UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLUMBIA

UNITED STATES OF AMERICA, Plaintiff, v. PHILIP MORRIS USA INC., f/k/a PHILIP MORRIS INC., *et al.*, Defendants.	)	Civil No. 99-CV-02496 (GK)

UNITED STATES' WRITTEN DIRECT EXAMINATION OF
DR. DAVID M. BURNS
SUBMITTED PURSUANT TO ORDER #471

Q: Please state your name for the record.

A: David Michael Burns, M.D.

Q: Dr. Burns, what is your current professional position?

A: I am a medical doctor, professor of medicine and professor of family and preventive medicine at the University of California, San Diego School of Medicine.

Q: Dr. Burns, do you recognize the document marked as U.S. Exhibit 78,526?

A: Yes, I do. This is a copy of my curriculum vitae. It provides a description of my additional qualifications, including my education, postgraduate training, teaching assignments, staff appointments, memberships and offices, awards, and publications.

Q: You been involved in the study of the health consequences of smoking for approximately 30 years, is that correct?

A: Yes.

Q: What is your understanding of the expertise for which you are being offered in this case?

A: The science of tobacco and health, including disease causation.

Q: I would like to begin by discussing your credentials and professional work experience. Please describe your professional education for the Court.

A: I received my doctorate in medicine at Harvard Medical School in 1972. I trained in Internal Medicine on the Harvard Medical Service at Boston City Hospital from 1972 through 1974. I received my training in Pulmonary Medicine at the University of California, San Diego School of Medicine from 1976 through 1979.

Q: How long have you held your current position at the University of California at San Diego?

A: I've been on the faculty at the University of California, San Diego since 1979, and I have been a Professor of Medicine there for about fourteen years.

Q: Are you presently licensed to practice medicine?

A: Yes, I am currently licensed to practice medicine in the State of California, I am board certified in Internal Medicine and Pulmonary Medicine and have a certificate of special competence in Critical Care Medicine.

Q: Are you currently practicing medicine?

A: Yes. My specialty is in lung disease, and I practice in consultative practice in the hospital on lung disease.

Q: Tell us a little bit about the scope of responsibilities that this job entails.

A: When I am on consultative service, I provide specific diagnostic and therapeutic advice to physicians in the hospital on patients with lung disease of a variety of different types, including critical care patients. I also interpret the pulmonary function studies in the hospital. As part of that responsibility, I teach medical students, interns, residents and fellows about the diagnosis and management of lung disease as well as the interpretation of pulmonary function studies.

Q: And in what areas do you teach?

A: I teach in a wide variety of areas that cover both my primary interest in lung disease and my interest in public health. In general, I teach third and fourth year medical students when they spend their time on clinical services in the hospital. I also teach interns and residents when they

rotate through the service or when I provide consults on individual patients. I teach on lung cancer; I teach on the diagnosis of chronic obstructive lung disease; on pulmonary physiology and pulmonary function testing, how it is interpreted, and how you use that evidence to make diagnostic judgments. I teach on the relationship between cigarette smoking and disease causation. I teach on the role of physicians in getting people to stop smoking. I teach on the role of society in terms of the effects of larger public policy change on changing people's smoking behavior.

Q: How do you divide your time among your various professional activities?

A: Well, about eighty-five percent or ninety percent of my time at the moment is spent on research activities that relate to a series of investigations. They include measurement of disease risks produced by smoking, evaluation of tobacco control programs at the state and national level, examination of the impact of public policy changes on smoking behaviors, and examination of the effect of the media on smoking behavior. I spend about five percent of my time on patient care and teaching, and about five percent of my time of late I've spent on things like preparing to testify in this case.

Q: Have you always divided your time in this manner?

A: No. For the first fifteen years of my time within the University, I spent about half of my time on clinical activities. That included attending and being responsible for patient care provided in the critical care unit, medical intensive care unit at our hospital. I also ran the respiratory therapy department at our medical center.

Q: What is the respiratory therapy department?

A: The respiratory therapy department is responsible for all of the mechanical ventilation, artificial ventilation that occurs within the hospital. It is also responsible for the delivery of oxygen, delivery of a variety of medications that are provided by an inhalational unit as well as various therapeutic modalities related to respiration.

Q: Can you describe the other positions that you have held at the University of California, starting when you arrived at the University?

A: In 1976, I came to UCSD to train in lung disease. I was a Fellow in chest medicine for three years and then joined the faculty as an Assistant Professor. I received an accelerated promotion to Associate Professor after six years, and after another six years was promoted to full Professor.

Q: How did you happen to choose the University of California, San Diego for your pulmonary fellowship?

A: I had been interested for some time in chest medicine, and UCSD was arguably the best training program in the country in lung disease at that time, and I applied and was accepted.

Q: Approximately how many patients over the years have you seen or been consulted on with regard to issues involving actual patient lung cancer or COPD?

A: I probably have seen thousands of people with chronic lung disease, chronic obstructive lung disease (also known as Chronic Obstructive Pulmonary Disease -- C.O.P.D.), and multiple hundreds of people with lung cancer.

Q: Do you have any additional responsibilities at the University of California, San Diego?

A: Yes. I also head the Tobacco Control Policies Project.

Q: What does that project involve?

A: The Tobacco Control Policies Project is a group that includes myself and five other professionals. We do a variety of activities that relate to examination of very large population based data sets and risk data sets. They include predicting lung cancer death rates from smoking behavior, examining the risks of smoking for various diseases, examining and collecting information on print media advertising and relating that to various smoking behaviors as well as other changes in cigarette use. We have done a substantial amount of work looking at various public policy changes, taxes, restrictions on the places people can smoke, physician advice, other kinds of activities, and the impact of those things on getting people to stop smoking or trying to prevent from people from starting. We've also done some work evaluating the global impact of state-wide tobacco control programs, both in California and Massachusetts, as well as looking at some of the issues in initiation and cessation nationally.

Q: How long has that project been in existence?

A: About ten years.

Q: And how long have you been affiliated with that project?

A: I started it, and I have headed it from the beginning.

Q: Have you been involved in epidemiological studies on smoking and health?

A: Yes. One of my principal interests has been in defining the risks that are produced by cigarette smoking.

Q: Have you studied cigarette design?

A: Yes, in the course of serving as senior scientific editor for the 1981 Surgeon General's Report and as part of my work as both an author and editor of NCI Monograph 13.

Q: Have you ever been asked to consult with any foreign governments or any worldwide organizations?

A: Yes. I have testified for the Attorney General of Canada in a lawsuit where, as I understand it, the tobacco companies sued to overturn the laws in Canada on restrictive advertising.

I was also part of an expert committee convened by the Canadian Minister to examine the question of whether low tar and nicotine cigarettes reduce disease risks and whether the terms light, ultralight, mild, et cetera, were misleading terms. I also served both as an expert and now as a member of the World Health Organization's Scientific Advisory Committee on Tobacco. As the Court is likely aware, the World Health Organization's responsibilities include dealing with the public health and disease burdens that occur throughout the world and trying to reduce the amount of disease and illness that occurs throughout the world.

