androgen-dependent. Others pointed to fluctuations in androgen receptor proteins, the length of which had been shown to vary by racial and ethnic group.[71] The most important hormone appeared to be testosterone, with an equally significant role for its metabolic byproduct, dihydrotestosterone. Data on serum testosterone levels was inconclusive, however. While some investigators reported higher levels in cancer patients versus controls, others found no difference, or even the reverse in younger men.[72]

Doctors hoped that work on the links among diet, hormones, and prostate cancer would help to explain the disease's racial differentials, which continued to intensify. In the United States, black men were 85% more likely than white men to receive a diagnosis of prostate cancer, and had a 114% greater chance of dying from the disease.[73] By 1993, the incidence rate for African American men reached 261.9 per 100,000, the highest of any racial or ethnic group in the world.[74] One set of studies that provided convincing

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[70] Meikle and Smith, 714.

[71] See Meikle and Smith, 714-715; Hugh McIntosh, "Why Do African-American Men Suffer More Prostate Cancer?" *Journal of the National Cancer Institute* 89, 3 (Feb. 5, 1997), 188-189.

[72] See, for instance, Meikle and Smith, 715.

[73] Ronald A. Morton, Jr., "Racial Differences in Adenocarcinoma of the Prostate in North American Men," *Urology* 44, 5 (Nov. 1994), 637.

[74] McIntosh, 188.

support for a relationship between race-based androgen levels and prostatic carcinoma came from Ronald K. Ross of the University of Southern California. He discovered that first-trimester levels of testosterone were 50% higher in pregnant African American women when compared with white women, a discrepancy which he thought could contribute to higher levels of the hormone in male offspring. Furthermore, levels of circulating testosterone in African American men surpassed those of whites by 10-15%; sustained over time, such a differential might explain a 70% higher incidence of prostate cancer in black men.[75]

Evidence was starting to suggest, also, that diet influenced the body's hormonal pathways, and increased consumption of fat could lead to elevated levels of androgens. Studies found that switching subjects to a vegetarian diet decreased plasma testosterone levels, and North American black men subsisting on vegetarian fare excreted fewer androgens and estrogens, lending theoretical support to a protective role for a low-fat, high-fiber diet.[76] The most compelling indication to date of a link between dietary fat and prostate cancer came in a 1995 study by lead author Alice S. Whittemore of the Stanford University School of Medicine. The team reported a positive association between prostate cancer risk and total fat intake among whites, blacks, Chinese Americans, and Japanese Americans in five cities in the United States and Canada. Overall caloric consumption was highest among blacks, lowest among Asian Americans,

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[75] R.K. Ross, L. Bernstein, and H. Judd et al., "Serum Testosterone Levels in Young Black and White Men," *Journal of the National Cancer Institute* 76 (1986), 45-48; B.E. Henderson, L. Bernstein, and R.K. Ross et al., "The Early In Utero Estrogen and Testosterone Environment of Blacks and Whites: Potential Effects on Male Offspring," *British Journal of Cancer* 57 (1988), 216-218; cited in Isaac J. Powell, "Prostate Cancer in the African American: Is This a Different Disease?" *Seminars in Urologic Oncology* 16, 4 (Nov. 1998), 224; McIntosh, 188-189.

[76] Kenneth J. Pienta and Peggy S. Esper, "Risk Factors for Prostate Cancer," *Annals of Internal Medicine* 118, 10 (May 15, 1993), 799.

and higher for cancer patients than for controls within each group. While all dietary fat was significant, saturated fat posed the strongest association with risk. Whittemore and her colleagues were quick to point out that consumption of saturated fat probably accounted for no more than 5-10% of the disease differential between blacks and whites and about 15% for whites and Asians, leaving room for the influence of "other environmental factors (such as diet during early adolescence) or nonenvironmental factors (such as genetically determined hormonal differences)."[77] Nonetheless, the team's discovery of a correlation between caloric intake and race and ethnicity mirrored the epidemiologic patterns of prostate cancer.

Whittemore's study may have relied on traditional epidemiologic methodology, but from the mid-1980s through the late 1990s, inquiries into the etiology of prostate cancer increasingly incorporated a serological framework. That is, researchers emphasized measurements of serum markers such as testosterone and prostate-specific antigen while paying less attention to the environmental variables on which colleagues had focused in the 1970s. The shift occurred partly as advancements in molecular biology influenced the techniques and approaches of other fields of biomedicine, including urologic oncology, and it was incomplete, as epidemiologists continued to examine socioeconomic criteria in their study of the disease. But the search for biochemical indicators, combined with prostate cancer's racial differentials, meant that the physicality of the body was becoming more critical to contemporary understandings of race. The body had internal mechanisms which could be measured and assessed,

