Tobacco Research Network on Disparities

TReND Toolkit

TReND
Tobacco Research Network on Disparities

Pre-Conference Workshop
14th World Conference on Tobacco or Health
March 8, 2009 ♦ Mumbai ♦ India
Toolkit

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Tobacco Research Network on Disparities (TReND)

Dear Colleague,

On behalf of the planning committee, I would like to thank you for participating in the Tobacco Research Network on Disparities' (TReND) pre-conference workshop, *Research to Reduce Tobacco-Related Inequalities: Worldwide Implications For and Exemplars of Tobacco Control.*

We explored how factors of social stratification (e.g. gender, socioeconomic class, race, ethnicity, and caste) play influential roles in tobacco-related outcomes that lead to inequalities in illness and mortality; and communication access, literacy, and use; and from effective health and policy interventions.

As a companion to this workshop, the enclosed toolkit contains a number of resources to provide you with some assistance as you start, or continue, your own work on tobacco-related health inequalities. This toolkit contains:

- Data set overviews and website links
- Media materials
- International tobacco control funding organizations – Bloomberg, Gates, Fogarty, Swedish, Canadian agencies
- Links to some international tobacco control organizations
- Exemplar papers on tobacco-related inequalities research (check to make sure this is true)
- Pre-conference Workshop Participant list
- TReND website link and membership list
- U.S. National Institute of Health Fogarty International Center's International Tobacco and Health Research and Capacity Building program abstracts and contact information

While attempts were made to include international sources, we acknowledge that the contents are not comprehensive and are drawn heavily from the U.S. The contents should be treated as the bricks to help you lay the foundation for your future research endeavors.

We look forward to working together to reduce tobacco-related inequalities.

Thank you,

Deborah L. McLellan, M.H.S.
TReND*, Workshop Chair

**For more information about TReND and the most up-to-date information on tobacco-related health inequalities, please visit our website at: www.tobaccodisparities.org.*

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Data Sets

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The Global Youth Tobacco Survey

The Global Youth Tobacco Survey (GYTS) was jointly developed by the World Health Organization (WHO) and Centers for Disease Control and Prevention's (CDC's) Office on Smoking and Health. The purpose of this survey is to track tobacco use among students aged 13-15 years across countries using a common methodology and core questionnaire. The objective of the GYTS surveillance system is to enhance the capacity of countries to design, implement, and evaluate tobacco control and prevention programs. Funding for the GYTS has been made possible by the Canadian Public Health Association, National Cancer Institute, United Nations Children Emergency Fund, and World Health Organization—Tobacco Free Initiative.

For more information on the survey methodology, or to view the questionnaires and datasets, please go here: http://www.cdc.gov/tobacco/global/gyts/index.htm.

The Global School Personnel Survey

The Global School Personnel Survey (GSPS) was also jointly developed by the WHO and the CDC. The purpose of this survey is to collect information from school staff concerning their tobacco use and their tobacco related school policies and programs.

For more information on the survey methodology, or to view a list of the core questionnaire components as well as the participating countries, please go here: http://www.cdc.gov/tobacco/global/gsps/intro.htm.

The Global Health Professions Student Survey

The Global Health Professions Student Survey (GHPSS) was developed by the WHO, the CDC and the Canadian Public Health Association. The survey's purpose is to collect information on tobacco use and cessation counseling among third-year health-profession students pursuing advanced degrees in dentistry, medicine, nursing, or pharmacy in all WHO-member states.

For more information on the survey methodology, or to view the questionnaire and data results, please go here http://www.cdc.gov/tobacco/global/ghpss/index.htm.

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The Global Adult Tobacco Survey

The Global Adult Tobacco Survey (GATS) was launched in February 2007 by the Bloomberg Global Initiative to Reduce Tobacco as a new component of the ongoing Global Tobacco Surveillance System (GTSS). It is a household survey designed to fill the data gap for measuring adult tobacco use globally and to optimize the reach and results of the GTSS. Countries can use results from the GATS to assist in the formulation, tracking and implementation of effective tobacco control interventions.

To learn more, please visit: http://www.cdcfoundation.org/sitefiles/Bloomberg_GATS.pdf.

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The International Tobacco Control Policy Evaluation Project

The International Tobacco Control Policy Evaluation Project (the ITC Project) is the first-ever international cohort survey of tobacco use. The project is comprised of annual surveys that are designed to address critical issues in global tobacco control by measuring the impact of policies (at the level of the individual smoker) in the Framework Convention on Tobacco Control (FCTC) that are being implemented in numerous countries worldwide. As a means of promoting strong, evidence-based implementation of the FCTC, research findings are widely disseminated to researchers, policymakers, advocates, and others involved in the global tobacco control community.

To access more information about the ITC Project and download a brochure, go to: http://www.itcproject.org/.

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The World Health Survey

The World Health Survey (WHS), developed and implemented by the World Health Organization (WHO), provides comprehensive baseline statistics on the health of populations in 70 countries and on the outcomes associated with the investment in health systems. With this survey, the WHO is able to offer valid, reliable and comparable information at a nominal cost that will allow policy-makers to monitor the achievement of the health systems' goals and give them the evidence needed to adjust their policies, strategies and programs.

Visit the link below for access to the following:

- Survey instruments and related documents
- Participating countries
- Sampling guidelines
- Training presentations

http://www.who.int/healthinfo/survey/en/index.html

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Media Materials

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Advertising and Promotion Resources

There are a large number of exemplar advertisements or promotional resources available on the World Wide Web pertaining to tobacco control. The following is a limited list of sites with links to materials you may find interesting and useful.

The Campaign for Tobacco Free Kids website hosts the "International Tobacco Control Library" which is a "treasure chest of tobacco control materials from around the world." Use this link to search the database: http://tobaccofreekids.org/campaign/global/library/. This organization also has an International Resource Center section of the site with links to advertising and promotional resources. That can be found here: http://www.tobaccofreecenter.org/resources/advertising_promotion/resources.

The Brazilian government has mandated new tobacco labeling measures. The graphic images associated with warning messages can be found on the Brazilian National Cancer Institute (INCA) website: http://www.inca.gov.br/english/cigarrete_packages.html.

The American Legacy Foundation launched the truth® campaign in February 2000. It is the largest youth smoking campaign in the United States and extremely successful. To read more about the campaign and find additional links, please visit: http://www.americanlegacy.org/truthnews.aspx.

Additional advertisements from the World Health Organization's Tobacco Free Initiative (TFI) program can be viewed on YouTube: http://www.youtube.com/view_play_list?p=BC60C5B504C8A01B.

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Reports and Studies

Tobacco Use Prevention Media Campaigns:

Lessons Learned from Youth in Nine Countries

The 2006 report, *Tobacco Use Prevention Media Campaigns: Lessons Learned from Youth in Nine Countries*, sponsored by the Centers for Disease Control (CDC), is a review of selected studies and successful mass media campaigns from various countries provided by tobacco control practitioners and researchers. Included in this report is a list of advertisements by country as well as states within the United States.

To read the report, please visit:
http://www.cdc.gov/tobacco/youth/00_pdfs/YouthMedia.pdf

Monograph 19: The Role of the Media in Promoting and Reducing Tobacco Use

The Role of the Media in Promoting and Reducing Tobacco Use is the 19th monograph in the U.S. National Cancer Institute's Tobacco Control Monograph Series. As mentioned on the webpage listed below, "Monograph 19 provides a critical, scientific review and synthesis of the current evidence regarding the power of the media, both to encourage and to discourage tobacco use. It is the most current and comprehensive summary of the scientific literature on media communication in tobacco promotion and tobacco control. Research included in the review comes from the disciplines of marketing, psychology, communication, statistics, epidemiology, and public health. All are vital to understanding how exposure to the media influences tobacco use."

To download a PDF of the full monograph or individual chapters, please visit this site:
http://cancercontrol.cancer.gov/TCRB/monographs/19/index.html.

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Funding Organizations

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Bloomberg Initiative to Reduce Tobacco Use

The Bloomberg Initiative to Reduce Tobacco Use is a $125 million initiative to combat tobacco use outside of the United States in low- and mid- income countries. Mayor Michael Bloomberg named five partner organizations to implement the initiative: the Campaign for Tobacco-Free Kids, Centers for Disease Control and Prevention Foundation, the Johns Hopkins Bloomberg School of Public Health, the World Health Organization, and the World Lung Foundation.

Visit the following links for more information about the Bloomberg Initiative from each of the five partners:

Campaign for Tobacco Free Kids:
http://tobaccofreecenter.org/about_us/bloomberg_initiative

Centers for Disease Control and Prevention Foundation:
http://www.cdcfoundation.org/programs/CDCFoundationinitiatives.aspx

Johns Hopkins Bloomberg School of Public Health:
http://www.jhsph.edu/global_tobacco/bloomberg_initiative/index.html

World Health Organization:
http://www.who.int/tobacco/communications/highlights/bloomberg/en/

World Lung Foundation: http://www.worldlungfoundation.org/news_bloombergfunds.php

Additional information can be found at these sites:
http://www.globaltobaccocontrol.org/
http://www.mikebloomberg.com/files/pdfs/initiative_whyuswhynow.pdf

Bloomberg Initiative to Reduce Tobacco Use
Why us? Why now?

The battle against tobacco is too important to lose, and the lives of those affected are too valuable not to act. Through increased education and awareness, we can empower governments with the tools they need and people with the knowledge they want to protect themselves against harmful tobacco and prevent up to a billion senseless deaths over the next century.

- ***Our Mission:*** Tobacco use is a *global* health problem that requires a *global* solution. Despite the awareness of this challenge, the battle against the dangerous and deadly effects of smoking is both under-funded and underexposed. Our mission is to work with governments and civil society to implement the strategies that are known to reduce tobacco use: monitoring tobacco use and tobacco-reducing policies, protecting people from the harms of tobacco smoke, offering help to smokers who want to quit, warning the public about the dangers of tobacco use, enforcing advertising bans, and raising taxes on tobacco. If implemented worldwide, these strategies have the potential to slash tobacco use and save many millions of lives over the next century.

- ***Our Commitment:*** Reducing the deadly effects of tobacco across the globe means an ongoing *global* commitment to tobacco prevention efforts. This uncompromising commitment to a cleaner, healthier world is reflected in the unprecedented resources we have pledged to this cause. With the addition of Bill and Melinda Gates to the Bloomberg Initiative, we are excited to announce that $500 million will be directed toward humanitarian efforts to turn the tide against harmful tobacco. Together, with our experience, resolve, and passion, we will turn these unparalleled resources into unparalleled success.

- ***Our Actions:*** We will employ tools uniquely targeted to the societies in which we work, ones that are proven to reduce tobacco use in a variety of settings. We will partner with governments to raise taxes on tobacco. We will devote millions of dollars toward restricting tobacco ads. And we will support NGO's with a variety of funding opportunities to strengthen their important work, providing them with the resources they need to conduct crucial tobacco-prevention work on the ground in low- and middle-income countries.

- ***Our Results:*** We have galvanized a movement, supporting organizations in many countries, such as Brazil, Indonesia, India, and Tanzania, in growing from one or a handful of people to nationwide forces. We have seen great progress in countries like China, which has gone smoke-free and has had large increases in favorable coverage of tobacco control efforts; Turkey, which has gone smoke-free; and Mexico, where Mexico City has gone smoke-free and important national legislation has been passed. And just recently, we were proud to play a role in the development and launch of the first ever World Health Organization Report on the Global Tobacco Epidemic. These accomplishments are integral to tobacco prevention, and we know that this progress will result in many lives saved. But it's only a start.

We know what must be done. Our role is to implement a global plan of action that has a long-term focus and immediate impact. Our mission is a healthy life right from the start – one that is free of smoke, free of addiction, and free of life-threatening tobacco-related diseases.

The Bloomberg Initiative will continue to apply unprecedented resources toward public education efforts, legislative action and enforcement, and monitoring rates of tobacco use. We will do whatever it takes to prevent the loss of life and protect still more from unnecessary suffering. It is not an easy challenge, but it's a challenge that we can and will meet.

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Bill & Melinda Gates Foundation

Based in Seattle, Washington, the Bill & Melinda Gates Foundation works to help all people, in both developed and developing countries, lead healthy, productive lives. The Gates Foundations has joined with New York Mayor Michael Bloomberg and his Bloomberg Initiative to combat the global tobacco epidemic. The foundation financially supports the MPOWER package, a set of six key tobacco control measures developed by WHO, and provides additional funding to build economic evidence to support tobacco control and public education.

Visit the Bill & Melinda Gates Foundation to read more:
http://www.gatesfoundation.org/topics/Pages/tobacco.aspx

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U.S. National Institutes of Health
John E. Fogarty International Center for Advanced Study in the Health Sciences

The overall mission of the Fogarty International Center (FIC), a part of the National Institutes of Health (NIH), it to support and facilitate global health research conducted by investigators worldwide, build partnerships between U.S. health research institutions and others abroad, and train the next generation of scientists to address health needs globally.

The FIC and various partnering institutions have established the International Tobacco and Health Research and Capacity Building Program (TOBAC). In 2002, the first 14 grants were awarded. Each project is designed to reduce the burden of tobacco consumption in low- and middle- income nations.

To read more about the FIC and the TOBAC, please visit:
http://www.fic.nih.gov/programs/research_grants/tobacco/index.htm

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Research for International Tobacco Control

The Research for International Tobacco Control is a program within the International Development Research Center's Social and Economic Policy program area. RITC is a funder of tobacco control research projects in developing countries that will lead to effective policies and programs. Currently, the RITC with support from the Bill & Melinda Gates Foundation, has launched an initiative to understand the determinants of tobacco control success in sub-Saharan Africa.

For more information on the Research for International Tobacco Control, please visit: http://www.idrc.ca/en/ev-83280-201-1-DO_TOPIC.html.

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Swedish International Development Cooperation Agency

The Swedish International Development Cooperation Agency (SIDA) is a governmental agency whose mission is to create opportunities for change and development that would reduce injustices and poverty among populations worldwide. SIDA has funded and partnered with Swedish organizations, companies, government agencies, societies and experts to carry out programs in at least 50 countries throughout Africa, Asia, Latin America and Eastern Europe. The agency makes more than 5,000 contributions yearly towards: education, health, private sector development, housing, rule of law, research, infrastructure and trade.

To learn more about SIDA and the work it does, please visit:
http://www.sida.se/sida/jsp/sida.jsp?d=121&language=en_US

Chapter 4

International Tobacco Control Organizations

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Campaign for Tobacco-Free Kids International Resource Center

The International Resource Center at the Campaign for Tobacco-Free Kids places its focus on low- and middle-income countries around the world and provides support to governments and non-governmental organizations within these countries in promoting and implementing public policies that are proven to reduce tobacco use.

Information and organizational resources for the following 15 countries with high burdens of tobacco use can be accessed by clicking on the links below:

Bangladesh	Brazil	China
Egypt	India	Indonesia
Mexico	Pakistan	Philippines
Poland	Russian Federation	Thailand
Turkey	Ukraine	Vietnam

To visit the International Resource Center homepage, go to:
http://tobaccofreecenter.org/

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International Tobacco Control Organizations*

The following is a grouping of tobacco control organizations from around the world. This is not an exhaustive list and should not be treated as such. We encourage you to expand this list as you see fit.

Name	**Action on Smoking and Health (ASH)**
Country	United Kingdom
URL	http://www.ash.org.uk/
Overview	The Action on Smoking and Health (ASH) is a public health charity that campaigns to eliminate the harm caused by tobacco. It works towards achieving the following six priorities: Advocacy & Policy Development, Information & Research, Networking & Enabling Networking, Governance, Resources & Sustainability, and Image & Communication. ASH receives funding from the British Heart Foundation and Cancer Research UK.

Name	**American Cancer Society (ACS)**
Country	United States
URL	http://www.cancer.org/docroot/home/index.asp
Overview	The American Cancer Society (ACS) is a nation-wide community-based voluntary health organization, but is emerging as a global leader in the fight against cancer. The society fosters in-country expertise in effective cancer control practices by providing advocacy, capacity building, and information.

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Name	**Asia-Pacific Association for the Control of Tobacco**
Country	Taiwan
URL	http://webbbs.mingdao.edu.tw/~foo/www6/h3.htm
Overview	The Asia Pacific Association for the Control of Tobacco was created in 1989 with the endorsement of the John Tung Foundation. This organization has joined forces with other Asia Pacific anti-tobacco groups to fight the international tobacco giants, and raise the standard of tobacco control and prevention in Taiwan.

Name	**Canadian Cancer Society**
Country	Canada
URL	http://www.cancer.ca/?sc_lang=en
Overview	The Canadian Cancer Society is a donor funded, national community-based organization of volunteers dedicated to cancer control. The organization focuses on five areas: research, advocacy, prevention, information, and support.

Name	**Healis Sekhsaria Institute for Public Health**
Country	India
URL	http://www.healis.org/healis/index.asp
Overview	The Healis Sekhsaria Institute for Public Health conducts research on tobacco use and its health effects specific to India's unique social and cultural settings. The organization helps assess the health situation and assists in guiding health services and programs. It works in collaboration with many other organizations worldwide leading to publications in peer-reviewed journals and key policy level actions.

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Name	**Institute for Global Tobacco Control (IGTC)**
Country	United States
URL	http://www.jhsph.edu/global_tobacco/
Overview	The Institute for Global Tobacco Control (IGTC) works through research, education and policy development to prevent tobacco related death and disease. It currently has a broad range of research projects in 40 countries around the world, as well as programs in education and training. The Institute was named a World Health Organization Collaborating Centre on Tobacco Control Surveillance and Evaluation in 2004.

Name	**International Development Research Centre (IDRC) – Research for International Tobacco Control (RITC)**
Country	Canada
URL	http://www.idrc.ca/en/ev-83280-201-1-DO_TOPIC.html
Overview	The Research for International Tobacco Control (RITC) is a program within IDRC's Social and Economic Policy program area. It funds multidisciplinary tobacco control research projects in developing countries that will lead to the implementation of effective tobacco control policies and programs.

Name	**International Network of Women Against Tobacco (INWAT)**
Country	Canada
URL	http://www.inwat.org/
Overview	The International Network of Women Against Tobacco (INWAT) is a global network of tobacco control specialists dedicated to addressing issues of tobacco use among women and young girls. There are over 1600 members in this network from 100 different countries.

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Name	**International Non-Governmental Coalition Against Tobacco**
Country	France
URL	http://www.ingcat.org/
Overview	The International Non-Governmental Coalition Against Tobacco was launched by members from three organizations: The International Union Against Tobacco and Lung Disease (The Union), the International Union Against Cancer (UICC), and the World Heart Federation (WHF). INGCAT's main purpose is to help its members take effective action on tobacco, principally within the context of the Framework Convention on Tobacco Control (FCTC), by providing information, help to locate resources, technical assistance, training, networking, and coordinating international campaigns.

Name	**International Union Against Cancer (UICC)**
Country	Switzerland
URL	http://www.uicc.ch/
Overview	The International Union Against Cancer (UICC) is a non-governmental organization comprised of cancer control organizations, professionals and volunteers dedicated to the global control of cancer. The union works in four areas: cancer prevention and control, tobacco control, knowledge transfer, and capacity building and supportive care.

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Name	**National Council Against Smoking (NCAS)**
Country	South Africa
URL	http://www.againstsmoking.org/
Overview	The National Council Against Smoking focuses on prevention, protection and cessation. To carry out its mission, the Council makes every effort to promote education, legislation, treatment and research.

Name	**Society for Research on Nicotine and Tobacco (SRNT)**
Country	United States
URL	http://www.srnt.org/
Overview	The Society for Research on Nicotine and Tobacco (SRNT) has over 1000 members from 20 different countries. This organization has three main aims: to sponsor scientific meetings and publications promoting the exchange of information on the biological, behavioral, social, and economic effects of nicotine; to encourage scientific research on the prevention of tobacco use; and provide the means for policymakers and public agencies to obtain expert advice on issues pertaining to tobacco use.

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Name	**World Health Organization (WHO)**
Country	Switzerland
URL	http://www.who.int/tobacco/en/
Overview	The World Health Organization (WHO) is the authority for health within the United Nations system. WHO provides leadership on global health matters, like tobacco control. It also shapes the agenda on health research, sets norms and standards, communicates evidence-based policy options, provides technical assistance to countries and monitors and measures health trends. One of the organization's programs is the Tobacco Free Initiative (TFI) which focuses on the global tobacco epidemic.

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Chapter 5

Publications

VOLUME 24 · NUMBER 14 · MAY 10 2006

JOURNAL OF CLINICAL ONCOLOGY

REVIEW ARTICLE

Between and Within: International Perspectives on Cancer and Health Disparities

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Submitted December 8, 2005; accepted March 1, 2006.

Supported by in part by National Center on Minority Health and Health Disparities Project EXPORT Grant No. P60 MD 000503 and Centers for Disease Control and Prevention Grant No. U48 CCU619515.

Authors' disclosures of potential conflicts of interest and author contributions are found at the end of this article.

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0732-183X/06/2414-2204/$20.00

DOI: 10.1200/JCO.2005.05.1813

ABSTRACT

The purpose of this article is to compare reasons for cancer health disparities in developing and developed countries. By 2010, approximately 60% of new cancer cases will occur in the developing world, higher than rates developed countries. However, disparities exist not only between countries but also within countries. Cancer epidemiology in developing countries is paradoxical: Increased incidence is partially due to increased development resulting in longer life expectancy and unhealthy lifestyle behaviors. Reduced mortality from infectious diseases results in relatively greater mortality from chronic diseases. However, infectious diseases are also risk factors for the leading causes of cancer mortality in these countries. While health disparities in developing versus developed countries are quantitatively worlds apart, they are qualitatively rather similar. They share common causes, such as environmental pollution, the need for social justice, large gaps between the rich and the poor, lack of access to cancer resources, and health services that are available to some but not to all. While industrialization and urbanization elevate a country's economic base while contributing to cancer incidence and mortality. Strategies to reduce international cancer disparities include country- and regional-level interventions, utilizing nongovernmental organizations, and developing long-term inter-institutional partnerships. Although economic aid is undoubtedly necessary, it is not sufficient to control cancer in the developing world. To address these problems, it will be necessary to focus attention on what can be done locally—within countries, not only between countries.

J Clin Oncol 24:2204-2208. © 2006 by American Society of Clinical Oncology

INTRODUCTION

> "There are only two families in the world, as my grandmother used to say: the haves and the have-nots."
>
> Sancho Panza in *Don Quixote de la Mancha*.[1]

This article compares sources of cancer health disparities in developing and developed countries. As used herein, developed or high-income countries are those of North America and Western and Northern Europe, Australia, New Zealand, and Japan. With a few exceptions, developing or low-income countries comprise the remainder, primarily nations in Africa, Asia, Central America, and South America.[2,3] Examples from developed countries come primarily from English-speaking nations. However, the authors will provide neither in-depth analysis of specific countries nor targeted solutions to international cancer health disparities. Rather, we believe that a better understanding of the general causes of international cancer inequities will facilitate efforts to reduce them.

INTERNATIONAL PERSPECTIVES

We often think of cancer as a disease of the developed world, where it is the second leading cause of death. However, cancer incidence in developing countries is rising, and survival outcomes vary dramatically around the globe. Approximately 50% of all cancer mortality occurs in developing countries, and approximately 60% to 70% of new cancer cases will occur in the developing world by 2020. Annually, nearly 7 million people die of cancer worldwide, growing to more than 10 million by 2020. Cancer kills approximately 13% of those who die and claims twice as many lives as AIDS. As living conditions around the world improve and the world population gradually ages, worldwide incidence of new cancer cases will increase 50% to 100%, from 10 million in 2000 to 15 to 20 million in 2020.[4]

When looking at health disparities between developed and developing countries, it is tempting to think in terms of "our society" in contrast to "their society." However, it is important to remember that disparities exist not only between countries but also within countries. There are differences even among developed countries. One study comparing Australia, Canada, Germany, New Zealand, the United Kingdom, and the United States found that the United States "stood out for high error rates, inefficient care coordination, and high out-of-pocket costs that serve as barriers to access."[5] These and other differences exist not only between countries

but also between cities, groups, hospitals, and even health care institutions within the same city.[6-8]

CANCER IN DEVELOPED COUNTRIES

In general, lower socioeconomic status (SES) groups have less access to care and are more unhealthy than higher SES groups. Consequently, the burden of cancer is not shared equally by all population segments. The common overlap of minority status with lower SES results in minorities' more frequently developing and dying as a result of cancer. However, even controlling for SES, minority and medically underserved groups tend to come into the health care system with late-stage disease, and consequently suffer higher morbidity and mortality than others. Although US cancer rates have gradually decreased since the National Cancer Act was passed in 1971, the "war on cancer" is clearly not over. Constant pressures of competing health priorities and limited resources make it difficult to address cancer and other health disparities.[9-12] Therefore, the National Cancer Institute (NCI) Center to Reduce Cancer Health Disparities (National Institutes of Health, Bethesda, MD), American Cancer Society (Atlanta, GA), Centers for Disease Control and Prevention (CDC; Atlanta, GA), Intercultural Cancer Council (Houston, TX), and many other agencies, institutions, and organizations have begun partnering to better address the unequal burden of cancer.[13-17]

New immigrants face additional problems as a result of linguistic, cultural, and economic barriers. However, some groups experience an interesting negative effect associated with the acculturation process itself, the so-called "Hispanic paradox."[18] Research has shown that Mexican immigrants of lower SES have comparable health to non-Hispanic whites of a higher SES. The health status of Mexican immigrants actually declines as they become more acculturated and adopt negative health behaviors such as smoking, alcohol consumption, and illicit drug use. Conversely, strong family ties and social behaviors seem to support better health status in less-acculturated immigrants. This observation requires more research, as it is not clear to what degree the finding may be a result of under-reporting and misclassification on birth and death certificates, or whether it is common across different immigrant groups.

Health disparities have been documented since the early 19th century in the United Kingdom, which has a large and growing black and minority ethnic population. Research shows that disparities similar to those in the United States exist and cannot be ascribed only to socioeconomic differences. Other variables include cultural differences, language and literacy, "newness" or user ignorance of the system, differential health care needs for different communities, location and mobility, and staff training needs in the area of cultural competence.[19] Therefore, the National Health Service (London, United Kingdom) and cancer patient support organizations now include in their missions outreach to ethnic and racial populations. These outreach efforts attempt to provide current information, advice, and support to people from black and minority communities representing as many as 12 different languages of origin.[20,21]

Half a world away, Australia has also identified the need to address striking health inequities between different population groups, including both indigenous and immigrant communities. Indigenous peoples have poorer overall cancer incidence and survival rates compared with nonindigenous Australians. Therefore, the Cancer Council of Australia (Camperdown, Australia) and the National Cancer Control Initiative (Carlton, Australia) have developed a national strategy for health professionals to address the needs of special populations. These efforts target Aboriginal peoples, those living in isolated areas, and other groups having disproportionate difficulty accessing care.[22,23] Assessing the problem is complex because many still have poor health even though health and nutrition in general have improved during the last 40 years. Data suggest that lifestyle-related diseases and other illnesses remain more common in the Aboriginal population than in the general Australian population. However, there is evidence that this is so only for a substantial minority of this minority community. Even with effective targeted efforts, the expectation is that it will take at least 10 years to achieve statistically significant results, and a generation to produce major change.[24]

Nearby New Zealand has also recognized the problem of health disparities among its residents. A 2005 report noted that the health gap between Maori and European New Zealanders is wider than that between American Indians and the majority white population in the United States.[25] From the mid-1980s until the mid-1990s, the mortality gap between the Maori/Pacific and non-Maori/non-Pacific populations grew because non-Maori/non-Pacific mortality rates were declining while Maori and Pacific mortality rates did not change.[26] The Health Research Council of New Zealand (Aukland, New Zealand) has consequently funded the Health Inequalities Research Programme to help reduce these inequalities, with specific attention to cancer.[27]

CANCER IN DEVELOPING COUNTRIES

Although the overall burden of cancer may be generally decreasing in developed countries, it is a growing problem in developing countries. Increased cancer incidence and prevalence are partially a result of the benefits of increased development (ie, longer life expectancy, with age being the primary risk factor for cancer) coupled with lifestyle behaviors such as smoking, unhealthy diet, and sedentary inactivity. Developed nations export lifestyles to developing countries, just as they do products. Consequently, those whose risk is increased the most are those who can least afford access to medical care.[28-31] Low-income countries understandably seek the benefits of industrialization, but high-income countries are more and more aware that development carries a hefty price in terms of both health and health disparities. Incidence of so-called Western cancers will increase in developing countries as improved immunization, sanitation, and food storage gradually bring infection under greater control (Fig 1). Similarly, as more people adopt industrialized lifestyles, they will also begin to suffer more from the cancers that are common in the developed countries. Meanwhile, the relative incidence of these cancers will decrease in some developed countries where tobacco use is declining and adoption of healthier lifestyles is rising. Thus, a growing percentage of cancer mortality will result from lung, breast, colon, and prostate cancers, which are not currently the most common cancers in low income nations.

In relative terms, reduced mortality from infectious diseases contributes to greater mortality from cancer and other chronic diseases. However, in the developing world, infectious diseases also contribute to cancer incidence. Unlike in developed countries, the leading causes

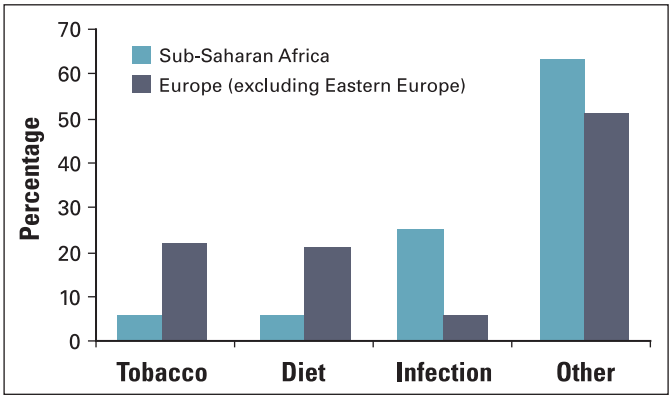

Fig 1. Percentages of cancer mortality resulting from major causes in Sub-Saharan Africa compared with Europe.[34]

of cancer mortality in the developing world are stomach, liver, and cervical cancers—all of which have been linked with infection.[32-34]

Low- and middle-income countries are faced with the difficult choices of where to allocate scarce financial resources along the continuum of cancer control—prevention, screening, early detection, treatment, and pain and palliative care.[35] These countries have less than 5% of the resources required for adequate cancer control, and more than 80% of their cancer patients will be incurable at the time of diagnosis. Long-term cancer survival rates are less than 20% in developing countries, 20% to 30% worldwide, and approximately 50% to 60% in high-income countries.[36] According to the International Atomic Energy Agency (Vienna, Austria), 85% of the world's population lives in developing countries, but is served by only approximately 30% of the world's radiotherapy facilities. Conversely, the developing countries, with 15% of the world's population, have 70% of these facilities. Approximately 15 countries in Africa, as well as several in Asia, do not have even one radiation therapy machine.[37]

ENVIRONMENTAL FACTORS: COMMONALITIES

Often, the disparities between low- and high-income nations share common causes. These include environmental factors, as well as the need for social justice, lack of access to cancer resources, and health services that are available to some but not to all. Equally often, as noted herein, disparities within countries may be greater than those between countries. Without oversimplifying conditions, cancer resources and access to care in developing versus developed countries may be worlds apart quantitatively, but qualitatively rather similar.[38] In 2001, 35% of worldwide cancer mortality was attributable to nine modifiable risk factors: alcohol, smoking, low fruit and vegetable intake, overweight and obesity, physical inactivity, urban air pollution, unsafe sex, contaminated injections in health care settings, and indoor smoke from household uses such as cooking or heating the home.[3] All but the last two are common in both high- and low-income countries.

The environment is invisible and encompassing, and it controls our lives in powerful ways, of which we are usually unaware—unaware, that is, until we are deprived of those influences (eg, fish out of water or humans breathing polluted air).[39] The environment has three components: the natural, the social, and the built. Industrialization, generally thought to be synonymous with development, can actually increase health risk because of resulting changes in the environment. In the natural environment, the result is pollution of the air, water, and soil. In the social environment, examples include increased sedentary lifestyles, the breakup of traditional family and support structures, and environmental injustice through placing the most polluting elements of the economy in the poorest neighborhoods and communities. In the built environment, construction materials, working conditions, and occupational exposure to carcinogens contribute to these problems. Population migration to more and more crowded urban areas increases with development in low-income countries. Urban concentration of population influences food availability, distribution, and safety.[6] As developing countries increase wealth, changes in modes of transportation, along with urbanization, contribute to decreasing health status. Walking gives way to riding bicycles, which gives way to riding mopeds and motor scooters, eventually culminating in driving automobiles with fewer and fewer occupants. This process contributes to a less active lifestyle while increasing exposure to pollutants.[40]

Despite these similarities and growing globalization, the gap between the poor and the rich nations may well continue to grow.[24,25] The developed countries have made extraordinary gains in technology and knowledge (eg, sequencing of the human genome, computers determining risk via gene expression, structural modeling, screening technologies, access to and understanding of information). However, these very gains serve to widen disparities that result from economics.[22] On the one hand, recent advances in telecommunication, transportation, and the global economy have facilitated information exchange and collaboration to improve health internationally. Teleconferencing and telemedicine allow direct aid from medical centers in developed countries to clinics in the poorest nations on the planet.[41] On the other hand, although economic growth has improved health care and reduced poverty in some countries, the economic gap between developing and developed countries also results in a "brain drain" of poor countries' best and brightest health professionals.[42,43]

MISCONCEPTIONS AND DILEMMAS

For 2005, 60% of all mortality worldwide were estimated to be due to chronic diseases, with cancer causing more deaths than HIV/AIDS, tuberculosis, and malaria combined (Fig 2). The WHO[44] notes that the neglect of prevention and control of cancer and other chronic diseases until now has been due, at least in part, to several misconceptions, including the beliefs that such diseases: (1) mainly affect high income countries and developed countries—in fact, low and middle

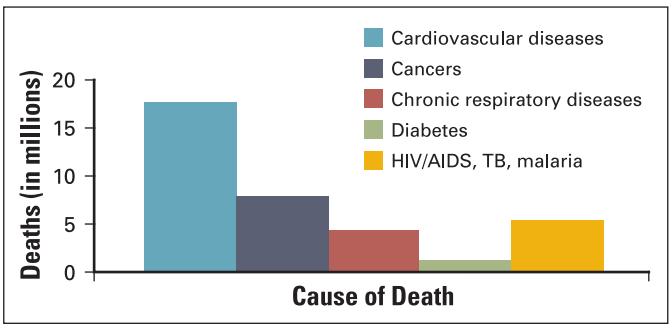

Fig 2. Projected global deaths for 2005 by cause (in millions). TB, tuberculosis.

income countries contribute 70% of related mortality worldwide; (2) require prevention and control efforts that are too expensive—in reality, many interventions are cost-effective when tailored to the socioeconomic circumstances of the country[45]; (3) cannot be prevented—more than 40% of cancers can in fact be prevented; (4) are the result of unhealthy lifestyles—healthy lifestyle choices are possible only when healthful options (eg, appropriate food, lodging, and health care) are available, and many of these are not yet available in developing countries; (5) affect primarily men—in reality, men and women are about equally affected, although they are impacted differently by different cancers; (6) mainly affect old people—it was once assumed that people in developing countries did not live long enough to develop cancer, but in actuality, 25% of deaths worldwide result from chronic disease occurring before age 60 years, and almost 50% before age 70 years; and (7) mainly affect rich people—but in the least developed countries, the poor are more likely to die as a result of cancer and other chronic diseases than are the wealthy.

Low- and moderate-income countries should control infectious diseases before chronic. As noted herein, chronic diseases are rising rapidly in developing countries as a result of risk factors related to development.

STRATEGIES TO REDUCE THE UNEQUAL BURDEN OF CANCER

The term "health disparity" is rarely used in the international literature. However, one author referred to the "yawning gap between poor and rich countries" in regard to the burgeoning cancer problem worldwide.[24] Eliminating cancer disparities will come not only from increased funding but also from better-targeted and -tailored prevention, and early detection, treatment, and care. Within the last decade, various agencies and organizations have called for different strategies to address the predicted global increase in cancer incidence.

The WHO defines cancer control as "the public health actions designed to reduce the incidence and mortality of cancer and improve quality of life of patients, through the systematic implementation of evidence-based strategies for prevention, early detection, diagnosis, treatment, and palliative care."[33] The group proposes both countrywide and regional cancer control strategies, developing nationwide databases to establish baseline data, and creating an international system to support such efforts. As described herein, the availability of such programs varies considerably between countries. Cancer is often a low public health priority for ministries of health, such that treatment approaches take precedence over prevention, early detection, and palliative care. A result is that most cancer patients in developing countries are diagnosed with late-stage disease.

Taking a different approach, the American Cancer Society proposes an initial focus on community-based cancer control initiatives. This approach utilizes nongovernmental organizations to develop effective and sustainable cancer control efforts. The strategy includes members of targeted communities in the design of interventions, addresses health issues relevant to the respective communities, and attempts to address potential sources of racial and ethnic disparities at both patient and system levels.[3,7,8,23]

The CDC Global Health Strategy has defined its goals in terms of public health surveillance and response, public health infrastructure and capacity building, disease prevention and control, applied research for effective health policies, and exchange of information and lessons learned. The CDC plans to develop partnerships with other institutions and pursuing long-term relationships with targeted countries to enhance productivity and sustain collaborations. Anticipated outcomes include improved country surveillance systems to identify critical public health problems, expanded research to generate new knowledge for application to those problems, identification of more partners in global collaboration, and health promotion programs seeking governmental policies to prevent tobacco use in youth.[46]

CONCLUSION

Access to cancer care resources is at the heart of cancer control in both developed and developing countries. In theory, solutions to cancer health disparities are deceptively straightforward: improved education, communication, and economics that provide better access to care. However, if theory were reality, the problem would evaporate. Cancer and other chronic diseases will increase extraordinarily over the next two decades. There is no cure yet for many—if not most—cancers, especially in the developing countries, where patients typically present with late-stage disease because of the lack of prevention, screening, and early detection. The most advanced technological therapeutics are not available in the poorest countries and, if available, they are beyond the socioeconomic reach of most citizens. Adoption of unhealthy behaviors such as smoking, certain dietary habits, and sedentary lifestyle will increase the cancer burden in countries that can least afford the costs of treatment. Therefore, prevention and control are the primary weapons of defense against cancer. Although industrialization and subsequent urbanization can elevate a country's development and economic base, they can also contribute to overall cancer incidence and mortality.

Making health care resources more equally available between the developed and the developing countries will continue to be critically important. However, true equalization of resources between countries will not happen in any foreseeable future. It has been noted that "the disparities hampering progress towards the [United Nations Millennium Development Goals] are systemic. They reflect complex hierarchies of advantage and disadvantage that are transmitted across generations—and they reflect public policy choices."[1] Therefore, although economic and material aid are undoubtedly necessary to alleviate the unequal burden of cancer in poor countries, they are not sufficient by themselves to control cancer in the developing world. To address these problems globally, it will be necessary to focus attention on what can be done locally—within countries, not only between countries.