Q: Other than working on the Tobacco Control Project, have you served in any additional capacities on issues relating to smoking and health?

A: Yes. I've served in a wide variety of capacities with a number of different organizations.

Q: Please highlight some of those experiences for the Court and explain your involvement with each.

A: Among my professional activities related to smoking and health, I was on the Research Advisory Board for Harvard University's John F. Kennedy School of Government Tobacco

Policies Project. That consisted of examining their research work and providing advice about how to conduct that work as well as what opportunities might exist to do new projects. I have served with the U.S. Consumer Product Safety Commission in an effort to define the risks, and more specifically the changes in risk, that might accrue with producing cigarettes that had less ignition potential – that is, cigarettes that wouldn't cause clothing and furniture to catch on fire if the cigarettes were left to burn unattended. I have served with the Environmental Protection Agency on their Indoor Air Quality Board, and specifically in relation to their assessment of environmental tobacco smoke exposure. I served with the National Cancer Institute on their Policy Advisory Committee for the COMMIT Trial.

Q: What is the COMMIT Trial?

A: The COMMIT Trial was a community intervention that tried to develop a broad-based community approach to smoking cessation and used eleven community pairs.

Q: Please explain briefly the results of the COMMIT Trial.

A: The COMMIT trial demonstrated that activation of mid-size communities around specific tobacco control channels was able to change smoking behavior in moderate but not heavy smokers.

Q: What, based on your participation with the Policy Advisory Committee for the COMMIT Trial, is its value for the design of smoking cessation programs?

A: This trial suggests that effective tobacco control strategies may require implementation across larger community or geographic groups, such as states or large metropolitan areas.

Q: What other smoking and health projects have you worked on?

A: I served on the Evaluation Committee for the State of California, Tobacco Control Program, and I also served on the Tobacco Research and Education Oversight Committee – the legislatively mandated oversight committee for the State of California's Proposition 99 funds – which is a statewide tobacco control program. I chaired an evaluation for the State of Massachusetts on the assessment of newer tobacco products. I worked with the American Cancer Society in a variety of different capacities on their tobacco and health committee. I served on the board of the state organization, I've chaired their Research Committee, and I've been president of the local organization. I've also served on boards of the local lung association, and have worked with the state and national organizations and helped them plan their tobacco control programs.

Q: Please tell the Court about the San Diego Tobacco Control Coalition.

A: I chaired the San Diego Control Coalition – that was immediately after the passage of Proposition 99 in California – to provide coordination and oversight to the activities of both the county and the grants that were awarded to individual community organizations within San Diego County, all of which were focused on trying to reduce the disease burden that smoking caused in San Diego County.

Q: Have you worked or consulted with the U.S. Department of Health and Human Services?

A: Yes. I have been a consultant with the Department of Health and Human Services for 27 years now, and that included consulting with the National Cancer Institute to help them design their tobacco control efforts. I have also chaired a number of grant reviews for the National

Cancer Institute and participated in others.

Q: Are you familiar with the published scientific literature on smoking and health related issues?

A: Yes. Smoking and health is one of the most studied subjects in the field of public health. The Smoking and Health Database, maintained by the Centers for Disease Control and Prevention, U.S. Department of Health and Human Services, is a bibliographic database – accessible via the internet – which covers over thirty years of information and abstracts with over 62,000 items on smoking and health. The medical literature is replete with extensive epidemiological studies, conducted over decades, comparing the disease and death rates of millions of smokers and nonsmokers. Every relevant population and demographic grouping has been examined. Examples of these studies are: the American Cancer Society Prevention Study I and II (CPS-I and CPS-II), the British Physicians Study, and the Dorn Study of U.S. Veterans. This body of literature has been reviewed and presented in Reports of the Surgeon General on Smoking and Health published in 1964, 1967, 1968, 1969, 1971, 1972, 1973, 1974, 1975, 1976, 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986, 1988, 1989, 1990, 1992, 1994, 1998, 2000, 2001 and 2004.

Q: What have you published relating to the topic of the health consequences of smoking?

A: I have published a substantial number of articles on that topic, as is reflected on my curriculum vitae. They include chapters in both of the principal textbooks of medicine. There are two major textbooks of medicine used in medical schools across the United States. One is

the Cecil Textbook of Medicine, and the other one is the Harrison Textbook of Medicine. I've authored chapters in both of those texts. I've also published work on the effects of smoking cessation in the elderly, the relationship between media campaigns and other tobacco control efforts, changes in smoking behavior, and a variety of other topics.

Q: Before we talk about your experience with preparing the U.S. Surgeon General Reports, please briefly describe the office of the U.S. Surgeon General for the Court.

A: The Surgeon General commonly referred to is the Surgeon General of the Public Health Service. Each service – the Army, Navy and Air Force as well as the Public Health Service – has a Surgeon General. The Surgeon General of the Public Health Service supervises the agencies of the Public Health Service, and has come to be the public spokesperson or family physician, if you will, of the United States. He or she is commonly the person we turn to to get advice on risks, on various health behaviors, and on health changes that are important to the American public.

Q: While the Court has heard some testimony on the development of certain of the Surgeon General Reports, can you briefly describe for the Court your view of what the Surgeon General Reports represent and how they came to be?

A: Yes. The Surgeon General's Reports are comprehensive reviews of the world's published literature on smoking and health. They have evolved over time in content. The very first Report was not a Report of the Surgeon General but rather a Report to the Surgeon General by an expert committee. The period of the mid-1950s was the period where the scientific community identified and came to the consensus that smoking caused lung cancer. There was a response by the tobacco industry at that time with a substantial media and disinformation campaign that

questioned whether the scientific community had reached that conclusion and questioned the validity of a great many of the scientific studies that had been published. They articulated that both to the press and to the Congress. As a result, in 1962, President Kennedy requested of the Surgeon General that he convene an expert committee to review all of this evidence and reach a judgment. That expert committee was selected from individuals who had no prior public position on whether smoking caused disease. That list was provided both to the tobacco industry and to the public health community, and they were allowed to veto any name on the list for any reason without question. No one did. And that group then convened and met for about thirteen months, reviewing all of the existing evidence, interacting with one another, synthesizing that evidence, and finally reaching a judgment that was summarized in the 1964 Report, and the judgment included the statement that cigarette smoking was a cause of lung cancer.

Following that Report, legislation was passed by Congress mandating that there be an Annual Report to Congress that synthesized the existing evidence on smoking and health. So for a series of years, the Report consisted of a review of one year's worth of scientific literature, everything that had been published. It was then distilled down and presented as a volume. The volume usually took on one additional topic as a special chapter.

Q: How did you first become involved with preparing the Surgeon General's Reports?

A: Between the two periods of my medical training, from 1974-1976, I was a medical officer for the U.S. National Clearinghouse for Smoking and Health at the Centers for Disease Control of the U.S. Public Health Service. The Clearinghouse was established by Congress in the mid-1960s and was responsible for several issues of public health, one of which was to collect all of

the world's literature in one place in a library on smoking and health, and to collect that continuously on an ongoing basis. They were also given the responsibility under law to prepare the Surgeon General's Report on Smoking and Health. My responsibilities at that time included the preparation of the 1975 U.S. Surgeon General's Report on the Health Consequences of Smoking and editing the 1976 U.S. Surgeon General's Report. I also was involved in the development of surveys of smoking behavior for the National Clearinghouse, and I worked on a number of health education projects for the Bureau of Health Education.