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[77] Alice S. Whittemore, Laurence N. Kolonel, Anna H. Wu, Esther M. John, Richard P. Gallagher, Geoffrey R. Howe, J. David Burch, Jean Hankin, Darlene M. Dreon, Dee W. West, Chong-Ze The, and Ralph S. Paffenbarger, Jr., "Prostate Cancer in Relation to Diet, Physical Activity, and Body Size in Blacks, Whites, and Asians in the United States and Canada," *Journal of the National Cancer Institute* 87, 9 (May 3, 1995), 652-661. Quotation is from p. 660.

yielding precise numbers ready for comparison. Given all that remained unknown about carcinogenesis in humans, most researchers were careful to cite a multifactorial etiology, including gene-environment interaction, for prostatic carcinoma. But the possibility of uncovering quantifiable biochemical differences between races retained a certain allure, and would be strengthened by the ascendancy of the PSA test in the 1990s.

The PSA Era

The powerful, irrefutable correlation between race and class in the United States not only formed the foundation of nearly every epidemiologic study involving African Americans and prostate cancer; it underpinned work on etiology, as well. Whether explicitly or implicitly, the disease's striking racial differentials presented a problem for researchers. Some responded by attempting to disaggregate race from class, focusing instead on what was happening underneath the skin, in the body's lymphatic, endocrinologic, and hematologic systems. Others chose to examine the issue of race and socioeconomic status directly. Despite the obvious differences in methodology, scientific discipline, and intellectual bias between these two approaches, each one played an important role in shaping an ongoing conversation about the meanings and definitions of race. By the end of the 1990s, the accumulation of work within biomedicine would force a realignment of the concepts of race, genes, and disease, with prostate cancer research making a substantial contribution to the shift.

Studies of the relationships of race and socioeconomic status to prostate cancer in the late 1980s and the 1990s differed little in design from those of the previous twenty years. As always, the goal lay in determining the relative influence of each in the

development, treatment, and prognosis of disease. Data sources deviated slightly from those used earlier, as scientists now had access to at least ten years' worth of figures from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program. SEER had begun collecting data on patient demographics, stage of cancer at diagnosis, and patient survival in 1973 in five states and the metropolitan areas of Detroit and San Francisco-Oakland, and gradually incorporated additional sites over the following years, including some specifically chosen to increase coverage of nonwhites.[78] In addition, improvements in geographic mapping meant that the measurement of socioeconomic indicators could be slightly more sophisticated. Whereas prior work took into account poverty levels, average number of years of education, and median rent and income for designated tracts, researchers now added variables including household crowding and median home values. Surprisingly, no one offered a description of the system of racial classification used by their sources of data. Race could either be a mark of self-identification, a box that one checked off on a census survey or a hospital admittance form, or a category to which others assigned someone, perhaps in the process of filling out a death certificate. While the ways in which one saw oneself racially would undoubtedly define the boundaries of one's social, cultural, and economic life, researchers may have skipped this step because of the convergence in most cases of internal and external perceptions of an individual's race.

Results overwhelmingly indicated that race remained a significant factor in the epidemiology of prostate cancer. For both morbidity and mortality, racial differences persisted after controlling for socioeconomic status, regardless of the criteria by which it

[78] http://seer.cancer.gov/about/ (accessed August 14, 2007).

was measured. One study found that SES accounted for differential incidence in sites including breast, cervix, esophagus, stomach, and pancreas, but failed to explain those for bladder, uterine corpus, multiple myeloma, and prostate.[79] Another reported that blacks in Suffolk County, New York, where the median family income in 1980 equalled 155% of the figure for African Americans overall and 94.1% of that for American whites, died from prostatic cancer at a lower rate than poorer blacks, but more frequently than whites.[80] A third analysis of racial differences in the stage of cancer at presentation reported "substantial racial gradients in the distribution of diagnostic stage" even after adjustment for socioeconomic status.[81] The authors discovered that black patients were diagnosed at younger ages than whites, and were more likely to have aggressive tumors and present with later-stage cancers.

Rather than isolating socioeconomic status as a crucial factor in the epidemiology of prostatic carcinoma, these studies were instead showing that it had little effect on rates for African Americans.[82] At every income level, blacks could be expected to develop more cases of the disease than whites. It appeared, moreover, that SES did not impact

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[79] William P. McWhorter, Arthur G. Schatzkin, John W. Horm, and Charles C. Brown, "Contribution of Socioeconomic Status to Black/White Differences in Cancer Incidence," *Cancer* 63, 5 (Mar. 1, 1989), 982-987.

[80] Anthony P. Polednak, "Cancer Mortality in a Higher-Income Black Population in New York State; Comparison With Rates in the United States as a Whole," *Cancer* 66, 7 (Oct. 1, 1990), 1654-1660.