REFERENCES

1. Inequality and human development, in Watkins K: Human Development Report 2005: International cooperation at a crossroads—Aid, trade and security in an unequal world. New York, NY, United Nations Development Programme, 2005

2. Parkin DM, Bray FI, Devesa SS: Cancer burden in the year 2000: The global picture. Eur J Cancer 37:S4-S66, 2001 (suppl)

3. Danaei G, Vander Hoorn S, Lopez AD, et al: Causes of cancer in the world: Comparative risk assessment of nine behavioural and environmental risk factors. Lancet, 366:1784-1793, 2005

4. International Union Against Cancer: Introducing UIUC Global Cancer Control. Geneva, Switzerland, International Union Against Cancer, 2005

5. Schoen C, Osborn R, Huynh PT, et al: Taking the pulse of health care systems: Experiences of

patients with health problems in six countries. Health Aff (Millwood) November 28, 2005 [epub ahead of print]

6. Ramirez AG, McAlister A, Villarreal R, et al: Prevention and control in diverse Hispanic populations: A national initiative for research and action. Cancer 83:S1825-S1829, 1998 (suppl)

7. Pérez-Stable EJ, Ramirez AG, Villarreal R, et al: Cigarette smoking behavior among US Latino men and women from different countries of origin. Am J Public Health 91:1424-1430, 2001

8. Ramirez AG, Suarez L, Chalela P, et al: Cancer risk factors among men of diverse Hispanic or Latino origins. Prev Med 39:263-269, 2004

9. Edwards BK, Brown ML, Wingo PA, et al: Annual report to the nation on the status of cancer, 1975-2002, featuring population-based trends in cancer treatment. J Natl Cancer Inst 97:1407-1427, 2005

10. Haran C: Vince DeVita: The view from the top. Cancer World, May-June 2005, p 38-43

11. Haynes MA, Smedley BD The Unequal Burden of Cancer: An Assessment of NIH Research and Programs for Ethnic Minorities and the Medically Underserved. Washington, DC, National Academies Press, 1999

12. Smedley BD, Stith AY, Nelson AR Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care. Washington, DC, National Academies Press, 2003

13. National Cancer Institute Center to Reduce Cancer Health Disparities. http://crchd.nci.nih.gov/introduction/

14. American Cancer Society: Research proposals directed at poor and underserved populations. http://www.cancer.org/docroot/RES/content/RES_5_2x_Targeted_Grants_for_Research_Directed_at_Poor_and_Underserved_Populations.asp

15. Centers for Disease Control and Prevention: Cancerhealthdisparities.http://www.cdc.gov/cancer/minorityawareness/overview.htm

16. Centers for Disease Control and Prevention: Eliminating racial & ethnic health disparities. http://www.cdc.gov/omh/AboutUs/disparities.htm

17. Intercultural Cancer Council. http://www.iccnetwork.org/

18. Hajat A, Lucas J, Kington R: Health outcomes among Hispanic subgroups: United States, 1992-95. Hyattsville, MD, National Center for Health Statistics, 2000

19. Szczepura A: Access to health care for ethnic minority populations. Postgrad Med J 81:141-147, 2005

20. CancerBACUP: Annual Review 2003-4. http://www.cancerbacup.org.uk/Aboutus/Annualreports/2003-2004/annual_review04.pdf

21. Peters J, Jackson M: Accessibility and use of touchscreens by black and ethnic minority groups in the three cities project. Ethn Health 10:199-211, 2005

22. Dugdale A, Watlemaro I: Aboriginal health: A sick minority skews statistics. Aust J Rural Health 9:111-115, 2001

23. Clinical Oncological Society of Australia, Cancer Council of Australia, National Cancer Control Initiative 2002: Optimising Cancer Care in Australia. http://www.ncci.org.au/projects/care/optimise_c_care.htm

24. Cancer Council of Australia: Cancer control in indigenous communities: Ways forward. http://www.cancer.org.au/content.cfm?randid=358285

25. Bramley D, Hebert P, Tuzzio L, et al: Disparities in indigenous health: A cross-country comparison between New Zealand and the United States. Am J Public Health 95:844-850, 2005

26. Blakely T, Ajwani S, Robson B, et al: Decades of disparity: Widening ethnic mortality gaps from 1980 to 1999. N Z Med J 117:U995, 2004

27. University of Otago: Research into health inequalities gains HRC programme funding. http://www.otago.ac.nz/news/news/2005/25d-05-05_press_release.html

28. Magrath I, Litvak J: Cancer in developing countries: Opportunity and challenge. J Natl Cancer Inst 85:862-874, 1993

29. Sikora K: Developing a global strategy for cancer. Eur J Cancer 35:24-31, 1999

30. Sikora K: Developing a global strategy for cancer. Eur J Cancer 35:1870-1878, 1999

31. World Cancer Research Fund, American Institute for Cancer Research: Food, Nutrition, and the Prevention of Cancer: A Global Perspective. Washington, DC, American Institute for Cancer Research, 1997, pp 549-557

32. American Cancer Society: The case for global action: The global cancer burden. http://www.cancer.org/docroot/AA/content/AA_2_5_1x_Case_for_Global_Action.asp?sitearea=AA

33. Jones SB: Cancer in the developing world: A call to action. BMJ 319:505-508, 1999

34. World Health Organization, International Union Against Cancer: Global Action Against Cancer. http://www.who.int/cancer/media/en/GlobalActionCancerEnglfull.pdf

35. Institute of Medicine Board on Global Health: Cancer Control in Low- and Middle-Income Countries. Washington, DC, Institute of Medicine, 2005

36. World Health Organization: Global Programming Note 2005-2007: Call for Resource Mobilisation and Engagement Opportunities. http://www.who.int/nmh/donorinfo/chronicdiseases/chp_intensifying_action_against_cancer.pdf

37. International Atomic Energy Agency. Millions of cancer victims in developing countries lack access to life-saving radiotherapy. http://www.iaea.org/NewsCenter/PressReleases/2003/prn200311.html

38. Yach D, Beaglehole R: Globalization of risks for chronic diseases demands global solutions. Perspectives on Global Development and Technology 2:1-21, 2004

39. McLuhan M, Fiore Q, Agel J: The Medium Is the Massage: An Inventory of Effects. San Francisco, CA, Hardwired, 1996

40. Hanna K, Coussens C (eds): Rebuilding the Unity of Health and the Environment: A New Vision of Environmental Health for the 21st Century. Washington, DC, National Academies Press, 2001

41. Ladd H: SATTELLIFE: Handhelds for Life—SATELLIFE's Experience in Asia and Africa. http://pda.healthnet.org/download/pdapaper1.pdf

42. Aiken LH, Buchan J, Sochalski J, et al: Trends in international nurse migration. Health Aff (Millwood) 23:69-77, 2004

43. Kaiser Family Foundation: Asia-Pacific region not on track to reach MDG targets, report says. http://www.kaisernetwork.org/daily_reports/rep_index.cfm?hint=4&DR_ID=32435

44. World Health Organization: Preventing chronic diseases: A vital investment. http://www.who.int/chp/chronic_disease_report/contents/en/index.html

45. Goldie SJ, Gaffikin L, Goldhaber-Fiebert JD, et al: Cost-effectiveness of cervical-cancer screening in five developing countries. N Engl J Med 353:2158-2168, 2005

46. Centers for Disease Control and Prevention: Working with partners to improve global health: A strategy for CDC and ATSDR. http://www.cdc.gov/ogh/

Authors' Disclosures of Potential Conflicts of Interest

The authors indicated no potential conflicts of interest.

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Eliminating Tobacco-Related Health Disparities: Directions for Future Research

Certain groups in the United States remain at high risk and suffer disproportionately from tobacco-related illness and death despite progress made in reducing tobacco use. To address gaps in research on tobacco-related disparities and develop a comprehensive agenda aimed at reducing such disparities, representatives from funding agencies, community-based organizations, and academic institutions convened at the National Conference on Tobacco and Health Disparities in 2002. Conference participants reviewed the current research, identified existing gaps, and prioritized scientific recommendations. Panel discussions were organized to address research areas affecting underserved and understudied populations. We report major research recommendations made by the conference participants in several scientific domains. These recommendations will ultimately help guide the field in reducing and eliminating tobacco-related disparities in the United States. (*Am J Public Health*. 2004;94:211–217)

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ACCORDING TO THE 2000 surgeon general's report *Reducing Tobacco Use,* eliminating health disparities related to tobacco use is a major public health challenge.[1] Members of racial/ethnic minority groups, individuals of low socioeconomic status, and other groups remain at high risk for tobacco use and suffer disproportionately from tobacco-related illness and death.[2–4] Health disparities exist within and between populations, and little is known about the causes of population differences observed in exposure and susceptibility to, and the consequences of, tobacco use, addiction, and related diseases.

Consequently, the evidence base for designing and implementing effective prevention and cessation interventions aimed at understudied and historically underserved populations in the United States is limited. Additional scientific investigation is needed to identify optimal entry points to address health inequities and to understand how to interrupt increasing disparities among certain populations.

The challenge of eliminating disparities requires concerted efforts by scientists, health professionals, policymakers, and others to address inequities in health as well as to amend the scientific research agenda. Such efforts will help to raise critical questions that will generate empirical data, identify action steps for research, and facilitate the development of strategic plans and interventions to address tobacco-related disparities in present and future initiatives. Few efforts to date have systematically addressed research initiatives for populations in which disparities are present or have identified key scientific domains upon which to base future inquiry.

The National Conference on Tobacco and Health Disparities (NCTHD), held in December 2002, represented the first scientific gathering to convene researchers and practitioners to review the current research, identify gaps, and develop a research agenda to eliminate tobacco-related health disparities. Collaborators who supported and developed the meeting content and agenda included representatives from the National Cancer Institute, the Centers for Disease Control and Prevention, the American Legacy Foundation, the Robert Wood Johnson Foundation, the Campaign for Tobacco-Free Kids, the American Cancer Society, the National Latino Council on Alcohol and Tobacco Prevention, and the National African American Tobacco Prevention Network, along with the planning committee members.

The NCTHD planning committee defined tobacco-related health disparities as follows: "differences in the patterns, prevention, and treatment of tobacco use; the risk, incidence, morbidity, mortality, and burden of tobacco-related illness that exist among specific population groups in the United States; and related differences in capacity and infrastructure, access to resources, and environmental tobacco smoke exposure." This definition was employed by conference participants from multiple disciplines to advance our understanding of tobacco-related disparities in 11 specific research domains[5]: epidemiology, surveillance, psychosocial, basic biology, harm reduction, marketing, policy, community and state, prevention of tobacco use, treatment of nicotine addiction, and research capacity and infrastructure (Table 1).

Research in these scientific domains can contribute enormously to our understanding of tobacco use, addiction, and related diseases; help to identify interventions to reduce tobacco use; and help to determine the resources and infrastructure needed to implement interventions (Figure 1). In this article, we outline the research recommendations developed by conference participants in each of the scientific domains.

RECOMMENDATIONS FOR RESEARCH

More than 100 research recommendations focusing on high-risk, underserved, and understudied populations were generated at the NCTHD. The major recommendations are reported in Table 2.

Epidemiology

Epidemiology is a critical scientific domain that can greatly influence our understanding of

TABLE 1—Definitions of Key Scientific Areas

Scientific Area	Definition
Epidemiology[a]	Assesses the relationships between tobacco use, behavioral factors, biological/genetic factors, the environment, and disease outcomes in populations
Surveillance[a]	Monitors and evaluates trends in tobacco use, tobacco-related risk factors, behaviors, and health services, and the influence of these trends on disease incidence, morbidity, mortality, and survival
Psychosocial research[a]	Identifies the behavioral, cultural, psychological, and social factors that influence tobacco use behaviors and studies to develop and test interventions aimed at changing tobacco-related behaviors
Basic biology[a]	Assesses the effects of tobacco and tobacco products on cell biology and cellular mechanisms of action, including the role of tobacco and tobacco by-products in the initiation and promotion of disease and the biological and health effects of exposure to tobacco
Harm reduction[b]	Minimizes the net damage to health associated with the use of tobacco products, constituents of tobacco products, and other substitutes for tobacco products
Marketing[c]	Examines audience segmentation, distribution patterns, brands, advertisements, promotion, and production of tobacco products
Policy[a]	Assesses the effects of worksite, community, state, and federal policies on tobacco initiation and use
Community and state[a]	Focuses on the impact and dissemination of single and multiple interventions on the prevention and cessation of tobacco use among large groups of people
Prevention of tobacco use[a]	Identifies and tests interventions designed to prevent the initiation of tobacco use and nicotine addiction
Treatment of nicotine addiction[a]	Identifies, tests, and effectively disseminates interventions to treat tobacco users addicted to nicotine
Research capacity and infrastructure[a]	Ensures the availability of necessary human, financial, and technological resources; systems for collaboration; and mechanisms for information dissemination feedback to provide a strong foundation upon which effective research can be designed and conducted, and public health initiatives can be developed and implemented

[a]Adapted from *Tobacco Research Implementation Plan: Priorities for Tobacco Research Beyond the Year 2000*.[5]
[b]K. Warner, unpublished data presented at Reducing Tobacco Harm Conference, May 10, 2001.
[c]Definition developed by conference planning committee.

tobacco use, addiction, and variations in disease. A 1998 report of the surgeon general, *Tobacco Use Among U.S. Racial/Ethnic Minority Groups,* concluded that few studies have examined the relationships between tobacco use and known health effects in racial/ethnic groups, and, furthermore, few extant databases contain sufficient information to conduct the types of analyses needed.[3]

This general assessment also applies to other underserved and understudied groups. For example, it was not until 1980 that causal relationships between tobacco use and tobacco-related diseases were established for women.[6] Cross-sectional and cohort studies of many populations are lacking or inadequately designed to answer key research questions. The Black Women's Health Study[7] is one of the few empirical investigations to elucidate the associations between risk factors and disease outcomes in understudied populations. The conference participants' recommendations in regard to addressing these gaps were to support the research needs of small populations, examine the sociological context of tobacco use, implement longitudinal studies, and explore the social context and factors associated with tobacco use and quitting behavior among burdened populations.

Surveillance

Surveillance research helps us monitor trends to identify disparities in use behaviors, diseases, and deaths associated with tobacco. Although many national and state surveillance systems monitor trends in tobacco use, most do not specifically focus on disparities and do not collect or report data on smaller population groups. For example, certain tobacco products such as Iq'mik, a form of chewing tobacco used by Alaska Natives, are not measured on national surveys (C. C. Renner, unpublished data presented at the NCTHD, December 12, 2002). Furthermore, national surveys do not regularly ask questions about sexual orientation, yet a recent review study of lesbian, gay, and bisexual populations indicates that their smoking prevalence rates may be as high as 50%,[8] suggesting that there is a need to include questions focusing on sexual orientation. Data on Asians are typically reported in an aggregate manner, masking high rates of smoking among Southeast Asian men (e.g., Vietnamese, Laotian, and Cambodian men)[9,10]; when sample sizes are small, it is not possible to conduct multilevel analyses. Such differences in smoking suggest that there are important intra-ethnic, nativity, and regional variations[3,9,11,12] that should be considered in developing interventions.

The recommendations from conference participants were to address the needs of small populations and communities, explore study designs and methodologies, determine the appropriateness of survey measures, and address the comprehensiveness of surveillance systems.

Psychosocial Research

Understanding tobacco use also requires knowledge of the context of specific cultural vari-

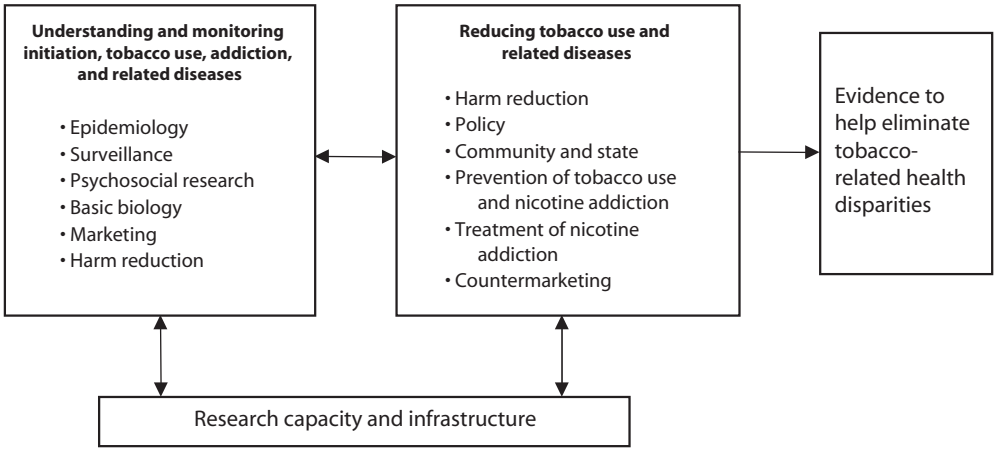

FIGURE 1—Scientific areas to be addressed in efforts to eliminate tobacco-related health disparities.

ables, norms, values, and community determinants and how these factors vary and interact within and between groups. Depending on context or other environmental attributes, variables such as acculturation may operate as risk factors in some populations and as protective factors in others. An understanding of how constructs are operationalized requires qualitative empirical strategies that provide context to the experiences and processes associated with tobacco use, as well as quantitative inquiry, which provides data on the relationships between multiple variables.

The field is just beginning to recognize the symbiotic nature between these types of investigations, which will enable us to comprehend the complexity of interactions among individual and community factors that influence tobacco use, cessation, and relapse. Also of note is researchers' tendency to operationalize psychosocial variables mainly in terms of the individual, with less focus on the community as the unit of analysis. The recommendations of the NCTHD participants were to identify risk and protective factors, develop appropriate measures for constructs, address systems issues, and examine the context of tobacco use behavior among racial/ethnic groups; immigrant populations; prison populations; the mentally ill; lesbian, gay, bisexual, and transgender populations; and faith-based communities.

Basic Biology

While observational and field research can help increase our understanding of tobacco use, basic biological research could provide additional information necessary to explain differences in tobacco-related disease outcomes among populations and the role of menthol in tobacco use and tobacco-related disease. Basic biological research may also explain the relationship between green tobacco sickness (an occupational illness resulting from transdermal nicotine exposure) and nicotine exposure among Latino tobacco farm workers.[13] Research in basic biology may require greater interdisciplinary collaborations to better integrate the science so that researchers can explore multiple biological pathways. The recommendations of the conference participants included the following: continue laboratory research, expand networks, and examine multiple interactions between risk factors.

Harm Reduction

While new to the tobacco research agenda, the study of harm reduction products will add to our understanding of tobacco product use and help to identify methods to reduce tobacco use and associated negative health consequences. Harm reduction, used as an intervention strategy in drug abuse research,[14–17] has become increasingly accepted in tobacco control research since the Institute of Medicine published *Clearing the Smoke*[18] in 2000, which explored this topic and provided tobacco control recommendations. The connection between harm reduction and health disparities is still evolving; however, given the availability of products with harm reduction claims, the use of strategies to reduce numbers of cigarettes smoked, and consumer practices in regard to brand preferences, it remains important to assess, monitor, and evaluate harm reduction's relevance to disparities research and impact on tobacco use. The recommendations of the NCTHD participants were to improve networking practices, investigate regulations associated with harm reduction claims, and conduct additional research.

Marketing

Marketing and countermarketing research can help increase our understanding of tobacco use and strategies to reduce rates of use among populations with disparities. The tobacco industry has a long history of targeting its marketing efforts toward populations such as women, youths, young adults, African Americans, Hispanics, Asian American/Pacific Islanders, Native Americans, and lesbian, gay, bisexual, and transgender populations.[3,4,19–24] It is still not clear how point-of-purchase advertising affects consumption patterns or how best to mobilize retailers in underserved communities to reject incentives and change promotional practices. Furthermore, additional evidence is needed to clarify the effects of countermarketing campaigns on understudied and underserved populations. The NCTHD participants recommended increased research on communications, countermarketing, and the tobacco industry itself (e.g., industry documents).

Policy

Public clean indoor air regulations, tobacco excise taxes, and home and car restrictions on smoking are strategies that protect nonsmokers and help reduce smoking prevalence rates in many states. Despite funding cuts to tobacco control budgets, many states, such as Delaware, California, Connecticut, Massachusetts, and New York, have been successful in passing clean indoor

TABLE 2—Summary of Major Recommendations in Each Scientific Domain

Domain	Recommendations
Epidemiology	Continue and expand research on prevalence and effects of environmental tobacco smoke in small/special populations; develop and implement longitudinal studies focusing on small, understudied, and underserved populations; examine and collect data on tobacco use and individual and community variables that may influence use and quitting behaviors; investigate mechanisms mediating the effects of low socioeconomic status on tobacco use behaviors; increase the study of socioeconomic status in smoking initiation and cessation; conduct more qualitative research on aspects of socioeconomic status, stress, locus of control, cultural affinity, and sexual orientation and their relationship to tobacco use; fund research to examine the effects of acculturation, stress, coping, racism, and discrimination on the etiology of smoking, trajectories, quitting, and disease onset; examine predictors of cessation among African Americans and other ethnic minority adult populations
Surveillance	Develop culturally and ethnically appropriate sampling, methods, and measures to obtain larger samples and to assess the smoking behavior of small populations at the national, regional, and local levels; include questions about the use of menthol, light, and other types of cigarettes in national and state surveys; provide further analysis of data collected from national surveys to obtain information on populations in which there are disparities and ethnic communities; expand national and state surveys to include questions relevant to tobacco use behaviors of understudied populations, including questions on smokeless tobacco and other methods of using tobacco
Psychosocial research	Convene grant review committees that recognize and value different perspectives and worldviews on research and health; use special peer review groups for grants that examine innovative qualitative and quantitative research needs of small populations; encourage ongoing funding for pilot projects for exploratory studies that develop innovative theories and methods from the perspective of the target population; promote an understanding that psychosocial issues are broader than the individual and encompass that individual's social context, experience in society, culture, history, and so forth; support research that examines the role of cultural beliefs, perceptions, and behaviors in tobacco use and exposure within multiple populations and channels; develop and assess reliable measures, protocols, methods, and models to assess cultural dimensions; investigate how social support networks can be used to help reduce tobacco use
Basic biological	Increase the use of current technologies to determine molecular changes resulting from tobacco exposure to provide clues on biological mechanistic pathways; explore the combination of constituents in tobacco (e.g., menthol) and the effects of differing levels (within products or brands) on addiction and subsequent health effects; conduct larger studies to assess whether observed differences in nicotine addiction and tobacco-related diseases are correlated with genetic variations in racially or ethnically classified social groups; systematically analyze the effects of interactions among tobacco constituents, genetic factors, and other environmental risks on health; create broader multidisciplinary teams to develop a comprehensive perspective on tobacco use, addiction, and health consequences
Harm reduction	Create a network of researchers to facilitate communications and collaboration regarding harm reduction studies and recommendations; investigate the impact of products with "harm-reducing" claims on cessation practices of underserved populations; assess patterns of use with ecological momentary assessment in special populations; expand Food and Drug Administration regulation of tobacco product content and claims
Marketing	Encourage the use of marketing research to develop communication strategies that address prevention, cessation interventions, and maintenance of quit behaviors; conduct countermarketing research using social marketing techniques and principles with specific application to populations in which there are disparities; develop funding for research on the tobacco industry, including industry documents, and integrate this research into tobacco control practice
Policy	Conduct research on different tobacco industry pricing practices in communities, especially low-income neighborhoods; study the impact of tax increases on consumption patterns, quitting, or reductions in numbers of cigarettes smoked in specific minority groups or communities in which there are disparities; conduct research on differential consequences of tobacco use policies in underserved neighborhoods; conduct research in underserved communities on the enforcement of existing tobacco policies (i.e., environmental tobacco smoke), voluntary policies, and the synergy between them; prioritize funding for community-based policy research to build the case for innovative tobacco control policy activities related to populations in which there are disparities; improve use of research, including basic biological research, in formulating policy; improve understanding of how tobacco control programs, tax increases, and smoking restriction policies together affect changes in tobacco use or quit rates among populations in which there are disparities; collect data on the economic impact of environmental tobacco smoke policies on restaurants, bars, the gaming industry, and so forth in minority communities; conduct research on how to obtain support for environmental tobacco smoke policies in communities in which there are disparities; disseminate information about research findings back to communities
Community and state	Conduct community assessment or capacity studies to determine optimal strategies for building, strengthening, and developing tobacco control initiatives; provide sustained funding for long-term community research; document state-level best practices for tobacco policy and control in communities in which there are disparities; build a network for state and community researchers to facilitate effective communication and collaboration; encourage research on the sustainability and effectiveness of anti-tobacco coalitions in addressing tobacco issues in communities of color; develop a system of accountability that ensures funds are spent and priorities are set in accordance with data regarding disparities
Prevention of tobacco use	Create a repository of tobacco control resources developed for populations in which there are disparities; ensure that evidence-based programs are culturally appropriate and effective; develop surveys and intervention materials in the native (non-English) language of survey respondents, intervention participants, communities, and so forth; fund randomized controlled trials of comprehensive community-based adolescent prevention programs; fund more studies to develop interventions to reduce children's exposure to environmental tobacco smoke in homes

Continued

TABLE 2—*Continued*

Domain	Recommendations
Treatment of nicotine addiction	Develop and evaluate interventions to promote delivery and use of treatment for nicotine addiction in various groups, including substance abusers and the mentally ill; conduct research on how evidence-based treatment programs are adopted, implemented, and maintained in health care systems, schools, and so forth; develop novel therapies and innovative ways of treating nicotine dependence; examine treatment in other fields for relevance to the treatment of nicotine dependence; examine barriers to use of behavioral and pharmacological treatments at the individual, organizational, and community levels and assess ways of improving access to treatments
Research capacity and infrastructure	Expand and fund mentorship programs, such as minority supplements, with mentor stipends to increase opportunities for training in community-based intervention research; fund the training and mentoring of minority researchers; train researchers in effective community research and skills related to building and maintaining relationships, negotiation, and group facilitation; develop strategies to facilitate change in the culture of research in academic and federal settings to make research more receptive to diverse perspectives; develop community-based research processes based on establishing and maintaining long-term relationships within the community; disseminate scientific data in a usable manner for academic research, community researchers, community members, and the general public; develop and explore funding mechanisms that incorporate methods to increase the likelihood that tobacco prevention efforts are culturally relevant and evidence based; develop funding mechanisms that allow for co-principal investigator structure between researchers and community organizations and develop mechanisms to ensure equity in terms of resources; incorporate principles of community participatory research and outcomes into studies and databases; develop funding mechanisms to promote collaborations between investigators at minority-serving institutions and investigators at larger research institutions

air ordinances.[25,26] Twenty-one states raised excise taxes in 2002, and past research suggests that increases in excise taxes decrease consumption rates among the general population.[27–29] However, little research has been conducted to assess the impact of policy interventions on tobacco use, cessation, and relapse behavior or to assess the economic impact of interventions such as taxes on low-income groups, blue-collar workers, farmers, service workers, and other understudied populations. Additional studies focusing on views and perceptions of tobacco control policies are also needed among populations with disparities.[30] NCTHD participants' recommendations focused on examining the effects of policies and perceptions of policies in underserved communities.

Community and State

Given that the states have received funds from the Master Settlement Agreement (MSA) to implement tobacco control initiatives, it is prudent to conduct community and state research to evaluate and assess what is being done and to determine the efficacy of community and state efforts. Despite monetary allocations from states, the lack of infrastructure and resources has affected communities' and states' ability to conduct and sustain educational programs, clinical services, media programs, and policy interventions in a variety of settings. It is unclear how effective MSA-funded programs have been in reaching communities or the extent of involvement of low-income, rural, and racial/ethnic communities in the development of these programs. Many of the conference participants' recommendations were not directed at developing new interventions, instead focusing on building networks, synthesizing research, and developing tools for conducting community- and state-level research to reach underserved and understudied populations.

Prevention of Tobacco Use

Tobacco use prevention research has primarily focused on young people, but for many populations, such as African Americans and Asian ethnic groups, targeted prevention programs may be necessary to prevent adult onset of tobacco use. However, little research is available to clarify the risk and protective factors along the age continuum or at different developmental stages, which would provide information regarding the ways in which disparities evolve in high-risk groups. Additional youth-focused studies are needed to help prevent exposure to environmental tobacco smoke in the home and to target groups of young people with multiple risks or problem behaviors. Additional work is also needed to help ensure the effectiveness of research prevention protocols, materials, and interventions aimed at populations in which disparities exist.[31] The recommendations of the NCTHD participants were to fund intervention research and synthesize existing evidence on prevention efforts.

Treatment of Nicotine Addiction

Additional epidemiological, surveillance, marketing, and psychosocial research may inform the treatment of nicotine addiction in populations exhibiting disparities. The limited knowledge of treating tobacco use, addiction, and related diseases provides little information on which to base effective intervention programs for underserved and understudied populations. Several recent reviews of cessation interventions for African Americans concluded that more studies are needed to identify successful smoking cessation interventions.[32,33] While some progress has been made in assessing treatment efficacy among populations in which disparities are present,[34] it is not clear how evidence-based treatments are adopted, implemented, and maintained in certain community systems (e.g., rural residents; the elderly; substance abusers; mentally ill individuals; lesbian, gay, bisexual, and transgender populations; low-income individuals; and racial/ethnic groups). The recommendations stemming from the NCTHD were to investigate therapies and interventions for treatment and to identify treatment barriers.

Research Capacity and Infrastructure

The scientific domains just described cannot be addressed without building the capacity and developing the infrastructure at multiple levels to conduct research involving understudied and underserved populations. In recent years, funding agencies have paid more attention to building infrastructure at multiple levels to support research and advocacy efforts, particularly in the areas of training, community-based participatory research, and dissemination. In addition, many national organizations, including the National Cancer Institute, the Robert Wood Johnson Foundation, the Centers for Disease Control and Prevention, and the American Legacy Foundation, have funded training programs to increase the pool of researchers from minority and underrepresented groups, cross train researchers in multiple disciplines, and work with community organizations to build capacity and conduct research.

Building research infrastructure allows researchers to raise incisive questions relevant to underserved and understudied populations, work more effectively with these populations, and develop community relationships to facilitate intervention and evaluation research. The conference participants recommended that support be provided in the areas of research training and mentoring, building collaborations between communities and academic institutions, and facilitating community-based research.

DISCUSSION

Embedded in the challenge to eliminate health disparities is the need to address tobacco use and related diseases among underserved and understudied populations. Addressing each of the 11 scientific domains described here will contribute to our understanding of tobacco use and related diseases and ultimately help to identify the entry points necessary to reduce tobacco-related disparities by enhancing our collective capability to intervene in burdened populations. Many of the recommendations stemming from the NCTHD focus heavily on expanding our understanding of tobacco use, addiction, and related diseases, thus reflecting the viewpoint that the tobacco control field is minimally prepared to develop effective interventions for populations exhibiting disparities. A more solid evidence base is needed, one that involves both quantitative and qualitative data upon which to develop interventions for populations in which disparities are present. How we move forward to build this evidence base represents an additional challenge to the field.

Several barriers, including limited financial resources, may affect the implementation of a research agenda on tobacco and health disparities. Many state organizations, as well as some private organizations, have eliminated or severely curtailed tobacco control programs and have diverted funds to other priority areas. If this trend continues, fewer resources will be available to reduce tobacco use and disease in underserved and understudied populations. No single organization can fully support these efforts; however, creative funding options and collaborative projects among researchers could help minimize the effects of recent changes.

The recent focus on disparity-based research in public health represents a major change in conceptualizing and understanding differences in health behavior, risks, and outcomes. The viewpoint that differences in health status are linked to economic, social, and political inequalities is a fundamental aspect of this perspective. Equally important is the proposition that research on disparities demands methodologically sound scientific innovations, professional dedication, and courage. These steps partly entail acknowledging the shortcomings and inequities involved in our scientific disciplines and rethinking what we decide to study. In addition, these recommendations are intended to encourage researchers, funders, and policymakers to discuss, debate, amend, and work to meet the challenges of this research agenda.

The process of collaboration between stakeholders such as researchers, practitioners, and community organizations with respect to research on disparities indicates the need for alternative viewpoints. In this regard, the work of Friere[35] and others has much to offer to the proposed research agenda on tobacco-related disparities, especially concerning community perceptions about research as "another structure of domination." Thus, the proposed recommendations for a national agenda to address disparities in tobacco use and related diseases require the research community to consider novel and practical approaches to the process of conducting research focusing on tobacco prevention and control. Disparity-based research will compel investigators not only to focus on quantitative and qualitative differences between groups but to examine the underlying reasons for these differences, adopt appropriate measures and standards of comparison, address the needs of smaller populations, conduct intragroup research, and translate research findings into policy.

Finally, moving forward with these recommendations requires that the research community and funding organizations allow for a certain degree of creativity and risk taking among investigators who use innovative research strategies to address tobacco-related health disparities. The trial-and-error process of using rigorous scientific standards, albeit protracted, has proven to be effective in advancing public health. In addition, the commitment to this challenge must be undergirded with the fortitude and patience to build upon conventional and long-established approaches to empirical investigations and develop new ideas and research strategies. ■

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This article was accepted October 6, 2003.

Contributors

P. Fagan summarized the recommendations, revised drafts of the article, and led the writing team. G. King and D. Lawrence contributed to the recommendation synthesis and editing of the article. S. Petrucci contributed to editing and to developing the recommendations section. R. Robinson, D. Banks, and S. Marable contributed to editing and to development of the recommendations and discussion sections. R. Grana assisted with the background research, synthesis of conference notes, and editing of the article.

Acknowledgments

The National Cancer Institute, the Centers for Disease Control and Prevention, the American Legacy Foundation, the Robert Wood Johnson Foundation, the Campaign for Tobacco-Free Kids, and the American Cancer Society provided funding for the National Conference on Tobacco and Health Disparities.

Note. This article represents the views of the authors and conference participants and does not reflect the respective policy positions of the funding agencies.

Human Participant Protection

No protocol approval was needed for this study.

References

1. *Reducing Tobacco Use.* Atlanta, Ga: Centers for Disease Control and Prevention; 2000.

2. *The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace.* Atlanta, Ga: Centers for Disease Control and Prevention; 1985.

3. *Tobacco Use Among U.S. Racial/Ethnic Minority Groups.* Atlanta, Ga: Centers for Disease Control and Prevention; 1998.

4. *Women and Smoking.* Atlanta, Ga: Centers for Disease Control and Prevention; 2001.

5. *Tobacco Research Implementation Plan: Priorities for Tobacco Research Beyond the Year 2000.* Bethesda, Md: National Cancer Institute; 1998. DHHS publication T644.

6. *The Health Consequences of Smoking for Women.* Atlanta, Ga: Centers for Disease Control and Prevention; 1980.

7. Russell C, Palmer JR, Adams-Campbell LL, Rosenberg L. Follow-up of a large cohort of black women. *Am J Epidemiol.* 2001;154:845–853.

8. Ryan H, Wortley PM, Easton A, Pederson L, Greenwood G. Smoking among lesbians, gays, and bisexuals: a review of the literature. *Am J Prev Med.* 2001;21:142–149.

9. Ma GX, Shive S, Tan Y, Toubbeh J. Prevalence and predictors of tobacco use among Asian Americans in the Delaware Valley region. *Am J Public Health.* 2002;92:1013–1020.

10. Moeschberger ML, Anderson J, Kuo YF, Chen MS, Wewers ME, Guthrie R. Multivariate profile of smoking in Southeast Asian men: a biochemically verified analysis. *Prev Med.* 1997;26:53–58.

11. King G, Polednak AP, Bendel R. Regional variation in smoking among African Americans. *Prev Med.* 1999;29:126–132.

12. King G, Polednak AP, Bendel R, Hovey D. Cigarette smoking among native and foreign-born African Americans. *Ann Epidemiol.* 1999;9:236–244.

13. Arcury TA, Quandt SA, Preisser JS, Bernert JT, Norton D, Wang J. High levels of transdermal nicotine exposure produce green tobacco sickness in Latino farmworkers. *Nicotine Tob Res.* 2003;5:315–321.

14. Berridge V. Histories of harm reduction: illicit drugs, tobacco, and nicotine. *Subst Use Misuse.* 1999;34:35–47.

15. Decorte T. Quality control by cocaine users: underdeveloped harm reduction strategies. *Eur Addict Res.* 2001;7:161–175.

16. Hilton BA, Thompson R, Moore-Dempsey L, Janzen RG. Harm reduction theories and strategies for control of human immunodeficiency virus: a review of the literature. *J Adv Nurs.* 2001;33:357–370.

17. Pollack HA. Ignoring 'downstream infection' in the evaluation of harm reduction interventions for injection drug users. *Eur J Epidemiol.* 2001;17:391–395.

18. Institute of Medicine. *Clearing the Smoke: Assessing the Science Base for Tobacco Harm Reduction.* Washington, DC: National Academy Press; 2001:23.

19. *Preventing Tobacco Use Among Young People.* Atlanta, Ga: Centers for Disease Control and Prevention; 1994.

20. Geobel K. Lesbians and gays face tobacco targeting. *Tob Control.* 1994;3:65–67.

21. Ling PM, Glantz SA. Why and how the tobacco industry sells cigarettes to young adults: evidence from industry documents. *Am J Public Health.* 2002;92:908–916.

22. Yerger VB, Malone RE. African American leadership groups: smoking with the enemy. *Tob Control.* 2002;11:336–345.

23. Robinson RG, Barry M, Bloch M, et al. Report of the Tobacco Policy Research Group on marketing and promotions targeted at African Americans, Latinos, and women. *Tob Control.* 1992;1:S24–S30.

24. Robinson RG, Pertschuk M, Sutton C. Smoking and African Americans: spotlighting the effects of smoking and tobacco promotion in the African American community. In: Samuels SE, Smith MD, eds. *Improving the Health of the Poor.* Menlo Park, Calif: Henry J. Kaiser Family Foundation; 1992.

25. Americans for Nonsmokers' Rights. Municipalities with 100% smokefree ordinances. Available at: http://www.no-smoke.org/100ordlisttabs.pdf. Accessed March 31, 2003.

26. Americans for Nonsmokers' Rights. Municipalities with 100% clean indoor air ordinances. Available at: http://www.no-smoke.org/100ordgraph.pdf. Accessed March 31, 2003.

27. Centers for Disease Control and Prevention. Cigarette smoking before and after an excise tax increase and an antismoking campaign—Massachusetts, 1990–1996. *MMWR Morb Mortal Wkly Rep.* 1996;45:966–970.

28. Centers for Disease Control and Prevention. Responses to increases in cigarette prices by race/ethnicity, income, and age groups—United States, 1976–1993. *MMWR Morb Mortal Wkly Rep.* 1998;47:605–609.

29. Zhang P, Husten C, Giovino G. Effect of the tobacco price support program on cigarette consumption in the United States: an updated model. *Am J Public Health.* 2000;90:746–750.

30. King G, Mallett RK, Kozlowski LT, Bendel RB. African Americans' attitudes toward cigarette excise taxes. *Am J Public Health.* 2003;93:828–834.

31. Robinson RG. Community development model for public health applications: overview of a model to eliminate population disparities. *J Health Educ Pract.* In press.

32. Lawrence D, Graber JE, Mills SL, Meissner HI, Warnecke R. Smoking cessation interventions in U.S. racial/ethnic minority populations: an assessment of the literature. *Prev Med.* 2003;36:204–216.

33. Peterson LL, Ahluwalia JS, Harris KJ, McGrady GA. Smoking cessation among African Americans: what we know and don't know about interventions and self quitting. *Prev Med.* 2000;31:23–38.

34. Boyd NR, Sutton C, Orleans CT, et al. Quit today! A targeted communications campaign to increase use of the Cancer Information Service by African American smokers. *Prev Med.* 1998;27:S50–S60.

35. Friere P. *Pedagogy of the Oppressed.* New York, NY: Continuum; 1970.

doi:10.1111/j.1360-0443.2007.01952.x

Identifying health disparities across the tobacco continuum

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ABSTRACT

Aims Few frameworks have addressed work-force diversity, inequities and inequalities as part of a comprehensive approach to eliminating tobacco-related health disparities. This paper summarizes the literature and describes the known disparities that exist along the tobacco disease continuum for minority racial and ethnic groups, those living in poverty, those with low education and blue-collar and service workers. The paper also discusses how work-force diversity, inequities in research practice and knowledge allocation and inequalities in access to and quality of health care are fundamental to addressing disparities in health. **Methods** We examined the available scientific literature and existing public health reports to identify disparities across the tobacco disease continuum by minority racial/ethnic group, poverty status, education level and occupation. **Findings** Results indicate that differences in risk indicators along the tobacco disease continuum do not explain fully tobacco-related cancer consequences among some minority racial/ethnic groups, particularly among the aggregate groups, blacks/African Americans and American Indians/Alaska Natives. The lack of within-race/ethnic group data and its interactions with socio-economic factors across the life-span contribute to the inconsistency we observe in the disease causal paradigm. **Conclusions** More comprehensive models are needed to understand the relationships among disparities, social context, diversity, inequalities and inequities. A systematic approach will also help researchers, practitioners, advocates and policy makers determine critical points for interventions, the types of studies and programs needed and integrative approaches needed to eliminate tobacco-related disparities.

Keywords Disparities, education, poverty, race/ethnicity, tobacco.

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All authors have no conflicts of interests.

INTRODUCTION

Tobacco is the leading cause of preventable death in the United States and contributes to increased incidence, morbidity and mortality from cancer, heart disease, stroke, complications of pregnancy and respiratory illness [1–3]. Approximately 438 000 people die annually from tobacco-related diseases [4], and some US minority racial and ethnic populations bear a disproportionate burden of tobacco-related health disparities [1]. Tobacco-related health disparities (TRHD) are differences in exposure to tobacco, tobacco use initiation, current use, the number of cigarettes smoked per day (cpd), quitting, treatment, relapse and the subsequent health consequences among specific population groups, and include differences in capacity and infrastructure as well as access to resources [5]. Some populations may not experience disparities along the entire 'tobacco disease continuum'. However, others may experience severe and devastating effects along the entire causal pathway to diseases such as cancer [1,6,7].

The independent investigation of differences in the prevalence of tobacco use and exposure and the incidence and mortality of diseases has produced limited progress in eliminating tobacco-related diseases and deaths. For example, previous studies suggest that higher smoking prevalence rates explain higher rates of tobacco-related cancers among black/African American men compared to white men [1]. Historically, the differences in smoking prevalence between black/African

American men and white men were small, such that for the past 10 years rates have been fairly similar. Black/African American adolescents have historically had substantially lower smoking rates than white youth [8,9] and experience delayed onset of smoking [10,11]. Black/African American adults and adolescents smoke substantially fewer cigarettes per day, and adults have lower quit rates [12] and younger age of diagnosis of lung cancer compared to whites [13]. Thus, existing tobacco data do not explain why black/African American men have the highest incidence and death rates from lung/bronchus, oral/pharynx, pancreatic, esophageal and larynx cancers [14]. Black/African American women have historically had similar smoking rates to white women and rates lower than American Indian/Alaska Native women. However, lung cancer incidence is increasing among black/African American women and they have the highest incidence and second highest lung/bronchus death rates of all women (see Tables 1 and 2). New paradigms for examining disparities may help to increase our knowledge on how to eliminate disparities in minority racial/ethnic groups.

Few frameworks have addressed work-force diversity, inequities and inequalities as part of a comprehensive approach to eliminating tobacco-related health disparities (see Fig. 1). In fact, diversity and disparities have often been approached separately, although a relationship exists. Diversity, which includes integration and inclusion, can create helpful environments that increase researchers' understanding of disparities. National reports have suggested that 'increasing racial and ethnic diversity among health professionals is important because the evidence indicates that diversity is associated with improved access to care for racial and ethnic minority patients . . .' [15]. Addressing inequities, injustice or unfairness in science may help to improve scientific practice and the capacity to address understudied research questions. Inequalities, the quality of being unequal, exist in the quality of and access to care, patient experiences and opportunities for preventive care [16]. Addressing inequalities may increase minority and racial and ethnic groups' access to cessation and cancer treatment services. Approaches that address simultaneously TRHD, work-force diversity and empowerment, inequities in research and inequalities in access to care may provide the paradigm shift needed to accelerate progress in reducing disparities among minority racial and ethnic groups (see Fig. 1).

This paper summarizes the known disparities that exist along the tobacco disease continuum for minority racial and ethnic groups, those living in poverty, those with low education and blue-collar and service workers. This paper also describes how addressing diversity, inequities in research and inequalities in care in tandem with disparities may help us reduce and eliminate TRHD. Minority racial and ethnic groups have suffered traditionally from or experienced social, cultural, educational, political and economic circumstances that greatly impact disparities [16]. Minority racial and ethnic groups have a long history of discrimination, oppression and social marginalization within the United States, and the fact that health status varies by race/ethnicity is partly a consequence of history and social and cultural systems [17]. Race and ethnicity may interact with socio-economic indicators such as poverty, education and occupation to increase or decrease disparities [16] at any point along the tobacco disease continuum. Minority racial and ethnic groups are also under-represented in the health research work-force, and there are inequities in research that impact our understanding of the multiple disparities from which these groups suffer. Understanding the relationship between TRHD among minority racial/ethnic groups and social and structural systems that impact disparities will help researchers, practitioners, advocates and policy makers determine optimal points along the tobacco disease continuum to intervene and integrative approaches needed to address this systemic epidemic.

METHODS

We searched the scientific literature using PubMed and examined major scientific reports (e.g. Surgeon General Reports, National Cancer Institute monographs and reports, Agency for Health Care Quality and Research, Morbidity and Mortality Weekly Reports, US Census Bureau) to delineate known disparities along the tobacco disease continuum, including data on second-hand smoke or environmental tobacco smoke (ETS), tobacco use initiation, current use, number of cigarettes smoked per day, quitting, treatment, relapse and tobacco-related cancer morbidity and mortality. We reviewed existing national survey data and state and local survey data when national data were not available for small racial and ethnic groups. National data used to report estimates of tobacco use [i.e. National Health Interview Survey (NIHS), the Youth Risk Behavioral Survey (YRBS), Monitoring the Future Survey (MTF), the National Survey of Drug Use and Health (NSDUH)] were reviewed for the most recent data and trends. Data from the Centers for Disease Control and Prevention National Program of Cancer Registry and the National Cancer Institute's Surveillance Epidemiology and End Results Program were used to report tobacco-related cancers. Because eliminating tobacco use would almost eliminate lung cancer, the leading cause of cancer deaths (see Table 1), and tobacco is related causally to 10 other cancers, we used tobacco-related cancers as our main indicators of disease consequences. In the following sections, we summarize the

Table 1 SEER tobacco-related cancer death rates and trends by sex and race/ethnicity, 1992–2002.