Q: And you have been involved with the preparation of numerous Surgeon General's Reports since then, correct?

A: Yes. I have been an author, editor or reviewer for each of the annual Reports of the U.S. Surgeon General on the Health Consequences of Smoking since 1975. Specifically, I authored the 1975 Report and was the editor for the 1976 Report. I also authored multiple chapters in the 1979 Report and assisted in editing that volume. I was Consulting Scientific Editor for the Reports from 1980 through 1983, and I was the Senior Scientific Editor for the Reports from 1984 through 1986. Since that time, I have been a Senior Reviewer for each of the Reports, including the "Health Consequences of Smoking: Addiction" in 1988; "Health Consequences of Smoking: Twenty-five Years of Progress" in 1989; "Health Benefits of Smoking Cessation" in 1990; "Preventing Tobacco Use Among Young People" in 1994; "Tobacco Use Among U.S. Racial/Ethnic Minority Groups" in 1998; "Reducing Tobacco Use" in 2000; "Women and Smoking" in 2001; and "The Health Consequences of Smoking" in 2004.

Q: Please explain your roles as Consulting Scientific Editor and Senior Scientific Editor

of multiple Reports of the Surgeon General.

A: As consulting scientific editor, I would work with the other editors to edit sections of the text and incorporate reviewers' comments. As senior scientific editor, I was also responsible for developing a detailed outline of the issues to be addressed in the Report, working with the chapter authors to develop their sections, coordinating the integration of reviewer's comments and was also responsible for the overall scientific integrity of the document.

Q: What responsibilities did you have as a Senior Reviewer of the Surgeon General's Reports?

A: It was my responsibility to read through the entire volume to make sure that all of the conclusions and statements in the volume were supported by existing evidence and reflected the consensus of scientific thought in the United States, that the Report was balanced in terms of its presentation of evidence, that its tone in terms of expressing the conclusions were correct relative to the scientific evidence that existed, and then to send that information back, along with any specific recommendations that I had for areas that might be improved in the volume, to the Office on Smoking and Health.

Q: Do some of the Reports you have been directly involved with relate to the issue of addiction?

A: Yes.

Q: Do they relate to cigarette smoking and nicotine?

A: Yes. That is a critical element to understanding smoking behavior and its consequences.

Q: Do they relate to the issues of attempts to quit smoking?

A: Yes. The issue of cessation is discussed extensively through many of the different Reports.

Q: We will come back to the issue of cessation. Continuing with our discussion of the various Surgeon General's Reports, do the Reports you have been involved with relate to lung cancer caused by cigarette smoking?

A: Yes, they do.

Q: And do they also relate to lung disease, including chronic obstructive pulmonary disease or emphysema?

A: Yes, absolutely.

Q: Do you have an opinion about the conclusions presented in each of these Reports?

A: Yes. It is my opinion that the scientific conclusions presented in each of the Reports of the Surgeon General are based on the consensus of then-existing scientific understanding.

Q: How is consensus reached?

A: Consensus is reached by reviewing all of the scientific evidence available; examining that evidence for its strength and consistency, and then reaching a judgment as to whether the data support a conclusion – for example, a causal relationship between smoking and a disease. That conclusion and the evidence upon which it is based are then sent through the multi-level peer review process of the Surgeon General's Reports to ensure that the conclusion reflects the consensus of current scientific opinion.

Q: Are you familiar with the process by which the scientific conclusions in a Report of the Surgeon General are reached?

A: Yes, I have a detailed understanding, based on my knowledge of how these Reports have been developed and prepared since 1975.

Q: Please explain the process for the Court.

A: These Reports go through a very careful process to ensure that individual biases are not determining the conclusions or statements within the volume. That process occurs through a set of expert reviews of the Report at various stages in its preparation. Individual scientists, usually outside of the government, are first selected and asked to author chapters on a given topic. Sometimes the entire Report will be devoted to a specific topic, like cancer or heart disease or lung disease, but individuals are asked to offer chapters or sections on specific questions that relate to that, so that they can be assembled to cover the entire topic. The individual authors selected are extensively knowledgeable in the specific area that they are asked to write about, with the constraints that all of the pertinent scientific literature is to be considered and that conclusions of the chapter are to be based on the data presented in that literature rather than on the individual perspective of the author. Initial drafts of chapters are prepared for each Report by the individual authors, and the initial drafts are received by the editors and edited into chapters. Once the chapters are submitted, the editors make all subsequent changes and the chapters are not resubmitted to the authors for approval of those changes. The chapters are next sent out to a group of expert scientific reviewers for peer review of their scientific accuracy and completeness, as well as for balance, tone and appropriateness of the conclusions drawn from the scientific data. These comments are integrated into the volume, and the entire volume is sent out to a group of senior scientists in the academic community for review of the entire volume for its accuracy,

balance and tone. The Report is also formally reviewed by each of the agencies of the Public Health Service. Once these reviews are completed, the editors again integrate the comments into the text to strengthen the text and the science. Once that has happened, it is then submitted for formal clearance by the Centers for Disease Control, by the Assistant Secretary for Health and the Surgeon General, and then by the Secretary of Health and Human Services. Once it is cleared, it is transmitted as a formal requirement of the law to Congress as the official position of the Department on the issue. It is also released to the public and the press.

Q: How long does this process take, typically?

A: In the first fifteen years or so, that process was accomplished within about a one-year interval. Over the last decade or the last fifteen years, perhaps, given the vast expansion in that body of literature, it has required two to three years to accomplish that task.

Q: Why is this review process utilized?

A: The extensive review process is utilized because this is a very serious public health issue, and it is quite important that the science be accurate. This represents the official position of the U.S. Public Health Service and, therefore, the U.S. Government. We take great pains to then make sure that the information represents the true consensus of scientific thought at the time, not the perspective of a single individual who may have a biased approach to a given topic.

Q: You have had involvement with peer reviewed publications outside of the context of the Surgeon General's Report, is that right?

A: Yes.

Q: How does the review process for the Surgeon General Reports compare to the

typical peer review process?

A: The review process for the Surgeon General Reports is much more robust both in terms of the objectivity and in terms of the extensiveness. It has three different layers of review. The review is conducted by a larger number of individuals at each layer, and the authors of the individual sections do not get to incorporate the changes suggested by the reviewers. That is done by the editors rather than the individual authors. It is a much more objective process, and it is one that is much more robust in terms of its scope and the number of individuals involved.

Q: Are the scientists who work on the Surgeon General's Report invited to do so?

A: Yes. They are selected. The editors, authors and reviewers are all invited.

Q: Are the reviewers paid for their work?

A: No. All of the reviewers provide that service voluntarily because of the importance of this volume for public health.

Q: How are individuals selected to work on the Surgeon General's Report?