[81] Samuel C. Ndubuisi, Vincent Y. Kofie, Jacob Y. Nadoh, and Eugene M. Schwartz, "Black-White Differences in the Stage at Presentation of Prostate Cancer in the District of Columbia," *Urology* 46, 1 (July 1995), 71-77. Quotation is on p. 71.

[82] In the epidemiologic studies I examined that assessed race, prostate cancer, and socioeconomic status and were published between 1940 and 2000, I found only two, by the same lead author, in which controlling for SES removed the racial differential in incidence or mortality. Those studies were authored by Hari H. Dayal; see full citations above. A review article by lead author William Dale corroborated my findings, although the researchers limited their survey to articles published between mid-1985 and July 1994. See William Dale, Srinivasan Vijayakumar, Edward F. Lawlor, and Katie Merrell, "Prostate Cancer, Race, and Socioeconomic Status: Inadequate Adjustment for Social Factors in Assessing Racial Differences," *The Prostate* 29, 5 (Nov. 1996), 271-281.

survival in a statistically significant way. Regardless of access to and utilization of quality health care services, blacks died from prostate cancer at greater rates than whites of similar socioeconomic background. The failure of socioeconomic variables to explain racial differentials raised a number of questions. First, what did socioeconomic status as a category of analysis actually measure and stand for? Second, if socioeconomic indicators could not account for higher morbidity and mortality from among African Americans, then what could? Finally, how did these results elucidate or complicate understandings of racial difference?

Researchers had been using socioeconomic status as a rough substitute for both race and what they considered its associated corollaries of lifestyle. As one surgeon put it, "race may be seen as a gross variable for culture….These shared elements lead to common lifestyle, attitudes, and behavior." Poverty, which was so prevalent among black Americans, acted "through the prism of culture," which could either accentuate or diminish its negative effects by encouraging people to mimic the choices of those around them.[83] Those who employed SES as a stand-in for race were essentially saying that race not only largely determined income, years of schooling, and the neighborhood in which one would live, but could be measured behaviorally, in diet, levels of cigarette smoking, alcohol consumption, and physical activity.[84] But race, it turned out, was not reducible to socioeconomic variables. Perhaps, speculated the authors of a study on race and socioeconomic factors, "measures of SES are only indirect indicators which may not

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[83] Harold Freeman, "Race, Poverty, and Cancer," *Journal of the National Cancer Institute* 83, 8 (Apr. 17, 1991), 526.

[84] See, for instance, He Yu, Randall E. Harris, and Ernst L. Wynder, "Case-Control Study of Prostate Cancer and Socioeconomic Factors," *The Prostate* 13, 3 (1988), 317-325; and Freeman, 526-527.

accurately represent the real risk factors….[T]here may be different etiologic factors operating at different intensities in whites and blacks."[85]

If the exploration of socioeconomic characteristics offered limited opportunity to identify these "different etiologic factors," then prostate-specific antigen assays gave scientists another shot at isolating the real effects of race on prostate cancer. The Federal Drug Administration approved the PSA test in 1986 as a means to monitor the progression of prostate cancer in those who were already receiving therapy. Many doctors, however, were using it as a cancer screening procedure well before its additional approval as such by the FDA in 1994.[86] Controversy continued to surround the PSA test, even into the late 1990s. Although the American Cancer Society endorsed widespread PSA screening as a tool in the diagnosis of prostate cancer, other organizations, such as the American College of Physicians, recommended instead that the test be instituted only on a case-to-case basis.[87] The National Cancer Institute did not sanction its use at all. Improvements in specificity and sensitivity had increased its effectiveness at finding tumors which were confined to the prostate—the type most easily treated and most likely to become fatal if left undisturbed—while lessening the chances of finding latent or insignificant cancers. Furthermore, some doctors reported favorable outcomes in using the PSA test as a prognostic indicator among men with the disease, as higher PSA values indicated more advanced cancers that would probably not respond well to treatment.[88]

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[85] Yu et al., 321-322.

[86] Graham J. McDougall, Jr., Bryan A. Weber, Timothy W. Dziuk, and Richard Heneghan, "The Controversy of Prostate Screening," *Geriatric Nursing* 21, 5 (Sep.-Oct. 2000), 245-248.

[87] Bill Berlin, "Prostate Cancer: Is the PSA Test the Answer?" *New Jersey Medicine* 95, 3 (Mar. 1998), 53.

[88] Ibrahim Abdalla, Paul Ray, and Srinivasan Vijayakumar, "Race and Serum Prostate-Specific Antigen Levels: Current Status and Future Directions," *Seminars in Urologic Oncology* 16, 4 (Nov. 1998), 208.