	Whites			*Blacks/African Americans*			*Asian Americans/ Pacific Islanders*			*American Indians/ Alaska Natives*			*Latinos/Hispanics**		
Sex/cancer site or type	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*
Male															
Lung and bronchus	79.3	−1.7**	−13.3	109.1	−2.5**	−25.4	41.1	−1.9**	−8.9	50.0	−2.3**	−9.9	40.4	−1.5**	−3.9
Urinary bladder	8.0	−0.5**	−0.3	5.9	−2.0**	−1.1	2.9	0.6	−0.2	2.5	¶	¶	4.2	−0.7	−0.2
Leukemia	10.6	−0.6**	−0.6	9.3	−1.1**	−0.6	5.4	−1.1	−0.7	5.1	−3.3	−0.8	6.7	0.2	0.0
Oral cavity and pharynx	4.2	−2.3**	−0.8	8.2	−4.6	−3.7	3.8	−2.8	−1.3	3.6	−1.5	−0.5	3.2	−4.0	−1.2
Kidney and renal pelvis	6.2	−0.1	−0.1	6.2	0.1	−0.1	2.7	−0.2	−0.3	6.8	0.8	−0.2	5.5	0.7	0.2
Stomach	6.3	−3.6**	−2.1	14.1	−3.0**	−3.5	12.6	−3.9**	−5.6	7.4	−1.7	−2.7	10.0	−2.1**	−2.1
Pancreas	12.0	0.0	0.2	16.4	−1.5**	−2.8	8.4	−2.1**	−0.4	6.3	1.4	0.9	9.4	−0.8	−0.8
Esophagus	7.2	1.6**	1.1	12.9	−4.4**	−4.9	3.6	−3.7**	−2.1	4.8	1.5	0.8	4.5	−1.2**	−0.5
Larynx	2.4	−2.3**	−0.6	5.7	−2.9**	−1.5	0.9	−1.2	−0.4	1.9	¶	¶	2.2	−3.2**	−0.6
Female															
Lung and bronchus	41.1	0.7**	3.4	39.3	0.4**	1.7	19.0	−0.4	−1.0	26.4	1.0	4.4	14.9	0.1	0.0
Urinary bladder	2.3	−0.3	0.0	3.0	−0.6	0.0	1.1	−2.2	−0.3	1.1	¶	¶	1.3	1.6	0.5
Leukemia	6.0	−0.5**	−0.4	5.5	−0.7**	−0.3	3.4	−1.6	0.6	3.4	−1.2	0.4	4.3	−0.6	−0.3
Oral cavity and pharynx	1.7	−2.3**	−0.4	2.1	−3.5	−0.6	1.5	−1.2	0.2	1.3	¶	0.1	0.9	−1.2	−0.4
Kidney and renal pelvis	2.9	−0.4	−0.1	2.8	0.0	0.2	1.2	0.3	0.2	3.4	−1.1	1.2	2.4	−0.5	0.0
Stomach	3.0	−2.9**	−0.9	6.7	−2.1**	−1.4	7.5	−3.5**	−2.4	4.2	0.0	0.0	5.5	−1.5**	−0.9
Pancreas	8.9	0.0	0.0	12.9	−0.8**	−1.0	6.7	0.9	1.1	6.1	0.1	−0.3	7.6	0.2	−0.1
Esophagus	1.6	0.7**	0.1	3.5	−3.3**	−1.2	0.9	−1.1	−0.1	1.1	¶	¶	0.9	1.7	0.3
Larynx	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Cervix uteri	2.7	−2.7**	−0.7	6.2	−4.9**	−3.1	2.9	−3.3**	−1.1	3.2	−4.7**	−1.9	3.8	−3.0**	−1.2

Table adapted from table 4 printed in Edwards *et al.* [14] with permissions from the author and Oxford University Press. Sources of data are the Surveillance, Epidemiology, and End Results (SEER) Program registries that include Connecticut, Hawaii, Iowa, Utah, and New Mexico; the metropolitan areas of San Francisco, Detroit, Atlanta, Seattle-Puget Sound, San Jose-Monterey and Los Angeles; rural Georgia and Alaska Natives in Alaska (i.e. SEER 13). AC = absolute change; APC = annual percentage change; N/A = not available. *Data for Hispanics/Latino/Hispanics excludes cases diagnosed in Detroit, Hawaii, Alaska Natives and rural Georgia. †Rates are per 100 000 people and are age-adjusted to the 2000 US Standard Population—using 19 age groups, with data provided from US Bureau of the Census, Current Population Reports, Series P25–1130. ‡APC is based on rates that were age-adjusted to the 2000 US. Standard Population—using 19 age groups, with data provided from US Bureau of the Census, Current Population Reports, Series P25–1130) (39). §AC was calculated as the difference in the age-adjusted rate for 2002 minus age-adjusted rate for 1992. ¶Statistic could not be calculated. APC based on fewer than 10 cases for at least 1 year within the time interval. **APC is statistically significantly different from zero (two-sided $P < 0.05$).

Table 2 SEER tobacco-related cancer incidence rates and trends by sex and race/ethnicity, 1992–2002.

Sex/cancer site or type	*Whites*			*Blacks/African Americans*			*Asian Americans/ Pacific Islanders*			*American Indians/ Alaska Natives*			*Latinos/Hispanics**		
	% rate†	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*	*% rate†*	*APC‡*	*AC§*
Males															
Lung and bronchus	81.5	−2.1	−18.5	122.8	−2.5¶	−35.7	61.2	−1.4¶	−14.0	49.8	−5.4¶	−20.3	47.2	−2.0¶	−7.2
Urinary bladder	39.7	−0.1	−2.5	20.2	−0.2	1.5	16.8	1.1	2.8	8.3	**	**	19.0	−0.2	0.0
Leukemia	17.4	−1.1¶	−2.6	12.9	−1.6	−3.6	9.8	−0.3	−0.2	5.5	**	**	11.8	−0.6	0.1
Oral cavity and pharynx	16.2	−1.5¶	−2.3	20.7	−3.1	−6.0	12.6	−1.5	−0.9	11.3	−9.2¶	−11.8	10.1	−2.8¶	−2.3
Kidney and renal pelvis	15.9	1.6¶	2.0	18.5	2.2¶	4.3	8.7	0.1	−0.1	15.6	−4.0¶	−4.2	14.7	2.0¶	2.5
Stomach	11.3	−2.1¶	−2.1	19.5	−2.8¶	−5.1	23.1	−3.3¶	−7.7	14.6	**	4.8	18.3	−2.5¶	−3.1
Pancreas	12.5	0.1	−0.1	17.5	−2.5	−5.4	10.7	−2.8¶	−1.3	7.8	**	**	10.8	−0.9	−1.0
Esophagus	7.4	1.5¶	0.5	12.4	−5.7¶	−7.4	5.1	−2.0	−0.5	6.1	**	**	5.9	−1.2	−1.2
Larynx	7.1	−3.3	−2.6	12.7	−3.2¶	−5.8	3.5	−2.5¶	−0.8	2.3	**	**	5.6	−1.6¶	−1.2
Female															
Lung and bronchus	51.3	−0.1	−0.6	53.6	0.5	2.8	28.4	0.0	−1.2	25.8	−2.8¶	−10.2	24.1	−1.5¶	−3.4
Urinary Bladder	9.9	−0.2	−0.2	7.5	0.9	1.0	4.2	−1.3	−1.7	2.0	**	**	5.2	0.3	0.05
Leukemia	10.1	−0.5	−0.6	8.0	−1.2	−1.1	6.2	−2.2¶	−0.8	4.5	**	**	7.7	−0.6	−0.4
Oral cavity and pharynx	6.7	−1.2¶	−0.4	6.5	−1.7¶	−0.3	5.8	−0.1	−0.7	4.0	**	**	4.0	−1.3	0.0
Kidney and renal pelvis	7.9	1.5¶	0.8	9.1	2.5	3.3	4.1	2.8¶	1.3	7.9	**	−3.4	7.8	2.7¶	2.7
Stomach	5.1	−1.0	−0.7	9.7	0.0	0.4	12.9	−2.9¶	−4.0	7.9	**	**	10.2	−0.6	−0.2
Pancreas	9.6	−0.3	−0.1	14.7	−1.6¶	−0.9	8.3	2.0¶	1.5	7.3	**	**	9.4	0.2	1.0
Esophagus	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Larynx	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Cervix uteri	9.3	−2.2	−2.4	12.6	−3.8	−4.4	10.5	−4.9	−4.3	6.6	−6.9¶	6.3	17.3	−3.3¶	6.3

Table adapted from Table 3 printed in Edwards *et al.* [14] with permissions from the author and Oxford University Press. Sources of data are the Surveillance, Epidemiology, and End Results (SEER) Program registries that include Connecticut, Hawaii, Iowa, Utah and New Mexico; the metropolitan areas of San Francisco, Detroit, Atlanta, Seattle-Puget Sound, San Jose-Monterey and Los Angeles; rural Georgia and Alaska Natives in Alaska (i.e. SEER 13). AC = absolute change; APC = annual percentage change; N/A = not available. *Data for Hispanics/Latino/Hispanics excludes cases diagnosed in Detroit, Hawaii, Alaska Natives and rural Georgia. †Rates are per 100 000 people and are age-adjusted to the 2000 US Standard Population—using 19 age groups, with data provided from US Bureau of the Census, Current Population Reports, Series P25–1130. ‡APC is based on rates that were age-adjusted to the 2000 US Standard Population—using 19 age groups, with data provided from US Bureau of the Census, Current Population Reports, Series P25–1130) (39). §AC was calculated as the difference in the age-adjusted rate for 2002 minus age-adjusted rate for 1992. ¶APC is statistically significantly different from zero (two-sided $P < 0.05$). **Statistic could not be calculated. APC based on fewer than 10 cases for at least 1 year during the time interval.

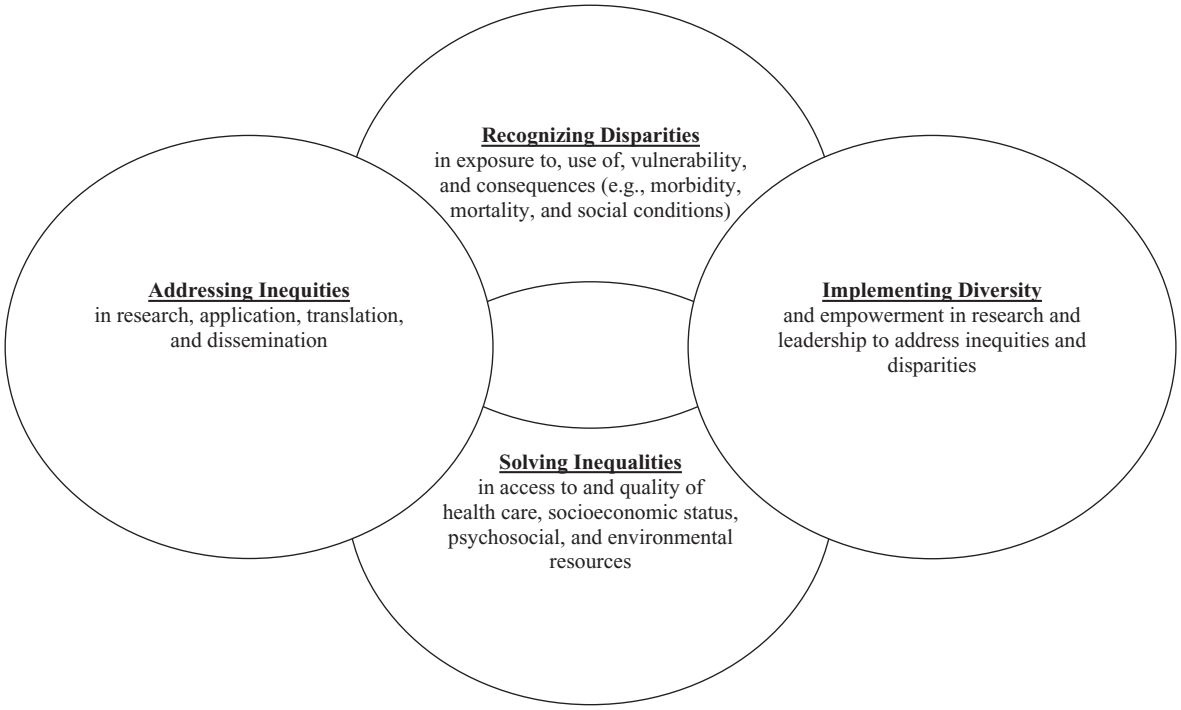

Figure 1 Strategy for eliminating tobacco-related health disparities

known disparities for each minority racial/ethnic group and by socio-economic indicators, and discuss the roles of diversity, inequity and inequalities in reducing disparities.

RESULTS

Disparities

A 'disparity' is defined as the condition or fact of being unequal in age, rank or degree [18], and is used to describe unequal health conditions or indicators (e.g. tobacco use initiation, ETS, current use, cpd, quitting, treatment, relapse, cancer). Data reported in the 1998 Surgeon General Report, Tobacco Use Among US Racial/Ethnic Minority Groups, suggests that there are unequal tobacco-related health indicators among racial and ethnic groups. Some differences are small, while others are relatively large and unexplainable for Latinos/Hispanics, blacks/African Americans, American Indian and Alaska Natives, and Asian Americans and Pacific Islanders. Because of small sample sizes, most data on minority racial/ethnic groups are reported in aggregate form, but some within-group data have been documented.

Latinos/Hispanics and tobacco disease continuum indicators

Latinos/Hispanics are the largest minority racial/ethnic group in the United States. In 2000, 12.5% of US citizens reported being Latino/Hispanic of any race [19]. Of these, 58.5% reported Mexican, 4.8% Central American, 3.8% South American, 9.6% Puerto Rican, 3.5% Cuban, 2.2% Dominican and 17.3% other Latino/Hispanic [19]. Latinos/Hispanics have the third highest poverty rate (21.9%) (see Fig. 2) [20] and third lowest median income of all the racial and ethnic groups ($34 241) [20]. In 2003, only 57.0% of Latinos/Hispanics graduated from high school [21] (see Fig. 3). Of those in the labor force, 18.0% reported management/professional; 21.0% service; 23.1% sales/office; 2.7% farming, fishing and forestry; 9.6% construction, extraction and maintenance; and 13.2% production, transportation and material moving occupations [22] (see Fig. 4).

Initiation and ETS

Smoking initiation and ETS exposure among minority racial and ethnic groups are poorly understood. Regular onset of smoking occurs primarily before age 18 and Latinos/Hispanics have earlier onset than Asians/Pacific Islanders and blacks/African Americans [11,23,24]. The YRBS data indicate that age of initiation among Latinos/Hispanics is higher but similar to that of whites [10]. Latinos/Hispanics are more likely to have ever smoked a whole cigarette compared to black/African American students and initiation diverges from black/African American students at ages 11–12 [10].

ETS exposure is low among Latinos/Hispanics in California [25] and is lower in Latino/Hispanic homes compared to other racial/ethnic groups except Somali

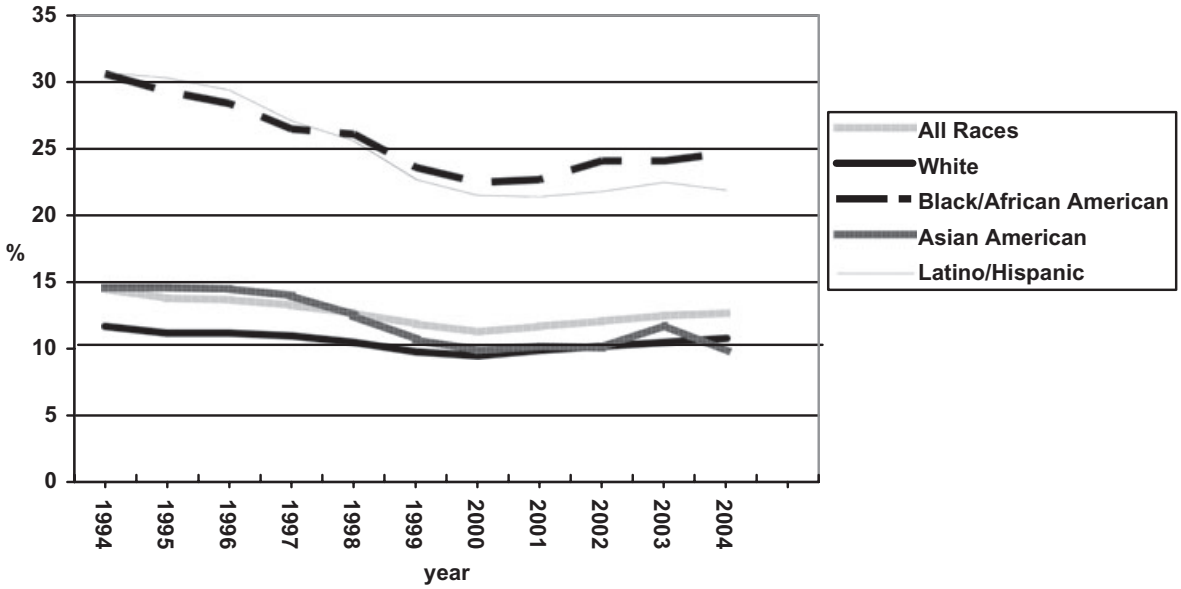

Figure 2 Percentage of all people below poverty by race/ethnicity in the United States, 1994–2004.
Table adapted from table B2 in United Status Census Bureau [20]. Source: US Census Bureau, Current Population Survey (CPS), 1960–2005 Annual Social and Economic Supplements. Consistent with 2001 data through implementation of Census 2000-based population controls and a 28 000 household sample expansion. For 1999, figures are based on Census 2000 population controls. For 2001 and earlier years, the CPS allowed respondents to report only one race group. The reference race groups for 2001 and earlier poverty data are white, non-Hispanic white, black and Asian and Pacific Islander. Black alone refers to people who reported black and did not report any other race category. Asian alone refers to people who reported Asian and did not report any other race category. Data for American Indian and Alaska Natives and Native Hawaiian and Other Pacific Islanders were not included in the above table. The Census Bureau estimates that 23.9% of American Indian and Alaska Natives were living in poverty in 2002–2003 and that 24.4% were living in poverty in 2003–2004. An estimated 14.4% of Native Hawaiian and Other Pacific Islanders were living in poverty in 2002–2003; 12.9% in 2003–2004. Source: Income, Poverty, and Health Insurance Coverage in the United States: 2004. United States Census Bureau, August 2005, p. 12

immigrants [26]. One study among children found that ETS exposure varies among ethnic groups; 10.3% of Mexican, 35.5% of Puerto Rican, 10.8% of Dominican and 16.4% of other Latinos/Hispanics were exposed to ETS compared to 45.8% of blacks/African Americans and 12.7% of whites [27].

Current use

MTF 30-day prevalence trends among 8th, 10th and 12th graders indicate that Latino/Hispanic smoking has declined since 2000 [9], but increased slightly from 2003 to 2005. Data from the 2003 YRBS indicate that 18.4% of Latino/Hispanic high school students smoked [8], but in 2005, 22% smoked [28]. Disaggregate and pooled data reported from 1999 to 2001 NSDUH indicate that 10.8% of Latino/Hispanic youth ages 12–17 smoked, and among ethnic groups, 11% of Mexicans, 10.8% of Puerto Ricans, 9.6% of Central or South Americans and 12.4% of Cubans smoked [29]. Combined data from the 1996–2000 MTF indicate that 30-day prevalence rates among 12th graders were 25.7% among Mexicans, 30.1% among Cubans, 29.6% among Puerto Ricans and 25.2% among other Latin American youth [30].

Current smoking has declined among adult Latinos/Hispanics since the 1970s [1]. Data from the 2004 NHIS indicate that 15% of Latino/Hispanic adults smoked; this rate is the second lowest of all racial and ethnic groups (see Table 3) [4]. According to the aggregate data reported using the 1999–2001 NSDUH, 23.1% of Latino/Hispanic adults smoked. The disaggregate data indicate that 23% of Mexican adults, 30.4% of Puerto Ricans, 21.3% of Central or South Americans and 19.2% of Cubans smoked [29]. Other data also report that current smoking rates are highest among the Puerto Rican ethnic group [1,31].

CPD

Latinos/Hispanics in general tend to be light smokers. Only 4.6% of Mexican, 11.3% of Cuban, 11.5% of Puerto Rican and 4.8% of other Latin American youth in the 12th grade smoked a half-pack or more per day [30]. The percentage of light (< 15 cpd) adult smokers has, in general, increased since the mid-1970s [1]. At least 65% of Latino/Hispanic smokers smoked < 15 cpd [1]. Data from the Multiethnic Cohort Study indicate that 59.0% of Latino/Hispanic men smoked ≤ 10 cpd and 27.8%,

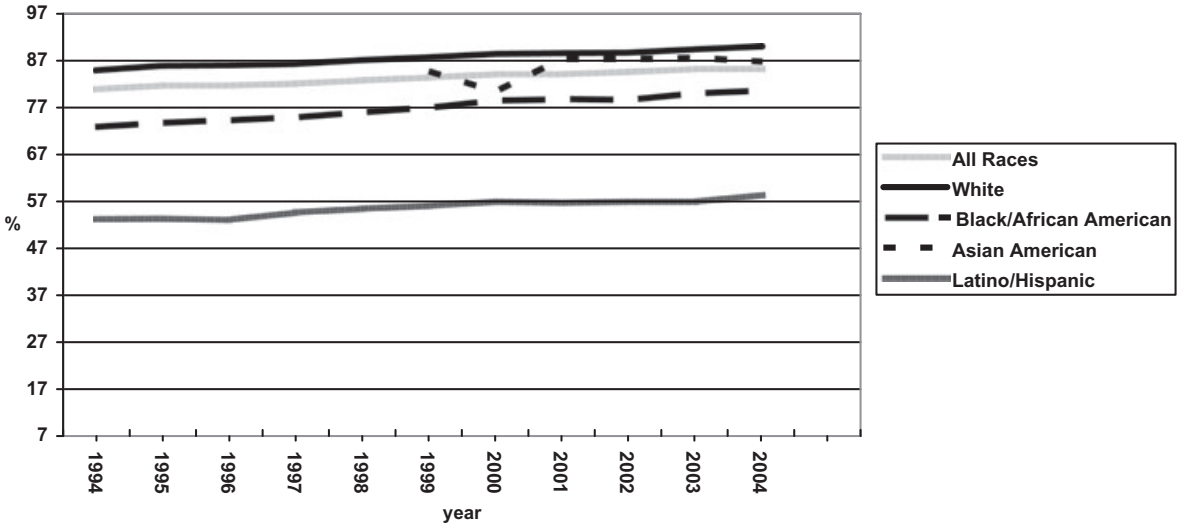

Figure 3 Percentage of people aged 25 years and over who have completed high school.
Consistent with 2001 data through implementation of Census 2000-based population controls and a 28 000 household sample expansion. For 1999, figures are based on Census 2000 population controls. For 2001 and earlier years, the Current Population Survey (CPS) allowed respondents to report only one race group. The reference race groups for 2001 and earlier poverty data are white, non-Hispanic white, black and Asian and Pacific Islander. Black alone refers to people who reported black and did not report any other race category. Asian alone refers to people who reported Asian and did not report any other race category. Source: US Census Bureau, Current Population Survey, 1960–2005 Annual Social and Economic Supplements. United States Census Bureau (March 2005). Percent of People 25 years and Over Who Have Completed High School or College, by Race, Hispanic Origin and Sex: Selected Years 1940–2004. Available at: http://www.census.gov/population/socdemo/education/tabA-2.pdf (accessed 9 February 2006). Until recently, the Census Bureau did not break out separate information for this category for Asians. The figures in this column were obtained from yearly reports issued by the Census Bureau from 1999 to 2004. United States Census Bureau (March 2005) 'Educational Attainment of the Population 25 years and Over, by Citizenship, Nativity and Period of Entry, Age, Sex, Race and Hispanic Origin. (1999–2004). Available at: http://www.census.gov/population/socdemo/education/cps2004/tab10-05.pdf (accessed 9 February 2006)

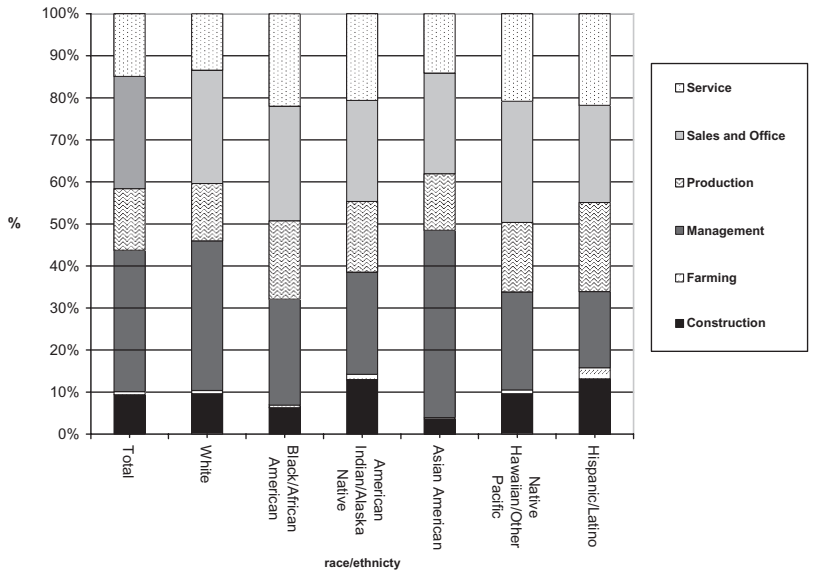

Figure 4 Selected occupational groups by race and hispanic origin, US 2000. Data based on a sample. For information on confidentiality protection, sampling error, non-sampling error and definitions, see http://www.census.gov/prod/cen2000/doc/sf3.pdf

11–20 cpd. Among women, 74.9% smoked ≤ 10 cpd and 18.4%, 11–20 cpd [32].

Quitting, relapse and treatment

Very little is known about quitting behaviors among Latino/Hispanic youth or adults. Data from the 2005 YRBS indicate that 53.4% of Latino/Hispanic youth tried to quit smoking [28]. Quitting smoking has increased among Latino/Hispanic adults [1]. Data from the 2000 NHIS indicate that 61.9% of Latinos/Hispanics want to quit and 42.9% who have ever smoked quit [12]. According to the 1998 Health Outcomes Survey (HOS), 65.4% of

Table 3 Percentage of people ≥ 18 years of age who were current smokers, by race/ethnicity, poverty and education, United States, National Health Interview Survey, 1994–2004.*

	1994	*1995*	*1996†*	*1997*	*1998*	*1999*	*2000*	*2001*	*2002*	*2003*	*2004*
Race/Ethnicity‡											
White	26.3	25.6	NA	25.3	25.0	24.3	24.1	24.0	23.6	22.7	22.2
Black/African American	27.2	25.8	NA	26.7	24.7	24.3	23.2	22.3	22.4	21.5	20.2
Hispanic/Latino	19.5	18.3	NA	20.4	19.1	18.1	18.6	16.7	16.7	16.4	15.0
American Indian/Alaska Native§	42.2	36.2	NA	34.1	40.0	40.8	36.0	32.7	40.8	39.7	33.4
Asian/Pacific Islander¶	13.9	16.6	NA	16.9	13.7	15.1	–	–	–	–	–
Asian American	–	–	NA	–	–	–	14.4	12.4	13.3	11.7	11.3
Poverty											
At or above	24.1	23.8	NA	24.6	23.5	23.4	22.9	23.0	22.2	21.7	20.6
Below	34.7	32.5	NA	33.3	32.3	33.1	31.7	31.4	32.9	30.5	29.1
Unknown	28.8	23.5	NA	20.5	22.5	20.2	21.4	19.3	19.7	18.4	19.0
Education (years)**											
≤ 8	23.7	22.6	NA	22.5	21.9	18.3	20.0	18.6	19.3	17.6	16.7
9–11	38.2	37.5	NA	35.4	36.8	37.7	33.9	34.3	34.1	34.0	34.0
0–12 (no diploma)	–	–	NA	–	–	28.4	28.2	28.4	27.6	26.6	26.2
12 (no diploma)	–	–	NA	–	–	26.0	32.7	29.5	31.0	29.3	25.5
GED diploma	–	–	NA	–	–	44.4	47.2	47.8	42.3	44.4	39.6
12 (diploma)	29.8	29.5	NA	28.4	27.4	26.3	27.2	26.1	25.6	25.4	24.0
Associates degree	–	–	NA	–	–	22.8	21.1	21.6	21.5	19.8	20.9
Some college	–	–	NA	–	–	25.3	23.5	24.2	23.1	21.9	22.2
Undergraduate degree	–	–	NA	–	–	11.9	13.2	12.3	12.1	12.3	11.7
Graduate degree	–	–	NA	–	–	7.8	8.4	9.5	7.2	7.5	8.0
13–15	25.7	23.6	NA	25.1	24.6	–	–	–	–	–	–
≥ 16	12.3	14.0	NA	11.6	11.3	–	–	–	–	–	–
Total	25.5	24.7	NA	24.7	24.1	23.5	23.3	22.8	22.5	21.6	20.9

Tables adapted from National Health Interview Survey data reported in [4] accessed at http://www.cdc.gov/tobacco/data_statistics/tables/adult/table_2.htm. The NHIS in 1997 was redesigned and trend analysis and comparison with data years before 1997 should be conducted with caution. *Current smokers include those who smoked ≥ 100 cigarettes per day and who smoked every day or some days. †Data not collected in 1996. ‡All racial ethnic groups are non-Hispanics except those categorized as Hispanic. §In 1997, the Office of Management and Budget changed its data collection guidelines to require that Native Hawaiian and Other Pacific Islander data be collected separately from Asian. ¶Data on American Indians/Alaska Natives are small and data for a single year may be unstable or unreliable. **Additional categories were added to education in 1997.

Latinos/Hispanics in Medicare managed care indicate that they received advice to quit from a doctor or health care provider in the last 12 months, a rate lower than whites and blacks/African Americans [33].

Cancer outcomes

The current data indicate that Latinos/Hispanics as an aggregate group suffer few disparities in tobacco-related cancers compared to other racial/ethnic groups. However, Latino/Hispanic women have the highest incidence rates of cervical cancer [14]. Latino/Hispanic men and women have the lowest incidence and death rates of lung cancer, and lung cancer incidence rates decreased for Latino/Hispanic women along with American Indians/Alaska Natives, while these rates remained stable for other women. Rates of lung cancer have declined among Latino/Hispanic men [14]. The incidence and death rates of oral/pharynx cancer were lowest among Latino/Hispanic men compared to other men (see Tables 1 and 2).

Blacks/African Americans and tobacco disease continuum indicators

In the 2000 Census, 12.2% of Americans reported being black or African American as their only race/ethnicity [34]. Four per cent of blacks/African Americans reported being foreign-born, most whom originate from the Caribbean and Africa [35]. Compared to other racial and ethnic groups, blacks/African Americans have the highest poverty rates (24.7%) (see Fig. 2), the lowest median income ($30 134) [20] and 80.0% completed a high school education [21] (see Fig. 3). Of those in the labor force, 25.2% reported management/professional; 22.0% service; 27.3% sales/office; 0.4% farming, fishing

and forestry; 6.5% construction, extraction and maintenance; and 18.6% production, transportation and material moving occupations [35] (see Fig. 4).

Initiation rates and ETS

Smoking initiation rates have declined among blacks/African Americans [1] and delayed onset has been documented consistently as a phenomenon among black/African American smokers. Blacks/African Americans begin regular smoking primarily after the age of 18 [11,23,36–42], and overall have lower smoking initiation rates compared to whites [43] and Latinos/Hispanics during adolescence [10]. Data from YRBS show similarities in initiation rates at ages 9–10 for blacks/African Americans, whites and Latinos/Hispanics, but at ages 11–12, black/African American initiation rates were substantially lower and diverged from whites and Latinos/Hispanics [10]. One study indicated that black/African American youth had higher initiation rates than whites, but lower rates of regular use compared to whites and Latinos/Hispanics [44]. Existing data do not indicate within group differences in smoking onset; the mean age of smoking onset among Caribbean-born blacks was 18.6 years, Haitian-born, 19.5 years and US-born blacks 18.3 years [42].

Some studies suggest that ETS exposure among black/African American children is greater than all other ethnic groups [26,27]. Studies have found higher levels of serum cotinine [26]and hair cotinine in black/African American children compared to white children, despite lower levels of reported ETS exposure [45,46]. Higher levels of serum cotinine were found among black/African American children compared to Latino/Hispanic children after controlling for ETS exposure [47]. Thirty-seven per cent of black/African American adults compared to 37.4% of whites and 35.1% of Mexican Americans reported exposure to ETS [48].

Current use

Data from MTF indicate that 30-day prevalence rates among youth have decreased steadily since 1998 [9]. Trends in 30-day prevalence indicate that rates overall have been consistently and substantially lower among black/African American youth compared to other racial/ethnic groups [1,9,36,43,49]. The 2005 YRBS indicate that 12.9% of black/African American high school students smoked [28]. The 1999–2001 NSDUH data indicate that 7% of black/African American youth smoked, which was lower than all racial/ethnic groups [29]. Combined data from the 1996–2000 MTF indicate that 14.3% of black/African American 12th graders smoked [30].

Among adults, smoking rates have also decreased. In 1965, rates were 45.8% [50] and in the 2004 NHIS, 20.2% of black/African American adults reported smoking, with rates slightly lower than the rate among whites (22.2%) (see Table 3) [4]. Current smoking rates among black/African American men were higher than white men in 1965 but rates have declined among black men and women since the mid-1970s [1]. In 2004, 23.9% of black/African American men and 17.2% of black/African American women smoked [4]. Haitian-born and Caribbean-born blacks report lower rates of current smoking than American-born blacks [42,51]; this study found that 7.1% of Caribbean-born, 9.7% of Haitian-born, and 23.4% of US-born blacks smoked [42]. Trend data that delineate smoking rates for foreign-born versus native-born blacks have not been published.

CPD

Similar to Latinos/Hispanics, blacks/African Americans tend to be light smokers. Only 2.6% of 12th grade black/African American youth smoked a half-pack or more per day [30]. Data from YRBS also indicate that blacks/African Americans and Latinos/Hispanics smoked fewer cpd than white students [10]. The percentage of black/African American adults who smoked < 15 cpd has increased since the 1970s [1,52]. More than 60.0% of black/African American adults smoked < 15 cpd and smoked fewer cpd than whites [53]. Less than 10.0% of blacks/African Americans smoked ≥ 25 cpd [1]. Data from the Multiethnic Cohort Study indicate that 42.9% of black/African American men smoked ≤ 10 cpd and 40.0%, 11–20 cpd, and among women, 59.7% smoked ≤ 10 cpd and 30.6%, 11–20 cpd [32]. Despite the fact that blacks/African Americans in general are light smokers, black/African American adolescents show higher cotinine-to-cigarette per day ratios than white youth [54] and black/African American adults also have higher serum cotinine than white [55–58] and Mexican American adult smokers, who smoke the same number of cpd [1,56,58].

Quitting, relapse and treatment

Little is know about quitting among black/African American youth; however, data from the 2005 YRBS indicate that 61.8% of black/African American youth tried to quit [28]. Quit rates have increased since 1978 among black/African American adults, but were lower than whites and other racial and ethnic groups [52,59,60]. In the 2000 NHIS, 68.4% of blacks/African Americans desired to quit, but only 37.3% who have ever smoked quit. This quit rate was lower than that of any

other racial or ethnic group [12]. Data from the Multiethnic Cohort Study indicate that rates of quitting were significantly lower among black/African American compared to white men (42.2% versus 55.5%), but were not significantly different from Japanese, Native Hawaiian or Latino/Hispanic men [32]. Some data suggest that there are few differences in mean quit attempts among blacks/African Americans, whites and Latinos/Hispanics [7]. In a 1998 survey of Medicare managed care enrolled smokers, 67.8% of blacks/African Americans reported receiving advice to quit [33], and studies have found that younger blacks/African Americans were less likely than whites to receive advice to quit [61].

Cancer outcomes

Blacks/African Americans have the highest overall incidence and death rates of all cancers (see Tables 1 and 2). Although rates are declining, black/African American men have the highest lung cancer incidence and death rates and there is an enormous disparity between black/African American men and Latino/Hispanic men, who have the lowest incidence and death rates. Black/African American men also have a younger age of lung cancer diagnosis than other groups [13]. Deaths from stomach cancer are more than 200% higher and for esophageal and oral cavity cancer, 75% higher among black/African American compared to white men [14]. Lung cancer incidence and death rates are lower among black/African American women compared to black/African American men, but black/African American women have the highest incidence rates of lung cancer and the second highest death rates after white women [14]. Lung cancer death rates have not declined for black/African American women [14].

American Indians, Alaska Natives and tobacco disease continuum indicators

In 2000, American Indians and Alaska Natives, not including those who report one or more race, comprised 0.9% of the US population [62]. Over 500 federally recognized tribes, villages and communities exist in the United States. The Cherokee community is the largest, with about 302 569 members [62], but many tribes have less than 1000 members [63]. Based on 3-year averages of aggregate data, data indicate that 24.3% of American Indians/Alaska Natives live in poverty and they have the second lowest median income ($33 132) of all racial/ethnic groups [20]. Although poverty rates vary among tribes, rates of poverty among all American Indian/Alaska Natives exceed that of whites and Asian Americans [62]. Seventy-one per cent have at least a high school education, which varies by tribe [62]. Of American Indians/Alaska Natives in the labor force, 24.3% reported management/professional; 20.6% service; 24.0% sales/office; 1.3% farming, fishing and forestry; 12.9% construction, extraction and maintenance; and 16.8% production, transportation and material moving occupations [62] (see Fig. 4). Some studies report data on American Indians separately from Alaska Natives and few report regional or tribal differences. We report differences where possible and use the label American Indians/Alaska Natives to represent the aggregate category reported in surveys.

Initiation and ETS

Although smokeless tobacco use is not the focus of this paper, it is worth noting that smokeless tobacco use rates range from 18 to 60% among youth and that initiation can begin as early as pre-school and kindergarten years among Alaska Natives [64–66]. Studies also report that 68.9% of girls and 79.1% of boys at Indian Health Service sites experiment with smokeless tobacco [67]. Among American Indians, smoking initiation rates have been reported in middle school [68]. In addition, the use of Ikmik, a smokeless form of tobacco, is used at all ages from youth to adulthood [69].

Very little is known about ETS exposure among American Indians and Alaska Natives. Studies indicate that in a combined category of Native American and South-east Asian children, ETS exposure was higher than Latino/Hispanic and Somali immigrants but lower than Black/African American children [26]. The US Women's Determinants Study found that home ETS exposure is higher among American Indians/Alaska Natives than other racial/ethnic groups [70].

Current use

American Indian/Alaska Native youth and adults have the highest rates of tobacco use in the United States [29]. Although many studies indicate that ceremonial use of tobacco is common among American Indians, recent studies indicate that non-traditional use of tobacco occurs frequently among American Indians [68]. Recent data from the YRBS and MTF are not published for American Indian/Alaska Native youth and very little trend data are available. From 1990 to 1994, the current smoking rate among 12th grade American Indians/Alaska Natives was 41.1%, but from 1995 to 1999 rates were 42.3% [71]. Data from the 1999–2001 NSDUH indicate that 27.9% of American Indian/Alaska Native high school students were current smokers [8]. Combined data from the 1996–2000 MTF indicate that 46.1% of American Indian 12th graders smoked [30].

Prevalence rates among adults have not declined consistently since the 1970s [1]. According to the 2004

NHIS, 33.4% of American Indian/Alaska Native adults smoked (see Table 3) [4]. Rates are higher among men compared to women (37.3% versus 28.5%) [4]. Higher rates have been reported in Alaska and the Northern Plains and lower rates reported in the South-west [1,72]. Forty-four per cent of those in the Northern Plains, 39% in Alaska, 31.9% in the East, 30.9% in the Pacific Coast and 21.2% in the South-west smoked [72].

The 1997 BRFSS data from Alaska indicate that 41.3% American Indians/Alaska Natives smoked [73]. Alaska Native adult current use rates range from 39 to 50% and among women, 36–68% [74]. Pregnant Alaska Native women have smoking prevalence rates of 39–44% [74]. Alaska women in rural areas have higher rates of tobacco use than women in urban areas [74]. Urban American Indians report higher smoking rates than rural American Indians [75].

CPD

Seventeen per cent of American Indian 12th graders smoked a half-pack or more per day; this rate was higher than that among white 12th graders (15.9%) [30]. The percentage of light smokers (< 15 cpd) has increased among adults [1]. About 50% of American Indians/Alaska Natives smoked < 15 cpd [1] and men were more likely than women to be heavy smokers (≥ 25 cpd) [1]. Heavy smoking among American Indians/Alaska Natives, however, was lower than white adults [1]. The number of cpd varies by region, but in all regions, light smoking exceeded 70.9% [1]. American Indian adults report smoking on average 10 cpd [68].

Quitting, relapse and treatment

Very little is known about quitting among American Indians and Alaska Natives. Quit rates have fluctuated since the 1970s [1]. In the 2000 NHIS, 69.8% reported a desire to quit and 40.9% of American Indians/Alaska Natives who ever smoked quit [12]. In the 1998 survey among Medicare managed care smokers, 72.2% of American Indians/Alaska Natives reported that they had received advice to quit, a rate equivalent to that of whites, who report the highest rates of receiving advice to quit [33].

Cancer outcomes

American Indian/Alaska Native men and women have the second lowest lung cancer incidence rates of all racial/ethnic groups and the third highest lung cancer death rates (see Tables 1 and 2). These same data indicate that lung cancer incidence rates decreased for American Indian/Alaska Native women, but lung cancer deaths did not decline among American Indian/Alaska Native men [14]. No declines were observed in cancers of the stomach and larynx among American Indians/Alaska Natives [14].