A: Selections are based on the scientific knowledge and stature of the individual, and the individuals are selected because of their record of publishing and being broadly knowledgeable on the specific issues that the Report is covering.

Q: Have you been involved in the preparation of other reports prepared by the Public Health Service on the issue of smoking and health?

A: Yes, I have. I was Senior Scientific Editor and contributing author for a series of tobacco control Monographs for the National Cancer Institute ("NCI"). I was also on the Science Advisory Board that reviewed the Environmental Protection Agency report, "Respiratory Health

Effects of Passive Smoking: Lung Cancer and Other Disorders" released in 1993.

Q: Please briefly explain the NCI tobacco control Monographs series.

A: Between 1990 and 2003 the National Cancer Institute produced 15 Smoking and Tobacco Control Monographs. These Monographs each focused on a specific issue or aspect of tobacco control and provided a vehicle to synthesize and disseminate information on tobacco issues. Of theses 15 Monographs, I was the editor for 10 of them and also contributed chapters or other sections of these volumes, as well as having been responsible with the National Cancer Institute in developing the entire series and helping choose some of the topics of the Monographs.

Q: Please describe the topics of each of the Monographs in which you were involved.

A: The ten monographs that I edited are: Monograph 1: Strategies to Control Tobacco Use In the United States: A Blueprint for Public Health Action In the 1990's (December 1991); Monograph 5: Tobacco and the Clinician: Interventions for Medical and Dental Practice (January 1994); Monograph 6: Community-Based Interventions for Smokers: The COMMIT Field Experience (August 1995); Monograph 8: Changes in Cigarette-Related Disease Risks and Their Implications for Prevention and Control (February 1997); Monograph 9: Cigars: Health Effects and Trends (February 1998); Monograph 11: State and Local Legislative Action to Reduce Tobacco Use (June 2000); Monograph 12: Population Based Smoking Cessation (November 2000); Monograph 13: Risks Associated with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine (October 2001); Monograph 14: Changing Adolescent Smoking Prevalence (November 2001); and Monograph 15: Those Who Continue To Smoke (September 2003).

Q: Are the Monographs, like Surgeon General's Reports, intended to provide a statement of the current scientific consensus on their topics?

A: In some cases, yes; in others, no. Some of these Monographs are intended to be comprehensive reviews of the existing literature coupled new analyses of smoking data with the goal of answering specific questions (Monographs 1, 9, 12, 13 and 15), while others are intended to be comprehensive analyses of available data on specific topics (Monographs 5, 6, 8, 11, and 14). Several have been intended to develop a scientific consensus on specific topics (Monographs 1, 9, 13).

Q: How were the Monographs you have been involved with prepared?

A: Each of the Monographs described above was prepared by groups of experts authoring sections and chapters, and each was peer reviewed. The extent of the peer review varied with the purpose of the Monograph. Those presenting comprehensive analyses of existing data underwent peer review similar to that conducted by most scientific journals, while those intended to answer specific questions underwent a two level review and clearance similar to that of the Surgeon General's Reports. Notable differences in the review process were that the authors participated in or approved the revisions of the chapters in response to the reviewers' comments and the clearance of the volumes was limited to the National Cancer Institute. This peer review and clearance process ensures that the conclusions expressed in the monographs express the consensus of current scientific thought.

Q: What memberships have you maintained in professional associations or societies?

A: I'm a Member of the American College of Chest Physicians, the American Thoracic

Society, the California Thoracic Society, the Society For Research on Nicotine and Tobacco, the San Diego Pulmonary Society, and the American Public Health Association, among others.

Q: I would like to talk briefly about honors or awards that you have received recognizing your work in the field of smoking and health. Have you ever been recognized by the American College of Chest Physicians?

A: Yes. I'm a Fellow of the American College of Chest Physicians, and have received the Alton Ochsner Award from the American College of Chest Physicians.

Q: What is the Oschner Award?

A: Alton Oschner was one of the founders of thoracic surgery in the United States who devoted a great deal of his energy to educating the public about the relationship between smoking and disease. The Oschner Award is granted for conducting distinguished research in smoking and health.

Q: What is the Joseph Cullen Memorial Award?

A: Dr. Cullen was one of the Associate Directors at the National Cancer Institute, and he quite rightly is given credit for encouraging and facilitating the large body of research that the National Cancer Institute has funded in the area of tobacco and health. He tragically died early in life from brain cancer, and the American Society of Clinical Oncology has named an award in distinguished service and preventive oncology in his name. I received that award as well.

Q: I understand that you also received the American Lung Association Life and Breath Award?

A: Yes. That is an award that I received for my work on environmental tobacco smoke in

1989.

Q: Are there any other awards that you received that you would like to tell the Court about?

A: Yes. Perhaps the award that I am most proud of is the Surgeon General's Medallion. I was awarded the medallion by C. Everett Koop, who was the Surgeon General at the time. And it was for my contributions nationally toward educating the public and the scientific community about issues relating to smoking and health.

Q: You have received a number of grants to study smoking and health issues, is that correct?

A: Yes, I have.

Q: What are some of those grants?

A: I have received funding from grants from the National Cancer Institute, from the Tobacco-Related Disease Research Program at the University of California, and also from the American Legacy Foundation to do research on tobacco. The specific issues include defining the disease risks of tobacco and examining questions of the relationship of marketing to smoking behavior.

Q: Dr. Burns, when you got involved in smoking and health issues some 30 years ago, was that for litigation purposes?

A: No. My work in smoking and health has been developed and implemented for purposes of science to try to understand these issues.

Q: But over the years, you have been called as an expert to testify in many courts on

issues of smoking and health, is that right?

A: That's correct.

Q: About how many times?

A: I have probably testified more than 30 times over the last twenty years in courts of law.

Q: Have you also been accepted by the courts in those cases as an expert with regard to low tar cigarettes, including the health effects of low tar cigarettes relative to full-flavor cigarettes?

A: Yes.

Q: Have you also been accepted by the courts in those cases as an expert with regard to Environmental Tobacco Smoke and its health effects?

A: Yes.

Q: Have you been accepted by the courts in those cases or in a number of those cases as an expert on nicotine addiction from cigarettes?

A: Yes, I have.

Q: And the effects of nicotine and cigarette smoking on lung cancer and chronic obstructive pulmonary disease?

A: Yes.

Q: You are familiar with the various diseases caused by smoking, correct?