In the years since its introduction, PSA screening appeared to have affected prostate cancer's rate of incidence, a measure, according to Andrew Farkas, the lead author of an article assessing the test's first ten years, of an effective screening procedure. The number of men diagnosed with the disease actually increased from 96,000 in 1987 to 132,000 in 1992, after which rates began to decline.[89] Farkas and his colleagues found, moreover, a downward shift in the patient's age and stage of cancer at diagnosis. In 1985, the mean age at diagnosis stood at seventy-two for white men and seventy for African Americans. By 1994, SEER data showed that the ages had decreased to sixty-nine and sixty-seven, respectively. The authors used the Gleason tumor grading system to illustrate the trend in grade at diagnosis; adopted in 1984, the Gleason scale classified tumors as well-, moderately-, or poorly differentiated, corresponding to scores of 2 to 4, 5 to 7, and 8 to 10. Since "the advent of the PSA era," most new diagnoses of the disease were of prostate-confined, moderately differentiated tumors with Gleason scores of 5 to 7, clinically significant cancers which generally reacted favorably to therapy.[90]

While Farkas highlighted the effectiveness of the PSA test as a screening tool, others were documenting numerous variations in PSA levels between African Americans and whites. One retrospective analysis by lead author June A. Kim reported mean pre-treatment PSA levels for prostate cancer patients at 31.7 nanograms per milliliter (abbreviated as "ng/mL") for whites and 71.6 for blacks, well above the normal range of 0 to 4.0 ng/mL. 73% of black patients, compared with 58% of whites, showed PSA readings greater than 10.0. After treatment, PSA levels among African American

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[89] See Berlin, 53; and A. Farkas, D. Schneider, M. Perrotti, K.B. Cummings, and W.S. Ward, "National Trends in the Epidemiology of Prostate Cancer, 1973 to 1994: Evidence for the Effectiveness of Prostate-Specific Antigen Screening," *Urology* 52, 3 (Sep. 1998), 444-448.

[90] Farkas et al., 445, 446-447.

subjects remained higher, with 60% continuing to show levels above 4.0, in contrast to just 40% of whites. The authors also discovered a greater percentage of black men with poorly differentiated tumors; 40% presented with Gleason scores of 8 to 10, compared with 26% of whites.[91] Others had similar findings, observing higher PSA values at diagnosis in African American versus white men with prostate cancer, even after adjustment for age, stage of the disease, tumor grade, and tumor volume.[92]

Overall, researchers found elevated pre- and post-treatment PSA levels in African Americans, along with higher Gleason scores, which indicated more aggressive tumors, a greater rate of treatment failure, and poorer prognosis. Studies documented higher serum PSA levels even in blacks without prostate cancer. One analysis, reported in a review article by lead author Ibrahim Abdalla, showed mean serum PSA values of 1.9 ng/mL in whites and 2.6 in African Americans. In many investigations, black men were found to have larger prostate volumes than whites, translating to higher PSA levels regardless of cancer status. A second study which controlled for prostate volume in non-cancerous men put PSA levels at 4.3 ng/mL for whites and 7.9, or 54% higher, for African Americans.[93]

As the evidence showed, African Americans consistently recorded higher PSA levels than whites. Among those with prostate cancer, black men showed higher readings than white men both at diagnosis and after treatment. Even African Americans with no

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[91] June A. Kim, Deborah A. Kuban, Anas M. El-Mahdi, and Paul F. Schellhammer, "Carcinoma of the Prostate: Race as a Prognostic Indicator in Definitive Radiation Therapy," *Radiology* 194, 2 (Feb. 1995), 545-549.

[92] Judd W. Moul, "Use of Prostate-Specific Antigen in Black Men: Age-Adjusted Reference Ranges for Maximal Cancer Detection," *Journal of the National Medical Association* 90, 11 (Supplement, Nov. 1998), S710-S712.

[93] Abdalla et al., 208-209.

clinical signs of prostatic carcinoma had elevated PSA values when compared with whites. Prostatic tumors in black men were distinctive, as well—more aggressive, less well-differentiated, and with a higher volume than similar-stage tumors in Caucasians. The differences in PSA readings among whites and blacks were consequential enough that one researcher proposed adopting more nuanced ranges for normal PSA values, which would be adjusted to take into account not only age, but also race.[94]

Serum PSA, like testosterone, appeared to be a biochemical marker that displayed distinct racial patterns. Although scientists had reached consensus that, as such, it offered a new method to quantify racial differences, they disagreed on the underlying reasons for the disparate readings among whites and blacks. One urologist suggested that cellular production of PSA might differ by ethnic group, with secretion highest among African Americans.[95] Abdalla and his colleagues pointed to higher serum testosterone levels in African Americans as a potential factor, as well as lower socioeconomic status and its corollaries of lack of health insurance and decreased access to medical care.[96] Kim and her coauthors identified "genetic predisposition, hormonal milieu, immune status, diet, and exposure to carcinogens" as additional factors for further study.[97] But socioeconomic variables also determined lifestyle factors, such as neighborhood of residence and patterns of physical activity, which could affect one's health in subtle, unmeasurable ways. While most researchers posited a combination of socioeconomic factors and serological indicators, they had to guess at the relative importance of each.