Lung cancer death rates reported by Indian Health Service (IHS) areas parallel smoking rates in those areas [1]. Data on lung cancer mortality from 1994 to 1998 indicate that American Indians/Alaska Natives in Alaska (78.1) and in the Northern Plains (96.9) have higher rates of lung cancer compared to the total US population of all racial/ethnic groups (57.8); rates were substantially lower among American Indians/Alaska Natives in the East (37.0), the Pacific Coast (39.5) and the South-west (14.1) [72]. Stomach cancer mortality from 1994 to 1998 was significantly higher in Alaska (16.3), the Northern Plains (8.9) and the South-west (10.3) compared to the total population of all racial/ethnic groups (5.1) [72].

Another study indicated that Alaska Natives have higher tobacco-related cancers including lung, oral/pharynx, stomach, kidney, cervix and pancreatic cancers than whites [76]. Lung cancer 5-year survival rates were 5% lower among Alaska Natives compared to whites from 1984 to 1999 and also lower for esophageal, pancreatic, stomach, oral, pharynx, kidney and cervical cancers [76].

Asian Americans, Hawaiians/Pacific Islanders and tobacco disease continuum indicators

In 2000, Asian Americans comprised 3.6% and Native Hawaiians/Pacific Islanders 0.1% of the US population [77]. Asians and Pacific Islanders consist of Asians born in the continental United States; Asian immigrants and refugees; and natives of Hawaii, Guam, American Samoa, Federated States of Micronesia, Republic of the Marshall Islands, Republic of Palau and the Commonwealth of the Northern Miriana Islands. Hawaiians make up the largest Pacific Islander group [77] and Chinese the largest Asian American group in the United States [78]. The aggregate group Asian American/Pacific Islander consists of more than 32 ethnic and language groups. Over 500 languages and dialects are represented among the aggregate group [1]. Asian Americans have the lowest poverty rate of all racial and ethnic groups (9.8%) (see Fig. 2) and the highest median income ($57 518). Native Hawaiians and Pacific Islanders have the second highest median income ($56 664) and a poverty rate of 13.2%. Because of small sample sizes, the Native Hawaiian and Pacific Islander income estimates are based on 3-year averages [20]. Eighty-eight per cent of Asian Americans and [21] 82.0% of Hawaiians and Pacific Islanders have graduated from high school [79] (see Fig. 3). Of Asian Americans in the labor force, 44.6% reported management/professional; 14.1%

service; 24.0% sales/office; 0.3% farming, fishing and forestry; 3.6% construction, extraction and maintenance; and 13.4% production, transportation and material moving occupations [22] (see Fig. 4). Of Hawaiians and Pacific Islanders in the labor force, 23.3% reported management/professional; 20.8% service; 28.8% sales/office; 0.9% farming, fishing and forestry; 9.6% construction, extraction and maintenance; and 16.5% production, transportation and material moving occupations [22]. We report ethnic differences where possible.

Initiation and ETS

Little is known about differences in initiation rates among Asian ethnic groups, but existing data suggest that there are within-group differences. Asian American youth are less likely to begin smoking in early adolescence [80,81]. Data from the 2000 NYTS indicate that smoking initiation rates were much lower among middle school Asian American (39.5%) compared to Hawaiians/Pacific Islanders (44.0%), blacks/African Americans (47.6%), Latinos/Hispanics (45.8%) and whites (47.2%). However, among high school students, smoking initiation was highest among Asian American youth (42.5), followed by Latinos/Hispanics (33.9%), whites (33.2%), blacks/African Americans (31.5%) and Hawaiians/Pacific Islanders (24.9%) [82]. Chinese and Koreans initiate later in life than whites [39] and Asian Americans/Pacific Islanders were more likely to start smoking regularly after the age of 18 [11]. Little is available on ETS exposure, but one study indicated that 31% of Vietnamese women were exposed daily to ETS [83].

Current use

Recent trend data from the YRBS and MTF have not been published for Asian American/Pacific Islander youth. Depending on the survey, current smoking rates vary among Asian Americans/Pacific Islanders. Data from the 1999–2001 NSDUH indicate that 8.1% of Asian American youth and 11.0% of Hawaiian/Pacific Islander youth smoked [29]. For Asian ethnic groups, current smoking was 10.6% among Koreans, 8.7% among Asian Indian, 7.4% among Filipino, 5.8% among Chinese, 5.2% among Japanese and 6.8% among Vietnamese youth [29]. Combined data from the 1996–2000 MTF indicate that 20.4% of Asian American 12th graders smoked [30]. Data from the 2000 NYTS indicate that 32% of Hawaiian and Pacific Islander high school students smoked [82]. Smoking rates have been reported as high as 38% among Vietnamese male high school students [84]. Another study reported that 8.6% of Filipino 8.3% of Korean, 7.4% of Japanese, 7.2% other Asian American and 2.8% of Chinese youth in California smoked [80].

Smoking rates decreased among Asian American/Pacific Islander adults from 2000 to 2004 (see Table 3). According to the 2004 NHIS, Asian American adults report lower current smoking than all racial and ethnic groups (11.3%) and rates were particularly low among Asian American women (4.8%) [4]. Data from the 1999–2001 NSDUH indicate that 16.2% of Asian Americans adults smoked [29]. Among ethnic groups, 12.3% of Chinese, 12.6% of Asian Indian, 14.8% of Filipino, 19.0% of Japanese, 26.5% of Vietnamese and 27.2% of Korean adults currently smoked [29]. Asian immigrants from South-east Asia (Vietnam, Cambodia and Laos) have reported rates ranging from 34 to 43% [1]. Current rates among Vietnamese men in California have been reported from 35 to 57% [85]. Korean men in California report high smoking rates (38.7%); however, Korean women report low rates (6.0%) [86]. The 1997 BRFSS data indicate that 17.3% of Asians/Pacific Islanders in Hawaii smoked [73]. Combined data among Native Hawaiians indicate that 28.9% of men and 27.6% of women smoked [87].

CPD

Few data have been published on the number of cigarettes smoked per day among Asian Americans/Pacific Islanders. Only 4.9% of Asian American 12th graders smoked a half-pack or more per day [30]. The percentage of adult light smokers (< 15 cpd) has increased [1]. More than 70% of Asians/Pacific Islanders smoked < 15 cpd [1]. Data from the Multiethnic Cohort indicate that 22% of Japanese American men smoked ≤ 10 cpd and 43.6%, 11–20 cpd, and among Native Hawaiians, 25.2% smoked ≤ 10 cpd and 39.7%, 11–20 cpd [32]. Among women, 53% of Japanese Americans smoked ≤ 10 cpd and 34.9%, 11–20 cpd, and among Native Hawaiians, 44.3% smoked ≤ 10 cpd and 37.1%, 11–20 cpd [32]. One study among smokers showed slower clearance of and reduced intake of nicotine among Chinese Americans compared to whites [88].

Quitting, treatment and relapse

Quit rates have increased since the 1970s [1]. In 2000 NHIS, 67.8% of Asian Americans desired to quit and 44.7% who have ever smoked quit [12]. In the 1998 survey of Medicare managed care enrolled smokers, 54% of Asian Americans/Pacific Islanders reported receiving advice to quit [33]. Data on Hawaiians indicate that 35.6% of men and 27.6% of women quit smoking [87]. Compared to Cambodian men, Laotian and Vietnamese men have lower rates of quitting [89]. Little is known about relapse behavior among Asian Americans/Pacific Islanders.

Cancer outcomes

Asian American/Pacific Islander men and women have the third lowest lung/bronchus cancer incidence and the second lowest death rates (see Tables 1 and 2). Asian Americans/Pacific Islanders have the highest incidence rates of stomach cancer [14]. Incidence rates for oral cancer decreased among all men except Asian Americans/Pacific Islanders [14]. Data from the California Multiethnic Cohort Study indicate that Native Hawaiian (263.9) lung cancer incidence rates were higher than whites (158.3), Japanese Americans (121.4) and Latinos/Hispanics (79.2), but equivalent to blacks/African Americans (263.9) [32]. Data from SEER 1988–99 indicate that the incidence and death rates of cancer among Hawaiians is lower than that of black/African American and white males [1].

Since the 1998 Surgeon General Report, Tobacco Use Among US Racial/Ethnic Minority Groups was published, new data have been published and provide additional insight on the tobacco disease continuum indicators. Much of the data combine prevalence, incidence and mortality rates across years to develop stable estimates for minority groups and data from states such as California help to increase our understanding of within ethnic group differences. However, gaps in knowledge still remain and inhibit our ability to understand the link between smoking and tobacco-related cancer outcomes. Additional information is needed on smoking initiation, ETS exposure, quitting and relapse patterns among minority racial/ethnic groups with a specific focus on within-group differences.

Other factors may also help to explain existing disparities. Poverty, education and occupation may impact TRHD independently and are likely to interact with race and ethnicity to impact disparities [90] at various points along the tobacco disease continuum. Race and ethnicity play important roles in understanding how minorities participate in systems which affect their poverty, levels of education and where they work [91]. Data on these interactions and stratified data are lacking and difficult to obtain because of small sample sizes.

Poverty and tobacco disease continuum indicators

Poverty is defined as a measure of family income less than the Census Bureau's official family money income threshold [20]. Family income thresholds vary by family size, but the official poverty thresholds are not adjusted to geographic location [20]. Cumulative adverse health effects result from living in poverty [92]. Population attributable death rates due to poverty increased between the 1970s and 1990s, especially among blacks/African Americans [93]. Poverty is an important variable to examine in tobacco-related health disparities because it is associated with multiple measures along the tobacco continuum; rates of poverty are more than twice as high among blacks/African Americans, American Indians/Alaska Natives and Latinos/Hispanics than whites, Asian Americans and Pacific Islanders; and the condition of poverty coupled with tobacco use might lead to multiple and cumulative disparities across the life-span.

Initiation and ETS

Low socio-economic status has predicted smoking initiation in longitudinal studies [94]. Studies have found that children in low-income areas are more likely to begin smoking than those in higher-income areas [95]. Children from ethnically diverse low-income and high-poverty neighborhoods are exposed to ETS [4], and exposure is as high as 89% among low SES children whose mothers' smoked [96]. Furthermore, ETS exposure among adults has been associated inversely with income [97]. However, some studies indicate that ETS exposure is low in low-income housing [98].

Current use

Individuals below poverty have consistently had higher rates of smoking than those at or above poverty and the disparities gap has not changed in the last 10 years (see Table 3). In 2004, 29.1% of adults below poverty were current smokers, with rates slightly higher among men (31.9%) compared to women (27.1%) [4].

Quitting, relapse and treatment

Individuals below poverty are less likely to quit smoking [99]. In 2000, 33.6% of ever smokers who were below poverty quit smoking compared to 49.9% of those at or above poverty [12]. Pregnant women below poverty were less likely to report a quit attempt [100]. Lower rates of quitting may be explained by unequal access to valid treatment [90]. Only half of Medicaid recipients in 1998 were covered for one or more tobacco dependence treatment [101] and few studies have investigated the effectiveness of cessation treatments for the poor [102].

Cancer outcomes

Disparities exist in lung cancer incidence and mortality among poor men and women. SEER trends indicate that the lung cancer incidence rate for men in high-poverty counties was at least 12% greater than men in low-poverty counties from 1975 to 1999. Lung cancer incidence from 1988 to 1992 increased with increasing census tract poverty rates for white and black/African American men and women and Asian American/Pacific Islander men. On the contrary, lung cancer incidence rates were higher in low-poverty census tracts than in

high-poverty census tracts for Latino/Hispanic men and women and American Indian/Alaska Native women. Trends in female lung cancer indicate that the incidence rate in high poverty counties was 11% greater than the rates for women in counties with poverty levels between 10 and 20% [103]. Compared to men in low-poverty counties, the lung cancer mortality for men in high-poverty counties was 7% greater in 1975 and 25% greater in 1999 [103]. Differences in lung cancer mortality among women diminished among area poverty groups from 1975 to the 1990s, and differences were no longer significant in 1999. Trends from 1975 to 1999 indicate that men in high-poverty areas experienced at least 18% higher lung cancer mortality than men in low-poverty areas [103].

Education and tobacco disease continuum indicators

Education, like poverty, has also been associated with health outcomes. Studies indicate widening gaps in mortality by education [104,105]. High levels of education, in general, are associated with lower risk of ill health and deaths due to cancer and cardiovascular disease across multiple cultures [106] and the life-span [107]. Cigarette smoking is associated with lower education and studies show differences in smoking by educational status.

Initiation and ETS

Education has been associated independently with initiation and exposure [52,53]. Studies show that children of parents with low education were more likely to try smoking [108–110]. One European study found that among women, higher education was associated with a higher relative risk of smoking initiation [111]. Low education has also been associated significantly with ETS exposure among young females [112] and ETS inversely associated with education among adults [97]. One study among women 40 and over found that higher ETS exposure was associated with lower education [70].

Current use

Thirty-day prevalence rates among children of parents with low education have decreased since 1999 [9]. Children of parents with low education are more likely to be current smokers [9,113–116], with the exception of children of parents with the lowest levels of education. Among 12th graders, 19.8% of those with the lowest parental education smoke compared to 18% of those with the highest parental education [9]. Among adults, smoking is more prevalent among the lower educated [23,112] and smoking rates have not been shown to have a direct inverse relationship to educational attainment [4,9]. Current smoking rates are highest among those with a General Education Development (GED) (39.6%) and lowest among those with a graduate degree (8%) (see Table 3). Data from the 2004 NSDUH indicate that among adults, 34.8% of those who had not completed a high school education, 30.4% of high school graduates who did not attend college, 29% of people with some college and 13.6% of college graduates smoked [117]. Full-time college students are less likely to smoke than 18–22-year-olds not enrolled full-time in college [117].

CPD

Little is known about the number of cpd among educational groups. Those with higher levels of education smoke fewer cpd [1]. One study found that as maternal education increased, the number of cigarettes smoked by adolescent offspring decreased [118].

Quitting, relapse and treatment

No direct inverse relationship exists between quitting smoking and educational attainment, but overall those with less education are less likely to quit successfully [52,59,119,120]. In the 2000 NHIS, ever quit rates were lowest among those with a GED certificate (33.6%), 55.8% among those with < 8 years of education, 53.7% among those with an associate degree and highest among those with a graduate degree (74.4%). Those with higher education are more likely to report higher mean quit attempts than lower education groups [7]. Although few studies have examined smoking relapse among the lower-educated, one study among lung cancer patients found that low education predicted current smoking and shorter time to relapse [121]. In general, advice to quit is lower among the less educated [122].

Cancer outcomes

Cancer rates are generally higher among those with low education, and lung cancer risk [123] and mortality is also higher among those with low education compared to high education groups [121]. Lung cancer mortality of men with elementary education or less was 32% higher than better educated men [124]. Another study also found a strong inverse education gradient for lung cancer [106]. Data from the California Multiethnic Cohort indicate that people attending some college have a decreased risk of lung cancer [32].

Occupation and tobacco disease continuum indicators

In addition to poverty and education, tobacco use coupled with occupational exposure to certain chemicals, dust, pesticides, radiation and industrial processes increases the risk for tobacco related diseases [6,125]. Nearly 65% of US citizens participate in the labor

force [126]. A larger proportion of Asian Americans and whites are employed in management/professional jobs compared to blacks/African Americans, American Indians/Alaska Natives, Hispanics/Latinos and Hawaiians/Pacific Islanders, many of whom are employed in service, sales and office positions. Differences in occupations may impact racial and ethnic differences in tobacco-related disparities.

Initiation and ETS

The work-place is a major source of environmental tobacco smoke exposure and about 40 000 deaths are due to environmental tobacco smoke each year [4]. ETS can decrease the productivity of the worker and cause employers to face liability for non-smoker's health [127]. Furthermore, ETS exposure among adults has been associated inversely with occupational class [97]. Blue-collar and service workers are significantly less likely to be protected by smoke-free policies than white-collar workers [128–130]. Bartenders and waiters/waitresses are less likely to be covered by a smoke-free policy and are more likely to be exposed to ETS even when covered by such a policy [129]. Migrant, seasonal and other workers who are exposed to tobacco leaves may suffer from green tobacco sickness, an occupational illness resulting from transdermal nicotine exposure [131]. The work-place may also contribute to smoking initiation. One-third of youth begin smoking regularly at work [132] and those in blue-collar and service position are more likely to initiate smoking earlier in life than white-collar workers [6,133,134].

Current use

One in three blue-collar and service workers smoke compared to one in five white-collar workers [135] and, in general, smoking rates are higher among blue-collar workers [6,133,136]. Rates vary within the blue-collar occupation [137]. In specific trades, such as construction workers, the prevalence can be as high as 40% [136].

CPD

Blue-collar workers (27.5%) and farm workers (27.0%) were more likely to smoke $\geq$ 25 cpd and estimates were significantly lower among white-collar (18.0%) and service workers (16.7%) [133]. Eight per cent of adolescent workers in service positions reported smoking > 10 cpd [138].

Quitting, relapse and treatment

Blue-collar and service workers are not as successful in quitting smoking as white-collar workers [6,138] even though they make the same number of quit attempts [135]. Fifty-one per cent of white-collar, 36.8% of blue-collar, 41.4% of farm workers and 32.8% of service workers quit successfully [133].

Cancer outcomes

Occupational exposures place workers at high risk for lung cancer, and some of these occupational agents are synergistic with smoking, increasing lung cancer risks [6,125,139] and diseases such as restrictive and chronic obstructive lung disease [4]. Studies in multiple countries indicate an occupational class gradient in lung cancer mortality among men [140].

In summary, those in poverty, with lower education and in service and blue-collar occupations generally fare worse across the tobacco disease continuum. Those with a GED certificate have higher smoking and lower quit rates than all educational levels. The effects of poverty on cancer vary by gender and racial ethnic group and among Latinos/Hispanics, those in the lowest poverty census tracts have lower cancer incidence than those in the highest poverty census tracts. Data on the number of cigarettes smoked per day by poverty level and education are lacking. While social factors may help to explain differences observed among racial/ethnic groups, focusing only upon 'disparities' in health is problematic because it is sanitized from any connection to structural inequality or other social contexts as producers of inequalities in health [141]. The question remains as to how to eliminate TRHD with the data that we have. Examining workforce diversity, inequities in research and inequalities in health care may help resolve challenges in eliminating disparities.

DIVERSITY

'Diversity' describes the integration and inclusion of races, ethnicities, genders and groups from different geographies, cultures and social classes into organizations, decision-making tiers, institutions and systems from which they are and have historically been excluded. Diversity creates a climate where there is variety in the quantity and quality of interactions [15] and equitable power brokering among stakeholders at multiple levels. Diversity may create research environments where the composition and group exchanges influence the generation and validation of research ideas, philosophy, conceptualization, theoretical approaches, design, implementation and interpretation. When health care organizations are diversified, health providers better understand medical conditions and treatments [142], and in classroom settings, diversity results in greater gains in learning and citizen engagement [143]. Students from diverse

backgrounds in training settings may help learners to challenge assumptions and broaden perspectives regarding racial, ethnic and cultural differences [142,144]. The integration and inclusion of various groups into different environments, structures and decision-making bodies may alter scientific thinking and approaches to tobacco health disparities research.

Diversity may mediate health outcomes [15,145] and is associated with improved access to care for minority patients and greater patient choice and satisfaction [15]. When provided with a choice, minority patients are more likely to select health care professionals of their own race or ethnicity [15] and are generally more satisfied with the care they receive from minority professionals [146,147]. Language-concordant physicians contribute to improved patient communications and healing [148]. Furthermore, racial and ethnic minority health care providers [149] and minority psychologists [150] are significantly more likely than their white peers to serve and practice in minority and medically underserved communities. Cantor *et al*. [151] found that minority and women physicians and those from lower socio-economic backgrounds are more likely to serve minority, low-income and Medicaid populations—those who often suffer from disparities.

Contact with minority patients may improve our understanding of the causes and consequences of racial/ethnic health disparities. Health providers and researchers from diverse backgrounds bring cultural perspectives and experiences to research and provider teams, which can increase the likelihood that socio-cultural issues influencing health outcomes will be addressed in research design and inquiry [145]. For example, some researchers have a wealth of knowledge on the relevance of cultural norms and experiences with tobacco, historical experience with mainstream cultures and researchers, healing practices, resiliencies and strengths, community social norms and historical relationships with tobacco (e.g. working in tobacco companies and on farms, receiving funding for social and cultural events). Health professionals with this knowledge may help to increase the acceptability and value of community experiences and practices. These professionals may also increase the acceptability of community-based participatory research and help to increase enrollment of minority patients into clinical trials [15] and intervention studies that seek to reduce or prevent tobacco use or exposure and improve time to diagnosis and treatment of tobacco-related cancers.

INEQUITIES

Attending to diversity in research and health organization practice may help us to address related inequities in research and intervention practice. 'Inequities' represent injustice and unfairness [18] in scientific practice and knowledge allocation. Inequities in research include inequities in scientific inquiry; validation and value for research and unique methodologies in small and traditionally underserved and understudied populations; the social integration of a health disparities agenda into mainstream tobacco control research; the application of research to populations affected by tobacco disparities; representation of underserved groups in research; the translation and delivery of effective interventions and services; and the development of applicable and relevant treatments for tobacco-related conditions.

Research priorities in non-mainstream cultures differ from priorities in mainstream cultures [152], which may contribute to an under-representation of tobacco-related research questions relevant to minorities. For example, in general, why do Alaska Native/American Indian youth and adults have the highest rates of tobacco use but lower rates of tobacco-related cancers compared to blacks/African Americans? While numerous community and institutional factors may impact the capacity to explore this question, understudied research may be impacted by perceived bias in the peer review system toward grants addressing disparities [152], the availability of funding mechanisms, interest of journals and acceptance of relevant peer-reviewed publications, and the value and reward within academic institutions and other organizations for tobacco-related disparities research.

Minority groups are typically under-represented in research [153]. For example, the National Health Interview Survey which reports annual tobacco prevalence in the United States does not collect data on US territories, Hawaiians and Pacific Islanders [4]. Under-representation inhibits the ability to analyze and detect between- and within-race/ethnic group differences and conduct multi-level analyses that incorporate social variables such as poverty, education and occupation. Minority groups are also under-represented because standard sampling methods may not capture prevalence rates among small minority groups such as lesbian, gay, bisexual or transgender (LGBT) minorities. Researchers are often slow to accept or even test alternative sampling methods (i.e. respondent-driven sampling) that may hold promise [154]. The lack of these data inhibits the development of prevention and cessation interventions for populations with the greatest risks for tobacco use (i.e. Alaska Natives, LGBT).

The gold standard, randomized controlled study designs, may not be feasible for some populations and the rejection of other designs suggests that there is a lack of sensitivity among grant reviewers for the context of research in racial/ethnic groups [152]. For example, when communities are small and closely connected, the

randomized control design may not be suitable. Cessation interventions among racial/ethnic groups are lacking and most are quasi-experimental designs [155]. Furthermore, few recommended interventions (e.g. advice to quit, nicotine replacement therapy) have proven effectiveness in reducing disparities among racial/ethnic groups. Inequities in scientific practice and knowledge allocation impact our ability to systemically address disparities related to tobacco.

INEQUALITIES

'Inequalities' are defined as the quality of being unequal or uneven [18]. Inequalities lie in access to care, quality of health care, socio-economic indicators that impact health care, and psychosocial and environmental resources (see Fig. 1). With the exception of psychosocial resources, multiple US national surveys track many of these inequalities.

The National Health Care Disparities Report, 2005 indicates that disparities in access to and quality of care among minority racial/ethnic and poor groups still exist [90]. For example, poor hospitalized smokers compared to high-income hospitalized smokers have increasing disparities related to receipt of advice to quit from health providers; this same inequality is observed among Latinos/Hispanics compared to non-Hispanic whites [90]. Access to health insurance from 1999 to 2003 was much lower among blacks/African Americans and American Indians/Alaska Natives compared to whites; among poor compared to high-income people; and among people with high school education or less compared to those with some college education. Latinos/Hispanics of every income and education level are less likely than non-Hispanic whites to have health insurance [90]. Even with access to care, inequalities still exist. Black/African American lung cancer patients are less likely to receive invasive staging for lung cancer, and even when there is staging, black lung cancer patients are less likely to receive recommendation for surgery [156]. Overall, blacks/African Americans, Latinos/Hispanics and American Indians/Alaskan Natives have poorer quality of care and worse access to care compared to whites [90] and factors such as discrimination and social prejudices may contribute to the disparities among racial/ethnic groups.

DISCUSSION

This paper suggests that in addition to addressing disparities, there is a need to address diversity, inequities and inequalities in efforts to eliminate TRHD. Data from national, state and local studies indicate that disparities represent relatively large or small differences between racial/ethnic groups and vary along the tobacco disease continuum. Based on the dose–response model for lung cancer risk [125], one might assume that duration of regular smoking and number of cigarettes smoked per day might support disease outcomes among racial/ethnic groups. Available evidence on blacks/African Americans, American Indians and Alaska Natives are not consistent with the model. Socio-economic differences among the racial/ethnic groups may not explain these inconsistencies fully. Additional data, strategies and models are needed to resolve the disparities puzzle among some minority racial/ethnic groups.

We should observe similar if not higher cancer incidence and death rates among American Indians/Alaska Natives compared to blacks/African Americans. American Indians/Alaska Natives begin using tobacco earlier in life, current smoking rates are substantially higher, the percentage of light smoking is lower, but quit rates slightly higher compared to blacks/African Americans. Smoking prevalence rates have consistently and substantially declined among blacks/African Americans, but not among American Indians/Alaska Natives. Only a small percentage of blacks/African Americans and American Indians/Alaska Natives smoke heavily; whites have substantially higher rates of heavy smoking compared to other racial/ethnic groups [1]. Socio-economic indicators for American Indians/Alaska Natives and blacks/African Americans are quite similar, yet we observe more devastating outcomes among blacks/African Americans.

Part of the challenge is that most American Indian/Alaska Native tobacco continuum indicators are reported in aggregate form, which impacts our ability to fully understand how consistent the epidemiological data are with the tobacco disease causal paradigm. Alaska Native cancer data indicate higher cancer incidence rates of six different tobacco-related cancers when compared to whites, [76] and regional differences in smoking parallel lung cancer rates among American Indians/Alaska Natives. Misclassification of American Indians/Alaska Natives in cancer registries may underestimate their cancer incidence and overestimate cancer among whites. One study found that 52.2% of cases in the Oregon, Washington and Idaho cancer data registries were misclassified; 92% were coded as white [157]. None of these factors explain differences in cancer outcomes among American Indians/Alaska Natives compared to blacks/African Americans.

Asian Americans/Pacific Islanders and Latinos/Hispanics experience few tobacco-related cancer disparities. Adult smoking rates are lower in these groups compared to other racial/ethnic groups, and other indicators along the tobacco disease continuum are similar for Asian Americans/Pacific Islanders and Latinos/Hispanics. One might assume that as both aggregate groups have the

lowest smoking rates, but substantially different socio-economic indicators, that Latinos/Hispanics might fare worse than Asian American/Pacific Islanders; but this is not the case. The higher lung cancer incidence rate for Latinos/Hispanics in low-poverty census tracts compared to high-poverty census tracts is puzzling. Focusing on variations in smoking among Puerto Ricans, Hawaiians, Pacific Islanders and South-east Asian men is needed to understand more clearly the dose–response model for these groups within their socio-economic context.

Occupation distribution, and therefore exposure to toxic chemicals, dust and fumes, may partially explain existing disparities [158]. One study found racial differences in exposure to environmental and occupational hazards [159] and another found increased risk for lung cancer among black/African American male farm workers, auto mechanics, painting machine operators, furnace operators, garbage collectors and cutting machines operators in Michigan [160]. Few studies have compared environmental risks for lung cancer and how work environment exposure may be experienced differentially among racial/ethnic groups. It is not known if occupational exposure to lung carcinogens varies when work-sites or work areas are predominately minority.

The experience, longevity and depth (e.g. 125%, 200%) of poverty may vary by race/ethnicity, geography, rural/urban, type of housing and neighborhood conditions, thereby resulting in differential effects that impact the tobacco disease continuum. Cumulative effects of economic deprivation may also be experienced differentially across the life-span [161,162]. For example, poverty is quite prevalent among the employed; 38.9% of those employed were below poverty [126]. The effects of economic deprivation across the life-span may vary for the working and non-working impoverished whose eligibility for and access to health, educational and social resources differ. The experience of poverty may also differ for racial/ethnic groups such as Latinos/Hispanics, who despite overall poor social indicators, suffer few tobacco-related cancer disparities.

Education only partially explains current smoking and quitting among blacks/African Americans and American Indians/Alaska Natives compared to whites [1]. High school dropout rates and misclassification of smoking are not likely to explain lower smoking rates among black/African American adolescents [1]; this may also be true for Latino/Hispanic youth who have the highest school dropout rates [71]. Even after controlling for parental socio-economic status, black/African American youth are still less likely to smoke compared to other racial/ethnic groups [1,49]. However, the experience of education may differ. In 2002, blacks/African Americans earned the lowest average incomes compared to other racial/ethnic groups with equivalent educational attainment, and the income gap increased with increasing educational attainment [21]. For blacks/African Americans, there may be an interactive effect of education–income–race/ethnicity across the tobacco disease continuum that may not be observed among some other racial/ethnic groups. In addition, because of the long history of oppression and racism in the United States against blacks/African Americans, one cannot rule out the differential effects of discrimination on blacks/African Americans compared to other minority racial/ethnic groups across the life-span.

Insufficient data are available to compare ETS exposure or hair cotinine levels among racial/ethnic groups, but some studies suggest that serum cotinine contributes to disparities through biological processes. Studies suggest that ETS exposure in children of light smokers is associated with the biologically effective dose of two known carcinogen–protein adducts (4-ABP and PAH) and general measures of genetic damage (SCEs), thereby suggesting that ETS exposure may cause molecular or genetic damage at any point in the life cycle [47]. Blacks/African Americans may metabolize nicotine more slowly than whites and Latinos/Hispanics [55,58], and a number of gene polymorphisms are being examined (i.e. CPY2A6) which might explain differences in enzyme levels and activity and thus differences in cotinine levels [55,163–168]. However, small differences in the prevalence of CYP2A6 polymorphisms may not explain fully the differences between cotinine levels and health outcomes [46]. Hypotheses about biological processes and gene polymorphisms may be critical steps to exploring alternative disease models for minority groups, but these hypotheses should be approached with caution; the quest for racial classification of genes will do little to reduce disparities among racial/ethnic groups.

Several caveats must be noted. A more comprehensive review of data on smoking among racial/ethnic groups prior to 1998 is reported in the 1998 Surgeon General Report. Psychosocial and cultural factors, community level influences, dependence/addiction, biological processes [169], menthol cigarettes, familial genes and susceptibility [170,171], gene polymorphisms and other factors may partially explain cancer disparities that exist, although we do not provide an exhaustive discussion of these factors. Such indicators are not normally part of surveillance systems and our goal here was to approach disparities from an epidemiological perspective using key national indicators. Several of these topics are discussed in other papers within this journal supplement. Biopsychosocial factors are discussed in Hammons *et al.* [172], dependence/addiction is discussed in Moolchan *et al.* [173] and community-based partnerships are discussed in Báezconde-Garbanati *et al.* [174]. The cancer risks of smoking mentholated compared to non-mentholated cigarettes have not been demonstrated [175], but studies

are examining how menthol contributes to carcinogenesis through increased toxicity, addictiveness and altered patterns of inhalation [176]. Unemployment was not examined as a social variable; however, national estimates indicate that 35% of the unemployed smoke [177] and unemployment is higher among blacks/African Americans, American Indians/Alaska Natives and Latinos/Hispanics compared to whites. Unemployment has been associated with smoking-related mortality [178,179]. Tobacco marketing, type of cigarette smoked (i.e. light), other forms of tobacco, other drugs, alcohol or dietary factors may interact with cigarette use or occur concurrently. Use of other tobacco is low in most racial/ethnic groups [1]. Among all cigarette users, 4.8% use smokeless tobacco [117]. Geographic region was not a focus, but tobacco use in cities and neighborhoods may far exceed national averages. For example, 44% of those sampled in Harlem were current smokers [180]. One final caveat is that we do not report specifically on LGBT racial/ethnic minorities because the epidemiological data are almost non-existent.

CONCLUSIONS

Approaches that address simultaneously TRHD, workforce diversity and empowerment, inequities in research and inequalities in access to care may provide the paradigm shift needed to accelerate progress in reducing disparities among minority racial and ethnic groups. Available data on indicators in the tobacco disease continuum challenge our assumptions about the dose–response model for minority racial/ethnic and low socioeconomic status groups and it is clear that alternative models are needed to understand more clearly the relationship between tobacco use and exposure and health consequences among these groups. A number of recommendations have been made in previous reports on how to reduce tobacco-related health disparities among minority racial/ethnic groups [1,181], yet few recommendations have been implemented. It is not our intention to reiterate those recommendations, but to encourage collective action to adopt a framework which focuses simultaneously on disparities, diversity, inequities and inequalities. We therefore recommend the following:

1 Support and create multi-ethnic and inclusive coalitions of researchers, practitioners, community organizations, families and policy makers who work collectively at the local, state and national levels to develop comprehensive solutions to eliminate TRHD in minority racial/ethnic groups.
2 Develop long-term concrete plans on how to increase, support and retain the future pipeline of researchers, practitioners and advocates and support intergenerational collaborations and shared leadership among stakeholders.
3 Work with policy makers, health systems, community service organizations, businesses and others to develop strategies that increase access, demand, availability and quality of care for underserved minority racial/ethnic groups.
4 Develop research–community partnerships that help to strengthen our ability to address inequities in research, application, translation and dissemination.
5 Work with coalitions at multiple levels to develop specific strategies to address social factors such as:
(a) reducing individual and community level poverty among minority racial/ethnic groups with the highest rates,
(b) increasing high school graduation rates among minority racial/ethnic groups and creating opportunities to support higher education, and
(c) developing strategies in work-places to protect workers in all social strata from environmental hazards.

While these recommendations merely scratch the surface, they serve to encourage collective action to eliminate the leading cause of preventable death in the United States and its causal factors. Furthermore, while research and public health efforts aim to reduce tobacco use among everyone, these recommendations serve as a specific call to action to eliminate disparities among minority racial and ethnic groups.

Acknowledgements

We extend our gratitude to the National Cancer Institute and the American Legacy Foundation who fund the Tobacco Research Network on Disparities. We thank Julie Volkman and D. B. Consulting for assisting with the preparation of the manuscript and Drs Cathy Backinger and Bradford Hesse for reviewing the manuscript.

References

1. U.S. Department of Health and Human Services. *Tobacco Use Among U.S. Racial/Ethnic Minority Groups.* Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease and Health Prevention and Health Promotion, Office on Smoking and Health; 1998.
2. U.S. Department of Health and Human Services. *Reducing Tobacco Use.* Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease and Health Prevention and Health Promotion, Office on Smoking and Health; 2000.
3. U.S. Department of Health and Human Services. *The Health Consequences of Smoking.* Atlanta, GA: U.S.

Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease and Health Prevention and Health Promotion, Office on Smoking and Health; 2004.

4. Centers for Disease Control and Prevention. *Percentage of Adults Who Were Current, Former, or Never Smokers, Overall and by Sex, Race, Hispanic Origin, Age, Education, and Poverty Status: National Health Interview Surveys, Selected Years—United States, 1965–2004.* Available at: http://www.cdc.gov/tobacco/data_statistics/tables/adult/table_2htm (accessed 14 June 2007).
5. Fagan P., King G., Lawrence D., Petrucci S. A., Robinson R. G., Banks D. *et al.* Eliminating tobacco-related health disparities: directions for future research. *Am J Public Health* 2004; **94**: 211–17.
6. U.S. Department of Health and Human Services. *The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace.* Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease and Health Prevention and Health Promotion, Office on Smoking and Health; 1985.
7. U.S. Department of Health and Human Services. *Women and Smoking.* Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease and Health Prevention and Health Promotion, Office on Smoking and Health; 2001.
8. Centers for Disease Control and Prevention. Cigarette use among high school students—United States, 1991–2003. *MMWR* 2004; **53**: 499–502.
9. Johnston L. D., O'Malley P. M., Bachman J. G., Schulenberg J. E. *Decline in Teen Smoking Appears to Be Nearing its End.* Ann Arbor, MI: University of Michigan News and Information Services; 2005. Available at: http://www.monitoringthefuture.org (accessed 3 January 2006).
10. Everett S. A., Warren C. W., Sharp D., Kann L., Husten C., Crossett L. S. Initiation of cigarette smoking and subsequent smoking behavior among U.S. high school students. *Prev Med* 1999; **29**: 327–33.
11. Trinidad D. R., Gilpin E. A., Lee L., Pierce J. P. Do the majority of Asian-American and African-American smokers start as adults? *Am J Prev Med* 2004; **26**: 156–8.
12. Centers for Disease Control and Prevention. Cigarette smoking among adults—United States, 2000. *MMWR* 2002; **51**: 642–5.
13. Stewart J. H. IV. Lung cancer carcinoma in African Americans. A review of the current literature. *Cancer* 2001; **91**: 2476–82.
14. Edwards B. K., Brown M. L., Wingo P. A., Howe H. L., Ward E., Ries L. A. G. *et al.* Annual report to the Nation on the status of cancer, 1975–2002, Featuring population-based trends in cancer treatment. *J Natl Cancer Inst* 2005; **97**: 1407–27.
15. *National Healthcare Disparities Report.* Rockville, MD: Agency for Healthcare Research and Quality; 2004. Available at: http://www.qualitytools.ahrq.gov/disparitiesreport (accessed 10 May 2006).
16. Institute of Medicine. In: Smedley B. D., Butler A. S., Bristow L. R., editors. *In the Nation's Compelling Interest. Ensuring Diversity in the Health-Care Workforce.* Washington, DC: The National Academies Press; 2004.
17. Nickens H. W. The health status of minority populations in the Unites States. *West J Med* 1991; **155**: 27–32.
18. Berube M. S., Jost D. A., Severynse M., Ellis K., editors. *Webster's New College Dictionary.* Boston, MA: Houghton Mifflin Company; 1999.
19. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *The Hispanic Population: 2000, Census 2000 Brief.* May 2001.
20. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *Income, Poverty and Health Coverage in the United States: 2004.* August 2005.
21. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *Educational attainment in the United States: 2003.* Population Characteristics, June 2004.
22. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *Occupations: 2000, Census 2000 Brief.* August 2003.
23. Griesler P. C., Kandel D. B. Ethnic differences in correlates of adolescent cigarette smoking. *J Adolesc Health* 1998; **23**: 167–80.
24. Headen S. W., Bauman K. E., Deane G. D., Koch G. G. Are the correlates of cigarette smoking initiation different for black and white adolescents? *Am J Public Health* 1991; **81**: 854–7.
25. Gilpin E. A., Emery S. L., Farkas A. L., Distefan J. M., White M. M., Pierce J. P. *The California Tobacco Control Program; a Decade of Progress, Results from the California Tobacco Surveys, 1990–1998.* La Jolla, San Diego, CA: University of California; 2001.
26. Sexton K., Adgate J. L., Church T. R., Hecht S. S., Ramachandran G., Greaves I. A. *et al.* Children's exposure to environmental tobacco smoke: using diverse exposure metrics to document ethnic/racial differences. *Environ Health Perspect* 2004; **112**: 392–7.
27. Freeman N. C. G., Schneider D., McGarvey P. Household exposure factors, asthma, and school absenteeism in a predominately Hispanic community. *J Expo Anal Environ Epidemiol* 2003; **13**: 169–76.
28. Centers for Disease Control and Prevention. Youth risk behavior surveillance—United States, 2005. *MMWR* 2006; **55**: 1–108.
29. Centers for Disease Control and Prevention. Prevalence of cigarette use among 14 racial ethnic populations—United States, 1999–2001. *MMWR* 2004; **53**: 49–52.
30. Wallace J. M. Jr, Bachman J. G., O'Malley P. M., Johnston L. D., Schulenberg J. E., Cooper S. M. Tobacco, alcohol, and illicit drug use: racial and ethnic differences among U.S. high school seniors, 1976–2000. *Public Health Rep* 2002; **117**: S67–75.
31. Kerner J. F., Breen N., Tefft M. C., Silsby J. Tobacco use among multi-ethnic Latino populations. *Ethn Dis* 1998; **8**: 167–83.
32. Haiman C. A., Stram D. O., Wilken L. R., Pike M. C., Kolonel L. N., Henderson B. E. *et al.* Ethnic and racial differences in the smoking-related risk of lung cancer. *N Engl J Med* 2006; **354**: 333–42.
33. Centers for Disease Control and Prevention. Receipt of advice to quit smoking in medicare managed care—United States, 1998. *MMWR* 2000; **49**: 797–801.
34. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *The Black Population: 2000, Census Brief.* August 2001.
35. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *We the People:*

Black Population in the United States, Census. Special Reports. August 2005.
36. National Cancer Institute. *Changing Adolescent Smoking Prevalence.* Smoking and Tobacco Control Monograph No. 14. Bethesda, MD: US Department of Health and Human Service, National Institutes of Health, National Cancer Institute; 2001. NIH Pub. No. 02-5086.
37. Moon-Howard J. African American women and smoking: starting later. *Am J Public Health* 2003; **93**: 418–20.
38. Geronimus A., Neidert L., Bound J. Age patterns of smoking in U.S. black and white women of childbearing age. *Am J Public Health* 1993; **83**: 1258–64.
39. Trinidad D. R., Gilpin E. A., Lee L., Pierce J. P. Has there been a delay in the age of regular smoking onset among African Americans? *Ann Behav Med* 2004; **28**: 152–7.
40. Robinson L. A., Kleges R. C., Zbikowski S., Glaser R. Predictors of risk for different stages of adolescent smoking in a biracial sample. *J Consult Clin Psychol* 1997; **65**: 653–62.
41. Royce J., Hymowitz N., Corbertt K., Hatrwell T., Orlandi M. Smoking cessation factors among African Americans and whites. COMMIT Research Group. *Am J Public Health* 1993; **83**: 220–6.
42. Taylor K. L., Kerner J. F., Gold K. F., Mandelblatt J. S. Ever vs. never smoking and an urban, multiethnic sample of Hatian-, Carribean-, and U.S.-born Blacks. *Prev Med* 1997; **26**: 855–65.
43. Kandel D. B., Kiros G. E., Schaffran C., Hu M. C. Racial/ethnic differences in cigarette smoking initiation and progression to daily smoking: a multilevel analysis. *Am J Public Health* 2004; **94**: 128–35.
44. Ellickson P. L., Orlando M., Tucker J. S., Klein D. L. From adolescence to young adulthood: racial/ethnic disparities in smoking. *Am J Public Health* 2004; **94**: 293–9.
45. Knights J. M., Eliopoulos C., Klein J., Greenwald M., Koren G. Passive smoking in children. Racial differences in systemic exposure to cotinine by hair and urine analysis. *Chest* 1996; **109**: 446–50.
46. Wilson S. E., Kahn R. S., Khoury J., Lanphear B. P. Racial differences in exposure to environmental tobacco smoke among children. *Environ Health Perspect* 2005; **113**: 362–7.
47. Tang D., Warburton D., Tannenbaum S. R., Skipper P., Santella R. M., Cereijido G. S. *et al.* Molecular and genetic damage from environmental tobacco smoke in young children. *Cancer Epidemiol Biomarkers* 1999; **8**: 427–31.
48. Pirkle J. L., Flegal K. M., Benert J. T., Brody D. J., Etzel R. A., Maurer K. R. Exposure of the U.S. population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991. *J Am Med Assoc* 1996; **275**: 1233–40.
49. Blum R. W., Beuhring T., Shew M. L., Bearinger L. H., Sieving R. E., Resnick M. D. The effects of race/ethnicity, income, and family structure on adolescent risk behaviors. *Am J Public Health* 2000; **90**: 1879–84.
50. Centers for Disease Control and Prevention. *Smoking Prevalence among U.S. Adults.* November 2005. Fact sheet. Available at: http://www.cdc.gov/tobacco/research_data/adults_prev/prevali.htm (accessed 11 May 2006).
51. King G., Polednak A. P., Bendel R., Hovey D. Cigarette smoking among native and foreign-born African Americans. *Ann Epidemiol* 1999; **9**: 236–44.
52. Novotny T. E., Warner K. E., Kendricks J. S., Remington P. L. Smoking by Blacks and Whites: socioeconomic and demographic differences. *Am J Public Health* 1988; **78**: 1187–9.
53. Watson J. M., Scarinci I. C., Kleges R. C., Muarry D. M., Weg M. V., DeBon M. *et al.* Relationships among smoking status, ethnicity, socioeconomic indicators, and lifestyle variables in a biracial sample of women. *Prev Med* 2003; **37**: 138–47.
54. Moolchan E. T., Franke F. H., Jaszyna-Gasior M. Adolescent nicotine metabolism: ethnoracial differences among dependent smokers. *Ethn Dis* 2006; **16**: 239–43.
55. Benowitz N. L., Perez-Stable E. J., Fong I., Modin G., Herrera B., Jacob P. III. Ethnic differences in N-glucuronidation of nicotine and cotinine. *J Pharmacol Exp Ther* 1999; **291**: 1196–203.
56. Caraballo R. S., Giovino G. A., Pechacek T. F., Mowery P. D., Richter P. A., Strauss W. J. Racial and ethnic differences in serum cotinine levels of cigarette smokers. Third National Health and Nutrition Examination Survey, 1988–1991. *J Am Med Assoc* 1998; **280**: 135–9.
57. King T. E. Jr, Brunetta P. Racial disparities in the rates of surgery for lung cancer. *N Engl J Med* 1999; **341**: 1231–3.
58. Perez-Stable E. J., Herrera M. S., Jacob P. III, Benowitz N. L. Nicotine metabolism and intake in black and white smokers. *J Am Med Assoc* 1998; **280**: 152–6.
59. Hatziandreau E. J., Pierce J. P., Lefkopoulou M., Fiore M. C., Mills S., Novotny T. E. *et al.* Quitting smoking in the United States in 1986. *J Natl Cancer Inst* 1990; **82**: 1402–6.
60. McWhorter W. P., Boyd G. M., Mattson M. E. Predictors of quitting smoking: the NHANES I follow-up experience. *J Clin Epidemiol* 1990; **43**: 1399–405.
61. Doescher M. P., Saver B. G. Physicians' advice to quit smoking. The glass remains half empty. *J Fam Pract* 2000; **49**: 543–7.
62. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *We the People: American Indians and Alaska Natives in the United States—Census 2000 Special Reports.* February 2006.
63. Norton I. M., Manson S. M. Research in American Indian and Alaska Native communities: navigating the cultural universe of values and process. *J Consult Clin Psychol* 1996; **64**: 856–60.
64. Bruerd B. Smokeless tobacco use among Native American school children. *Public Health Rep* 1990; **105**: 196–201.
65. Etzel R. A., Jones D. B., Schlife C. M., Lyke J. R., Dunaway C. E., Middaugh J. P. Saliva cotinine concentrations in young children in rural Alaska. *Arctic Med Res* 1991; **Suppl.**: 566–7.
66. Centers for Disease Control. Smokeless tobacco use in rural Alaska. *MMWR* 1987; **36**: 140–3.
67. Backinger C. L., Bruerd B., Kinney M. B., Szpunar S. M. Knowledge, intent to use, and use of smokeless tobacco among sixth grade schoolchildren in six selected U.S. sites. *Public Health Rep* 1993; **108**: 637–42.
68. Eichner J. E., Cravatt K., Beebe L. A., Blevins K. S., Stoddart M. L., Bursac Z. *et al.* Tobacco use among American Indians in Oklahoma: an epidemiologic view. *Public Health Rep* 2005; **120**: 192–9.
69. Renner C. C., Enoch C., Patten C. A., Ebbert J. O., Hurt R. D., Moyer T. P. *et al.* Iqmik: a form of smokeless tobacco used among Alaska natives. *Am J Health Behav* 2005; **29**: 588–94.