A: Yes. The complete list of diseases caused by smoking is extensive. The 2004 Surgeon General's Report (U.S. Exhibit 88,621) concluded that smoking causes numerous forms of cancer, cardiovascular and respiratory disease, and a wide variety of other diseases. These

diseases are listed on pages 4-8 of the Report. As indicated on page 4 of the Report, smoking causes bladder cancer, cervical cancer, esophageal cancer, kidney cancer, laryngeal cancer, leukemia, lung cancer, oral cancer, pancreatic cancer and stomach cancer. It also causes abdominal aortic aneurysm, atherosclerosis, cerebrovascular disease and coronary heart disease. Pages 5-7 of the Report list a large number of respiratory diseases caused by smoking, including: chronic obstructive pulmonary disease, pneumonia, a reduction of lung function in infants, respiratory effects in childhood and adolescence (impaired lung growth during childhood and adolescence; early onset of lung function decline during late adolescence and early adulthood; respiratory symptoms in children and adolescents, including coughing, phlegm, wheezing, and dyspnea; and asthma-related symptoms (i.e., wheezing) in childhood and adolescence), and respiratory effects in adulthood (premature onset of and an accelerated age-related decline in lung function). Smoking also causes fetal deaths and stillbirths, reduced fertility in women, low birth weight, and complications of pregnancy (premature rupture of the membranes, placenta previa, placental abruption, preterm delivery and shortened gestation), cataract, hip fractures, low bone density, peptic ulcer disease, and adverse surgical outcomes due to poor wound healing and respiratory complications.

Q: Dr. Burns, I have handed you what is marked as U.S. Exhibit 58,700. Could you please identify this document?

A: Yes. It is a copy of Monograph 13, "Risks Associated with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine."

Q: Did you play a role in the creation of Monograph 13?

A: Yes. I authored two of the chapters, and I was one of two editors for that volume.

Q: Which chapters did you author?

A: I authored Chapter 1 and Chapter 4 of the Monograph.

Q: When was Monograph 13 published?

A: It was published in November of 2001.

Q: What was the purpose of Monograph 13?

A: The Federal Trade Commission ("FTC") requested that the National Cancer Institute ("NCI") and the Food and Drug Administration ("FDA") conduct a comprehensive review of the scientific evidence on the risks of smoking lower yield cigarettes to determine whether or not the evidence supported the conclusion that low tar and nicotine cigarettes are less harmful to smokers than full-flavor cigarettes. There had been considerable concern in the public health community that the information provided by the FTC tar and nicotine testing method was being inaccurately interpreted, that its original purpose – as a means of measuring the difference in disease risks of different cigarettes – was not valid; that the light and low tar cigarettes were indeed not less risky. That concern grew, and the FTC was receiving substantial criticism for its continued maintenance of the FTC Method of measuring tar and nicotine. The FDA and the NCI elected to use the monograph process to conduct and provide a review of the science.

Q: Do you have an understanding of why the FTC Method was first implemented?

A: Yes, I do. Prior to 1967, the different cigarette manufacturers were making claims about the tar deliveries of their cigarettes which could not be easily reconciled because they were based on numerous different testing methods. This resulted in consumer confusion. In order to reduce

the confusion produced by the competing claims, in 1967 the FTC promulgated standards for measurement of tar and nicotine using a standard machine smoking protocol, which is commonly referred to as the FTC Method.

Q: On what do you base your understanding of why the FTC Method was implemented?

A: As part of my work with the Surgeon General's Reports I examined how the FTC measured tar and nicotine yields of cigarettes and the history of selecting that method for identifying relationships of those yields to disease risks. Among other sources, this testing method is set out in NCI Monograph 7 (U.S. Exhibit 78,714), in Chapter 2.

Q: Do you have an understanding of what the FTC Method was intended to do?

A: It was recognized early on that there was an increase in lung cancer risk with increasing smoke exposure, and it had been demonstrated that most of the carcinogenicity of tobacco smoke was in the particulate phase of the smoke. That led to a suggestion by several leading public health experts that cigarettes which delivered less tar to smokers might also deliver less risk. Neither the FTC nor the public health community expected that the machine measured values by the FTC protocol would be accurate estimates of the smoke exposure of individual smokers smoking individual cigarettes, because of the well recognized variability among smokers in how they smoked individual cigarettes. What measurements under the FTC protocol were expected to do was to allow smokers to select brands of cigarettes that would deliver less tar and nicotine to the smokers who smoked them. The expectation was that smokers who switched from a high tar brand to cigarettes with a low tar measurement would actually be exposed to less tar when they

smoked, and that the lower tar exposure would result in less risk.

Q: How was the subject matter of Monograph 13 determined?

A: The FTC asked the NCI to address the specific question of whether there was a reduction in disease risks for the use of low tar and nicotine cigarettes.

Q: Did you choose yourself to be the scientific editor for Monograph 13?

A: No. I was chosen by the FDA and NCI.

Q: When were you first approached to participate in this Monograph?

A: I do not recall the exact day, but my recollection is it was sometime in 1998. Representatives of the National Cancer Institute and the Food and Drug Administration approached me and asked me if I would edit that review along with Dr. Neal Benowitz.

Q: Who were some of the other scientists who were involved in Monograph 13?

A: There were a number of distinguished scientists. In addition to Dr. Benowitz, who was the co-editor with me, Dr. Lynn Kozlowski and Dr. Dietrich Hoffman were among those involved. As a group, the authors were selected because they were among the world's preeminent scientists in areas related to the topics of the Monograph.

Q: Who selected the scientists that were involved with Monograph 13?

A: The Food and Drug Administration and the National Cancer Institute selected me and also selected Dr. Benowitz to be the editors of the volume. The others were selected by the National Cancer Institute in consultation with myself and Dr. Benowitz.

Q: Please describe briefly for the Court the process by which Monograph 13 was completed.

A: Monograph 13 reflects the complexity of integrating all of the lines of evidence required to develop scientific conclusions. The Monograph went through an extensive internal review. The completed chapter drafts were sent out for review and revised based on the comments received. The completed volume was then sent out for a second review and once again the reviewers' comments were incorporated into the text. The volume was then submitted to the NCI for review and once again comments of the reviewers were discussed, responded to and the text was again revised. Concerns were raised by Richard Peto, a distinguished tobacco epidemiologist, about the interpretation of trends over time in lung cancer rates at young ages in Great Britain. An additional external discussion of that issue was conducted at a meeting in Toronto which included Richard Peto and several other experts, in order to ensure that all of the differing perspectives on this complex issue were considered in framing the language and content of the conclusions. The volume was again subjected to a detailed external review, and the text was once again revised to include the improvements suggested in that review. Once the chapters were in final form, careful consideration was given to the specific language for the conclusions of the chapters and the overall conclusions of the volume. After several iterations language for the conclusions was agreed upon which most accurately expressed the evidence discussed in the volume. This final text and conclusions were then submitted for formal clearance and approval by the National Cancer Institute as the official NCI position on the science.

Q: Dr. Burns, why was there such a long review process for the Monograph?

A: The extensive review and prolonged consideration of Monograph 13 reflects both the complexity of the science being considered and the care taken by the NCI and the scientists

involved to ensure that the most accurate and complete description of the evidence was provided. This careful consideration of the issues and data by multiple individuals as part of a formal review and clearance process was intended to, and did, accomplish the objective of developing conclusions that are based solidly on existing evidence and that express the understanding of that evidence as clearly and as completely as possible.

Q: Each of the chapters in Monograph 13 had conclusions; is that correct?

A: That's correct.

Q: I'd like to start by discussing the two Chapters that you authored in the Monograph, Chapters 1 and 4. Please briefly explain the conclusions reached in Chapter 1 for the Court.