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[94] See Moul, S710-S712.

[95] Powell, "Prostate Cancer in the African American," 223.

[96] Abdalla et al., 209-210.

[97] Kim et al., 548.

As Abdalla wrote, "[w]hether these differences are due to biological or sociological reasons or a combination of both is not known at this time."[98]

Advances in genetics provided another set of possible explanations. One team of researchers linked hereditary prostate cancer to a gene on chromosome 1, called HPC1, and found that African Americans were twice as likely as whites to have the associated form of the disease.[99] Other work connected an increased risk of prostate cancer to a relatively low number of CAG repeats on an androgen receptor gene.[100] Fewer CAG repeats correlated with increased androgen stimulation and a diagnosis of prostate cancer at a younger age. Compared with whites, African Americans had shorter CAG repeat lengths on the androgen receptor gene.[101] As with other theories on the etiology of the disease, however, the final significance of CAG repeats, admitted Abdalla, "is not known."[102]

By the end of the 1990s, shifts in avenues of investigation for the etiology of prostate cancer largely reflected broader changes in the direction of mainstream scientific study. New breakthroughs and new technologies led scientists in all areas of biomedicine to explore previously unknown or overlooked paths. Advancements in the field of genetics, in particular, reshaped the field of cancer research. The Human Genome Project, begun in 1990 and completed thirteen years later, provided investigators with a chromosomal map that seemed certain to revolutionize the practice of medicine.

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[98] Abdalla et al., 211.

[99] Powell, "Prostate Cancer in the African American," 224.

[100] "C," "A," and "G" stand for cytosine, adenine, and guanine, which, along with thymine, are the four chemical bases that make up DNA.

[101] Abdalla, 210; Powell, "Prostate Cancer in the African American," 224.

[102] Abdalla, 210.

Observers touted gene therapy as the frontier of the twenty-first century, hoping some day to be able to offer treatments for disease which could be customized to suit a patient's particular genotype.

Although researchers had made a measure of progress in determining the causes of prostate cancer, the disease's origins, for the most part, remained a mystery. It was clear that neither socioeconomic status nor biochemical indicators alone could account for the complexity of the disease. The effects of socioeconomic variables were still mediated through one's genetic makeup, as in the well-known example of the lifelong smoker who never got lung cancer. Serological markers such as prostate-specific antigen and hormonal levels appeared to be determined by genes, as well.

Scientists had, in short, devised a new way of looking at race, and discoveries from work on prostate cancer provided the medical foundation for it. Prostate cancer researchers had determined that genetic differences between groups of people not only did exist, but were crucial to one's health. These disparities went beyond inconsequential variables, such as hair color and eye color, to encompass internal mechanisms that affected the development and progression of disease. Genes governed both one's physical appearance and one's biochemical composition. They affected whether one would have high or low testosterone levels, abnormal cholesterol readings, or a predisposition to cancer.

That this new conception of race centered on genes was not, in itself, significant to contemporary understandings of it. What mattered, instead, were the ways in which genotype aligned with phenotype. In other areas of disease, one group of subjects might weigh more or have higher average blood pressures than another. With prostate cancer,

however, differences in PSA readings and testosterone levels correlated with existing racial boundaries: race could act as a predictor of an individual's relative PSA and testosterone levels. This presented the illusion that race had a biological basis; that blacks, whites, and Asians differed from one another in ways which remained unchanged from one generation to the next. Race now indicated not just external disparities, but internal ones, as well. Genetic characteristics, determined by the concentration of certain traits within a population, now appeared to equate to innate biological ones.

Rather than refuting the idea that race was more than skin deep, scientific advancements in molecular biology and diagnostic technologies had instead confirmed it. The new conception of race complicated decades of disavowals by racial activists, social scientists, and liberal policymakers that race was a primarily social category with no grounding in the body's internal networks, for it seemed, now, that race influenced every cell of one's body, inside and out. As the twentieth century drew to a close, racial discourse lay mired in an obfuscation of genes and biology, of genotype and phenotype, with medical science unable to provide a meaningful resolution to the longstanding entanglements of race and disease.