70. Stamatakis K. A., Brownson R. C., Luke D. A. Risk factors for exposure to environmental tobacco smoke among ethnically diverse women in the United States. *J Women Health Gen B* 2002; **11**: 45–51.
71. U.S. Department of Health and Human Services. National Institutes of Health. National Institute on Drug Abuse. *Drug Use Among Racial/Ethnic Minorities.* NIH publication No. 03-3888, 2003.
72. Centers for Disease Control and Prevention. Cancer mortality among American Indians and Alaska Natives—United States, 1994–1998. *MMWR* 2003; **52**: 704–7.
73. Bolen J. C., Rhodes L., Powell-Griner E. E., Bland S. D., Holtzman D. State-specific prevalence of selected health behaviors, by race and ethnicity—Behavioral Risk Factor Surveillance System, 1997. *Morb Mortal Wkly Rep CDC Surveill Summ* 2000; **49**: 1–60.
74. Kaplan S., Lanier A., Merritt R. K., Siegal P. Z. Prevalence of tobacco use among Alaska Natives: a review. *Prev Med* 1997; **26**: 460–5.
75. Hodge F. S., Fredericks L., Kipnis P. Patient and smoking patterns in northern California American Indian Clinics. Urban and rural contrasts. *Cancer* 1996; **1**: 1623–8.
76. Lanier A. P., Holck P., Kelly J., Smith B., McEvoy T. *Alaska Native Cancer Survival Report.* Anchorage, AK: Alaska Native Health Board; 1999.
77. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *The Native Hawaiian and Other Pacific Islander Population: 2000, Census 2000 Brief.* December 2001.
78. U.S. Census Bureau. Department of Commerce, Economics and Statistics Administration. *The Asian Population: 2000, Census 2000 Brief*. February 2002.
79. U.S. Census Bureau. *Facts for Features. Asian/Pacific American Heritage Month*, 2005. Available at: http://www.census.gov/PressRelease/www/releases/archives/facts_for_features_special_editions/004522.html (accessed 11 May 2006).
80. Chen X., Unger J. B., Cruz T. B., Johnson C. A. Smoking patterns of Asian American youth in California and their relationship with acculturation. *J Adolesc Health* 1999; **24**: 321–4.
81. Chen X., Unger J. B. Hazards of smoking initiation among Asian American and non-Asian adolescents in California: a survival model analysis. *Prev Med* 1999; **28**: 589–99.
82. Appleyard J., Messeri P., Haviland M. L. Smoking among Asian American and Hawaiian/Pacific Islander youth: data from the 2000 National Youth Tobacco Survey. *Asian Am Pac Isl J Health* 2001; **8**: 5–14.
83. Lew R., Yu J.-H. *A Policy Framework for Preventing and Reducing Tobacco Use in the Asian American and Pacific Islander Community.* Oakland, CA; APPEAL; 2002.
84. Wiecha J. M. Differences in patterns of tobacco use in Vietnamese, African-American, Hispanic, and Caucasian adolescents in Worcester, Massachusetts. *Am J Prev Med* 1996; **12**: 29–37.
85. Centers for Disease Control and Prevention. Cigarette smoking among Chinese, Vietnamese, and Hispanics—California 1989–1991. *MMWR* 1992; **41**: 362–7.
86. Lew R., Moskowitz J. M., Wismer B. A., Min K., Kang S. H., Chen A. M. *et al.* Correlates of cigarette smoking among Korean American adults in Alameda County, California. *Asian Am Pac Isl J Health* 2001; **9**: 49–60.
87. Maskarinec G., Dhakal S., Pagano I., Carlin L., Goodman M. L., Marchand L. L. *et al.* Ethnic difference in trend and determinants of cigarette smoking in Hawaii. *Ethn Dis* 2005; **15**: 316–23.
88. Benowitz N. L., Perez-Stable E. J., Herrera B., Jacob P. III. Slower metabolism and reduced intake of nicotine from cigarette smoking in Chinese-Americans. *J Natl Cancer Inst* 2002; **16**: 108–15.
89. Moeschberger M. L., Anderson J., Kuo Y. F., Chen M. S., Wewers M. E., Guthrie R. Multivariate profile of smoking in Southeast Asian men: a biochemical verified analysis. *Prev Med* 1997; **26**: 53–8.
90. *National Healthcare Disparities Report.* Rockville, MD: Agency for Healthcare Research and Quality; 2005. Available at: http://www.ahrq.gov/qual/nhdr05/nhdr05.htm (accessed 11 May 2006).
91. Smedley A., Smedley B. D. Race as biology is fiction, racism as a social problem is real. *Am Psychol* 2005; **60**: 16–26.
92. Kuh D., Ben-Shlomo Y., editors. *A Life Course Approach to Chronic Disease Epidemiology.* Oxford: Oxford University Press; 1997.
93. Hahn R. A., Eaker E. D., Barker N. D., Teutsch S. M., Sosniak W. A., Krieger N. Poverty and death in the United States. *Int J Health Serv* 1996; **26**: 507–19.
94. Conrad K. M., Flay B. R., Hill D. Why children start smoking cigarettes: predictors of onset. *Br J Addict* 1992; **87**: 1711–24.
95. Semmer N. K., Cleary P. D., Dwyer J. H., Fuchs R., Lippert P. Centers for Disease Control (CDC) Psychosocial predictors of adolescent smoking in two German cities: the Berlin-Bremen Study. *MMWR* 1987; **36**: 3S–10S.
96. Cornelius M. D., Goldschmidt L., Dempsey D. A. Environmental tobacco smoke exposure in low-income 6-year-olds: parent report and urine cotinine measures. *Nicotine Tob Res* 2003; **5**: 333–9.
97. Whitlock G., MacMahon S., Vander Hoorn S., Davis P., Jackson R., Norton R. Association of environmental tobacco smoke exposure with socioeconomic status in a population of 7725 New Zealanders. *Tob Control* 1998; **7**: 276–80.
98. Berman B. A., Wong G. C., Bastani R., Hoang T., Jones C., Goldstein D. R. *et al.* Household smoking behavior and ETS exposure among children with asthma in low-income, minority households. *Addict Behav* 2003; **28**: 111–28.
99. Flint A. J., Novotny T. E. Poverty status and cigarette smoking prevalence and cessation in the United States, 1983–1993: the independent risk of being poor. *Tob Control* 1997; **6**: 14–18.
100. Yu S. M., Park C. H., Schwalberg R. H. Factors associated with smoking cessation among U.S. pregnant women. *Matern Child Health J* 2002; **6**: 89–97.
101. Schauffler H. H., Barker D. C., Orleans C. T. Medicaid coverage for tobacco-dependence treatments. *Health Aff (Millwood)* 2001; **20**: 298–303.
102. Hahn E. J., Rayens M. K., Chirila C., Riker C. A., Paul T. P., Warnick T. A. Effectiveness of a quit and win contest with a low-income population. *Prev Med* 2004; **39**: 543–50.
103. Singh G. K., Miller B. A., Hankey B. F., Edwards B. K. *Area Socioeconomic Variations in U.S. Cancer Incidence, Mortality, Stage, Treatment, and Survival, 1975–1999.* NCI Cancer Surveillance Monograph Series, Number 4. Bethesda, MD: National Cancer Institute; 2003. NIH Publication No. 03-5417.
104. Duleep H. O. Mortality and income inequality among

economically developed countries. *Soc Secur Bull* 1995; **58**: 34–50.
105. Pappas G., Queen S., Hadden W., Fisher G. The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *N Engl J Med* 1993; **329**: 103–9.
106. Steenland K., Henley J., Thun M. All-cause and cause-specific death rates by educational status for two million people in two American Cancer Society cohorts, 1959–1996. *Am J Epidemiol* 2002; **156**: 11–21.
107. Evans T., Whitehead M., Diderichsen F., Bhuiya A., Wirth M. *Challenging Inequities in Health: From Ethics to Action*. New York: Oxford Press, Rockefeller Foundation; 2001.
108. Ary D. V., Biglan A. Longitudinal changes in adolescent cigarette smoking behavior: onset and cessation. *J Behav Med* 1988; **11**: 361–82.
109. Harrell J. S., Bangdiwala S. I., Deng S., Webb J. P., Bradley C. Smoking initiation in youth: the roles of gender, race, socioeconomics, and developmental status. *J Adolesc Health* 1998; **23**: 271–9.
110. Waldron I., Lye D. Employment, unemployment, occupation, and smoking. *Am J Prev Med* 1989; **5**: 142–9.
111. Crone M. R., Reijneveld S. A., Burgmeijer R. J., Hirasing R. A. Factors that influence passive smoking in infancy: a study among mothers of newborn babies in The Netherlands. *Prev Med* 2001; **32**: 209–17.
112. Scarinci I. C., Watson J. M., Slawson D. L., Klesges R. C., Murray D. M., Eck-Clemens L. H. Socioeconomic status, ethnicity, and environmental tobacco exposure among non-smoking females. *Nicotine Tob Res* 2000; **2**: 355–61.
113. Defacto. *Stivoro for a Smoke-Free Future.* Annual report 1999; 25 years Stivoro: a good start. The Hague: Defacto; 2000.
114. Fagan P., Brook J. S., Rubenstone E., Zhang C. Parental occupation, education, and smoking as predictors of offspring tobacco use in adulthood: a longitudinal study. *Addict Behav* 2005; **30**: 517–29.
115. Cavelaars A. E., Kunst A. E., Geurts J. J., Crialesi R., Grotvedt L., Helmert U. *et al.* Educational differences in smoking: international comparison. *Br Med J* 2000; **320**: 1102–7.
116. Defacto. *Stivoro for a Smokefree Future. 100 000 Heroes*. Annual report Stivoro 2000. The Hague: Defacto; 2002.
117. Substance Abuse and Mental Health Services Administration. *Results from the 2004 National Survey on Drug Use and Health.* (Office of Applied Studies, NSDUH Series H-27, DHHS Publication No. SMA 05-4061).Rockville, MD: 2005.
118. Conwell L. S., O'Callaghan M. J., Andersen M. J., Bor W., Najman J. M., Williams G. M. Early adolescent smoking and a web of personal and social disadvantage. *J Ped Health* 2003; **39**: 580–5.
119. Fiore M. C., Jorenby D. E., Baker T. B., Kenford S. L. Tobacco dependence and the nicotine patch: clinical guidelines for effective use. *J Am Med Assoc* 1990; **263**: 2760–5.
120. Winkleby M. A., Feldman H. A., Murray D. M. Joint analysis of three U.S. community intervention trials for reduction of cardiovascular disease. *J Clin Epidemiol* 1997; **50**: 645–58.
121. Huisman M., Kunst A. E., Mackenbach J. P. Inequalities in the prevalence of smoking in the European Union: comparing education and income. *Prev Med* 2005; **40**: 756–64.
122. Tomar S. L., Husten C. G., Manley M. W. Do dentists and physicians advise tobacco users to quit? *J Am Dent Assoc* 1996; **127**: 259–65.
123. Muscat J. E., Stellman S. D., Richie J. P. Jr, Wynder E. L. Lung cancer risk and workplace exposures in black men and women. *Environ Res* 1998; **76**: 78–84.
124. Martikainen P., Lahelma E., Ripatti S., Albanes D., Virtamo J. Educational differences in lung cancer mortality in male smokers. *Int J Epidemiol* 2001; **30**: 264–7.
125. International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Tobacco Smoke and Involuntary Smoking*, Vol. 83. Lyon: World Health Organization Marketing and Dissemination; 2004.
126. U.S. Census Bureau. U.S. Department of Commerce, Economics and Statistics Administration. *Employment Status: 2000, Census 2000 Brief.* August 2003.
127. U.S. Environmental Protection Agency. *Integrated Risk Information System.* Washington, DC: Office of Health and Environmental Assessment, Office of Research and Development, Environmental Protection Agency; 1994.
128. Gerlach K. K., Shopland D. R., Hartman A. M., Gibson J. T., Pechacek T. F. Workplace smoking policies in the United States: results from a national survey of more than 100 000 workers. *Tob Control* 1997; **6**: 199–206.
129. Shopland D. R., Anderson C. M., Burns D. M., Gerlach K. K. Disparities in smoke-free workplace policies among food service workers. *J Occup Environ Med* 2004; **46**: 347–56.
130. Wortley P. M., Caraballo R. S., Perderson L. L., Pechacek T. F. Exposure to secondhand smoke in the workplace: serum cotinine by occupation. *J Occup Environ Med* 2002; **44**: 503–9.
131. Arcury T. A., Quandt S. A., Preisser J. S., Bernert J. T., Norton D., Wang J. High levels of transdermal nicotine exposure produce green tobacco sickness in Latino farm workers. *Nicotine Tob Res* 2003; **5**: 315–21.
132. Borland R., Chapman S., Owen N., Hill D. Effects of workplace smoking bans on cigarette consumption. *Am J Public Health* 1990; **80**: 178–80.
133. Centers for Disease Control and Prevention. *Work, Smoking, and Health: A NIOSH Scientific Workshop.* Washington, DC: Department of Health and Human Servicer Publication # 2002-148; 2000.
134. Giovino G. A. Epidemiology of tobacco use in the United States. *Oncogene* 2002; **21**: 7326–40.
135. Barbeau E. M., Krieger N., Soobader M. J. Working class matters: socioeconomic disadvantage, race/ethnicity, gender, and smoking in NHIS 2000. *Am J Public Health* 2004; **94**: 269–78.
136. Nelson D. E., Kirkendall R. S., Lawton R. L., Chrismon J. H., Merritt R. K., Arday D. A. *et al.* Surveillance for smoking-attributable mortality and years of potential life lost, by state—United States, 1990. *MMWR* 1994; **43**: 1–8.
137. Lee D. J., LeBlanc W., Fleming L. E., Gomez-Marin O., Pitman T. Trends in US smoking rates in occupational groups: the National Health Interview Survey 1987–1994. *J Occup Environ Med* 2004; **46**: 538–48.
138. Sorensen G., Fagan P., Hunt K. M., Stoddard A. M., Girod K., Eisenberg M. *et al.* Changing channels for tobacco control with youth: developing an intervention for working teens. *Health Educ Res* 2004; **19**: 250–60.
139. Saracci R., Boffetta P. Interactions of tobacco smoking and other causes of lung cancer. In: Samet J. M., editor.

Epidemiology of Lung Cancer. New York: Marcel Dekker; 1994, p. 465–93.
140. Kunst E. A., Groenhof F., Mackenbach J. EU Working Group on Socioeconomic Inequalities in Health. Occupational class and cause specific mortality in middle aged men in 11 European countries: comparison of population based studies. *Br Med J* 1998; **316**: 1636–41.
141. Riska E. Towards gender balance: but will women physicians have an impact on medicine? *Soc Sci Med* 2001; **52**: 179–87.
142. Whitla D. K., Orfield G., Silen W., Teperow C., Howard C., Reede J. Educational benefits of diversity in medical school: a survey of students. *Acad Med* 2003; **78**: 460–6.
143. Gurin P., Dey E. L., Hurtado S., Gurin G. Diversity in higher education: theory and impact on educational outcomes. *Harvard Educ Rev* 2002; **72**: 330–66.
144. Cohen J. J. The consequences of premature abandonment of affirmative action in medical school admissions. *J Am Med Assoc* 2003; **289**: 1143–9.
145. Institute of Medicine. In: Haynes A. M., Smedley B. D., editors. *The Unequal Burden of Cancer: An Assessment of NIH Programs and Research for Minorities and the Medically Underserved.* Washington, DC: National Academy Press; 1999.
146. Laveist T. A., Nuru-Jeter A. Is doctor-patient race concordance associated with greater satisfaction with care? *J Health Soc Behav* 2002; **43**: 296–306.
147. Saha S., Komaromy M., Koepsell T. D., Bindman A. B. Patient-physician racial concordance and the perceived quality and use of health care. *Arch Intern Med* 1999; **159**: 997–1004.
148. Perez-Stable E. J., Napoles-Springer A., Miramontes J. M. The effects of ethnicity and language on medical outcomes of patients with hypertension or diabetes. *Med Care* 1997; **35**: 1212–19.
149. Komaromy M., Grumbach K., Drake M., Vranizan K., Lurie N., Keane D. The role of black and Hispanic physicians in providing health care for underserved populations. *N Engl J Med* 1996; **334**: 1305–10.
150. Turner C. B., Turner B. F. Who treats minorities? *Cult Divers Ment Health* 1996; **2**: 175–82.
151. Cantor J. C., Miles E. L., Baker L. C., Barker D. C. Physician service to the underserved. Implications for affirmative action in medical education. *Inquiry* 1996; **33**: 167–80.
152. Shavers V., Fagan P., Lawrence D., McCaskill-Stevens W., McDonald P., Browne D. *et al.* Barriers to racial/ethnic minority investigator application and successful competition for NIH funding. *J Natl Med Assoc* 2005; **97**: 1063–77.
153. Nevid J. S., Javier R. A. Preliminary investigation of a culturally specific smoking cessation intervention for Hispanic smokers. *Am J Health Promot* 1997; **11**: 198–207.
154. Magnani R., Sabin K., Saidel T., Heckathorn D. D. Review of sampling hard-to-reach and hidden populations for HIV surveillance. *AIDS* 2005; **19**: S67–S72.
155. Lawrence D., Graber J. E., Mills S. L., Meissner H. I., Warnecke R. Smoking cessation interventions in U.S. racial/ethnic minority populations: an assessment of the literature. *Prev Med* 2003; **36**: 204–16.
156. Lathan C. S., Neville B. A., Earle C. C. The effect of race on invasive staging and surgery in non-small-cell lung cancer. *J Clin Oncol* 2006; **24**: 1–5.
157. Becker T. M., Bettles J., Lapidus J., Campo J., Johnson C. J., Shipley D. *et al.* Improving cancer incidence estimates for American Indians and Alaska Natives in the Pacific Northwest. *Am J Public Health* 2002; **92**: 1469–71.
158. Sterling T. D., Weinkam J. J. Comparison of smoking-related risk factors among black and white males. *Am J Ind Med* 1989; **15**: 319–33.
159. Miller W. J., Cooper R. Rising lung cancer death rates among black men: the importance of occupation and social class. *J Natl Med Assoc* 1982; **74**: 253–8.
160. Swanson G. M., Lin C. S., Burns P. B. Diversity in the association between occupation and lung cancer among black and white men. *Cancer Epidemiol Biomarkers* 1993; **2**: 313–20.
161. Kreiger N. Theories for social epidemiology in the 21st century: an ecosocial perspective. *Int J Epidemiol* 2001; **30**: 668–77.
162. Davey Smith G., editor. *Health Inequalities: Life Course Approaches*. Bristol: Policy Press; 2003.
163. Ahijevych K., Tyndale R. F., Dhatt R. K., Weed H. G., Browning K. K. Factors influencing cotinine half-life during smoking abstinence in African American and Caucasian women. *Nicotine Tob Res* 2002; **4**: 423–31.
164. Benowitz N. L., Herrera. B., Jacob P. III. Mentholated cigarette smoking inhibits nicotine metabolism. *J Pharmacol Exp Ther* 2004; **310**: 1208–15.
165. Marsit C. J., Wiencke J. K., Liu M., Kelsey K. T. The race associated allele of Semaphorin 3B (SEMA3B) T415I and its role in lung cancer in African-Americans and Latino/Hispanic-Americans. *Carcinogenesis* 2005; **26**: 1446–9. Epub 2005 April 14.
166. Tricker A. R. Nicotine metabolism, human drug metabolism polymorphisms, and smoking behaviour. *Toxicology* 2003; **183**: 151–73.
167. Wu X., Amos C. I., Kemp B. L., Shi H., Jiang H., Wan Y. *et al.* Cytochrome P450 2E1 DraI polymorphisms in lung cancer in minority populations. *Cancer Epidemiol Biomarkers* 1998; **7**: 13–18.
168. Wu X., Zhao H., Amos C. I., Shete S., Makan N., Hong W. K. *et al.* p53 genotypes and haplotypes associated with lung cancer susceptibility and ethnicity. *J Natl Cancer Inst* 2002; **94**: 681–90.
169. Richie J. P., Carmella S. G., Muscat J. E., Scott D. G., Akerkar S. A., Hecht S. S. Differences in urinary metabolites of tobacco-specific ling carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone in Black and White smokers. *Cancer Epidemiol Biomarkers* 1997; **6**: 783–90.
170. Bailey-Wilson J. E., Amos C. I., Pinney S. M., Petersen G. M., de Andrade M., Wiest J. S. *et al.* A major lung cancer susceptibility locus maps to chromosome 6q23–25. *Am J Hum Genet* 2004; **75**: 460–74.
171. Cote M. L., Kardia S. L., Wenzlaff A. S., Ruckdeschel J. C., Schwartz A. G. Risk of lung cancer among white and black relatives of individuals with early-onset lung cancer. *J Am Med Assoc*2005; **293**: 3036–42.
172. Fernander A. F., Shavers V. L., Hammons G. J. A biopsychosocial approach to examining tobacco-related health disparities among racially classified social groups. *Addiction* 2007; **102** (Suppl. 2): 43–57.
173. Moolchan E. T., Fagan P., Fernander A. F., Velicer W. F., Hayward M. D., King G. *et al.* Addressing tobacco-related health disparities. *Addiction* 2007; **102** (Suppl. 2): 30–42.
174. Báezconde-Garbanati L., Beebe L. A., Pérez-Stable E. J. Building capacity to address tobacco-related disparities

among American Indian and Hispanic/Latino communities: conceptual and systemic considerations. *Addiction* 2007; **102** (Suppl. 2): 112–122.

175. Carpenter C. L., Jarvik M. E., Morgenstern H., McCarthy W. J., London S. J. Mentholated cigarettes and lung cancer risk. *Ann Epidemiol* 1999; **9**: 114–20.
176. Henningfield J. E., Djordjevic M. V. Menthol cigarettes. Research needs and challenges. *Nicotine Tob Res* 2004; **6**: S11–16.
177. Fagan P., Shavers V., Lawrence D., Gibson J. T., Ponder P. Cigarette smoking and quitting behaviors among unemployed adults in the United States. *Nicotine Tob Res* 2007; **9**: 241–8.
178. Kivimaki M., Vahtera J., Virtanen M., Elovainio M., Pentti J., Ferrie J. E. Temporary employment and risk of overall and cause-specific mortality. *Am J Epidemiol* 2003; **158**: 663–8.
179. Pasarin M. I., Borrell C., Brugal M. T., Diaz-Quijano E. Weighing social and economic determinants related to inequalities in mortality. *J Urban Health* 2004; **81**: 349–62.
180. Northridge M. E., Morabia A., Ganz M. L., Bassett M. T., Gemson D., Andrews H. *et al.* Contribution of smoking to excess mortality in Harlem. *Am J Epidemiol* 1998; **147**: 250–8.
181. National Cancer Institute. *Eliminating Tobacco-Related Health Summary Report*, NIH Publication No. 05-5283. Washington, DC: DHHS; 2005.

International Journal for Equity in Health

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The burden of non communicable diseases in developing countries

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Published: 14 January 2005

International Journal for Equity in Health 2005, **4**:2 doi:10.1186/1475-9276-4-2

Received: 27 July 2004
Accepted: 14 January 2005

This article is available from: http://www.equityhealthj.com/content/4/1/2

Abstract

Background: By the dawn of the third millennium, non communicable diseases are sweeping the entire globe, with an increasing trend in developing countries where, the transition imposes more constraints to deal with the double burden of infective and non-infective diseases in a poor environment characterised by ill-health systems. By 2020, it is predicted that these diseases will be causing seven out of every 10 deaths in developing countries. Many of the non communicable diseases can be prevented by tackling associated risk factors.

Methods: Data from national registries and international organisms are collected, compared and analyzed. The focus is made on the growing burden of non communicable diseases in developing countries.

Results: Among non communicable diseases, special attention is devoted to cardiovascular diseases, diabetes, cancer and chronic pulmonary diseases. Their burden is affecting countries worldwide but with a growing trend in developing countries. Preventive strategies must take into account the growing trend of risk factors correlated to these diseases.

Conclusion: Non communicable diseases are more and more prevalent in developing countries where they double the burden of infective diseases. If the present trend is maintained, the health systems in low-and middle-income countries will be unable to support the burden of disease. Prominent causes for heart disease, diabetes, cancer and pulmonary diseases can be prevented but urgent (preventive) actions are needed and efficient strategies should deal seriously with risk factors like smoking, alcohol, physical inactivity and western diet.

Background

For centuries, communicable diseases were the main causes of death around the world. Life expectancy was often limited by uncontrolled epidemics. After the second World War, with medical research achievements in terms of vaccination, antibiotics and improvement of life conditions, non communicable diseases(NCDs) started causing major problems in industrialized countries. Heart diseases, cancer, diabetes, chronic pulmonary and mental diseases became a real burden for health systems in developed countries. For a while, these diseases were associated with economic development and so called diseases of the rich. Then, by the dawn of the third millennium, NCDs appeared sweeping the entire globe, with an increasing trend in developing countries (Table 1) where, the transition imposes more constraints to deal with the double burden of infective and non-infective diseases in a poor environment characterized by ill-health systems. In 1990

Table 1: Evolution of NCDs in developing countries (in million) [1,8,9]

	Non-Communicable Diseases	Communicable Diseases + Maternal + Perinatal + Nutritional	Injuries	total
1990	18.7 (47%)	16.6 (42%)	4.2 (11%)	39.5 (100%)
2000	25.0 (56%)	14.6 (33%)	5.0 (11%)	45.0 (100%)
2020	36.6 (69%)	09.0 (17%)	7.4 (14%)	53.0 (100%)

the leading causes of disease burden were pneumonia, diarrhoeal diseases and perinatal conditions. By 2020, it is predicted that NCDs will account for 80 percent of the global burden of disease, causing seven out of every 10 deaths in developing countries, compared with less than half today[1,2].

Efficient (preventive)strategies are needed and urgent measures should be taken to control risk factors like tobacco, alcohol, obesity, blood pressure diet and inactivity. Otherwise, developing countries will be unable to provide their people with standard health care.

The costly and prolonged treatment of NCDs raises the equity problem between and within countries. As expressed by the WHO Director-General in his overview to the annual report[1], If a Japanese woman develop chronic diseases, excellent treatment and rehabilitation services will be available and she can expect to receive, on average, medications worth about US$ 550 per year and much more if needed. Meanwhile, a woman in Sierra Leone can expect, on average, medicines worth about US$ 3 per year and, if she survives middle age and develop chronic diseases then she will die prematurely as a consequence of inadequate treatment. The contrasts in opportunities of treatment exist also within developing countries; between poor and rich, cities and rural areas and also between men and women.

In previous papers, the authors proposed mathematical models dealing with the burden of diabetes and its complications[3], Dynamics of a disabled population[4], the effect of physical exercise[5] and a model of dengue fever[6]. The present paper is devoted to the burden caused by NCDs in developing countries. In order to reverse the increasing trend of this burden (or at least to control it), the focus is made on the risk factors associated with these diseases.

Different methods can be considered to quantify the burden of NCDs. In order to overcome the specific problems of each country, the most used method is the approach that measures the global burden of NCDs in terms of Disability Adjusted Life Years (DALYs) which is a combination of Years of Life Lost(YLL) through premature death, and Years Lived with Disability(YLD). Thus, DALY is thought of as one lost year of healthy life [7-9]]. For example, deaths from underweight every year rob the world's poorest children of an estimated total of 130 million years of healthy life[10]. According to this approach, the burden of adult NCDs account for 80% in developed countries and for 70% in middle-income countries. Even in the high-mortality regions of the world, almost 50% of the adult disease burden is attributable to NCDs.

Methods

Data from national registries and international sources are collected, compared and analyzed in order to show the trend of NCDs. Four diseases or cluster of diseases(Cardio-Vascular Diseases(CVDs), diabetes, cancers and chronic respiratory diseases) are considered to illustrate the growing burden of NCDs in developing countries. The main sources of data are the annual reports and regular publications released by the World Health Organization(WHO), World Heart Federation(WHF), Pan American Health Organization(PAHO), International Diabetes Federation (IDF), International Agency for Research on Cancer(IARC), Centre for Chronic Disease Prevention and Control(CCDPC), International Task Force for Prevention of Coronary Heart Disease and a multitude of websites and papers dealing with NCDs. The literature associated with these diseases in developed countries is abundant. However, despite the encouraging programmes and joint projects proposed by WHO and other organisms in the form of collaborative research agreements to developing countries, in order to support national registries, unreliable and insufficient data are still prevailing in most of these countries. Moreover, the release of health data is shadowed by the security vision in some countries. Extrapolations are needed in the case of missing or incomplete data. Consequently, more efforts are needed to convince health decision makers in low- and middle-income countries of the necessity to develop epidemiological studies that allow for preventive strategies making health policy at the centre of sustainable development.

Table 2: Deaths caused wordwide by specific diseases (× 10^3)

Deaths & % Disease	2002 [1]	1990 [8]
Ischaemic heart disease	7000 (12.6%)	6260 (12.4%)
Cerebrovascular disease	5400 (09.6%)	4380 (08.7%)
Lower Respiratory Diseases	3700 (06.6%)	4300 (08.5%)
COPD	2700 (04.8%)	2211 (04.4%)
Cancer(all types)	7100 (12.6%)	6200 (11.2%)
Diabetes	3200 (05.6%)	2400 (05.0%)

Results

According the World Health Organization's statistics, chronic NCDs such CVDs, diabetes, cancers, obesity and respiratory diseases, account for about 60% of the 56.5 million deaths each year and almost half of the global burden of disease. In 1990, 47% of all mortality related to NCDs was in developing countries, as was 85% of the global burden of disease and 86% of the DALYs attributable to CVDs. An increasing burden will be born mostly by these countries in the next two decades. The socio-economic transition and the ageing trend of population in developing countries will induce further demands and exacerbate the burden of NCDs in these countries. If the present trend is maintained, it is predicted that, by 2020, NCDs will account for about 70 percent of the global burden of disease, causing seven out of every 10 deaths in developing countries, compared with less than half today.

In 1990, approximately 1.3 billion DALYs were lost as a result of new cases of disease and injury, with the major part in developing countries. In 2002, these countries supported 80% of the global YLDs due to the double burden of communicable and non communicable diseases. Consequently, their people are not only facing higher risk of premature life(lower life expectancy) but also living a higher part of their life in poor health[1]. These remarks indicate that NCDs are exacerbating health inequities existing between developed and developing countries and also making the gap more profound between rich and poor within low and middle-income countries.

CVDs in developing countries

CVD is the name for the group of disorders of the heart and blood vessels and include hypertension (high blood pressure), coronary heart disease (heart attack), cerebrovascular disease (stroke), peripheral vascular disease, heart failure, rheumatic heart disease, congenital heart disease and cardiomyopathies. These diseases constitute the major contributor among NCDs (Table 2).

Worldwide, an estimated 17 million people die of these diseases, particularly heart attacks and strokes, every year. Once associated with industrialized countries, CVDs are now emerging or rapidely increasing in developing countries. Indeed, in 1998, 86% of the DALYs caused by CVDs were attributed to developing countries and in 1999 CVDs contributed to a third of global deaths with 78% in low- and middle-income countries. The trend is increasing, indicating that by the year 2010 CVDs will be the leading cause of death in developing countries as a consequence of lifestyle changes brought about by industrialization and urbanization in developing countries engaged in the socio-economic transition. CVDs are promoted by risk factors like tobacco use, alcohol, physical inactivity and unhealthy diet. Unfortunately, the harm caused by these risk factors affects the rise of life expectancy in developing countries[1,11,12].

The costly and prolonged care of CVDs in low-and middle-income countries often divert the scarce family and societal resources to medical care. Consequently, the lower socio-economic groups have greater prevalence of risk factors, higher incidence of disease and higher mortality.

Diabetes

The recent statistics released by the World Health Organization and the International Diabetes Federation are alarming[1,12](Table 3). The number of diabetes in the world is expected to increase from 194 Million in 2003 to 330 in 2030 with three in four living in developing countries. Moreover, in developed countries most people with diabetes are above the age of retirement, whereas in developing countries those most frequently affected are aged between 35 and 64 which makes the burden in terms of DALYs and YLDs heavier in poorer countries. Indeed, in some countries of the Middle East, one in four deaths in adults aged between 35 and 64 years is attributable to diabetes. The burden is exacerbated by the complications such as blindness, amputations and kidney failure for which diabetes is the leading cause, and the interfering action of CVDs which are responsible for between 50 and 80% of deaths in people with diabetes. The burden of pre-

Table 3: Diabetes prevalence (× 10^6) [13]

Country	2000		2030
India	31.7	India	79.4
China	20.8	China	42.3
United States	17.7	United States	30.3
Indonesia	8.4	Indonesia	21.3
Japan	6.7	Pakistan	14.9
Pakistan	5.2	Bangladesh	11.8
Russia	4.6	Brazil	11.3
Brazil	4.5	Japan	8.9
Italy	4.2	Italy	5.4
Bengladesh	3.2	Russia	5.3

Table 4: Cancer by types and numbers worldwide (× 10^3):

Cancer	**2000 [18] Incidence**	**%**	**2000 deaths**	**%**	**1990 Incidence**	**%**	**1990 [19] deaths**	**%**
Lung	1239	12.3	1103	17.8	1037	12.8	921	17.8
Breast	1050	10.4	373	6.0	796	9.8	314	6.1
Colorectal	945	9.4	492	8.0	783	9.7	437	8.4
Stomach	876	8.7	646	10.0	798	9.9	628	12.1
Liver	564	5.6	546	8.8	437	5.4	427	8.2
Prostate	543	5.4	204	3.3	396	4.9	165	3.2
Cervical	471	4.7	233	3.7	371	4.6	190	3.7
Oesophagus	413	4.1	337	5.4	316	3.9	286	5.5
Head&neck	390	3.9	207	3.3	306	3.8	162	3.1
Bladder	336	3.3	132	2.1	261	3.2	115	2.2
Other	3228	32.2	1934	31.0	2582	32.0	1537	30.0
Total	10055	100%	6209	53%	8083	100.0	5182	100.0

mature death from diabetes is similar to that of HIV/AIDS, yet the problem is largely unrecognised [13].

Studies in different countries have shown that diabetes is a costly disease accounting for between 2.5 and 15% of the total healthcare expenditure[3]. For the age category 20–79, the world annual direct cost is estimated to be over $153 billion and expected to double in 2025.

According to the National Institute of Diabetes and Digestive Kidney Disease(NIDDK) and the American Diabetes Association, diabetes was the sixth leading cause of death in 1999 with a direct cost of $44 billion and an indirect cost of $54 billion annually. In 2002, the direct and indirect cost totalled $132 billion.

In France, an estimation of $5.7 billion was given for the direct cost of diabetes, whereas, an equivalent cost of 5.2 billion, representing approximately 9% of the annual NHS budget, was given for UK in 2000.

The burden affects more and more developing countries as stressed by the different authors who attended the seventh congress of the Pan-African diabetes study group in 2001[14] and the Metabolic syndrome, type II diabetes, and atherosclerosis congress in 2004[15].

Cancer

Cancer is now a major cause of mortality throughout the world (Table 4). In the developed world, it is generally exceeded only by CVDs but developing countries are responsible for the globally increasing trend. Over 10 million new cases and over 7 million deaths from cancer occurred worldwide in 2000[1,2,16-19]]. The contribution of developing countries was 53% for incidence and 56% for deaths (Table 5). From 1990 to 2000, the incidence and deaths increased by 2.4% per annum.

Between 2000 and 2020, the total number of cases of cancer in the developed world is predicted to increase by 29% whereas, in developing countries an increase by 73% is

Table 5: Cancer in developing countries :incidence & deaths (× 10^3) in 2000 [18]

Cancer	Developing Incidence	%	Developing deaths	%
Lung	792	14.7	522	14.6
Breast	471	8.8	184	5.6
Colorectal	334	6.2	252	7.0
Stomach	543	10.1	417	11.7
Liver	457	8.5	443	12.4
Prostate	127	2.4	76	2.1
Cervical	379	7.0	194	5.4
Oesophagus	341	6.3	274	7.7
Head&neck	262	4.9	154	4.3
Bladder	124	2.3	65	1.8
Other	1546	71.2	992	27.8
Total	5376	100%	3563	57.4

expected (largely as a result of an increase in the number of old people and as a result of urbanization and change in dietary habits).

The incidence of cancers of the lung, colon and rectum, breast and prostate generally increases in parallel with economic development, while the incidence of stomach cancer usually declines with development[2].

Lung cancer

This is currently the most common cancer in the world. In developed countries, smoking causes over 80% of such cancers and generally, heavy smoking increases the risk by around 30-fold making lung cancer a major problem in developing countries where the consumption of tobacco is flourishing.

Breast cancer

According to the International Agency for Research on Cancer (IARC), there were over a million new cases in the world in the year 2000, making it the second most common in the world and the most common among women with 47% in developing countries. Although rates are five times higher in industrialized countries, the burden of disease is heavier in poorer countries because breast cancer is highly curable if detected early and, unfortunately, about 80% of the cases are detected at advanced stages in developing countries.

Colorectal cancer

Ranking at the third place, with incidence rates tenfold higher in developed than in developing countries, this type of cancer is assumed to be mainly related to dietary factors which account to up to 80% of the between-country differences in rates.

Stomach cancer

20 years ago, this cancer used to be the most common in the world. At the moment, it is the fourth most common in the world but the second most common in developing countries. Substantial evidence suggests that risk is increased by high intakes of some traditionally preserved salted food and that risk is decreased by high intakes of fruit and vegetables.

Liver cancer

Approximately 75% of cases occur in developed countries, the rate vary over 20fold between countries. In developing countries, ingestion of contaminated food is an important risk factor together with active hepatitis virus infection whereas, alcohol consumption is the main diet-related risk factor in the world.