A: Chapter 1 is the overall summary chapter for the rest of the volume. The first conclusion is that "[e]pidemiologic and other scientific evidence, including patterns of mortality from smoke-caused diseases, does not indicate a benefit to public health from changes in cigarette design and manufacturing over the last 50 years." The second conclusion is that "[f]or spontaneous brand switchers, there appears to be a complete compensation for nicotine delivery, reflecting more intensive smoking of lower-yield cigarettes." The third conclusion is that "[w]idespread adoption of lower yield cigarettes in the United States has not prevented the sustained increase in lung cancer among older smokers." The fourth conclusion is that "[m]any smokers switch to lower yield cigarettes out of concern for their health, believing these cigarettes to be less risky and to be a step toward quitting," and that "[a]dvertising and marketing of lower yield cigarettes may promote initiation and impede cessation, more important determinants of

smoking-related diseases." The fifth conclusion is as follows: "Measurements of tar and nicotine yields using the FTC Method do not offer smokers meaningful information on the amount of tar and nicotine they will receive from a cigarette. The measurements also do not offer meaningful information on the relative amounts of tar and nicotine exposure likely to be received from smoking different brands of cigarettes."

Q: Please direct your attention to Chapter 4 of Monograph 13. What were the conclusions reached in Chapter 4?

Conclusion number one is that "[c]hanges in cigarette design and manufacturing over the last fifty years have substantially lowered the sales-weighted, machine-measured tar and nicotine yields of cigarettes smoked in the United States." Number two, "[c]igarettes with low machine-measured yields by the FTC method are designed to allow compensatory smoking behaviors that enable a smoker to derive a wide range of tar and nicotine yields from the same brand, offsetting much of the theoretical benefit of a reduced-yield cigarette." Number three, "[e]xisting disease risk data do not support making a recommendation that smokers switch cigarette brands. The recommendation that individuals who cannot stop smoking should switch to low yield cigarettes can cause harm if it misleads smokers to postpone serious efforts at cessation." Number four, "[w]idespread adoption of lower yield cigarettes by smokers in the United States has not prevented the sustained increase in lung cancer among older smokers." Number five, "[e]pidemiologic studies have not consistently found lesser risk of disease, other than lung cancer, among smokers of low yield cigarettes. Some studies have found lesser risks of lung cancer among smokers of reduced yield cigarettes. Some or all of this reduction in lung

cancer risk may reflect differing characteristics of smokers of reduced-yield compared to higher-yield cigarettes." Number six, "[t]here is no convincing evidence that changes in cigarette design between 1950 and the mid 1980s have resulted in an important decrease in the disease burden caused by cigarette use either for smokers as a group or for the whole population."

Q: Have you concluded to a reasonable degree of scientific certainty whether light or low tar cigarettes have reduced the risks associated with smoking compared to the regular cigarettes for those who have smoked those products?

A: Yes, I have.

Q: What have you concluded?

A: I have concluded that the changes in cigarettes that resulted in a lowering of the FTC tar and nicotine yields over the past 50 years have not resulted in a reduction in the disease risks of smoking cigarettes for the smokers who use these cigarettes.

Q: Are the conclusions listed in Chapter 4 based on the new analyses done therein?

A: No. They are based on all the evidence presented from the published literature and cited in the monograph. The conclusions are based on all the evidence available and all of the chapters of the Monograph. The conclusions are not dependent on the new analyses presented. The new analyses are presented to provide examples of how issues raised in the volume could influence the analyses in epidemiological studies.

Q: Were these conclusions shared by the authors of Monograph 13?

A: Yes; these conclusions were specifically signed off on by all of the authors of this chapter and also by the National Cancer Institute.

Q: How did the scientists involved come to agree on the conclusions of Monograph 13?

A: As with many consensus statements, it was an iterative process. Language was written, individuals responded to that language, made suggestions for alternatives, raised concerns, expressed ideas, explored issues, and over time, and in this case it was the better part of about a year, these conclusions emerged as the consensus.

Q: Can you say, with a reasonable degree of scientific certainty, that these conclusions represent the consensus view of the scientific community as of the time that Monograph 13 was published and thereafter?

A: Yes, that represents the consensus of the scientific community on this issue.

Q: What are some of the main scientific publications that support your statement?

A: NCI Monograph 13 (U.S. Exhibit 58,700), the 2001 IOM publication "Clearing the Smoke" (U.S. Exhibit 20,919), and the 2004 Surgeon General's Report (U.S. Exhibit 88,621) are a few of the publications that demonstrate that the conclusions we have just discussed represent the consensus view of the scientific community.

Q: Was Monograph 13 the first time that a U.S. Government document had concluded that low tar cigarettes offered no health benefit?

A: That is the first time that it has been expressed that clearly, yes. An Institute of Medicine report which was done at the behest of the Government came to a similar conclusion about a year earlier but did not express it as clearly.

Q: I'd like you to take a look at U.S. Ex. 88,621. Can you identify this document?

A: Yes, it is a copy of the 2004 Surgeon General's Report.

Q: Directing your attention to page 25, what is the third "Major Conclusion" of the Report?

A: "Smoking cigarettes with lower machine-measured yields of tar and nicotine provides no clear benefit to health."

Q: Based on your scientific expertise, do you agree with this statement?

A: Yes, I do.

Q: Please turn to page 325 of the Report. What is listed on this page?

A: The conclusions to Chapter 2, which deals with cancer, are listed on this page.

Q: What is Conclusion 3 on this page?

A: "Although characteristics of cigarettes have changed during the last 50 years and yields of tar and nicotine have declined substantially, as assessed by the Federal Trade Commission's test protocol, the risk of lung cancer in smokers has not declined."

Q: Based on your scientific expertise, do you agree with this statement?

A: Yes, I do.

Q: Have any other bodies in the public health community reached conclusions similar to those in Monograph 13?

A: Yes. Monograph 13 is consistent with the recently expressed reviews of the evidence by the Institute of Medicine, The World Health Organization's Scientific Advisory Committee on Tobacco, and the Canadian Expert Committee.

Q: To your knowledge, have reviews by other public health agencies contradicted the conclusions of Monograph 13 that we just discussed?

A: Not to my knowledge.

Q: I want to talk about the way that your knowledge and that of the scientific community developed concerning the comparative disease risks of light and low tar cigarettes. How has scientific knowledge developed in this area, beginning in the 1960s?

A: Early on, we were operating from a dose response understanding of the generation of cancer. That is, if you receive less tar, you would get less cancer. Epidemiological studies from the late 1960's onward suggested that populations of smokers who chose to smoke lower tar or filtered cigarettes had lower lung cancer risks, but not lower risks of other diseases caused by smoking. These findings were particularly exciting at that time, since smokers had been smoking these reduced-yield cigarettes for only short periods of time. We expected that as more individuals used these products for longer periods of time, the reduction in disease risk would increase and national lung cancer death rates would fall.

Q: Can you point us to any scientific publications that discuss these early epidemiological studies?

A: Yes; these studies are discussed in NCI Monograph 7 (U.S. Exhibit 78,714), in the 1981 Surgeon General's Report (U.S. Exhibit 74,603), in the 2001 Institute of Medicine publication "Clearing the Smoke" (U.S. Exhibit 20,919), and in Monograph 13 (U.S. Exhibit 58,700).