Conclusion

The story of prostate cancer in the twentieth century illustrates a number of themes at the crossroads of race and medicine. First, the meanings of race within biomedicine can be deeply affected, if not determined, by scientific methodology. In the postwar period, changes in epidemiologic methodology shaped the criteria by which researchers organized their work, prompting a focus on socioeconomic status beginning

in the 1960s that would gradually grow more refined with an increasingly sophisticated database of indicators. At the same time, advances in biochemistry and molecular biology made new tools and knowledge available to researchers, who were now able both to measure serological markers where they previously couldn't and to examine disease on a genetic level. Second, prostate cancer's growing association with African Americans, a shift that probably resulted from a combination of increased diagnosis, longer life span, and enhanced medical scrutiny, guided biomedical research on etiology. Once investigators had documented racial disparities in the disease, they sought to explain them. But the racial distribution of prostate cancer shaped what scientists chose to study. Since they already knew that blacks had higher rates of incidence and mortality, they looked for ways in which blacks differed from whites, factors which might explain the disease's racial patterns. Thus, socioeconomic variables and serological indicators became two of the major categories of investigation.

But scientists could not ultimately resolve the question of prostate cancer's racial patterns because no one had figured out a way to account for all that race determined and meant. An evolving epidemiologic methodology and advancements in the cellular foundations of disease guided research on prostate cancer, but both socioeconomic status and serology proved to be insufficient ways of measuring race and quantifying its effects. Epidemiologists concerned with socioeconomic status believed that race was not a factor in itself, but mattered in the extent to which it determined income and lifestyle; they assumed that the influence of race could be assessed through an index of social indicators. The inability of socioeconomic variables to explain fully the racial patterns of prostatic carcinoma, as it became increasingly clear throughout the 1970s and into the

1980s, led scientists to other areas of biomedicine. The identification of prostate-specific antigen in 1979 and the PSA test as a method of diagnosis marked a turning point in research on the disease, as it provided evidence that race was more than a mere social category and gave scientists a way to quantify it in a biochemically measurable way.

The failure of both socioeconomic variables and serologic markers as complete etiologic explanations reveals the difficulty of finding a way to measure the real significance of race to health. Race is neither purely social nor purely biochemical; that differences in blood measurements conform to racial boundaries at all is a function of both genetic and social influences. As the history of prostate cancer shows, the most significant shift in the understanding of race within biomedicine in the postwar United States has been from a primarily social concept to one which emphasizes intracellular differences between racial groups. The trend today is toward scientific analyses which substantiate a bodily, biochemical foundation for race. Racial categories will likely retain their utility within medicine for some time as reasonable ones into which to divide research subjects. But their importance derives from the historical interaction of variables that remain too numerous, indistinct, and imprecise to measure.

Conclusion

The New Biology of Race

For centuries, we have looked to science to generate and clarify our understandings of race. Phenotypic racial markers have long suggested that its meanings would be revealed by medical assessments of the human body. Scientific advancements in techniques and methodologies, with their underpinnings of experimental and analytical rigor, have brought with them changes in how we perceive race and its bodily expressions. Although medical notions of race no longer encompass the idea of innate physiological differences among populations—what disparities exist are thought to be a result of genetics, not essential biology—the physical manifestations of race remain visible on the body. Thus, the scientific fields which study the body will continue to be seen as the logical places to search for answers regarding race.

In June 2005, the U.S. Food and Drug Administration caused a stir in medical and academic circles when it approved a medication called BiDil. Not a new drug at all, but an unpatented combination of two previously available generics which had been in use for over a decade, BiDil was the first race-specific drug to receive government sanction. In clinical trials, it was associated with a reduced risk of hospitalizations and deaths in African Americans with congestive heart failure. The Association of Black Cardiologists and the Congressional Black Caucus stepped in line behind the drug, while others lauded the FDA's decision as a move toward pharmacogenomics, in which pharmaceuticals and other treatments for disease could be tailored to each individual's specific genotype. Critics, however, pointed to the study's faulty scientific reasoning, warning that it

signaled a return to an era in which physicians reified race, ascribing its significance to immutable, unchangeable physical characteristics.

The controversy surrounding BiDil centered on the meanings of race within medicine. If the drug was really more successful at treating heart disease in one race, then why was it so? How could race, a social category with boundaries arbitrarily drawn along lines of skin color, alter the medication's effectiveness? Although BiDil had been tested only in African Americans, the exclusion of other racial groups from the study did not necessarily indicate that it would be an ineffective therapy for non-blacks. Some wondered if the drug's racial associations would make physicians reluctant to prescribe it for white, Asian, or Hispanic patients whom it could potentially help. Others questioned the composition of the subjects in the clinical trial. "Does the drug work more effectively," asked one observer, "on dark-skinned blacks than light-skinned, vanilla-colored vs. caramel, deep-chocolate-colored vs. high yellow? What level of black blood, DNA or gradation of complexion must African Americans have to be good candidates to receive the drug and have it work effectively?"[1]

The crucial detail about the clinical trial was that it drew its sample from self-identified African Americans: every patient in the study had described himself or herself as such and checked the appropriate box on the requisite medical questionnaire. This simple act of self-identification denoted much more than just skin color. Even if categorizing oneself as African American implied that one possessed genetic markers that were concentrated within the population due to generations of reproductive patterns, the

[1] Frank Harris III, "'Black' Drug for Heart Baffles the Mind," *Hartford Courant*, Nov. 15, 2004, A9; quoted in Keith Wailoo and Stephen Pemberton, *The Troubled Dream of Genetic Medicine* (Baltimore: Johns Hopkins University Press, 2006), 172.

more significant aspect of self-identification was that it affected how one chose to live one's life. It could impact where someone resided, went to school, and worked, and the people whom he or she befriended. It could also influence how one was perceived by others. In the field of medicine, this could alter doctor-patient interactions in ways that affected health outcomes but were too subtle and indistinct to measure.