Cervical cancer

80% of the new cases and deaths are occurring in developing countries where it constitutes a major health problem. In developed countries, screening programmes and early detection have led to a noticeable decline in cervical cancer incidence and mortality, whereas, the trend is stable or increasing in low- and middle-income countries owing to their limited health care resources but also to their ill-health systems generating inefficient (or no)strategies[20].

Oral cavity, pharynx and oesophagus

In developed countries these types of cancer are mainly correlated to alcohol and tobacco(up to 75% of such cancers are attributable to these two lifestyle factors).

In developing countries, around 60% of such cancers are thought to be a result of micronutrient deficiencies related to a restricted diet that is low in fruit and vegetables and animal products. There is also consistent evidence that

Table 6: Burden of disease and risk factors worldwide:year 2002 [1]

Risk factor	Deaths ($\times 10^3$)	% of total death	DALYs ($\times 10^3$)	% of total DALY
Hypertension	7141	12.8	64270	04.5
Tobacco	4907	08.8	59081	04.1
High cholesterol	4415	07.9	40437	02.8
Low fruit & veg	2726	04.9	26662	01.9
Overweight	2591	04.6	33415	02.3
Alcohol	1804	03.2	58323	04.0
Phys. inactivity	1922	03.4	19092	01.3

consuming drinks and foods at a very high temperature increases the risk for these cancers[2].

Pancreatic, endometrial, prostate and kidney cancers

These types of cancer are more common in industrialized countries. However, the fact that overweight/obesity is an established risk factor, their incidence is expected to increase in developing countries engaged in the socio-economic transition[2].

Chronic respiratory diseases

Chronic respiratory diseases represent a major burden for the health systems worldwide. Most developing countries have no standard protocols for assessing and managing chronic non communicable respiratory diseases such as Chronic Obstructive Pulmonary Disease (COPD) and Asthma. In these countries, the population afflicted by poverty and illiteracy, having very little (or no) access to health services, will die before the age of 40 years. They comprise 15% of the population in Latin America, 34% in Arab world, 45% in Sub-Saharan Africa and south-east Asia[21,22].

Respiratory diseases cause 15% of the global burden of disease. Worldwide, it is estimated that 600 million people suffer from COPD and 2.5 million deaths were attributed to these diseases in 2000. By 2020, COPD is expected to become the third most common cause of mortality in the world.

Discussion

Risk factors: the enemies

In the previous sections, we considered four classes of non communicable diseases, namely, CVDs, diabetes, cancer and chronic respiratory diseases. Despite some differences between these classes and into each class, they do have a common denominator which is the risk factors. Indeed, Tobacco, alcohol, high blood pressure, diet and physical inactivity were indicated, at different levels, as risk factors in the four classes of NCDs. Moreover, these risk factors are seen to affect people worldwide with an increasing tendency. (Table 6)

Globally, many of the risk factors for heart disease, diabetes, cancer and pulmonary diseases are due to lifestyle and can be prevented. Physical inactivity, western diet and smoking are prominent causes[23]. Tobacco is the enemy number one. It is the most important established cause of cancer but also responsible in CVDs and chronic respiratory diseases. Tobacco and diet are the principal risk factors, responsible for more than 40% of cancer deaths and incidence. Obesity and dietary habits are the principal risk factors for diabetes of type 2.

Tobacco[1,2,24]

In the 20 the century, approximately 100 million people died worldwide from tobacco-associated diseases such as cancers, chronic lung disease, diabetes and CVDs.

While tobacco consumption is falling in most developed countries, it is increasing in developing countries by about 3.4% per annum. Today, 80% of the 1.2 billion smokers in the world live in poorer countries where smoking prevalence among men is nearly 50% (48%) and 50% of the 5 million deaths attributed to smoking in 2000 occurred in developing countries, also responsible for the increase in deaths by more than one million during the last decade.

Tobacco remains the most important avoidable risk for the four classes of NCDs. It increases the risk of dying from coronary heart disease and cerebrovascular disease 2–3 fold. It increases the risk of many types of cancer, for lung cancer the risk is increased by 20–30fold. According to studies conducted in Europe, Japan and North America, 83–90% of lung cancers in men and 57–80 in women, are imputable to tobacco. Between 80 and 90 % of oesophagus, larynx and oral cavity are caused by tobacco and alcohol [17]. In developing countries, an estimated one-third of all cancer deaths was attributable to smoking in 1995.

Finally, tobacco exacerbates the conditions of people living with COPD and asthma.

Lifestyle[2,25-27]

Up to 80% of cases of coronary heart disease, and up to 90% of cases of types 2 diabetes, could potentially be avoided through changing lifestyle factors.

One-third of cancers could be avoided by eating healthily, maintaining normal weight, and exercising throughout life.

It was estimated that in high-risk populations, an optimum fish consumption of 40–60 grams per day would lead to approximately a 50% reduction in death from coronary heart disease. A recent study based on data from 36 countries, reported that fish consumption is associated with a reduced risk of death from all causes as well as CVD mortality. Unfortunately, the fish consumption is very low even in some countries known for their large fish stock like the north African region.

Daily intake of fresh fruit and vegetables in adequate quantity (400–500 grams per day), is recommended to reduce the risk of coronary heart disease, stroke and high blood pressure. But, once more, this is thwarted by the western lifestyle invading developing countries.

Overweight/Obesity[2,28]

Overweight and Obesity lead to adverse metabolic changes such as insulin resistance, increasing blood pressure and cholesterol. Consequently, they promote CVDs, diabetes and many types of cancer. Worldwide, overweight affects 1.2 billion of which 300 million are clinically obese. In some developed countries like USA, the prevalence reaches 60% but developing countries like Kuwait have also a very high prevalence. More and more children are suffering from overweight and obesity. However, the most contrasting phenomenon is to find Overweight/Obesity and malnutrition side by side in low- and middle-income countries and hence contributing to the growing burden afflicting these countries. According to the International Obesity Task Force (IOTF) and the WHO World Health report 2002, about 60% of diabetes globally can be attributable to overweight and obesity. In other respects, it is estimated that 60% of world's population do not do enough physical activity.

Alcohol[2]

Alcohol consumption has also increased in the last decades, with the major part of this increase imputable to developing countries. In 2000, Alcohol was responsible for nearly 2 million deaths in the world, representing 4% of the global disease burden. Moreover, alcohol was estimated to cause 20 to 30 % of oesophagus cancer, liver disease, epilepsy, motor vehicle accidents and other hazards.

Conclusions

Non communicable diseases are more and more prevalent in developing countries. These diseases are highly correlated to risk factors like smoking, alcohol, obesity, diet and inactivity. The World Health Organization and many other organisms and associations are urging health decision makers to develop efficient preventive strategies to halt the growing trend of NCDs through the control of risk factors. However, although most of developed countries have reacted by pragmatic measures, the trend remain globally passive mainly because developing countries have been, so far, satisfied with adopting national conventions and adhering to international recommendations instead of pragmatic decisions such as prohibiting smoking in public areas, controlling alcohol abusers, encouraging physical activity, promoting healthy diet and improving primary health care for screening and early detection of chronic diseases. In these countries, 2.8 billion people live with less than 2 dollars, 1.2 billion live with less than one dollar and 1.3 billion live on fragile and often remote rural ecosystems[29]. So, the behaviour can be partly explained by lack of means and poor budget affected to health care but, in general, bad management and absence of goodwill assume a large part of responsibility. For instance, many developing countries have signed the Framework Convention on Tobacco Control(FCTC) and voted laws that prohibits smoking in public areas but the laws are not executed. Also, in the absence of early detection, many people are diagnosed at advanced stages of cancer, cardiovascular diseases and diabetes complications. Also, in these countries, until recently, it was widely believed that economic development was a necessary prerequisite for improving a population health status and the health was often classified as a non productive sector. Now, politicians and health policy makers are timidly recognizing that investing in people's health is a necessary condition for economic development but energetic decisions are needed for the adoption of urgent and consequent strategies. The need for such strategies is enhanced by the fact that risk factors like cholesterol, tobacco, blood pressure, and obesity are no more a specificity of industrialized countries, they are becoming more prevalent in developing nations, where they double the burden of infectious diseases that have always afflicted poorer countries. Moreover, multinational companies have been competing fiercely to expand their sales in developing countries and western lifestyle is invading middle-income countries [2,30]. Adhesion to the Framework Convention on Tobacco Control(FCTC) and other international strategies must be taken seriously by developing countries facing the pandemics of NCDS.

Competing interests

The author(s) declare that they have no competing interests.

Authors' contributions

AB contributed by the collection of data concerning CVDs and diabetes and to English writing SB contributed by the collection of data concerning cancer and chronic pulmonary diseases.

The two authors contributed equally to the final version of the paper.

Acknowledgements

This paper is dedicated to Wiam Boutayeb for her sixteenth birthday of which fifteen years lived with diabetes.

References

1. The World health report: **Today's challenges.** [http://www.who.int/whr/2003/en.]. Geneva, World Health Organization
2. World Health Organisation: **Diet, Nutrition and the prevention of Chronic Diseases.** In *Technical report Series 916* Geneva, World Health Organization; 2003.
3. Boutayeb A, Twizell EH, Achouyab K, Chetouani A: **A mathematical model for the burden of diabetes and its complications.** *BioMedical Engineering Online* 2004, **3:**20.
4. Derouich M, Boutayeb A: **The effect of physical exercise on the dynamics of glucose and insulin.** *Journal of Biomechanics* 2002, **35:**911-917.
5. Boutayeb A, Chetouani A: **Dynamics of a disabled population in Morocco.** *BioMedical Engineering Online* 2003, **2:**2.
6. Derouich M, Boutayeb A, Twizell EH: **A model of dengue fever.** *BioMedical Engineering Online* 2003, **2:**4.
7. Mathers CD, Bernard C, Iburg KM, Inoue M, Fat DM, Shibuya K, Stein C, Tomijima N, Xu H: **Global Burden of Disease in 2002 data sources, methods and results.** . Paper 54 WHO
8. Burden of Disease Unit: **The global burden of disease in 1990.** [http://www.hsph.harvard.edu/organizations/bdu/GBDseries.html]. Harvard University Press
9. Hutubessy R, Chisholm D, Edejer TT: **Generalized cost-effectiveness analysis for national-level priority setting in health sector.** *Cost Eff Resour Alloc* 2003, **1:**8.
10. The World health report: **Reducing Risk: Promoting Health Life.** [http://www.who.int/whr/2002/en]. Geneva, World Health Organization
11. Lenfant C: **Can we prevent cardiovasculazr diseases in low- and middle-income countries?** *Bulletin of the World Health Organization* 2001, **79:**980-987.
12. Reddy KS: **Cardiovascular diseases in the developing countries: dimensions, determinants, dynamics and directions for public health action.** *Public Health Nutrition* 2002, **5:**231-237.
13. International Diabetes Federation(IDF): **Action Now: A joint initiative WHO and IDF.** [http://www.idf.org].
14. Belkhadir J: **Le diabète en milieu marocain en 2001.** [http://www.marocsante.com/html/diabetes].
15. MSDAC 2004: **Metabolic syndrome, type II diabetes, and atherosclerosis congress.** [http://http:/www.msdac.com].
16. World Health Organization and International Agency for Research on Cancer: **BIEANNIAL REPORT 2002–2003.** *International Agency for Research on Cancer, Lyon, France* 2003.
17. **Le code Européen contre le cancer** [http://telescan.nki.nl/code/fr_code.html]
18. GLOBOCAN: **Cancer Incidence, Mortality and Prevalence Worldwide.** 2000 [http://www-dep.iarc.fr./globocan/globocan.html].
19. Parkin DM, Pisani P, Ferlay J: **Global Cancer Statistics.** *CA CANCER J CLIN* 1999, **49:**33-64.
20. Sankaranrayanan R, Budukh AM, Rajkumar R: **Effective screening programmes for cervical cancer in low- and middle-income developing countries.** *Bulletin of the World Health Organization* 2001, **79:**954-962.
21. Ait-Khaled N, Enarson D, Bousquet J: **Chronic respiratory diseases in developing countries: the burden and strategies for prevention and management.** *Bulletin of the World Health Organization* 2001, **79:**971-979.
22. BPCO: **La Bronchopneumopathie Chronique Obstructive: le fardeau de la BPCO.** [http://www.doctissimo.fr/html/dossiers/bronchite.htm].
23. Alberti G: **Noncommunicable diseases: tomorrow's pandemics.** *Bulletin of the World Health Organization* 2001, **79:**906-1004.
24. This month's special theme: **Tobacco.** *Bulletin of the World Health Organization* 2000, **78:**866-948.
25. Stampfer MJ: **Primary prevention of coronary heart disease in women through diet and life style.** *N Engl J Med* 2000, **343:**16-22.
26. Diabetes Prevention Program Research Group: **Reduction in the incidence of type II diabetes with lifestyle intervention or metformin.** *N Engl J Med* 2002, **346:**343-403.
27. Key TJ: **The effect of diet on risk of cancer.** *Lancet* 2002, **360:**861-868.
28. Kenchaiah S, Evans JC, Levy D, Wilson PM, Benjamin EJ, Larson MG, Kannel WB, Vasan RS: **Obesity and the risk of heart failure.** *N Engl J Med* 2002, **347:**305-313.
29. The World Bank: **Sustainable development in a dynamic world.** In *World development Report 2003* The World Bank, Washington, DC; 2003.
30. Joossens L: **From public health to international law: possible protocols for inclusion in the Framework Convention on Tobacco Control.** *Bulletin of the World Helth Organization* 2000, **78:**930-937.

VOLUME 24 · NUMBER 14 · MAY 10 2006

JOURNAL OF CLINICAL ONCOLOGY

REVIEW ARTICLE

Patterns of Cancer Incidence, Mortality, and Prevalence Across Five Continents: Defining Priorities to Reduce Cancer Disparities in Different Geographic Regions of the World

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Submitted December 6, 2005; accepted January 30, 2006.

Supported in part by the Intramural Research Program of the National Institutes of Health/National Cancer Institute (Bethesda, MD).

Authors' disclosures of potential conflicts of interest and author contributions are found at the end of this article.

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0732-183X/06/2414-2137/$20.00

DOI: 10.1200/JCO.2005.05.2308

ABSTRACT

Efforts to reduce global cancer disparities begin with an understanding of geographic patterns in cancer incidence, mortality, and prevalence. Using the GLOBOCAN (2002) and Cancer Incidence in Five Continents databases, we describe overall cancer incidence, mortality, and prevalence, age-adjusted temporal trends, and age-specific incidence patterns in selected geographic regions of the world. For the eight most common malignancies—cancers of lung, breast, colon and rectum, stomach, prostate, liver, cervix, and esophagus—the most important risk factors, cancer prevention and control measures are briefly reviewed.

In 2002, an estimated 11 million new cancer cases and 7 million cancer deaths were reported worldwide; nearly 25 million persons were living with cancer. Among the eight most common cancers, global disparities in cancer incidence, mortality, and prevalence are evident, likely due to complex interactions of nonmodifiable (ie, genetic susceptibility and aging) and modifiable risk factors (ie, tobacco, infectious agents, diet, and physical activity). Indeed, when risk factors among populations are intertwined with differences in individual behaviors, cultural beliefs and practices, socioeconomic conditions, and health care systems, global cancer disparities are inevitable. For the eight most common cancers, priorities for reducing cancer disparities are discussed.

J Clin Oncol 24:2137-2150.

INTRODUCTION

Cancer health disparities reflect differences in cancer incidence, mortality, and prevalence among different populations.[1] Although the term health disparities is primarily used in the United States and health inequalities in other parts of the world, both terms can be similarly defined to indicate a difference or dissimilitude.[2] Cancer incidence is defined as the number of new cancer cases occurring in a defined population within a specified period of time.[3] Incidence is determined by exposure to etiologic factors and individual susceptibility and may be further affected by screening practices, health care access, and quality of care. Cancer mortality is influenced by cancer incidence, individual biologic factors, tumor characteristics and stage at diagnosis, and response to available treatment. Cancer prevalence represents the disease burden in a population at a specific time and is related to survivability, with the most curable or clinically controllable cancers comprising those with the highest prevalence.

In this article, we provide an overview of worldwide cancer incidence, mortality, and prevalence, along with a more extensive discussion of the eight most common cancers in the world. To identify priorities to reduce the global cancer burden, we review established risk factors and existing cancer prevention and control strategies for the major cancer sites.

MATERIALS AND METHODS

Overall Incidence, Mortality, and 5-Year Prevalence

Data for overall annual cancer incidence, annual mortality, and 5-year prevalence were obtained from the GLOBOCAN database for the year 2002.[4] Incidence rates were derived from cancer registries that encompass national populations or samples of populations from selected regions. Cause-specific mortality data were derived from national registration information. All incidence and mortality rates were age standardized to the World Standard Population and expressed per 100,000 person-years (PY). Five-year prevalence was expressed as the number of individuals alive 5 years after a diagnosis of cancer. As an indirect measure of cancer survival, a mortality-to-incidence rate ratio (MR:IR) was calculated by dividing the mortality rate by the incidence rate; MR:IR approaching 1.0 suggests a limited survival.

To identify regional disparities, we used geographic categories defined in GLOBOCAN, including world, more-developed countries (all regions of

Europe, Australia, New Zealand, North America and Japan), and less-developed countries (all regions of Africa, Central America, South America, all regions of Asia except Japan, Caribbean, Melanesia, Micronesia, and Polynesia). We also defined the following five geographic areas: Africa (Eastern Africa, Middle Africa, Northern Africa, Southern Africa, and Western Africa); Asia (Eastern Asia, South-Eastern Asia, South Central Asia, and Western Asia); Oceania (Australia and New Zealand); Central/South America (Central America and South America); Europe (Eastern Europe, Northern Europe, Southern Europe, and Western Europe); and North America (Northern America).

Age-Specific Incidence Patterns

Age-specific cancer incidence rates in 5-year age groups were obtained from Cancer Incidence in Five Continents database (CI5VIII) and expressed per 100,000 PY.[5] Using CI5VIII, we defined geographic areas to best approximate the five geographic areas in GLOBOCAN, (ie, Africa, Asia, Oceania, Europe, and North America). More-developed countries included all regions of Europe, Australia, New Zealand, North America and Japan; whereas less-developed countries included all regions of Africa, Central America, South America, and all regions of Asia except Japan. Age-specific incidence rate figures were generated with S-Plus 6.1 software (Insightful Corporation, Seattle, WA) using a log-log scale, as originally described by Armitage and Doll.[6,7]

Temporal Trends

Temporal patterns in selected geographic areas were assessed using incidence data from CI5 I-VIII. Incidence rates were plotted on a log-linear scale by five 5-year time periods (1973 to 1977, 1978 to 1982, 1983 to 1987, 1988 to 1992, and 1993 to 1997) and expressed per 100,000 PY, as previously described.[8] Geographic areas in each continent, except Africa, were selected based on availability of data over the specified time periods, and case numbers. No data were available from Africa. Geographic areas selected were United States (Surveillance, Epidemiology, and End Results database, white and black), United Kingdom (South Thames), Australia (South), Japan (Osaka Prefecture), Singapore (Chinese), Costa Rica, and Columbia (Cali). Data from each time period were not available for all geographic regions.[9]

WORLDWIDE OVERALL CANCER INCIDENCE, MORTALITY, AND PREVALENCE

In 2002, there were an estimated 10,864,499 new cancer cases (excluding skin cancer) worldwide (Fig 1), with 44.9% (n = 4,878,952) in Asia, 26.0% (n = 2,820,771) in Europe, 14.5% (n = 1,570,520) in North America , 7.1% (n = 766,575) in Central/South America, 6.0% (n = 649,760) in Africa, and 1.0% (n = 103,725) in Oceania. In general, the distribution of cancer deaths paralleled the distribution of cancer incidence. The number of 5-year prevalent cancer cases was highest in Asia, Europe, and North America. Geographic variation in overall incidence and mortality at least partly reflected the relative frequency of site-specific cancers occurring in more-developed compared with less-developed parts of the world.[10]

Worldwide incidence and mortality of site-specific cancers are depicted in Figure 2. Cancers of the lung, stomach, colon and rectum, liver, and esophagus are associated with the highest incidence worldwide, in addition to sex-specific malignancies of the female breast, uterine cervix, and prostate. Whereas cancers of the female breast and prostate are among the most common worldwide, mortality rates are comparatively low, yielding a low MR:IR, consistent with favorable survival. In contrast, cancers of the lung, liver, and esophagus are associated with high MR:IR approaching 1.00, indicative of poor survival. To more closely examine disparities among different geographic regions, next we consider the most common cancer sites individually.

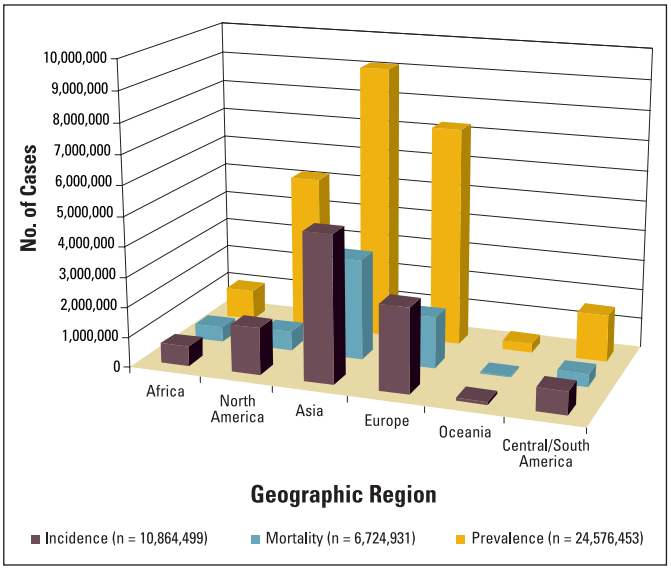

Fig 1. Worldwide overall annual cancer incidence, mortality, and 5-year prevalence (1993-2001).

Lung Cancer

With an estimated 965,446 new lung cancer cases per year among males and 386,875 cases per year among females, lung cancer is the most common cancer in the world and the leading cause of cancer-related mortality, accounting for 1,179,074 cancer deaths per year (Fig 2). During the years 1973 to 1997, lung cancer incidence rates either decreased or were stable among males in the geographic areas included in this study, except in Japan (Fig 3). In contrast, lung cancer rates generally increased among women in most regions (Fig 4), although this pattern may have moderated or ended in the late 1990s.[11]

Across continents, incidence rates varied from a low of 2.0 per 100,000 PY among females in Africa to a high of 61.2 per 100,000 PY among men in North America (Table 1). For both males and females, mortality rates were only slightly lower than incidence rates worldwide, yielding MR:IR ranging from 0.80 to 0.88. MR:IR rates were most favorable in North America and least favorable in Central/South America and Africa. Age specific incidence rate patterns were similar for both sexes in all parts of the world (Fig 5), increasing linearly with advancing age. Steadily rising age-specific incidence rates are consistent with long-term multistep carcinogenesis, purportedly requiring four to seven stochastic genetic changes over a lifetime of etiologic exposure.[12-14]

Cigarette smoking is by the far the most important risk factor for lung cancer.[15] Compared with nonsmokers, smokers have a 20-fold increased risk of developing lung cancer.[16] Other risk factors include occupational and indoor exposure to asbestos, radon progeny, and air pollution, as well as increasing age, genetic susceptibility, and perhaps low intake of fruits, vegetables, and micronutrients.[16-18]

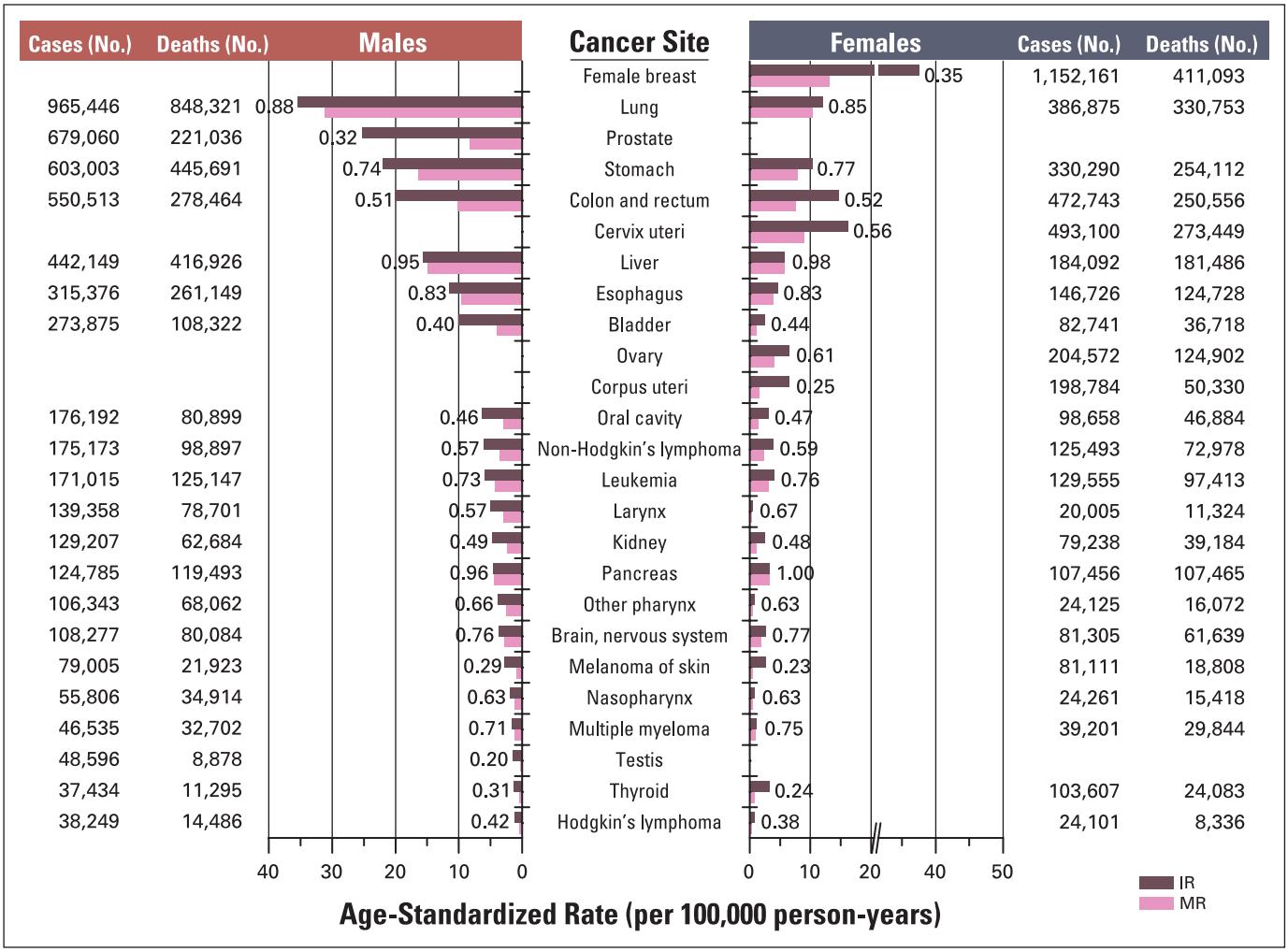

Fig 2. Worldwide annual number of cancer cases and cancer deaths, incidence rates (IRs), mortality rates (MRs), and mortality-to-incidence rate ratios (MR:IR; adjacent to bars) according to cancer site and sex (1993-2001). Reporting sources for IRs and MRs differ.

With a lag period of 20 to 30 years, patterns of lung cancer incidence closely follow smoking prevalence. Although lung cancer incidence currently is more common in the developed world, this pattern is expected to change in the next two decades. A dramatic rise in the incidence of lung cancer in China has been predicted, where smoking rates have markedly increased.[19] Estimates indicate that by 2030, 70% of tobacco-related deaths will occur in developing countries.[20]

Despite therapeutic advances, little gain has been achieved in overall lung cancer survival over the past 30 years, with approximately 15% 5-year survival rates for all stages combined.[21] Thus, conventional treatment remains an unsatisfactory means by which to decrease global lung cancer burden. Chemoprevention is theoretically possible for primary lung cancer prevention, although clinical trials have been disappointing to date.[22] Results from ongoing clinical trials to determine whether screening strategies will favorably impact lung cancer survival are awaited.[23]

Currently the most effective and important approach to reduce lung cancer burden worldwide is to reduce smoking rates through behavioral interventions and public health policy. Smoking cessation methods that are sensitive to country-specific needs and customs will be important to ensure successful outcomes.[20] To this end, in 2003 the World Health Assembly adopted the World Health Organization Framework Convention on Tobacco Control,[24] the first international treaty designed to enhance national and international coordination to control the tobacco epidemic. Entering into force in February 2005, the World Health Organization Framework Convention on Tobacco Control incorporates a variety of measures to counter the tobacco epidemic, including both concrete obligations or requirements and recommendations.[25] Requirements include restrictions on advertising, sponsorship, and promotion of tobacco products, and enforcement of packaging and labeling specifications. Recommendations include establishing clean indoor air controls and strengthening legislation against tobacco smuggling.

Female Breast Cancer

With an estimated 1,152,161 new cases each year, female breast cancer is the second most common cancer in the world and the most common cancer among women, accounting for 411,093 cancer deaths per year (Fig 2). Breast cancer incidence rates increased in all regions of the world included in this study during the years 1973 and 1997 (Fig 4), with the highest rates in Surveillance, Epidemiology, and

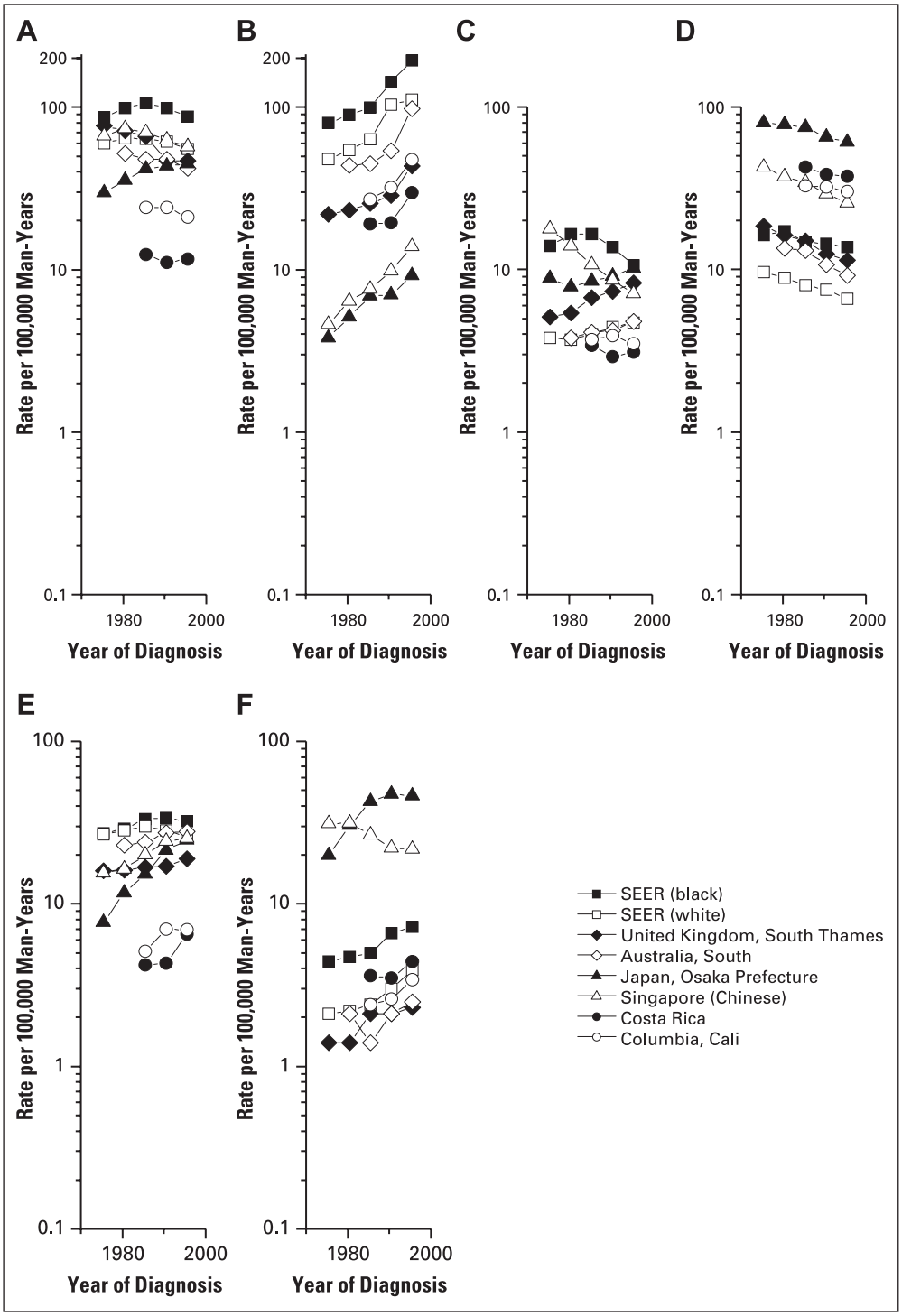

Fig 3. Age-standardized cancer incidence rates among males in various geographical areas according to specified cancer sites (1973-1997). (A) Lung; (B) prostate; (C) esophagus; (D) stomach; (E) colon; (F) liver. SEER, Surveillance, Epidemiology, and End Results database.

End Results (whites) and the lowest rates in Japan (Osaka Prefecture) and Costa Rica. Incidence rates were nearly three-fold higher in more-developed than less-developed geographic locations (67.8 to 23.8 per 100,000 PY; Table 1), whereas mortality rates were less than two-fold higher in more-developed than less-developed areas. Consequently, MR:IRs varied widely, from a low of 0.19 in North America to a high 0.69 in Africa.

Notwithstanding well-established reproductive risk factors such as early menarche, low parity, late age at first pregnancy, late menopause, and hormonal exposures,[26] the greatest risk factor for developing female breast cancer is aging.[27] However, while incidence rates for most epithelial cancers rise steadily with aging (Fig 5),[6,7] rates for female breast cancer increase rapidly until approximately age 50 years then rise more slowly. The midlife inflection in

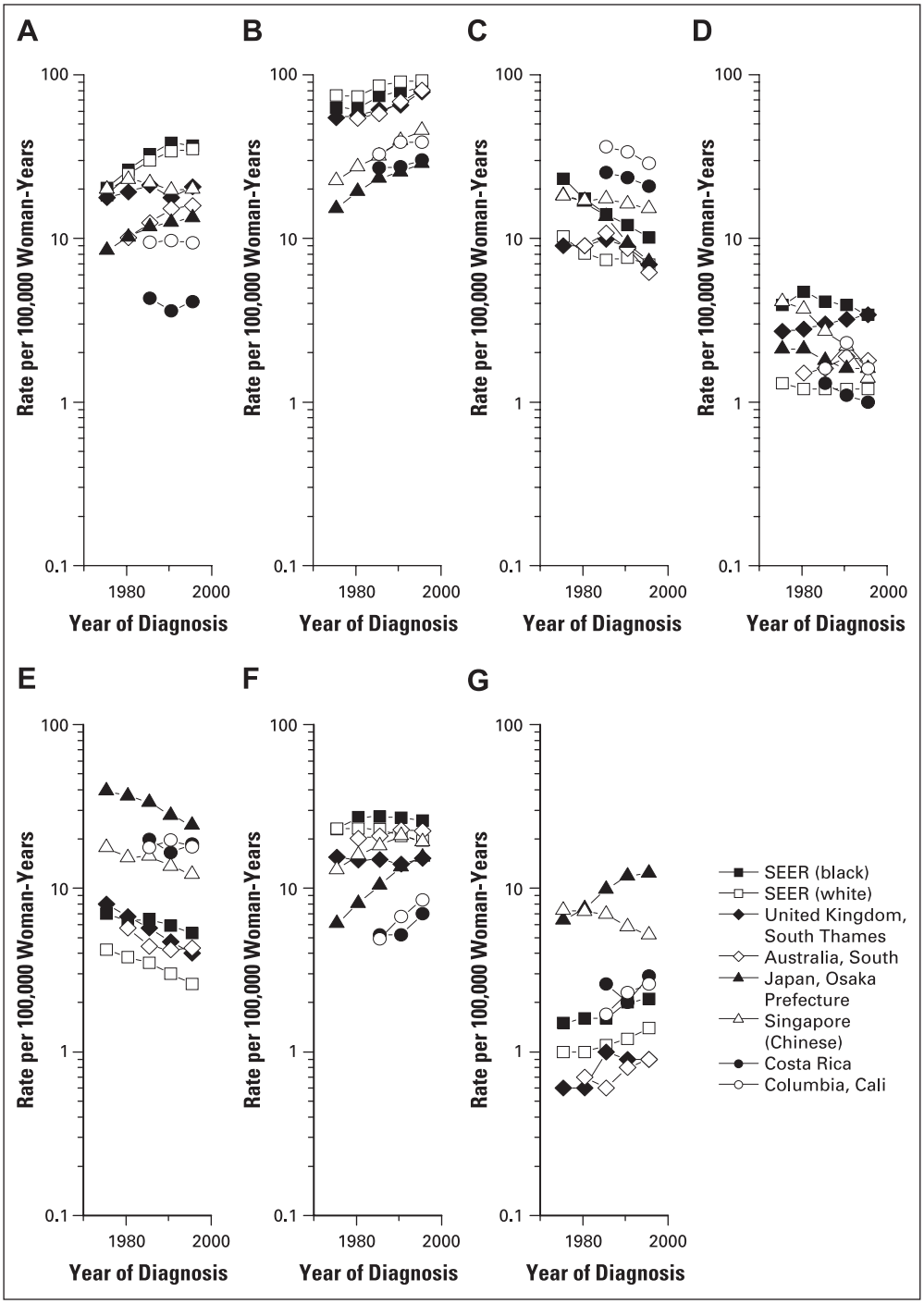

Fig 4. Age-standardized cancer incidence rates among females in various geographic areas according to specified cancer sites (1973-1997). (A) Lung; (B) breast; (C) cervix; (D) esophagus; (E) stomach; (F) colon; (G) liver. SEER, Surveillance, Epidemiology, and End Results database.

age-specific rates is termed Clemmesen's hook and has been attributed to menopause.[28-30]

Following Clemmesen's menopausal hook, breast cancer rate patterns among older women vary worldwide (Fig 5), continuing to rise more slowly in high-risk (or more-developed) areas and flattening or falling in low-risk (or less-developed) regions. Divergent age-specific rates after Clemmesen's hook have been attributed by some investigators to calendar-period and/or birth cohort artifacts,[31,32] whereas others have speculated the superimposition (or mixture) of two distinct rate patterns, corresponding to estrogen receptor (ER) -positive and ER-negative breast cancers.[33,34] Rates for ER-positive tumors increase rapidly until age 50 years then rise more slowly, similar to overall rates in high-risk countries. In contrast, rates for ER-negative tumors rise rapidly until age 50 years then plateau, similar to overall rates in low-risk countries.[34-36]

Accumulating molecular data also suggest the existence of two main breast cancer types according to ER expression.[37] Indeed, clinicians have long-suspected two types of breast cancer with age at onset being the major determinant of breast cancer outcome[38-41]; one type occurring in premenopausal women and characterized by aggressive

Table 1. Worldwide Age-Standardized Incidence Mortality Rates, and Mortality-to-Incidence Ratios for Selected Cancer Sites According to Sex (1993-2001)

	Males					Females				
	Incidence		Mortality			Incidence		Mortality		
Cancer	No.	Rate	No.	Rate	MR:IR*	No.	Rate	No.	Rate	MR:IR*
All sites, except skin										
World	5,802,531	209.6	3,796,383	137.7	0.66	5,061,968	161.5	2,928,548	92.2	0.57
More developed	2,698,175	314.1	1,503,060	169.6	0.54	2,317,939	228.0	1,185,412	102.5	0.45
Less developed	3,092,817	158.7	2,284,779	119.3	0.75	2,736,696	128.8	1,738,455	83.1	0.65
Continent										
North America	834,546	398.4	331,226	153.0	0.38	735,974	305.1	300,745	112.1	0.37
Oceania	56,119	349.7	24,812	149.1	0.43	47,606	280.3	19,611	103.4	0.37
Europe	1,499,642	290.7	958,248	180.8	0.62	1,321,129	209.7	743,224	103.6	0.49
Central/South America	365,497	198.9	221,243	122.6	0.62	401,078	182.2	215,395	99.2	0.54
Asia	2,697,813	167.0	1,983,473	124.3	0.74	2,181,139	126.1	1,372,455	79.9	0.63
Africa	311,363	126.0	251,099	104.1	0.83	338,397	121.0	255,013	92.6	0.77
Lung cancer										
World	965,446	35.5	848,321	31.2	0.88	386,875	12.1	330,753	10.3	0.85
More developed	481,950	54.9	423,507	47.6	0.87	194,731	17.0	161,472	13.6	0.80
Less developed	481,222	25.9	422,860	22.9	0.88	191,164	9.4	168,453	8.3	0.88
Continent										
North America	131,481	61.2	105,718	48.7	0.80	94,160	35.6	72,631	26.7	0.75
Oceania	6,539	39.1	5,848	34.7	0.89	3,285	17.4	2,820	14.6	0.84
Europe	296,364	56.8	270,832	51.1	0.90	78,396	11.3	70,765	9.8	0.87
Central/South America	38,587	21.8	35,615	20.3	0.93	15,646	7.3	15,291	7.2	0.99
Asia	472,791	30.1	411,414	26.3	0.87	188,559	11.0	162,648	9.5	0.86
Africa	14,752	7.0	14,172	6.7	0.96	4,776	2.0	4,559	1.9	0.95
Female breast cancer										
World	—	—	—	—	—	1,152,161	37.5	411,093	13.2	0.35
More developed	—	—	—	—	—	636,128	67.8	189,765	18.1	0.27
Less developed	—	—	—	—	—	514,946	23.8	221,028	10.4	0.44
Continent										
North America	—	—	—	—	—	229,631	99.4	48,239	19.2	0.19
Oceania	—	—	—	—	—	13,507	84.6	3,338	19.4	0.23
Europe	—	—	—	—	—	360,746	62.3	129,010	19.7	0.32
Central/South America	—	—	—	—	—	90,147	41.0	30,361	14.0	0.34
Asia	—	—	—	—	—	385,853	22.1	152,967	8.8	0.40
Africa	—	—	—	—	—	65,197	23.4	44,399	16.2	0.69
Colorectal cancer										
World	550,513	20.1	278,464	10.2	0.51	472,743	14.6	250,556	7.6	0.52
More developed	353,390	40.0	159,914	17.7	0.44	312,341	26.6	153,980	12.3	0.46
Less developed	196,093	10.2	118,042	6.2	0.61	159,730	7.7	96,222	4.7	0.61
Continent										
North America	94,745	44.4	33,421	15.3	0.34	88,728	32.8	32,939	11.6	0.35
Oceania	7,897	48.2	3,247	19.4	0.40	7,002	36.9	2,786	14.1	0.38
Europe	192,982	36.1	102,228	18.7	0.52	178,724	24.2	101,067	12.8	0.53
Central/South America	25,837	14.3	13,072	7.3	0.51	27,995	13.0	14,457	6.7	0.52
Asia	213,456	13.3	113,204	7.1	0.53	156,240	9.1	87,541	5.1	0.56
Africa	12,778	5.4	11,525	4.8	0.89	10,903	4.2	9,738	3.7	0.88
Stomach cancer										
World	603,003	22.0	445,691	16.3	0.74	330,290	10.3	254,112	7.9	0.77
More developed	195,782	22.3	128,721	14.5	0.65	115,372	10.0	83,515	6.9	0.69
Less developed	404,788	21.5	315,249	17.0	0.79	213,804	10.4	169,777	8.3	0.80
Continent										
North America	15,742	7.4	9,096	4.2	0.57	9,158	3.4	6,205	2.2	0.65
Oceania	1,632	9.9	1,005	6.0	0.61	828	4.2	603	3.0	0.71
Europe	104,620	20.0	83,280	15.7	0.79	69,394	9.5	57,626	7.6	0.80
Central/South America	39,480	22.0	29,400	16.5	0.75	25,597	11.8	19,528	9.0	0.76
Asia	425,146	26.9	307,755	19.6	0.73	211,520	12.3	157,340	9.2	0.75
Africa	13,836	6.2	13,002	5.8	0.94	12,350	4.9	11,633	4.6	0.94

(continued on following page)

Table 1. Worldwide Age-Standardized Incidence Mortality Rates, and Mortality-to-Incidence Ratios for Selected Cancer Sites According to Sex (1993-2001) (continued)

	Males					Females				
	Incidence		Mortality			Incidence		Mortality		
Cancer	No.	Rate	No.	Rate	MR:IR*	No.	Rate	No.	Rate	MR:IR*
Prostate cancer										
World	679,060	25.3	221,036	8.2	0.32	—	—	—	—	—
More developed	513,464	56.2	130,382	13.5	0.24	—	—	—	—	—
Less developed	165,401	9.4	90,550	5.2	0.55	—	—	—	—	—
Continent										
North America	257,943	119.9	36,447	15.8	0.13	—	—	—	—	—
Oceania	13,486	79.9	3,206	18.1	0.23	—	—	—	—	—
Europe	225,226	40.0	83,064	14.2	0.36	—	—	—	—	—
Central/South America	73,112	42.9	29,606	17.4	0.41	—	—	—	—	—
Asia	70,317	4.7	40,126	2.7	0.57	—	—	—	—	—
Africa	29,663	16.0	23,564	12.8	0.80	—	—	—	—	—
Liver cancer										
World	442,149	15.7	416,926	14.9	0.95	184,092	5.8	181,486	5.7	0.98
More developed	74,253	8.5	71,153	8.0	0.94	36,151	3.0	38,083	3.0	1.00
Less developed	365,923	18.4	343,956	17.4	0.95	147,210	7.1	142,728	6.9	0.97
Continent										
North America	11,058	5.3	9,229	4.4	0.83	5,152	1.9	5,319	1.9	1.00
Oceania	622	3.9	573	3.5	0.90	239	1.3	292	1.5	1.15
Europe	35,301	6.7	36,136	6.7	1.00	18,315	2.5	21,351	2.7	1.08
Central/South America	7,233	4.0	10,818	6.0	1.50	7,119	3.3	10,940	5.1	1.55
Asia	350,291	21.3	323,078	19.7	0.92	135,248	7.9	125,703	7.3	0.92
Africa	35,812	14.8	35,215	14.6	0.99	16,912	6.2	16,638	6.2	1.00
Cervical cancer										
World	—	—	—	—	—	493,100	16.2	273,449	9.0	0.56
More developed	—	—	—	—	—	83,437	10.3	39,512	4.0	0.39
Less developed	—	—	—	—	—	409,269	19.1	233,727	11.2	0.59
Continent										
North America	—	—	—	—	—	14,670	7.7	5,796	2.3	0.30
Oceania	—	—	—	—	—	1,063	7.4	330	2.0	0.27
Europe	—	—	—	—	—	59,931	11.9	29,812	5.0	0.42
Central/South America	—	—	—	—	—	65,493	29.0	29,524	13.4	0.46
Asia	—	—	—	—	—	265,744	15.4	142,679	8.4	0.55
Africa	—	—	—	—	—	78,897	29.3	61,671	23.1	0.79
Esophageal cancer										
World	315,376	11.5	261,149	9.6	0.83	146,726	4.7	124,728	3.9	0.83
More developed	57,889	6.8	50,295	5.8	0.85	15,986	1.3	14,827	1.2	0.92
Less developed	256,217	13.7	209,851	11.4	0.83	130,215	6.5	109,478	5.4	0.83
Continent										
North America	12,111	5.8	10,778	5.1	0.88	3,625	1.3	3,330	1.2	0.92
Oceania	905	5.5	801	4.8	0.87	465	2.3	370	1.8	0.78
Europe	33,070	6.5	29,659	5.7	0.88	9,970	1.3	9,474	1.2	0.92
Central/South America	10,479	5.9	9,631	5.4	0.92	3,748	1.8	3,516	1.7	0.94
Asia	241,301	15.3	193,465	12.4	0.81	120,443	7.1	99,908	5.9	0.83
Africa	16,289	7.8	15,659	7.5	0.96	8,058	3.4	7,743	3.2	0.94

NOTE. All rates are age standardized to the world population and expressed per 100,000 person-years.
Abbreviations: MR:IR, mortality-to-incidence rate ratio.
*MR:IR may exceed 1.00 because of different reporting sources for incidence and mortality rates.

and largely ER-negative features with a second type occurring in postmenopausal women and associated with indolent and mostly ER-positive features.