Q: Did lung cancer death rates go down as a result of low tar cigarettes?

A: No. The reduction in lung cancer rates expected based on the epidemiological studies has not occurred in national lung cancer death rate trends. Use of lower-yield cigarettes did grow until they were the dominant type of cigarette on the U.S. market, and 97 percent of the cigarettes

sold in the United States are currently filtered cigarettes. However, lung cancer rates continued to rise. Lung cancer death rates finally peaked in 1990 among white males, and they continue to rise among women in spite of their higher prevalence of low-yield cigarette use. These lung cancer trends are adequately explained by changes in smoking prevalence due to cessation and reduced initiation without postulating reductions in disease risks due to changes in cigarette design.

Q: What scientific publications do you rely upon to support your statements about lung cancer trends?

A: Monograph 13 (U.S. Exhibit 58,700), the 2001 IOM publication "Clearing the Smoke" (U.S. Exhibit 20,919) and a 2001 article in the journal *Lung Cancer* by Mannino and others all support this conclusion. Mannino, D.M., Ford, E., Giovino, G.A, Thun, M., Lung cancer mortality rates in birth cohorts in the United States from 1960 to 1994. *Lung Cancer* 31(2-3):91-99, 2001.

Q: What, if anything, has the evidence from prospective mortality studies conducted over long periods of time indicated?

A: Prospective mortality studies of smokers in the United States and the United Kingdom revealed an increase, rather than a decrease, in the risk of smoking over a period when tar and nicotine yields of cigarettes were declining. Data from two large prospective mortality studies conducted by the American Cancer Society ("ACS") more than 20 years apart (from 1959 to 1966 and from1982 to 1988) are particularly compelling. These two studies are referred to as CPS-I (indicating "Cancer Prevention Study") and CPS-II. Machine-measured tar and nicotine

yields of U.S. cigarettes declined dramatically in the interval between these two studies, and the machine-measured yields of the cigarettes actually smoked by the participants in these two studies were dramatically different as a result. Despite the substantive reduction in tar yield of the cigarettes smoked in CPS-II, lung cancer disease risks increased rather than decreased in comparison to CPS-I, even when controlled for differences between the two studies in number of cigarettes smoked per day and duration of smoking. Thus, there has been an increase rather than a decrease in the death and disease caused by smoking over the last several decades in the United States in spite of the reduction in tar yields and other design changes in the cigarettes manufactured over that interval.

Q: When did you first conclude that low tar cigarettes weren't any better for people?

A: That conclusion slowly emerged in my mind during the late 1990s.

Q: How did the 1981 Surgeon General's Report reach its conclusion that lower yield cigarettes might offer less risk for those who could not quit?

A: In 1980, when we were looking at the epidemiology on the risks of smoking low yield cigarettes for the 1981 Surgeon General's Report, we were concerned because lung cancer rates were still climbing in males and were climbing rapidly in females. Filtered cigarettes had by then been available for over twenty years with a substantial reduction in tar and nicotine for ten or more years. There should have been plenty of time for a reduction in deaths to have begun to occur. But the evidence that we had when I was working with the 1981 Surgeon General's Report suggested that the cigarettes delivered less tar and nicotine and the epidemiology supported a reduction in lung cancer risk for those smokers who used lower yield cigarettes.

Therefore, even though we were concerned, we felt that the science was sufficient to draw a conclusion that these cigarettes were less hazardous if people switched to them.

Q: What information was obtained after the 1981 Surgeon General's Report was published that informed this issue?

A: Subsequent to the 1981 Report, data was published that the amount of nicotine and amount of tobacco contained in a low tar and nicotine cigarette was similar to the amount of nicotine and the amount of tobacco in a higher tar and nicotine cigarette, that people smoked specifically based upon their addiction, that that populations of individuals who smoked cigarettes with widely different nicotine yields by the FTC method had similar amounts of nicotine in their bodies.

The understanding then progressed through the mid 1990s to show that people smoked low tar and nicotine cigarettes very differently. They blocked the ventilation holes, they drew bigger puffs from these cigarettes, they inhaled those puffs more deeply, they took more puffs on the cigarettes, and they took faster puffs. All of those changes increased the tar and nicotine delivery of the cigarettes that were on the market as lower yield cigarettes.

Studies of lung cancer rates among smokers who were examined during the 1960s were compared to those of smokers who were examined during the 1980s, a period of time during which the machine-measured tar and nicotine yield of cigarettes dropped by sixty percent, demonstrated that, instead of going down, lung cancer risks actually went up.

And finally, we obtained information on the design and testing of these cigarettes from the internal tobacco industry documents, and we discovered that they were designed and tested

for characteristics that would yield increasing amounts of tar and nicotine when smoked differently. They were tested under conditions where the holes were blocked and with increased puff volumes. We then had an understanding of what was going on scientifically sufficient to reach a conclusion that our earlier statement in the 1981 Surgeon General's Report had been erroneous. We had been in error in 1981 because we did not understand the characteristics of the cigarette and how people change their smoking pattern based on addiction as it interacted with the cigarette design characteristics.

As we came to understand that people fully compensated, we understood that they did not receive less tar and less nicotine from low yield cigarettes, and therefore, there was no reason to expect a risk reduction. That understanding allowed us to close the discrepancy between the existing epidemiologic studies which showed a decline in risk and the national death rates which did not show a decline.

Q: Did the conclusions reached in Monograph 13 come as a surprise to you?

A: It wasn't a surprise at that point in time.

Q: Why is that?

A: As I mentioned before, concern on my part and on many other scientists' part had been emerging since the mid 1990s for a variety of reasons, including the fact that we had expected that there would be a reduction in lung cancer by that time, because beginning in the 1950s, more than sixty percent of the population had switched to smoking filter cigarettes and smoking cigarettes with purportedly lower tar yields. By the 1980s, there was a sixty percent reduction in the machine-measured tar and nicotine yields. We therefore expected a reduction in lung cancer

rates in the 1970s, 1980s and early 1990s. We kept waiting to see it happen, and it didn't. A second line of evidence emerged. When studies were done at two points in time, a time when high tar and nicotine cigarettes were available and more recently when low tar and nicotine cigarettes were the ones that were being used, what we saw was not a reduction in the risk of smoking but actually an increase. That occurred in studies both here and in the United Kingdom. Those two lines of evidence made us concerned that the prior recommendations and the prior evidence were not valid. In the mid 1990s, we began to get access to internal tobacco industry documents that demonstrated how the cigarettes were actually designed and what testing was done on those products. And that filled in the missing link.

Q: Is the concern that you mention reflected in any of the Public Health Service publications between the 1981 Surgeon General's Report and the publication of Monograph 13 in 2001?

A: Some of the same concerns were expressed in the 1989 Surgeon General's Report.

Q: To your knowledge, did any of the defendants to this lawsuit inform consumers, either through advertising, warning labels, or other public statements, about the concern for disease risks of low tar cigarettes held by the Public Health Service and the mainstream scientific community during the time period we have discussed, 1981 to 2001?