The medical meanings of race have long been contested and fraught with controversy. As biomedical research on cancer reveals, scientists in the twentieth century employed numerous definitions of race in their struggle to explain racial patterns of incidence. The racial associations of prostate cancer, nasopharyngeal carcinoma, skin cancer, and cervical cancer each offered an opportunity to analyze the relationship between race and carcinogenesis by determining the medical significance of various racial traits. Doctors classified and reclassified subjects according to a range of physical and physiological criteria, hoping that each new characteristic would be the one to unlock the mysteries of cancer causation.

Racial categories and cancer etiologies, moreover, were mutually constitutive; doctors laboring to uncover the disease's pathways shaped racial categorizations through their interpretations of epidemiologic patterns, while particular definitions of race influenced investigative methodology. Disease identities showed them where to begin, and new diagnostic technologies and procedures enabled them to assess what they found. But while the interplay of disease identity, technology, and methodology guided the search for the roots of carcinogenesis, each successive measure of race that researchers adopted proved insufficient at exposing the reasons behind cancer's racial patterns. Nationality, culture, skin tone, physicality, genetics, socioeconomics, and biochemistry

offered partial explanations, but none could elucidate everything. As a result, scientists were forced to confront new ways of understanding race in their continual quest to determine why some racial groups developed cancer at higher rates than others.

In the 1920s and 1930s, cancer researchers considered race through the lens of civilization, a cultural designation which inferred race only indirectly. Through the use of death certificates and insurance records, they compiled statistics which served to both naturalize and rationalize racial categories of organization. Between about 1930 and 1955, shifting knowledge about the etiology of skin cancer, the form of the dread disease with the earliest visibility, led to changing classifications of subjects by race, ethnicity, and skin color. A research focus on culture and skin tone among "Negroes" gave way to an emphasis on complexion among whites after World War II, when the causal link between ultraviolet radiation and skin carcinomas gained broad acceptance. Cervical cancer researchers took a different approach, as skin tone had little physiological effect on the development of the illness. Instead, they defined race in behavioral terms, rendering it a risk factor for the disease because of the ways in which it appeared to shape culture and conduct, particularly the sexual activity that played a role, though still undetermined at the time, in cervical cancer etiology. Probing the mechanisms responsible for nasopharyngeal carcinoma led scientists to multiple definitions of "Chinese" and "Oriental." Categorizations based on ideology took precedence over medical ones; at the same time, researchers standardized as "Chinese" the habits and customs of those who traced their ancestry to Guangzhou province, as they constituted the most populous group among Chinese immigrants in the U.S. By the end of the twentieth century, prostate cancer had played the most significant role in influencing

racial discourse, as new techniques for testing the body's internal mechanisms appeared to confirm the existence of biochemical discrepancies between races, offering proof of essential differences between them and casting doubt on the notion of race as only a social category.

Like the narrative of the racial distribution of cancer in the twentieth century, the story of BiDil underscores the ways in which science continues to shape racial knowledge. Since discoveries in the field of genetics in the mid-twentieth century, race has gradually made its way back inside the body, bypassing skin color, physiology, and socioeconomics as inadequate measurements of its medical significance. The new racial biology portrays genes as determinative of medical outcomes, and biomedical data as evidence of the naturalness of racial groupings. The problem is not that discrete groups possess distinctive genotypes, for traits have indeed been distributed unequally across continents, a consequence of both migration and isolation. But when these genotypes align with phenotype—when internal genetic variations fall along the borders of the categories we have defined as races—then biological conceptions of race reemerge, cast in the language of genetics.