ER expression also varies by morphologic subtype of breast cancer.[42-45] For example, ER-negative tumors are most common in medullary breast carcinomas.[42] Notably, medullary carcinomas are rare tumors that have been associated with germline mutations in *BRCA1*,[46-61] and are more common in low-risk, native Japanese[35,44,62-65] and African[66] populations than in high-risk United States populations. On the other hand, tubular and lobular carcinomas have been associated with ER-positive breast cancers, germline mutations in *BRCA2*,[46,54,57,60,61] and are more common in high-risk populations such as the United States.[44,67]

A predominance of early-onset and aggressive ER-negative breast cancers in low-risk countries may, in part, account for the high MR:IR in less-developed regions such as Asia (0.40 per 100,000

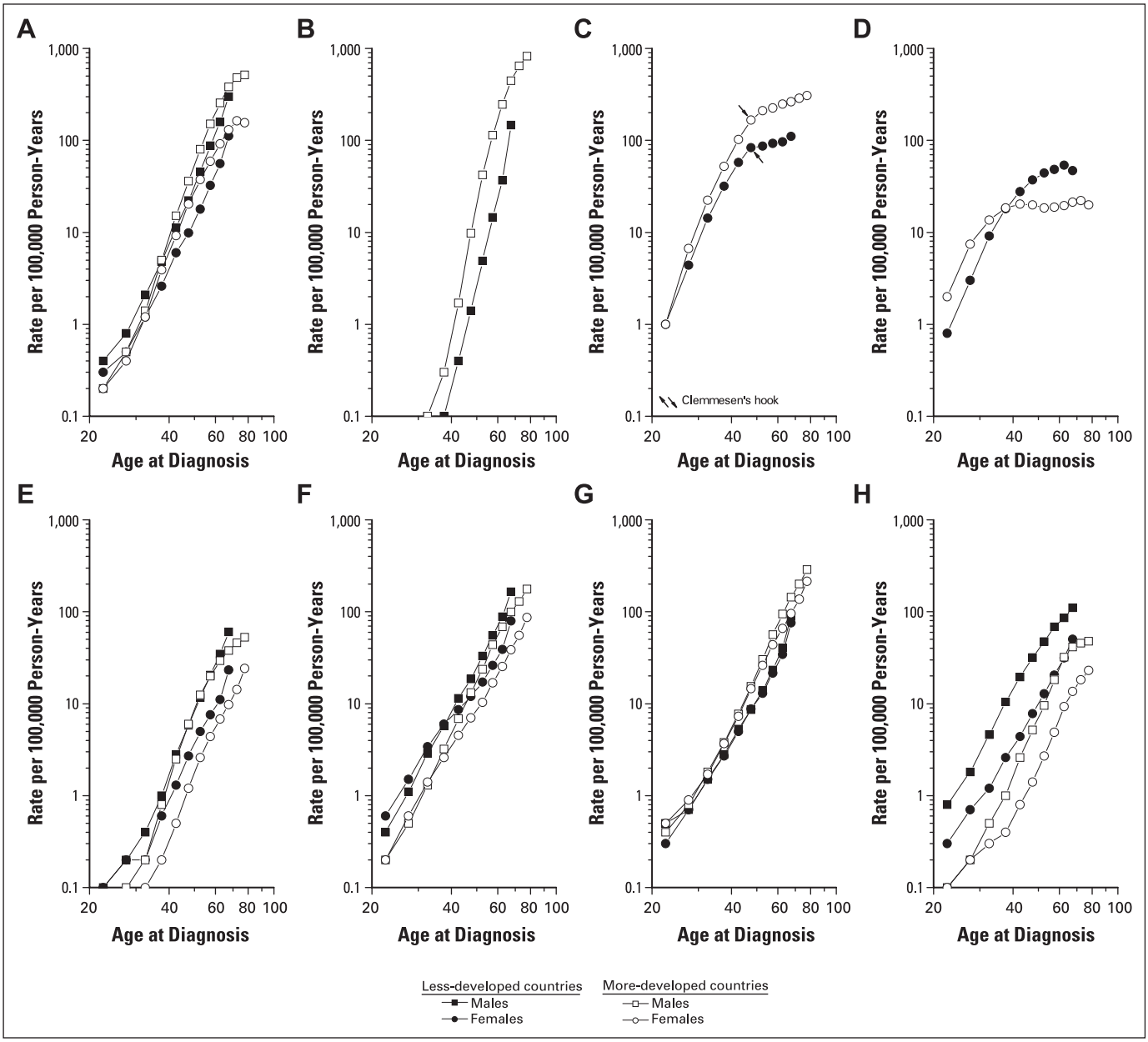

Fig 5. Age-specific cancer incidence rates in more-developed and less-developed countries according to specified cancer sites and sex, 1993-2001. (A) Lung; (B) prostate; (C) female breast; (D) cervix; (E) esophagus; (F) stomach; (G) colon; (H) liver. Incidence rates are age standardized to the world population.

person-years) and Africa (0.69 per 100,000 person-years; Table 1). In contrast, a larger fraction of late-onset and indolent ER-positive tumors in high-risk countries may explain the lower MR:IR in more-developed areas such as North America (0.19). In fact, Lawson et al[68] examined migration patterns according to ER expression between two populations with similar genetic background (ie, Japanese women from Hawaii and Japanese women from Sapporo, Japan.) Japanese women in Hawaii had higher breast cancer incidence and relatively more ER-positive tumors than native Japanese women, whereas native Japanese women had lower incidence but comparatively more ER-negative tumors, consistent with the hypothesis that ER-positive tumors are associated with geographic regions characterized by high breast cancer incidence rates.

Geographic disparities in breast cancer incidence and mortality are also related to differences in screening, chemoprevention, and treatment strategies in more-developed compared with less-developed regions of the world. Estimates from randomized clinical trials and population-based models from countries such as the United States and United Kingdom suggest that early detection and improved treatment might reduce breast cancer mortality rates by 25% to 30%.[69,70] Of note, differential detection of ER-positive tumors by screening mammography also accentuates disparities in breast cancer patterns, by yielding a higher incidence of more indolent tumors associated with more favorable survival.[71-73]

In sum, ER-negative and ER-positive breast cancers appear to be distinct breast cancer types with unique incidence and prognostic

patterns, molecular signatures, and morphologic features. With breast cancer screening detection rates, chemoprevention,[74] and treatment strategies[75] also varying by ER expression, a stratified breast cancer model may be an important conceptual framework to consider for reducing global breast cancer disparities.

Colorectal Cancer

Colorectal cancer (CRC) is the third most common cancer worldwide after lung and breast cancers, accounting for an estimated 1,023,256 new cancer cases and 529,020 cancer deaths per year (Fig 2). Between 1973 and 1997, colon cancer incidence rates increased in most parts of the world included in this study (Figs 3 and 4), except in the United States where incidence rates peaked in the mid-1980s and then began to decline.[76]

Among both men and women, incidence rates were nearly four-fold higher in more-developed compared with less-developed regions of the world (Table 1), whereas mortality rates were only two-and-a-half-fold higher. Across continents, MR:IRs were lowest in North America and highest in Africa, ranging from 0.34 in men in North America to 0.89 in men in Africa.

Colorectal carcinogenesis is a prototypical long-term multistep process with each step corresponding to key etiologic events.[6,7,14,77] Over a lifetime of accumulated carcinogenic events there is pathologic evolution from premalignant adenoma to invasive CRC. The adenoma-to-carcinoma (ACS) sequence results in a linear age-specific incidence rate pattern when rates are plotted on a log-log scale (Fig 5).

The complete ACS can take from 10 to 15 years or more.[78] During this time period, environmental exposures presumably promote the carcinogenic process, the latter also affected by individual genetic susceptibility. However, the dominant role of environmental risk factors in the ACS has been demonstrated in migrant studies, where CRC risk increases several fold among first generation immigrants emigrating from a country at low risk for developing CRC to one at high risk.[79,80] Consequently, there is a strong ecologic correlation between increased CRC incidence and dietary factors such as low fiber intake.[81] Some case-control and cohort studies have found modest associations (relative risks, < 2) between increased CRC risk and low intake of fiber and vegetables[82,83] and folate,[84] as well as high consumption of meat.[85,86] However, these relationships have been inconsistent across studies, and clinical trials thus far have failed to show a benefit for either dietary or supplemental fiber intake in reducing colorectal adenomas.[87] Closely related to dietary intake, excess body weight,[88,89] and physical inactivity[90,91] have also been linked with increased risk of CRC.

The decline in CRC rates during the 1980s and 1990s in the United States has been attributed to increasing CRC screening and therapeutic interventions, following President Ronald Reagan's diagnosis of CRC in 1985.[92] For all average risk individuals aged 50 years or older, the American Cancer Society currently recommends one of four routine CRC screening options: fecal occult blood testing (FOBT) every year; flexible sigmoidoscopy (FS) every 5 years; double contrast barium enema every five years; or colonoscopy every 10 years.[93]

The ultimate goal of CRC screening is to interrupt the ACS, and attempt to reduce CRC incidence and mortality. Annual home-based FOBT reduces CRC incidence by 20% and mortality by 16%.[94] FS directly visualizes the rectum and distal colon, but does not reach the proximal colon. Combined FOBT and FS are probably more effective than either test alone. In addition, colonoscopy performed in follow-up for abnormal FS yields a 40% to 80% reduction in CRC incidence.[95-99] Colonoscopy is the current gold standard for colorectal cancer screening and the preferred diagnostic test to pursue an abnormal CRC screening test. Despite widespread acceptance in the United States, CRC screening practices vary worldwide.[100]

Though not yet a clinical reality, chemopreventive agents targeting critical CRC pathways have successfully interrupted the ACS. Indeed, more than 40 candidate agents (nonsteroidal anti-inflammatory drugs, selenium, hormone replacement therapy, and calcium carbonate) show promise for CRC prevention.[101] However, given that chemopreventive agents are administered to ostensibly healthy individuals, the ultimate effectiveness of any agent will depend on establishing a favorable risk-to-benefit ratio. With the narrow therapeutic index for most chemopreventive agents, alone or in combination, the most effective strategies for reducing the worldwide CRC burden currently include primary prevention through modifiable risk factors and secondary prevention through screening.

Stomach Cancer

Stomach cancer is the fourth most common cancer worldwide, with 603,003 new cases among men and 330,290 new cases among women (Fig 2). Across continents, incidence rates vary from 3.4 per 100,000 PY among females in North America to 26.9 per 100,000 PY among males in Asia, whereas MR:IRs are consistently high in all parts of the world (Table 1). Overall 5-year relative survival rates approximate 20%[21,102,103] in most areas of the world, except in Japan where mass screening programs, staging systems, and treatment may contribute to superior 5-year survival rates of approximately 60%.[5,104,105]

Stomach cancers are anatomically classified as noncardia and cardia cancers. Because noncardia cancers constitute the majority of stomach cancer cases worldwide, overall stomach cancer incidence rates are predominated by this disease entity. Risk factors for noncardia cancers include *H pylori* infection,[106] low socioeconomic status,[107,108] smoking,[109] intake of salty and smoked food,[110,111] and low consumption of fruits and vegetables.[112] In the past century, the incidence of noncardia stomach cancer has declined several fold in more-developed regions of the world.[113] This decrease likely reflects a diminishing prevalence of *H pylori* infection due to improved sanitation, increasing availability of fresh fruits and vegetables, and decreasing use of salt- and smoke-based food preservation methods. However, noncardia stomach cancer remains common in many geographic regions, including China, Japan, Eastern Europe, and Central/South America.[113]

In contrast to noncardia cancers, incidence rates of gastric cardia cancers have either increased or remained constant in Western countries.[114-116] Risk factors for cardia cancer include male sex,[117] white race,[117] smoking,[118,119] obesity,[118,119] and gastroesophageal reflux.[120] The association between *H pylori* and cardia cancer is unclear.[121]

Major prevention strategies to reduce stomach cancer incidence include improved sanitation, higher intake of fresh fruits and vegetables, food preservation methods that are not salt- or smoke-based, avoidance of tobacco products, and maintenance of a normal body weight. To date there is insufficient clinical evidence to recommend endoscopic screening worldwide.

Prostate Cancer

Prostate cancer is the fifth most common cancer in the world and the second most common cancer among men (Fig 2). During the years 1973 to 1997, prostate cancer incidence rates increased in all parts of the world (Fig 3). Annual incidence rates were nearly six-fold higher in more-developed compared with less-developed regions (Table 1), ranging from 4.7 per 100,000 PY in Asia to 119.9 per 100,000 PY in North America. However, mortality rates were only 2.5 times higher in more-developed compared with less-developed parts of the world, and consequently MR:IRs ranged from 0.13 in North America to 0.80 in Africa. Age-specific incidence rates rose steadily with advancing age worldwide (Fig 5).

Diet has been implicated in the etiology of prostate cancer, but definitive etiologic evidence is lacking. High intake of animal fat,[122] meat,[122] and calcium[123] has been associated with an increased risk of prostate cancer, whereas high intake of vegetables,[123] selenium,[124,125] vitamin D,[126] vitamin E,[127,128] lycopene,[129] and omega-3 fatty acids[130] has been associated with reduced risk for prostate cancer. Obesity has been linked with increased risk of prostate cancer death.[131]

Differences in worldwide prostate cancer incidence rates may in part be due to variations in diet, but are also likely influenced by more vigorous screening with prostate-specific antigen testing in more-developed countries. Autopsy series have shown prostate cancer prevalence of approximately 80% in men who died in their 70s from other causes.[132,133] Therefore, prostate-specific antigen testing likely results in the detection of some biologically indolent prostate cancers. It is unclear whether superior survival in more-developed countries stems from earlier detection and improved treatment or reflects detection of biologically indolent disease. The United States Preventive Services Task Force finds insufficient evidence for prostate cancer screening.[134] However, additional evidence regarding screening recommendations is anticipated based on results from large randomized screening studies in both Europe and the United States.[135,136]

Based on current knowledge of risk factors, it is possible that a diet rich in intake of vegetables and low in meat and animal fat may reduce prostate cancer risk. Final disposition regarding the benefit of selenium and vitamin supplementation is pending completion of ongoing clinical trials, including National Cancer Institute–sponsored Selenium and Vitamin E Cancer Prevention Trial (SELECT).[137]

Liver Cancer

With 626,241 new cancer cases per year, liver cancer is the sixth most common cancer in the world. It is approximately 2.5 times more common among men than women (Fig 2). Between 1973 and 1997, incidence rates increased in all parts of the world included in this study, except Singapore (Figs 3 and 4). For both men and women, liver cancer incidence rates were higher in less-developed than more-developed regions (Table 1; Fig 5). Indeed, incidence rates ranged from 1.3 per 100,000 PY among females in Oceania to 21.3 per 100,000 PY among males in Asia. Survival is universally poor as reflected by MR:IRs approximating unity (Table 1).

The most important risk factors for liver cancer are hepatitis B virus (HBV),[138] hepatitis C virus (HCV),[139] and dietary aflatoxins.[140] Other well-established risk factors include alcoholic liver disease[141] and hemochromatosis.[142] Obesity,[143] diabetes,[144] steatosis,[144] smoking,[145] oral contraceptive use,[146] and inadequate intake of selenium[147] and antioxidants[148] also have been implicated as risk factors. High incidence rates and young age of onset in sub-Saharan Africa and eastern and southeastern Asia are primarily a result of HBV infection acquired at birth or during childhood and are also a result of high consumption of aflatoxins.[140] Doubling of liver cancer incidence rates in the United States and other developed countries over the past 30 years (Fig 5) is in part attributed to chronic HCV infection that occurred in the 1960s and 1970s.[149,150]

Large-scale immunization against HBV at birth is the most important step to prevent liver cancer worldwide. In 1992, the World Health Organization recommended that hepatitis B vaccine be integrated into the immunization program of all countries by 1997, and currently over 135 countries have done so.[151] Decreasing aflatoxin exposure will likely reduce the incidence of liver cancer and has already contributed to the decrease in incidence in Singapore (Figs 3 and 4) and Shanghai, China over the past two decades.[88] Screening blood products for HBV and HCV, moderating alcohol consumption, and maintaining normal body weight are also important measures to reduce liver cancer incidence rates.

Cervical Cancer

With 493,100 new cases per year, cervical cancer in the seventh most common cancer in the world, and the second most common cancer among women (Fig 2). During the years 1973 and 1997, cervical cancer rates decreased in most parts of the world included in this study (Fig 4). Incidence rates are almost two-fold higher in less-developed compared with more-developed countries, 19.1 and 10.3 per 100,000 PY, respectively (Table 1). Incidence was highest in Africa and Central/South America (approximately 29 per 100,000 PY) and lowest in Oceania and North America (approximately 7.5 per 100,000 PY; Table 1). Mortality rates vary more than 10-fold across continents and therefore MR:IRs range from 0.27 in Oceania to 0.79 in Africa (Table 1).

Almost all cervical cancer cases are caused by one of 15 types of oncogenic human papillomavirus (HPV), with HPV types 16 and 18, accounting for the majority of cervical cancer cases.[152-154] Other suggested etiologic cofactors include low socioeconomic status,[155,156] smoking,[157,158] low intake of vitamins and micronutrients,[159,160] multiple sexual partners, promiscuous sexual partner,[152] sexual relations at a young age,[152] and oral contraceptive use.[157,158] The presence of HPV infection is a sine qua non in cervical carcinogenesis, and most other factors mediate their effect via exposure to HPV or by affecting susceptibility to the carcinogenic effects of HPV.

Low incidence and mortality rates of cervical cancer in more-developed countries have been attributed to extensive cancer screening practices. Age-specific incidence rates (Fig 5) reveal unique patterns: in more-developed countries rates are high in younger individuals but plateau after the age 40; in less-developed countries, rates are low at young ages but subsequently increase and, after age 40 years, exceed those in more-developed countries. The pattern in more-developed countries may in part be attributed to screening practices.

The conventional method of cervical cancer screening with Papanicolau smears involves sequential testing and is difficult to ubiquitously implement in developing countries due to requirement for compliance with repeated testing, expertise in specimen preparation and diagnostic interpretation, and cost. However, new studies using computer-based models have suggested that HPV DNA testing once (at age 35) or twice (at ages 35 and 40) can be used as a cost-effective method to reduce cervical cancer rates in developing countries.[161,162]

Creation of papillomavirus-like particles in the laboratory in the early 1990s[163] made it possible to make and test anti-HPV vaccines, which have already shown nearly 100% efficacy in reducing HPV infection and cancer rates.[164,165] In the near future, widespread vaccination against oncogenic types of HPV may significantly reduce the global burden of cervical cancer.[161]

Esophageal Cancer

Esophageal cancer is the eighth most common cancer in the world (Fig 2). Overall incidence rates are two-fold higher in less-developed compared with more-developed geographic regions, with the highest rates occurring in Asia (Table 1). Incidence and mortality rates are two- to three-fold higher in males than females. Survival is universally poor as reflected in high MR:IR worldwide (Table 1). Incidence rates rise steadily with advancing age in more-developed and less-developed areas, and incidence patterns are similar between both sexes (Fig 5). However, between 1973 and 1997, regional temporal trends varied, likely reflecting differences in underlying histologic subtypes. Squamous cell carcinoma of the esophagus (ESCC) comprises the majority of cases worldwide followed by adenocarcinoma of the esophagus (EAC).[166] Whereas ESCC incidence has decreased over the past three decades in more-developed countries where annual incidence rates are generally less than 10 per 100,000 PY,[116] incidence has remained high in some less-developed countries, where incidence rates can exceed 100 per 100,000 PY, particularly in high-risk areas of China,[167] Iran,[168,169] and South Africa.[170]

Smoking and alcohol consumption are strong risk factors for ESCC, especially in Western countries.[171-173] In the United States, where ESCC is more common among Blacks,[116] more than 90% of cases are attributed to smoking and alcohol consumption.[171,172] In contrast, few cases in high-risk areas of less-developed countries[168,174] are attributed to smoking and alcohol consumption, and purported risk factors include low consumption of fruits and vegetables,[175,176] selenium[177] and zinc deficiencies,[178] vitamin E deficiency,[179] high exposure to polycyclic aromatic hydrocarbons,[180] and poor oral hygiene.[181,182]

In contrast to ESCC, EAC predominantly occurs in more-developed countries, with the rate of rise in incidence exceeding the rate of decline of ESCC in several Western countries, in both males and females.[116,183] A rare entity 30 years ago, EAC now constitutes approximately half of all esophageal cancer cases in Western countries,[116,117,184,185] reflecting the increasing prevalence of implicated risk factors, gastroesophageal reflux,[120] smoking,[186] and obesity.[187]

Eliminating risk factors that account for a majority of esophageal cancers,[118] including tobacco use, alcohol intake, low fruit and vegetable consumption, obesity, and gastroesophageal reflux, may result in lowering incidence rates. Developing a greater understanding of the etiology of ESCC in less-developed countries, in particular, is needed. Detection, continued surveillance and/or treatment of precursor lesions, such as squamous dysplasia in ESCC and Barrett's esophagus in EAC may decrease cancer burden, although surveillance and treatment measures are expensive and treatment is associated with morbidity and mortality. Chemopreventive agents need additional study but have shown promise for prevention of both ESCC[188-190] and EAC.[191,192]

CONCLUSION

Given our current state of knowledge, a variety of approaches can be used to reduce cancer disparities between countries, including primary and secondary prevention methods. Several effective primary prevention measures are possible due to identification of etiologic agents, including avoidance of tobacco for lung cancer prevention; eradication of *H pylori* for stomach cancer prevention; vaccination against HPV for cervical cancer prevention; and vaccination against HBV for liver cancer prevention. Indeed, an estimated 35% of overall cancer mortality has been attributed to only nine modifiable risk factors.[193] Identification of premalignant or precursor lesions through secondary prevention efforts with screening and early detection strategies has been possible, including mammography for breast cancer detection; FOBT, colonoscopy, and other tests for colorectal cancer detection; and prostate-specific antigen testing for prostate cancer detection. Notably not all screening tests have gained universal acceptance and more definitive information regarding effectiveness of testing is awaited from ongoing clinical trials. Although successful cancer therapies have been identified over the past 30 years, treatments are often toxic, expensive, and require highly trained staff and specialized facilities. Consequently, the most feasible methods to reduce global cancer disparities are to target etiologic factors and high-risk behaviors and to develop strategies for prevention.

REFERENCES

1. Trans-HHS Cancer Health Disparities Progress Review Group: Making cancer health disparities history. U.S. Department of Health and Human Services, 2004

2. Carter-Pokras O, Baquet C: What is a "health disparity"? Public Health Rep 117:426-434, 2002

3. Last JM: A Dictionary of Epidemiology (3rd Ed). Oxford, Oxford University Press, 1995

4. Ferlay J, Bray F, Pisani P, et al: GLOBOCAN 2002, Cancer incidence, mortality and prevalence worldwide, IARC CancerBase No. 5, version 2.0, IARC Press. Lyon, France. http://www-dep.iarc.fr/globocan/database.htm

5. Parkin DM, Whelan SL, Ferlay J, et al: Cancer incidence in five continents, Vol VIII. Lyon, France, IARC Scientific Publications No. 155, 2002

6. Armitage P, Doll R: A two-stage theory of carcinogenesis in relation to the age distribution of human cancer. Br J Cancer 11:161-169, 1957

7. Armitage P, Doll R: The age distribution of cancer and a multi-stage theory of carcinogenesis. Br J Cancer 8:1-12, 1954

8. Devesa SS, Donaldson J, Fears T: Graphical presentation of trends in rates. Am J Epidemiol 141:300-304, 1995

9. Parkin DM, Whelan SL, Ferlay J, et al: Cancer incidence in five continents, I to VIII. Lyon, IARC Cancer Base No. 7, 2005

10. Parkin DM, Bray F, Ferlay J, et al: Global cancer statistics, 2002. CA Cancer J Clin 55:74-108, 2005

11. Jemal A, Ward E, Thun MJ: Contemporary lung cancer trends among U.S. women. Cancer Epidemiol Biomarkers Prev 14:582-585, 2005

12. Bertram JS: The molecular biology of cancer. Mol Aspects Med 21:167-223, 2000

13. Ashley DJ: The two "hit" and multiple "hit" theories of carcinogenesis. Br J Cancer 23:313-328, 1969

14. Fearon ER, Vogelstein B: A genetic model for colorectal tumorigenesis. Cell 61:759-767, 1990

15. Ezzati M, Lopez AD: Estimates of global mortality attributable to smoking in 2000. Lancet 362:847-852, 2003

16. Alberg AJ, Samet JM: Epidemiology of lung cancer. Chest 123:21S-49S, 2003

17. Wardwell NR, Massion PP: Novel strategies for the early detection and prevention of lung cancer. Semin Oncol 32:259-268, 2005

18. Alberg AJ, Brock MV, Samet JM: Epidemiology of lung cancer: Looking to the future. J Clin Oncol 23:3175-3185, 2005

19. Yang G, Fan L, Tan J, et al: Smoking in China: Findings of the 1996 National Prevalence Survey. JAMA 282:1247-1253, 1999

20. Warner KE: The role of research in international tobacco control. Am J Public Health 95:976-984, 2005

21. Ries LAG, Eisner MP, Kosary CL, et al: SEER Cancer Statistics Review, 1975-2002, National

Cancer Institute. Bethesda, MD. http://seer.cancer.gov/csr/1975_2002/sections.html

22. Hirsch FR, Lippman SM: Advances in the biology of lung cancer chemoprevention. J Clin Oncol 23:3186-3197, 2005

23. Mulshine JL: New developments in lung cancer screening. J Clin Oncol 23:3198-3202, 2005

24. Roemer R, Taylor A, Lariviere J: Origins of the WHO framework convention on tobacco control. Am J Public Health 95:936-938, 2005

25. WHO framework convention on tobacco control (WHA56.1), Fifty-six World Health Assembly (agenda item 13), 2003

26. Henderson BE, Feigelson HS: Hormonal carcinogenesis. Carcinogenesis 21:427-433, 2000

27. Lilienfeld AM, Johnson EA: The age distribution in female breast and genital cancers. Cancer 8:875-882, 1955

28. Clemmesen J: Carcinoma of the breast. Br J Radiol 21:583-590, 1948

29. Thomas DB: Breast cancer in men. Epidemiol Rev 15:220-231, 1993

30. Anderson WF, Althuis MD, Brinton LA, et al: Is male breast cancer similar or different than female breast cancer? Breast Cancer Res Treat 83:77-86, 2004

31. Parkin DM, Bray FI, Devesa SS: Cancer burden in the year 2000: The global picture. Eur J Cancer 37:S4-66, 2001 (suppl 8)

32. Stevens RG, Moolgavkar SH, Lee JA: Temporal trends in breast cancer. Am J Epidemiol 115: 759-777, 1982

33. Anderson WF: Puberty and genetic susceptibility to breast cancer. N Engl J Med 349:1088-1089, 2003

34. Anderson WF, Chatterjee N, Ershler WB, et al: Estrogen receptor breast cancer phenotypes in the Surveillance, Epidemiology, and End Results database. Breast Cancer Res Treat 76:27-36, 2002

35. Yasui Y, Potter JD: The shape of age-incidence curves of female breast cancer by hormone-receptor status. Cancer Causes Control 10:431-437, 1999

36. Tarone RE, Chu KC: The greater impact of menopause on ER- than ER+ breast cancer incidence: A possible explanation (United States). Cancer Causes Control 13:7-14, 2002

37. Perou CM, Sorlie T, Eisen MB, et al: Molecular portraits of human breast tumours. Nature 406: 747-752, 2000

38. Moolgavkar SH, Lee JA, Hade RD: Comparison of age-specific mortality from breast cancer in males in the United States and Japan. J Natl Cancer Inst 60:1223-1225, 1978

39. Spratt JS Jr, Kaltenbach ML, Spratt JA: Cytokinetic definition of acute and chronic breast cancer. Cancer Res 37:226-230, 1977

40. Ershler WB: A gerontologist's perspective on cancer biology and treatment. Cancer Control 1:103-107, 1994

41. De Waard F, De Laive JWJ, Baanders-van Halewijn EA: On bimodal age distribution of mammary carcinoma. Br J Cancer 14:437-448, 1960

42. Anderson WF, Chu KC, Chang S, et al: Comparison of age-specific incidence rate patterns for different histopathologic types of breast carcinoma. Cancer Epidemiol Biomarkers Prev 13:1128-1135, 2004

43. Stalsberg H, Thomas DB: Age distribution of histologic types of breast carcinoma. Int J Cancer 54:1-7, 1993

44. Stalsberg H, Thomas DB, Noonan EA: Histologic types of breast carcinoma in relation to international variation and breast cancer risk factors. WHO Collaborative Study of Neoplasia and Steroid Contraceptives. Int J Cancer 44:399-409, 1989

45. Putti TC, El-Rehim DM, Rakha EA, et al: Estrogen receptor-negative breast carcinomas: A review of morphology and immunophenotypical analysis. Mod Pathol 18:26-35, 2005

46. Armes JE, Egan AJ, Southey MC, et al: The histologic phenotypes of breast carcinoma occurring before age 40 years in women with and without BRCA1 or BRCA2 germline mutations: A population-based study. Cancer 83:2335-2345, 1998

47. Ashida A, Fukutomi T, Tsuda H, et al: Atypical medullary carcinoma of the breast with cartilaginous metaplasia in a patient with a BRCA1 germline mutation. Jpn J Clin Oncol 30:30-32, 2000

48. Hedenfalk I, Duggan D, Chen Y, et al: Gene-expression profiles in hereditary breast cancer. N Engl J Med 344:539-548, 2001

49. Breast Cancer Linkage Consortium: Pathology of familial breast cancer: Differences between breast cancers in carriers of BRCA1 or BRCA2 mutations and sporadic cases. Lancet 349:1505-1510, 1997

50. Johannsson OT, Idvall I, Anderson C, et al: Tumour biological features of BRCA1-induced breast and ovarian cancer. Eur J Cancer 33:362-371, 1997

51. Karp SE, Tonin PN, Begin LR, et al: Influence of BRCA1 mutations on nuclear grade and estrogen receptor status of breast carcinoma in Ashkenazi Jewish women. Cancer 80:435-441, 1997

52. Lakhani SR, Gusterson BA, Jacquemier J, et al: The pathology of familial breast cancer: Histological features of cancers in families not attributable to mutations in BRCA1 or BRCA2. Clin Cancer Res 6:782-789, 2000

53. Lakhani SR, Jacquemier J, Sloane JP, et al: Multifactorial analysis of differences between sporadic breast cancers and cancers involving BRCA1 and BRCA2 mutations. J Natl Cancer Inst 90:1138-1145, 1998

54. Marcus JN, Page DL, Watson P, et al: BRCA1 and BRCA2 hereditary breast carcinoma phenotypes. Cancer 80:543-556, 1997

55. Robson M, Gilewski T, Haas B, et al: BRCA-associated breast cancer in young women. J Clin Oncol 16:1642-1649, 1998

56. Verhoog LC, Brekelmans CT, Seynaeve C, et al: Survival and tumour characteristics of breast-cancer patients with germline mutations of BRCA1. Lancet 351:316-321, 1998

57. Marcus JN, Watson P, Page DL, et al: Hereditary breast cancer: Pathobiology, prognosis, and BRCA1 and BRCA2 gene linkage. Cancer 77:697-709, 1996

58. Eisinger F, Jacquemier J, Charpin C, et al: Mutations at BRCA1: the medullary breast carcinoma revisited. Cancer Res 58:1588-1592, 1998

59. Phillips KA: Breast carcinoma in carriers of BRCA1 or BRCA2 mutations: Implications of proposed distinct histologic phenotypes. Cancer 83: 2251-2254, 1998

60. Marcus JN, Watson P, Page DL, et al: BRCA2 hereditary breast cancer pathophenotype. Breast Cancer Res Treat 44:275-277, 1997

61. Armes JE, Venter DJ: The pathology of inherited breast cancer. Pathology 34:309-314, 2002

62. Chabon AB, Takeuchi S, Sommers SC: Histologic differences in breast carcinoma of Japanese and American women. Cancer 33:1577-1579, 1974

63. MacMahon B, Morrison AS, Ackerman LV, et al: Histologic characteristics of breast cancer in Boston and Tokyo. Int J Cancer 11:338-344, 1973

64. Morrison AS, Black MM, Lowe CR, et al: Some international differences in histology and survival in breast cancer. Int J Cancer 11:261-267, 1973

65. Rosen PP, Ashikari R, Thaler H, et al: A comparative study of some pathologic features of mammary carcinoma in Tokyo, Japan and New York, USA. Cancer 39:429-434, 1977

66. Amaku EO: A review of 40 cases of breast cancer seen in Lagos Teaching Hospital. West Afr Med J Niger Pract 17:102-104, 1968

67. Klonoff-Cohen HS, Schaffroth LB, Edelstein SL, et al: Breast cancer histology in Caucasians, African Americans, Hispanics, Asians, and Pacific Islanders. Ethn Health 3:189-198, 1998

68. Lawson JS, Field AS, Tran DD, et al: Breast cancer incidence and estrogen receptor alpha in normal mammary tissue: An epidemiologic study among Japanese women in Japan and Hawaii. Int J Cancer 97:685-687, 2002

69. Blanks RG, Moss SM, McGahan CE, et al: Effect of NHS breast screening programme on mortality from breast cancer in England and Wales, 1990-1998: Comparison of observed with predicted mortality. BMJ 321:665-669, 2000

70. Humphrey LL, Helfand M, Chan BK, et al: Breast cancer screening: A summary of the evidence for the U.S. Preventive Services Task Force. Ann Intern Med 137:347-360, 2002

71. Narod SA, Dube MP: Biologic characteristics of interval and screen-detected breast cancers. J Natl Cancer Inst 93:151-152, 2001

72. Porter PL, El-Bastawissi AY, Mandelson MT, et al: Breast tumor characteristics as predictors of mammographic detection: Comparison of interval- and screen-detected cancers. J Natl Cancer Inst 91:2020-2028, 1999

73. Gilliland FD, Joste N, Stauber PM, et al: Biologic characteristics of interval and screen-detected breast cancers. J Natl Cancer Inst 92:743-749, 2000

74. Fisher B, Costantino JP, Wickerham DL, et al: Tamoxifen for prevention of breast cancer: Report of the National Surgical Adjuvant Breast and Bowel Project P-1 study. J Natl Cancer Inst 90:1371-1388, 1998

75. Early Breast Cancer Trialists' Collaborative Group: Effects of chemotherapy and hormonal therapy for early breast cancer on recurrence and 15-year survival: An overview of the randomised trials. Lancet 365:1687-1717, 2005

76. Ries LAG, Eisner MP, Kosary CL, et al: SEER Cancer Statistics Review, 1975-2002, National Cancer Institute. Bethesda, MD, 2005

77. Vogelstein B, Fearon ER, Hamilton SR, et al: Genetic alterations during colorectal-tumor development. N Engl J Med 319:525-532, 1988

78. Day DW: The adenoma-carcinoma sequence. Scand J Gastroenterol Suppl 104:99-107, 1984

79. King H, Haenszel W: Cancer mortality among foreign- and native-born Chinese in the United States. J Chronic Dis 26:623-646, 1973

80. McMichael AJ, Giles GG: Cancer in migrants to Australia: Extending the descriptive epidemiological data. Cancer Res 48:751-756, 1988

81. McKeown-Eyssen GE: Fiber intake in different populations and colon cancer risk. Prev Med 16:532-539, 1987

82. Trock B, Lanza E, Greenwald P: Dietary fiber, vegetables, and colon cancer: Critical review and meta-analyses of the epidemiologic evidence. J Natl Cancer Inst 82:650-661, 1990

83. Slattery ML, Curtin KP, Edwards SL, et al: Plant foods, fiber, and rectal cancer. Am J Clin Nutr 79:274-281, 2004

84. Giovannucci E, Stampfer MJ, Colditz GA, et al: Multivitamin use, folate, and colon cancer in women in the Nurses' Health Study. Ann Intern Med 129:517-524, 1998

85. Chao A, Thun MJ, Connell CJ, et al: Meat consumption and risk of colorectal cancer. JAMA 293:172-182, 2005

86. Giovannucci E, Rimm EB, Stampfer MJ, et al: Intake of fat, meat, and fiber in relation to risk of colon cancer in men. Cancer Res 54:2390-2397, 1994

87. Schatzkin A, Lanza E, Corle D, et al: Lack of effect of a low-fat, high-fiber diet on the recurrence of colorectal adenomas. Polyp Prevention Trial Study Group. N Engl J Med 342:1149-1155, 2000

88. Giovannucci E: Modifiable risk factors for colon cancer. Gastroenterol Clin North Am 31:925-943, 2002

89. Giovannucci E: Diet, body weight, and colorectal cancer: A summary of the epidemiologic evidence. J Womens Health 12:173-182, 2003