At the dawn of the twenty-first century, biomedicine is furnishing another way to understand race, but one which harkens back to a time of "natural," internal distinctions between human populations. We stand poised on the precipice of a dangerous new era of biologism, in which the ascendancy of genetics threatens to supplant decades of disavowal of innate racial differences and return us to an older notion of racial science. Misconceptions about the distinction between genes and biology risk obfuscating the difference between inherited traits and immutable ones, taking traits which differ among

individuals who are members of already defined ethnic or racial groups, and recasting them as immutable, biological distinctions. The new racial biology, furthermore, assumes that the major categories of self-identification, such as age, race, and gender, are the ones with relevance to biomedical research. By medicalizing the effects of social and political categories, this approach naturalizes social inequalities and imprints them upon a racialized body.[2]

The DNA era, widely hailed as an age when the idea of a biological basis to race would be dismantled once and for all, has instead engendered popular confusion. Rather than vanquishing the long-entrenched notion of physical differences as fixed, it has reinforced them by lending fresh credibility to old attitudes. We are amassing a new library of misuses of scientific knowledge about race. When DNA evidence appears to quantify preconceived attitudes toward race, then the dissimilarities appear natural and authentic. And once we agree that groups which we have defined as races differ from one another both physically and physiologically, then claims for other kinds of inborn disparities may not be far off. James Watson, the geneticist and Nobel Prize winner, made headlines in October 2007 when he declared that Africans were inherently less intelligent than whites, and predicted that the gene responsible for differences in intellectual capacity would be found within a decade.[3] While the assertion itself was not original, the scientific scaffolding for the claim was. Private companies, such as 23andMe, now offer genetic testing to consumers eager to find out their ancestry and

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[2] My thinking here has been influenced by Steven Epstein's *Inclusion: The Politics of Difference in Medical Research* (Chicago: University of Chicago Press, 2007), especially Chapter 10, "To Profile or Not to Profile: What Difference Does Race Make?".

[3] Cahal Milmo, "Fury at DNA pioneer's theory: Africans are less intelligent than Westerners," *The Independent*, Oct. 17, 2007, http://www.independent.co.uk/news/science/fury-at-dna-pioneers-theory-africans-are-less-intelligent-than-westerners-394898.html (accessed Apr. 15, 2008).

inherited risks for certain diseases. But such tests can raise more questions than they answer. Henry Louis Gates, Jr., for instance, discovered that his maternal ancestors were white, while James Watson's genetic test revealed the same amount of African DNA in the scientist as could be expected in someone with one African great-grandparent.[4] Continental origins do not necessarily correlate to skin color; even if they did, their meanings would still be historically determined.

Despite new technologies and diagnostic procedures, biomedicine cannot yet account for all that race determines and influences. Perhaps race, as we have historically understood it, is less important than racism in affecting health outcomes. Nancy Krieger, an epidemiologist who studies social inequalities in health, has found that Asian Americans who describe high levels of racial and ethnic discrimination are more likely to smoke than peers who report no unfair treatment; smoking, of course, is the greatest risk factor for lung cancer, which is the leading cause of cancer-related mortality among Asian Americans.[5] Neuroendocrinologist Bruce McEwen argues that feelings of frustration, anger, or powerlessness trigger the release of hormones which unleash a collection of detrimental bodily effects, ranging from increased blood sugar and heart rate to inhibited digestion. Since poor people, especially blacks and Latinos, are constantly

[4] Ron Nixon, "DNA Test Find Branches but Few Roots," *New York Times*, Nov. 25, 2007 (accessed online Apr. 15, 2008); John Schwartz, "DNA Pioneer's Genome Blurs Race Lines," *New York Times*, Dec. 12, 2007 (accessed online Apr. 15, 2008).

[5] David H. Chae, David T. Takeuchi, Elizabeth M. Barbeau, Gary G. Bennett, Jane Lindsey, and Nancy Krieger, "Unfair Treatment, Racial/Ethnic Discrimination, Ethnic Identification, and Smoking Among Asian Americans in the National Latino and Asian American Study," *American Journal of Public Health* 98, 3 (Mar. 2008), 485-492. See also Nancy Krieger, "Stormy Weather: Race, Gene Expression and the Science of Health Disparities," *American Journal of Public Health* 95, 12 (Dec. 2005), 2155-2160.

exposed to sources of stress, hormonal response might account for some of the differences in rates of disease among these populations.[6]

Its shortcomings notwithstanding, race will continue to be a viable category for medical research for the simple reason that it is the best one we have yet devised. Imperfect though it may be, race shapes health outcomes and responses to disease in numerous ways. It affects access to treatment, structures the interpersonal transactions of doctor-patient relationships, and determines the effectiveness of drug therapies. While it is a category with social meaning, medicine has shown us that it is more than that. Whatever else race is, it is a social category with somatic effects. As long as it exists as a social classification, it will beget bodily markers of non-medical variables. Race has always been, and will remain a reality for those whose everyday lives it structures, and it will not cease to shape disease outcomes. But until we can devise a dependable way to measure not only the tangible effects of race, but the intangible ones, as well, we will have to work to ensure that the medical meanings of race are not misused to affirm the adequacy of analytically inadequate boundaries among human populations.

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[6] See Helen Epstein, "Ghetto Miasma: Enough To Make You Sick?" *New York Times Magazine*, October 12, 2003.

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