90. Samad AK, Taylor RS, Marshall T, et al: A meta-analysis of the association of physical activity with reduced risk of colorectal cancer. Colorectal Dis 7:204-213, 2005

91. Schnohr P, Gronbaek M, Petersen L, et al: Physical activity in leisure-time and risk of cancer: 14-year follow-up of 28,000 Danish men and women. Scand J Public Health 33:244-249, 2005

92. Brown ML, Potosky AL: The presidential effect: The public health response to media coverage about Ronald Reagan's colon cancer episode. Public Opin Q 54:317-329, 1990

93. Smith RA, von Eschenbach AC, Wender R, et al: American Cancer Society guidelines for the early detection of cancer: Update of early detection guidelines for prostate, colorectal, and endometrial cancers. Also: Update 2001–testing for early lung cancer detection. CA Cancer J Clin 51:38-75, 2001

94. Towler BP, Irwig L, Glasziou P, et al: Screening for colorectal cancer using the faecal occult blood test, hemoccult. Cochrane Database Syst Rev:CD001216, 2000

95. Kavanagh AM, Giovannucci EL, Fuchs CS, et al: Screening endoscopy and risk of colorectal cancer in United States men. Cancer Causes Control 9:455-462, 1998

96. Muller AD, Sonnenberg A: Protection by endoscopy against death from colorectal cancer: A case-control study among veterans. Arch Intern Med 155:1741-1748, 1995

97. Muller AD, Sonnenberg A: Prevention of colorectal cancer by flexible endoscopy and polypectomy: A case-control study of 32,702 veterans. Ann Intern Med 123:904-910, 1995

98. Newcomb PA, Norfleet RG, Storer BE, et al: Screening sigmoidoscopy and colorectal cancer mortality. J Natl Cancer Inst 84:1572-1575, 1992

99. Selby JV, Friedman GD, Quesenberry CP Jr, et al: A case-control study of screening sigmoidoscopy and mortality from colorectal cancer. N Engl J Med 326:653-657, 1992

100. Hakama M, Hoff G, Kronborg O, et al: Screening for colorectal cancer. Acta Oncol 44:425-439, 2005

101. Hawk ET, Levin B: Colorectal cancer prevention. J Clin Oncol 23:378-391, 2005

102. Cunningham SC, Kamangar F, Kim MP, et al: Survival after gastric adenocarcinoma resection: Eighteen-year experience at a single institution. J Gastrointest Surg 9:718-725, 2005

103. Faycal J, Bessaguet C, Nousbaum JB, et al: Epidemiology and long term survival of gastric carcinoma in the French district of Finistere between 1984 and 1995. Gastroenterol Clin Biol 29:23-32, 2005

104. Tsubono Y, Hisamichi S: Screening for gastric cancer in Japan. Gastric Cancer 3:9-18, 2000

105. Roukos DH, Kappas AM: Perspectives in the treatment of gastric cancer. Nat Clin Pract Oncol 2:98-107, 2005

106. Helicobacter and Cancer Collaborative Group: Gastric cancer and Helicobacter pylori: A combined analysis of 12 case control studies nested within prospective cohorts. Gut 49:347-353, 2001

107. Smith GD, Hart C, Blane D, et al: Adverse socioeconomic conditions in childhood and cause specific adult mortality: Prospective observational study. Bmj 316:1631-1635, 1998

108. van Loon AJ, Brug J, Goldbohm RA, et al: Differences in cancer incidence and mortality among socio-economic groups. Scand J Soc Med 23:110-120, 1995

109. Tredaniel J, Boffetta P, Buiatti E, et al: Tobacco smoking and gastric cancer: Review and meta-analysis. Int J Cancer 72:565-573, 1997

110. Buiatti E, Palli D, Decarli A, et al: A case-control study of gastric cancer and diet in Italy. Int J Cancer 44:611-616, 1989

111. De Stefani E, Correa P, Boffetta P, et al: Dietary patterns and risk of gastric cancer: A case-control study in Uruguay. Gastric Cancer 7:211-220, 2004

112. Nouraie M, Pietinen P, Kamangar F, et al: Fruits, vegetables, and antioxidants and risk of gastric cancer among male smokers. Cancer Epidemiol Biomarkers Prev 14:2087-2092, 2005

113. Munoz N, Franceschi S: Epidemiology of gastric cancer and perspectives for prevention. Salud Publica Mex 39:318-330, 1997

114. Blot WJ, Devesa SS, Kneller RW, et al: Rising incidence of adenocarcinoma of the esophagus and gastric cardia. JAMA 265:1287-1289, 1991

115. Devesa SS, Fraumeni JF Jr: The rising incidence of gastric cardia cancer. J Natl Cancer Inst 91:747-749, 1999

116. Brown LM, Devesa SS: Epidemiologic trends in esophageal and gastric cancer in the United States. Surg Oncol Clin N Am 11:235-256, 2002

117. Devesa SS, Blot WJ, Fraumeni JF Jr: Changing patterns in the incidence of esophageal and gastric carcinoma in the United States. Cancer 83: 2049-2053, 1998

118. Engel LS, Chow WH, Vaughan TL, et al: Population attributable risks of esophageal and gastric cancers. J Natl Cancer Inst 95:1404-1413, 2003

119. Lindblad M, Rodriguez LA, Lagergren J: Body mass, tobacco and alcohol and risk of esophageal, gastric cardia, and gastric non-cardia adenocarcinoma among men and women in a nested case-control study. Cancer Causes Control 16:285-294, 2005

120. Lagergren J, Bergstrom R, Lindgren A, et al: Symptomatic gastroesophageal reflux as a risk factor for esophageal adenocarcinoma. N Engl J Med 340:825-831, 1999

121. Dawsey SM, Mark SD, Taylor PR, et al: Gastric cancer and H pylori. Gut 51:457-458, 2002

122. Kolonel LN: Fat, meat, and prostate cancer. Epidemiol Rev 23:72-81, 2001

123. Chan JM, Gann PH, Giovannucci EL: Role of diet in prostate cancer development and progression. J Clin Oncol 23:8152-8160, 2005

124. Duffield-Lillico AJ, Dalkin BL, Reid ME, et al: Selenium supplementation, baseline plasma selenium status and incidence of prostate cancer: An analysis of the complete treatment period of the Nutritional Prevention of Cancer Trial. BJU Int 91: 608-612, 2003

125. Clark LC, Dalkin B, Krongrad A, et al: Decreased incidence of prostate cancer with selenium supplementation: Results of a double-blind cancer prevention trial. Br J Urol 81:730-734, 1998

126. Giovannucci E: The epidemiology of vitamin D and cancer incidence and mortality: A review (United States). Cancer Causes Control 16:83-95, 2005

127. Virtamo J, Pietinen P, Huttunen JK, et al: Incidence of cancer and mortality following alpha-tocopherol and beta-carotene supplementation: A postintervention follow-up. JAMA 290:476-485, 2003

128. Weinstein SJ, Wright ME, Pietinen P, et al: Serum alpha-tocopherol and gamma-tocopherol in relation to prostate cancer risk in a prospective study. J Natl Cancer Inst 97:396-399, 2005

129. Giovannucci E: A review of epidemiologic studies of tomatoes, lycopene, and prostate cancer. Exp Biol Med 227:852-859, 2002

130. Leitzmann MF, Stampfer MJ, Michaud DS, et al: Dietary intake of n-3 and n-6 fatty acids and the risk of prostate cancer. Am J Clin Nutr 80:204-216, 2004

131. Freedland SJ: Obesity and prostate cancer: A growing problem. Clin Cancer Res 11:6763-6766, 2005

132. Haas GP, Sakr WA: Epidemiology of prostate cancer. CA Cancer J Clin 47:273-287, 1997

133. Breslow N, Chan CW, Dhom G, et al: Latent carcinoma of prostate at autopsy in seven areas. The International Agency for Research on Cancer, Lyons, France. Int J Cancer 20:680-688, 1977

134. Harris R, Lohr KN: Screening for prostate cancer: An update of the evidence for the U.S. Preventive Services Task Force. Ann Intern Med 137:917-929, 2002

135. Auvinen A, Curtis RE, Ron E: Risk of subsequent cancer following breast cancer in men. J Natl Cancer Inst 94:1330-1332, 2002

136. Kramer BS, Brown ML, Prorok PC, et al: Prostate cancer screening: What we know and what we need to know. Ann Intern Med 119:914-923, 1993

137. Lippman SM, Goodman PJ, Klein EA, et al: Designing the Selenium and Vitamin E Cancer Prevention Trial (SELECT). J Natl Cancer Inst 97:94-102, 2005

138. Lok AS: Prevention of hepatitis B virus-related hepatocellular carcinoma. Gastroenterology 127:S303-S309, 2004

139. Heathcote EJ: Prevention of hepatitis C virus-related hepatocellular carcinoma. Gastroenterology 127:S294-S302, 2004

140. Yu MC, Yuan JM: Environmental factors and risk for hepatocellular carcinoma. Gastroenterology 127:S72-S78, 2004

141. Morgan TR, Mandayam S, Jamal MM: Alcohol and hepatocellular carcinoma. Gastroenterology 127:S87-S96, 2004

142. Kowdley KV: Iron, hemochromatosis, and hepatocellular carcinoma. Gastroenterology 127: S79-S86, 2004

143. Caldwell SH, Crespo DM, Kang HS, et al: Obesity and hepatocellular carcinoma. Gastroenterology 127:S97-S103, 2004

144. Powell EE, Jonsson JR, Clouston AD: Steatosis: Co-factor in other liver diseases. Hepatology 42:5-13, 2005

145. Jee SH, Ohrr H, Sull JW, et al: Cigarette smoking, alcohol drinking, hepatitis B, and risk for hepatocellular carcinoma in Korea. J Natl Cancer Inst 96:1851-1856, 2004

146. Schlesselman JJ: Net effect of oral contraceptive use on the risk of cancer in women in the United States. Obstet Gynecol 85:793-801, 1995

147. Sakoda LC, Graubard BI, Evans AA, et al: Toenail selenium and risk of hepatocellular carcinoma mortality in Haimen City, China. Int J Cancer 115:618-624, 2005

148. Bjelakovic G, Nikolova D, Simonetti RG, et al: Antioxidant supplements for preventing gastrointestinal cancers. Cochrane Database Syst Rev: CD004183, 2004

149. Seeff LB: Introduction: The burden of hepatocellular carcinoma. Gastroenterology 127:S1-4, 2004

150. Hoofnagle JH: Hepatocellular carcinoma: Summary and recommendations. Gastroenterology 127:S319-S323, 2004

151. World Health Organization. Hepatitis B vaccine, http://www.who.int/vaccines/en/hepatitisb.shtml

152. Schiffman MH, Brinton LA, Devesa S, et al: Cervical cancer, in Schottenfeld D, Fraumeni JF Jr (eds): Cancer Epidemiology and Prevention. New York, Oxford University Press, 1996, pp 1090-1116

153. Bosch FX, de Sanjose S: Human papillomavirus and cervical cancer: Burden and assessment of causality. J Natl Cancer Inst Monogr 3-13, 2003

154. Munoz N, Bosch FX, de Sanjose S, et al: Epidemiologic classification of human papillomavirus types associated with cervical cancer. N Engl J Med 348:518-527, 2003

155. Fasal E, Simmons ME, Kampert JB: Factors associated wih high and low risk of cervical neoplasia. J Natl Cancer Inst 66:631-636, 1981

156. Brinton LA, Hamman RF, Huggins GR, et al: Sexual and reproductive risk factors for invasive squamous cell cervical cancer. J Natl Cancer Inst 79:23-30, 1987

157. Hellberg D, Stendahl U: The biological role of smoking, oral contraceptive use and endogenous sexual steroid hormones in invasive squamous epithelial cervical cancer. Anticancer Res 25:3041-3046, 2005

158. Castellsague X, Munoz N: Cofactors in human papillomavirus carcinogenesis: Role of parity, oral contraceptives, and tobacco smoking. J Natl Cancer Inst Monogr 20-28, 2003

159. Ziegler RG, Weinstein SJ, Fears TR: Nutritional and genetic inefficiencies in one-carbon metabolism and cervical cancer risk. J Nutr 132:2345S-2349S, 2002

160. Hernandez BY, McDuffie K, Wilkens LR, et al: Diet and premalignant lesions of the cervix: Evidence of a protective role for folate, riboflavin, thiamin, and vitamin B12. Cancer Causes Control 14:859-870, 2003

161. Schiffman M, Castle PE: The promise of global cervical-cancer prevention. N Engl J Med 353:2101-2104, 2005

162. Goldie SJ, Gaffikin L, Goldhaber-Fiebert JD, et al: Cost-effectiveness of cervical-cancer screening in five developing countries. N Engl J Med 353:2158-2168, 2005

163. Zhou J, Sun XY, Stenzel DJ, et al: Expression of vaccinia recombinant HPV 16 L1 and L2 ORF proteins in epithelial cells is sufficient for assembly of HPV virion-like particles. Virology 185:251-257, 1991

164. Koutsky LA, Ault KA, Wheeler CM, et al: A controlled trial of a human papillomavirus type 16 vaccine. N Engl J Med 347:1645-1651, 2002

165. Villa LL, Costa RL, Petta CA, et al: Prophylactic quadrivalent human papillomavirus (types 6, 11, 16, and 18) L1 virus-like particle vaccine in young women: A randomised double-blind placebo-controlled multicentre phase II efficacy trial. Lancet Oncol 6:271-278, 2005

166. Stewart BW, Kleihues P: World Cancer Report. Lyon, IARC Press, 2003

167. Blot WJ, Li JY, Taylor PR, et al: Nutrition intervention trials in Linxian, China: Supplementation with specific vitamin/mineral combinations, cancer incidence, and disease-specific mortality in the general population. J Natl Cancer Inst 85:1483-1492, 1993

168. Islami F, Kamangar F, Aghcheli K, et al: Epidemiologic features of upper gastrointestinal tract cancers in Northeastern Iran. Br J Cancer 90:1402-1406, 2004

169. Mahboubi E, Kmet J, Cook PJ, et al: Oesophageal cancer studies in the Caspian Littoral of Iran: The Caspian cancer registry. Br J Cancer 28:197-214, 1973

170. Somdyala NI, Marasas WF, Venter FS, et al: Cancer patterns in four districts of the Transkei region–1991-1995. S Afr Med J 93:144-148, 2003

171. Brown LM, Hoover RN, Greenberg RS, et al: Are racial differences in squamous cell esophageal cancer explained by alcohol and tobacco use? J Natl Cancer Inst 86:1340-1345, 1994

172. Brown LM, Hoover R, Silverman D, et al: Excess incidence of squamous cell esophageal cancer among US black men: Role of social class and other risk factors. Am J Epidemiol 153:114-122, 2001

173. Tuyns AJ: Oesophageal cancer in non-smoking drinkers and in non-drinking smokers. Int J Cancer 32:443-444, 1983

174. Tran GD, Sun XD, Abnet CC, et al: Prospective study of risk factors for esophageal and gastric cancers in the Linxian general population trial cohort in China. Int J Cancer 113:456-463, 2005

175. Brown LM, Swanson CA, Gridley G, et al: Dietary factors and the risk of squamous cell esophageal cancer among black and white men in the United States. Cancer Causes Control 9:467-474, 1998

176. Cook-Mozaffari PJ, Azordegan F, Day NE, et al: Oesophageal cancer studies in the Caspian Littoral of Iran: Results of a case-control study. Br J Cancer 39:293-309, 1979

177. Mark SD, Qiao YL, Dawsey SM, et al: Prospective study of serum selenium levels and incident esophageal and gastric cancers. J Natl Cancer Inst 92:1753-1763, 2000

178. Abnet CC, Lai B, Qiao YL, et al: Zinc concentration in esophageal biopsy specimens measured by x-ray fluorescence and esophageal cancer risk. J Natl Cancer Inst 97:301-306, 2005

179. Taylor PR, Qiao YL, Abnet CC, et al: Prospective study of serum vitamin E levels and esophageal and gastric cancers. J Natl Cancer Inst 95:1414-1416, 2003

180. Kamangar F, Strickland PT, Pourshams A, et al: High exposure to polycyclic aromatic hydrocarbons may contribute to high risk of esophageal cancer in northeastern Iran. Anticancer Res 25:425-428, 2005

181. Abnet CC, Qiao YL, Mark SD, et al: Prospective study of tooth loss and incident esophageal and gastric cancers in China. Cancer Causes Control 12:847-854, 2001

182. Sepehr A, Kamangar F, Fahimi S, et al: Poor oral health as a risk factor for esophageal squamous dysplasia in northeastern Iran. Anticancer Res 25: 543-546, 2005

183. Vizcaino AP, Moreno V, Lambert R, et al: Time trends incidence of both major histologic types of esophageal carcinomas in selected countries, 1973-1995. Int J Cancer 99:860-868, 2002

184. Walther C, Zilling T, Perfekt R, et al: Increasing prevalence of adenocarcinoma of the oesophagus and gastro-oesophageal junction: A study of the Swedish population between 1970 and 1997. Eur J Surg 167:748-757, 2001

185. Wijnhoven BP, Louwman MW, Tilanus HW, et al: Increased incidence of adenocarcinomas at the gastro-oesophageal junction in Dutch males since the 1990s. Eur J Gastroenterol Hepatol 14:115-122, 2002

186. Gammon MD, Schoenberg JB, Ahsan H, et al: Tobacco, alcohol, and socioeconomic status and adenocarcinomas of the esophagus and gastric cardia. J Natl Cancer Inst 89:1277-1284, 1997

187. Chow WH, Blot WJ, Vaughan TL, et al: Body mass index and risk of adenocarcinomas of the esophagus and gastric cardia. J Natl Cancer Inst 90:150-155, 1998

188. Limburg PJ, Wei W, Ahnen DJ, et al: Randomized, placebo-controlled, esophageal squamous cell cancer chemoprevention trial of selenomethionine and celecoxib. Gastroenterology 129:863-873, 2005

189. Kresty LA, Morse MA, Morgan C, et al: Chemoprevention of esophageal tumorigenesis by dietary administration of lyophilized black raspberries. Cancer Res 61:6112-6119, 2001

190. Shimizu M, Suzui M, Deguchi A, et al: Effects of acyclic retinoid on growth, cell cycle control, epidermal growth factor receptor signaling, and gene expression in human squamous cell carcinoma cells. Clin Cancer Res 10:1130-1140, 2004

191. Vaughan TL, Dong LM, Blount PL, et al: Nonsteroidal anti-inflammatory drugs and risk of neoplastic progression in Barrett's oesophagus: A prospective study. http://oncology.thelancet.com, 2005

192. Raj A, Jankowski J: Acid suppression and chemoprevention in Barrett's oesophagus. Dig Dis 22:171-180, 2004

193. Danaei G, Vander Hoorn S, Lopez AD, et al: Causes of cancer in the world: Comparative risk assessment of nine behavioural and environmental risk factors. Lancet 366:1784-1793, 2005

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The Tobacco Research Network on Disparities (TReND) was created in recognition of the need to progress scientific evidence toward reducing tobacco-related health disparities.

Despite impressive scientific progress in documenting tobacco-related health disparities by income, education, race/ethnicity, and other social determinants, many questions remain about the underlying causes of tobacco use, differential clustering within and across population groups, and the mediating mechanisms and processes that account for the linkages that exist between the causes of tobacco-related disease and the social determinants that produce disparities in disease outcomes.

TReND is transdisciplinary by design, and includes researchers from a wide range of academic disciplines who aim to stimulate new studies, challenge existing paradigms, address significant gaps in research on understudied and underserved populations, and work collaboratively to devise innovative solutions to address tobacco-related health disparities.

On the TReND Web site, you will find a list of projects, resources, news and discussions.

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Toolkit

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TReND
Tobacco Research Network on Disparities

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Chapter 7

Fogarty Program Abstracts and Contact Information

U.S. National Institutes of Health
JOHN E. FOGARTY INTERNATIONAL CENTER
FOR ADVANCED STUDY IN THE HEALTH SCIENCES

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Project Title	Increasing Capacity for Tobacco Research in Hungary
Project Number	TW007927-01
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Collaborating Countries	Hungary
Project Summary	DESCRIPTION (provided by applicant): Hungary has the highest percentage of smoking-related deaths in the European Union (EU) (21%). An estimated 34% of adults in Hungary smoke, including 40.5% of men and 28% of women. The goal of this research and capacity building project is to reduce the burden of tobacco through the advancement of scientific study of tobacco use and to broadly disseminate research findings that will result in innovative tobacco control interventions in Hungary. Scientists from Semmelweis University in Budapest and Wake Forest University in Winston Salem, NC USA will collaborate to achieve the following aims: 1. Create institutional capacity that will support scientists and advocates engaged in tobacco research and translate research findings into programs and policies to reduce tobacco use. 2. Conduct mentored research that has the potential to significantly reduce tobacco use at the local and national level. 3. Build individual capacity among Hungarian and U.S. research partners through formal in-country training and mentored research projects. Semmelweis University will engage students, academics, practitioners, and policy makers from a wide range of disciplines in an institutional capacity building process. Team leadership from Hungary and the U.S. will guide mentored scientists in strategic planning and logic model development that will inform the tobacco research mission in Hungary. U.S. investigators will offer formal training to Hungarian scientists in core competencies of research methodology, ethical conduct of research, tobacco control research, and grant writing. Leadership in the U.S. and Hungary will also embed the mentored research projects into the capacity building effort through 1-on-1 guidance and support. The research capacity of U.S. scholars will also be enhanced. The U.S. team will learn about the history and tradition of tobacco research Hungary, understand the gaps in tobacco knowledge that exist in Hungary, and collaborate with stakeholders and scientists engaged in the tobacco control movement. By implementing a co-learning process embedded in mutual learning and mutual respect, participating Hungarian and U.S. scientists will be better equipped to collaborate on scientifically rigorous and meaningful research and to make an impact on tobacco use in Hungary. There is dearth of tobacco science in Hungary and no current strategy to lead a comprehensive tobacco control program that is evidence-based. This research and capacity-building proposal will fill a very important need in Hungary. The Hungarian Ministry of Health and the World Health Organization have endorsed this project.

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Project Title	Tobacco Control Policy Analysis & Intervention Evaluation in China and Indonesia
Project Number	TW005938-06
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Collaborating Countries	China, Indonesia
Project Summary	DESCRIPTION (provided by applicant): China is the largest consumer and Indonesia is the fifth largest consumer of cigarettes in the world. Our current NIH/FIC findings were used by Chinese policymakers in their decision to ratify the Framework Convention of Tobacco Control (FCTC). However, tobacco control in China still faces a long road, as China has not stipulated increasing tobacco tax and it is unclear how well the FCTC will be implemented in China. In order to continue progress in China on tobacco control, additional studies will be needed to test potential tobacco control economic policies based on our research findings and to monitor the implementation of the FCTC. Indonesia faces an even longer road as it has yet to ratify the FCTC. Additional research is needed to make the economic case for tobacco control in Indonesia and convince policymakers to ratify the FCTC. In both China and Indonesia, more tobacco control researchers at the local level are needed to further build the knowledge base for tobacco control policies. This competing renewal has four goals: (1) Continue Chinese tobacco control research in five areas: development of tobacco tax policy alternatives, a crop substitution program for tobacco farmers, smoke-free environments in hospitals, a social marketing tobacco control campaign focused on maternal and child health, and monitoring China's tobacco industry transition under the FCTC. (2) Develop Indonesian tobacco control research in three areas: the economic costs of smoking, the tobacco and clove farming, and monitoring Indonesia's tobacco industry after market entry by Philip Morris International. (3) Train more tobacco control researchers in both China and Indonesia (4) Engage key economic policymakers in both China and Indonesia through policy working groups and policy conferences. A consortium of institutions, led by the Public Health Institute (U.S), will conduct these activities. Collaborating institutions include Sichuan University (China), the National Center for Disease Control (China CDC), the Chinese Association on Tobacco Control (CATC), and the University of Indonesia's School of Public Health and Demographic Institute.

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Project Title	Measuring Tobacco Mortality within the Million Death Study in India
Project Number	TW007939-01
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Collaborating Countries	India
Project Summary	DESCRIPTION (provided by applicant): The project will measure the mortality consequences of tobacco smoking and chewing among 1.3 million households (about 7.6 million people) who are already enrolled in the Indian Sample Registration System (SRS), and who will be re-surveyed every 6 months until 2014. India's SRS is a large continuous demographic survey of 7,597 small areas (4,433 rural and 3,164 urban) randomly selected from the 2001 Census. This project (among the world's largest prospective studies of adult health) is possible because of the success of our earlier NIH grant (#TW005991) which established reliable, routine, low-cost and long-term monitoring of causes of death, and which surveyed 150,000 deaths that occurred in 2001-2003. This first NIH grant also documented: (i) that 37% of males aged 25-69 smoke, with up to 9-fold variation by state; (ii) that illiterate men had a 4-fold higher risk of smoking bidis, and a 2.5 fold lower risk of smoking cigarettes than did those with grade 10 or higher education; (iii) smoking bidis or cigarettes already causes about 1 in 3 adult male deaths (a proportion equal to that seen in the United States about two decades ago); and (iv) smoking causes nearly half of tuberculosis deaths in India. Specific aims of this project (2006-2012) are to: Quantify risks for tuberculosis, heart attack, cancers and other causes in relation to male smoking and tobacco chewing and in relation to female tobacco chewing among 160,000 adult deaths at ages 25-69. > Quantify the effects of household male smoking and indoor air pollution among about 3,500 childhood respiratory deaths and about 7,500 adult female respiratory deaths. Study the trends and determinants of smoking and chewing, including cessation among 0.6 million male smokers, 0.5 million male chewers and 0.2 million female chewers. Study the correlations of tobacco with other risk factors for chronic diseases (obesity, blood pressure and lipids, diabetes) in a blood-based pilot study of 10,000 adults. Provide applied training to field staff, epidemiologists and scientists in tobacco epidemiology, including a "knowledge translation" program to convert research findings into policy and monitoring. The project builds sustainable capacity to monitor diverse tobacco hazards in a population of 1 billion at a unique scale (surveying about 0.7 million deaths from 2004-2014, of which about 0.4 million will be surveyed in the project period). Leverage of Government of India and other resources permits the study to be very low-cost (< $1/person/year). The blood-based pilot studies should enable large, representative and reliable genetic and biological epidemiological studies of premature mortality in the near future.

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Project Title	Responding to the changing tobacco epidemic in the Eastern Mediterranean Region
Project Number	DA024876-01
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Collaborating Countries	Syria
Project Summary	DESCRIPTION (provided by applicant): Tobacco use accounts for 4.9 million deaths annually, with 70% of 2020's 10 million predicted deaths occurring in developing nations. This shocking prediction highlights the need for developing nations to examine patterns and determinants of tobacco use, understand local tobacco use methods, develop effective cessation interventions, and train their own tobacco control scientists. In 2002, with NIH/FIC support, the Syrian Center for Tobacco Studies (SCTS) began this effort in the Eastern Mediterranean Region (EMR). Meanwhile, Syria and other EMR nations ratified the Framework Convention on Tobacco Control (FCTC). This application for continued SCTS support builds on four years of research and training momentum, and prepares Syria and the EMR to meet FCTC obligations. There are four specific aims. First, we seek to understand adolescent tobacco use patterns and determinants: a school-based longitudinal study will examine trends in tobacco use in 4000 Syrian youth. Second, we will examine waterpipe toxicant exposure, dependence, and risk in a laboratory study of 240 waterpipe users placed in three groups based on past-month use frequency. All users will be tested under conditions of 0- and 72-hour abstinence. Particulate matter emissions, a risk for non-users, will also be assessed. Third, we will conduct a randomized clinical trial (multi-site, double blind, placebo-controlled) of a smoking cessation intervention in 250 smokers, while simultaneously studying proximal (e.g., patient) and distal (health care center, staff, policy) factors that can influence the intervention's successful integration within the Syrian public health care system. Fourth, we will continue to build regional tobacco control capacity: our successful program of training Syrian researchers will be maintained, as we also organize and fund three, 1-week, training courses in years 1, 3, and 5 and develop and capitalize on our Research Assistance Matching project. SCTS research and FCTC obligations point to the need for understanding tobacco use determinants, waterpipe use, and cessation efficacy and integration, as well as training of tobacco control researchers across the EMR. This application seeks to build on the past success of the SCTS while helping Syria and the EMR meet their FCTC obligations. Relevance of this research to public health: The proposed work will help understand tobacco use determinants, waterpipe use, and cessation efficacy and integration, which will be instrumental in informing prevention and intervention efforts in Syria and the EMR. Research and training activities proposed will contribute to the building of professional capacity that can aid the successful implementation of the FCTC in the EMR.

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Project Title	Building Capacity of Tobacco Cessation in India & Indonesia
Project Number	TW007944-01
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Collaborating Countries	India, Indonesia
Project Summary	DESCRIPTION (provided by applicant): Tobacco cessation is a global health priority not yet addressed in low and low-middle income countries such as India and Indonesia. An important lesson gleaned from international health is that interventions proven effective in western countries cannot simply be exported to developing nations without significant cultural adaptation. Research is urgently needed to find the most effective means of promoting cessation in local contexts. A first step in gaining public attention about the harm of tobacco use and the need for cessation is to involve the medical community in tobacco cessation efforts. It has been well established that before tobacco use declines in the general population, health care providers must be at the forefront of tobacco cessation efforts. To do so, they must quit tobacco use themselves, routinely ask patients about tobacco use, and advise them to quit. At present, there is little involvement of physicians in tobacco cessation efforts in India and Indonesia. Based on four years of experience during Project Quit Tobacco International (QTI) (under the previous Fogarty initiative), the proposed project unfolds as a four-step process to develop a cohort of tobacco cessation researchers in India and Indonesia, using local medical schools as a hub for research activities in both the health care community and the community at large. Medical schools will become centers for recruiting and training tobacco cessation researchers as well as sites for implementing pilot studies and mobilizing local tobacco cessation research networks. The specific objectives of the project are to (1) increase knowledge about the risks of tobacco use and the importance of cessation through dissemination of a model tobacco education curriculum for medical schools in India and Indonesia; (2) recruit and train tobacco researchers concurrently with introducing tobacco education in nine medical schools in each country; (3) involve partner medical schools in tobacco cessation-related community-based research pilot studies, and (4) build capacity in tobacco-related research in both countries by creating tobacco cessation research networks in the six states (three in each country) where project activities will take place. Developed over the past four years, the QTI tobacco cessation research centers in India and Indonesia have begun to engage the communities in which they are located. In-country researchers have gained the skills and confidence to take the next step toward building research capacity beyond their institutions in these two culturally diverse nations. The proposed project will leverage lessons learned during QTI and provide an infrastructure for training a next generation of local tobacco researchers to meet the challenges of tobacco cessation within their own countries.

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Project Title	Tobacco Control Research and Training in South America
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Collaborating Countries	Argentina
Project Summary	DESCRIPTION (provided by applicant): This is a competitive renewal application for a Fogarty International Center Tobacco control research and capacity building project focused in Argentina. This project is based in the Southern Cone of South America that has the highest attributable mortality to tobacco in the Americas. In the first funded cycle, we developed methods to recruit and survey a cohort of 3,500 13 to 15 year old adolescents from Jujuy, a province in the Northwest of Argentina with a predominantly indigenous population. We have completed a baseline and two follow-up surveys and will be able to generate smoking initiation and transition rates that will include 3 years of follow up with a final survey in 2007. In this proposal we aim to 1) Use the cohort data to develop an intervention to prevent tobacco use among diverse youth in Northwest Argentina; 2) Implement and evaluate a system-based smoking cessation intervention using a randomized trial design among physicians to promote smoking abstinence and quit attempts in their patients who smoke over a one-year period; 3) Continue to follow the cohort for three additional years or until age 20; 4) Develop policy interventions to promote smoke free indoor space and regulation of tobacco products advertising by continuing to analyze the tobacco industry documents on Argentina, using data generated from the heart disease policy model, and evaluation of tobacco use in popular films in Argentina; and 5) Build research capacity by expanding training opportunities in Argentina at all levels, selecting 2 scholars annually to visit UCSF for development of tobacco control research methods, and support the application of promising scientists in Latin America to the Tobacco Control Research Fellowship at UCSF.

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Project Title	Advancing Cessation of Tobacco In Vulnerable Indian Tobacco consuming Youth
Project Number	TW007933-01
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Collaborating Countries	India
Project Summary	DESCRIPTION (provided by applicant): The goal of this study is to test the efficacy of a comprehensive, community-based tobacco control intervention, among disadvantaged youth (10-19 years) living in low income communities of Delhi. The specific aims of the project are:(1) To conduct a cluster randomized trial, in slum-dwelling and other low income residential communities of Delhi, of non-pharmacologic community led behavioral intervention intended to promote cessation of tobacco use by adolescent consumers of tobacco, in order to evaluate the effectiveness and cost-effectiveness of such interventions; (2) To identify the demographic and psychosocial factors associated with the uptake and cessation of tobacco products by adolescents residing in such communities; (3) To increase the capacity of Indian investigators, research staff, partner NGOs, community leaders and youth self help groups to implement tobacco cessation programs in low resource community settings; (4) To increase the capacity of US investigators to adapt research models and methods validated in developed countries for application in a developing country. Twenty slum clusters of Delhi would be selected to form ten matched pairs, one of each of which would be randomly allocated to a behavioral intervention for tobacco cessation and the other to a control group receiving free eye check-up. The intervention group would receive training for youth peer leaders, adult community leaders and NGO personnel, followed by implementing interactive community based activities that would aim to enhance motivation to quit tobacco use, encourage quit attempts, promote continued abstinence, enhance knowledge around tobacco consumption and tobacco control, develop advocacy skills etc. over a 24 months period. The main outcome measure would compare tobacco cessation rates, between the two trial groups, of young persons (who have remained tobacco free for at least 30 days) and reduce the prevalence rate between the baseline and end- line surveys. Cost-effectiveness (cost per quality-adjusted life year added due to the intervention) would also be assessed. Capacity building activities would include training workshops (for partners and other participants in this research) and preparation of training modules which would be widely disseminated to public health professionals in India. The results of this study are expected to provide information on effectiveness of non-drug community based behavioral interventions for promoting tobacco cessation in disadvantaged youth of low income countries.

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Project Title	Epidemiology & Intervention Research for Tobacco Control in China
Project Number	TW007949-06
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Collaborating Countries	China
Project Summary	DESCRIPTION (provided by applicant): China is a particularly critical country for global tobacco control. It has the world's largest number of smokers, approximately 350 million, and the immense market afforded by the 60% of men who smoke and the 95% of women who do not smoke represents a prize target for the multinational tobacco companies. China has ratified the Framework Convention for Tobacco Control (FCTC) and now needs to implement its provisions across a large and diverse population. This application builds on a 10-year collaboration with investigators in China, including Dr. Gong-Huan Yang, now Deputy Director of the China CDC. It extends formative work carried out over the last five years with support from the Fogarty International Center. In this application, we propose a program of evidence-based interventions to be implemented at the province and local levels with the overall objective of developing an approach for dissemination and implementation across China. The study aims to improve the Strength of Tobacco Control (SOTC) at the province level in China by improving capacity in program effectiveness, using resources of the China CDC and Peking Union Medical College. Capacity building activities will be undertaken using distance-based approaches and an intervention study will be conducted involving 10 provinces, five participating in a China CDC-led initiative and five continuing with the approaches generally in place. A systematic mix of approaches will be implemented based on experiences from our work in China and from the American Stop Smoking Intervention Study for Cancer Prevention (ASSIST). In the intervention provinces, the China CDC will team with the province-level CDCs to assess capacity for tobacco control and the current status of tobacco control. This systematic characterization will highlight resource, infrastructure, capacity and program needs. The intervention program will then be developed, based on the SOTC assessment and the evidence already gathered in our current Fogarty International Center funding cycle. With this evidence-based approach, the provincial CDCs should be able to identify the optimum mix of tobacco control programs and policies for its residents. The interventions will address both urban and rural locations, as the majority of Chinese population still living in rural areas. A three-year intervention phase will be followed by an evaluation. We have the overall objective of preparing the China CDC to disseminate and implement a proven approach to tobacco control across all provinces.

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Project Title	Network for Tobacco Control among Women in Parana, Brazil
Project Number	DA024875-01
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Collaborating Countries	Brazil
Project Summary	DESCRIPTION (provided by applicant): Brazil is the second largest producer of tobacco in the world, and 96% of the tobacco is produced is in the three Southern states (including the state of Parana - the proposed geographic setting for this application). Parana also has the highest prevalence of cigarette smoking among women in the country, and smoking initiation is higher among girls than boys. Recent guiding documents have identified the understanding of women and tobacco-related issues and the need for the development of gender-relevant tobacco control efforts as priorities, particularly in developing countries (e.g., WHO Framework Convention on Tobacco control, the Research for International Tobacco Control agenda for Latin America and the Caribbean). Therefore, we propose the development of a Network for Tobacco Control among Women in Parana, Brazil in order to establish community and institutional capacity to promote gender-relevant tobacco control efforts among Brazilian women through community-based participatory research and training. The goals of the "Network" are to reduce tobacco use and exposure to environmental tobacco smoke among Brazilian women, and to develop a cadre of well-trained researchers in tobacco control. These goals will be accomplished based on the principles of Community-Based Participatory Research and the Empowerment Model, and will be guided by a multi-level approach that will address four target levels: individuals, organizational/policy systems, and agents of change. The program will consist of six phases: (a) Network establishment; (b) Capacity building in which partners will train and empower each other to carry forward the network mission; (c) Needs/assets assessment phase in which network partners will assist in the identification of needs and assets regarding tobacco control among women as well as needs in training at the professional and academic levels, and will establish priority areas for intervention; (d) Development of a Community Action Plan (CAP) which will consist of a comprehensive tobacco control intervention targeting women; (e) Implementation of CAP; and (f) Dissemination and sustainability of the network. In synergism with these efforts, we will implement a Career Development and Research Training program in tobacco control targeting academicians, professionals, and students to promote research, which will lead to the development of a cadre of well-trained researchers who will continue to address comprehensive tobacco control strategies at all levels long after the study has ended.

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Project Title	The Political Economy of Tobacco Control in Southeast Asia
Project Number	TW007924-01
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Collaborating Countries	Cambodia, Malaysia, Thailand, Vietnam, Laos, Indonesia and the Philippines
Project Summary	DESCRIPTION (provided by applicant): We propose to study the political economy of tobacco in low- and middle-income countries in Southeast Asia. Through a regional research and capacity building program, we seek to enable those in the region to respond more effectively to the challenge of tobacco use for the long term and on their own terms. By political economy, we refer to the interrelationships between political processes and economic variables that influence the course of tobacco control policy. From taxes to trade of tobacco, these issues are inherently challenging-transdisciplinary in nature, often regional in scope and implications, and not bounded by only health concerns in the larger context of development. This program represents a unique partnership that builds upon the legacy of the Rockefeller Foundation's Trading Tobacco for Health initiative, leverages the policy reach of the Southeast Asian Tobacco Control Alliance (SEATCA) regional network, and builds synergy with the American Cancer Society's Tobacco Control Surveillance Program. By Southeast Asia, we will refer primarily here to seven countries in that region: Cambodia, Malaysia, Thailand, Vietnam, Laos, Indonesia, and the Philippines. The specific aims of this project are to: 1. Conduct research that examines the political economy of tobacco control and its impact on health in Southeast Asia. Through regional meetings, targeted research grants, and subsequent smaller workshops, we would cultivate, train and resource a group of country-level researchers. Selected projects will receive expert technical assistance as well as support for editing, policy translation and dissemination. 2. Support studies that situate the impact of tobacco into the larger context of sustainable development. Reframing tobacco as more than a public health concern may yield greater policy change. 3. Build capacity and networking of researchers in Southeast Asia to enable a strong, local evidence base for tobacco control and to encourage effective translation of research into policy. These country researchers would participate in regional and in-country meetings, periodic conference calls, and an on-line collaborative workspace. For there to be a community of researchers sharing interest in tobacco control, few countries in Southeast Asia have sufficient critical mass to mount a network of their own. Scaling this up to a regional network affords multiple advantages: the opportunity for cross-country comparison and cross-border learning, the potential for building up research centers of excellence that could serve a region, and most importantly, a community of colleagues to provide evidence for tobacco control policy in Southeast Asia.

*Some of the information may have changed since this toolkit was created.

Pre-Conference Workshop
14th World Conference on Tobacco or Health
March 8, 2009 ♦ Mumbai ♦ India
Toolkit*

Project Title	SMS Turkey: Harnessing the power of TXT messaging to promote smoking cessation
Project Number	TW007918-01
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Collaborating Countries	Turkey
Project Summary	DESCRIPTION (provided by applicant): Smoking has long been recognized as a significant public health concern associated with great morbidity and mortality. Although smoking rates have decreased in high income countries, these trends have not been noted for tow and middle income countries. This is especially true in Turkey, which was ranked 6th in the world in smoking consumption. Between 51-63% of Turkish men and 24-26% of Turkish women 15 years of age and older are smokers. With lung cancer the leading cancer-related cause of death for both men and women, effective and accessible smoking cessation interventions are needed. Cell phone technology represents a unique opportunity to deliver evidenced-based smoking cessation behavioral treatments through a delivery mechanism already widely adopted by adults. An estimated 35 million Turks were using cell phones, making them 1.8 times more common than land lines. Cell phone interventions are a unique delivery method because of their 'always on' capability. The rapid uptake of cell phones allows us to potentially reach those who might otherwise not utilize smoking cessation services. In this response to Request for Applications (RFA-TW-06-006), we propose to design and evaluate an evidenced-based smoking intervention that takes advantage of technologies adopted by adult smokers in Turkey. Based upon STOMP (Stop smoking by Mobile Phone) New Zealand, a TXT messaging-based smoking cessation program, SMS (Smoking Management System) Turkey will be a community-based intervention that has both wide reach and high impact. To do so, we bring together a multi-national team of smoking and technology health experts dedicated to building the capacity of smoking cessation research expertise in Turkey to reduce the public health burden associated with smoking in Turkey. Using qualitative methods to inform design and content and quantitative methods to assess the feasibility and potential intervention effect, we propose to develop SMS Turkey, an innovative smoking cessation program that uses technology widely adopted by adults, an under-targeted population, to deliver a proactive, cognitive behavioral therapy (CBT)-based, theoretically grounded intervention. Simultaneously, we will build the capacity for future smoking cessation efforts in Turkey by training two medical fellows to become smoking cessation researchers, and continuing to strengthen international research ties. The high reach of cell phones allows us to potentially reach smokers who would otherwise not utilize traditional smoking cessation services, representing the possibility of a large public health impact.

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