A: No, the defendants almost uniformly denied that any cigarettes caused any disease risk at all.

Q: How do you explain the early epidemiological findings in light of what you have seen in lung cancer mortalities since that time?

A: The reasons for the disparity between the epidemiological studies and what actually occurred in the population of smokers relate to differences in the individuals who choose to smoke cigarettes marketed as delivering less tar and risk, the tendency of many smokers to compensate for the reduced yield by smoking cigarettes more intensely, and the use of cigarettes smoked per day as a measure to control for difference in smoke exposure in epidemiological studies which results in an artifactual risk reduction in the studies.

Q: Dr. Burns, I'd like to ask you some questions about the passage that appears at the bottom of page 1, in the Preface of Monograph 13. To start, can you read that passage for the Court?

A: Yes. It states that "This monograph is unique in another important aspect. For the first time, the authors who have prepared the various chapters have had extensive access to the information gleaned from the internal documents of the tobacco companies. The tobacco industry files that are open to the public and available on the Internet constitute some 33 million pages of formal and informal memos, meeting notes, research papers and similar corporate documents. Included are marketing strategies that express the growing concern among the various tobacco companies of the potential loss of new recruits. This concern over the potential loss of market was due to the evolving public opinion that smoking is harmful to health and that it is related to many of the illnesses that smokers experience over the course of their lives."

Q: Are the conclusions that you described from Chapters 1 and 4 based in part on a review of those documents?

A: Yes. They include information that came from that review.

Q: How you reach conclusions in Monograph 13 from this review of tobacco industry documents?

A: The expectation has been that low tar and nicotine cigarettes reduced disease risk because the smoker would actually get less tar when they smoked those products. When we looked at the internal tobacco industry documents, we were able to understand that these cigarettes had been designed specifically to produce a low level when measured by machine but to have an elasticity of delivery when they were smoked by individual smokers. The internal tobacco industry documents demonstrate the design characteristics, the design intent for developing these products, the testing that was done, the conditions under which they were tested, and the results of that testing demonstrate that what was happening was that cigarettes were intentionally being designed to vary the yield when smoked differently, and that the reason for that design was to satisfy the nicotine ingestion required by individual smokers. So the product could produce a very low level when smoked by machine; but when smoked by the individual, the individual could derive from that cigarette as much nicotine as they needed to satisfy their addiction. That piece filled in one missing link that allowed us to bridge what had been a confusing conflict in the scientific evidence; that some of the epidemiologic studies showed a reduction in disease risk, whereas we didn't see that reduction when we looked at populations, and we didn't see that when we looked at different groups examined at different points in time. So all of that evidence accumulated over the late 1990s, and we then were asked to review all of that evidence in total. And when you look at what we know about the cigarette, what we know about addiction, what we know about compensation, what we know about the epidemiologic data, and what we know

about disease risks, we were able to reach the conclusion that there is not evidence that establishes any benefit to these products.

Q: And based upon your review of those documents, you're giving us your interpretation of the information that was available to the industry based upon what was in those documents, correct?

A: Yes. Specifically, what I am giving you is my understanding that when an experiment is conducted that examines conditions that are similar to the way a cigarette is smoked in real life but dissimilar to the way the cigarette is smoked by the FTC method, the understanding of the characteristics being sought in the engineering studies are characteristics that enhance the differences between what the FTC method reports and what the smokers is actually likely to receive. That is the design intent of that examination.

Q: You mentioned that low tar cigarettes had been designed specifically to produce a low level when measured by machine but to have an elasticity of delivery when they were smoked by individual smokers. What would be the point in doing that?

A: As stated by Monograph 13 (U.S. Exhibit 58,700), the IOM Report (U.S. Exhibit 20,919) and the 2004 Surgeon General's Report (U.S. Exhibit 88,621), the industry used low machine-measured tar and nicotine levels to reassure the public that smoking filtered or "low tar" cigarettes was safe, or at least safer than unfiltered or high tar products.

Q: What were some of these design changes?

A: One of the earliest modifications of the cigarette marketed to reassure smokers about the disease risks of smoking was the filter. However, industry scientists understood that smokers

were smoking to obtain nicotine and that they would change their smoking behavior to preserve their intake of nicotine, eliminating any beneficial effect of filters. Another is filter ventilation, where tiny holes are placed in the cigarette filter to lower the machine-measured tar and nicotine levels. If you have a machine pulling smoke through the cigarette and you poke holes in the filter, what do you suppose happens? When the machine draws on the filter, part of what comes in is the smoke and a large part of what comes in is just the air drawn in through those holes. This reduces the amount of smoke measured by machine. The holes are placed so the machine won't cover them. So when the machine pulls on that cigarette, it gets air, but humans are not smoking to get air. So when a smoker puffs in on the cigarette, if what he or she gets is a lot of air through the holes, he or she changes the way he or she smokes because the smoker wants nicotine. What smokers then do is block those holes with their lips or they inhale faster and harder and deeper until they work the cigarette so that they can get from it the nicotine they need. When they do that, they don't get one milligram of tar they get a full dose of tar. They get the same dose of tar that they would get from a full-flavored, regular cigarette.

Q: Do smokers consciously know that they are attempting to get more nicotine?

A: No. There is a receptor in the back of your throat that senses the nicotine coming in. Nicotine doesn't go directly to your brain from that sensor, but it tells you how much nicotine is coming in. You have a perception of how much nicotine is coming into your mouth and, therefore, how much is going to hit your brain seven or eight seconds later. And so you automatically – because you want the nicotine hitting your brain – you automatically explore and change and experiment with how you draw on that cigarette. Smokers, when given one of these

cigarettes, will change the way they smoke that cigarette on the first puff.

Q: They compensate in order to get more nicotine?

A: They compensate in two fashions. One, on the first puff they compensate. And then they learn how to work the cigarette, blocking the holes with their fingers, blocking the holes with their lips, drawing harder, drawing faster, until they get from their cigarette the same amount of tar and nicotine that they would get with a higher tar cigarette.

Q: Have you heard the proposition that compensation is temporary?

A: Yes.

Q: Will you please explain that proposition to the Court?

A: When smokers first switch to lower delivery cigarettes, they adopt a variety of changes in smoking pattern to increase the delivery of nicotine. They take bigger puffs, faster puffs, more puffs and block the holes. As they continue to smoke the cigarettes, they modify these behaviors and some of the differences are reduced or eliminated. These changes occur over the first days of using a new cigarette and revert back quickly leading some investigators to assume that the compensatory changes are only temporary. However, what happens in reality is that smokers experiment with different patterns of smoking to see which patterns provide the most effective delivery of nicotine and persist in using those patterns. The most common persistent change is an increased puff volume.

Q: Do you believe the theory that compensation is temporary to be a valid one?

A: No.

Q: Why not?

A: Studies of smokers who use cigarettes with markedly different nicotine yields by machine testing have similar levels of nicotine in their body when studied with natural use of cigarettes. This shows that compensatory behavior persists in terms of total nicotine delivery even though some of the individual changes in puffing pattern may not persist.

Q: I assume you have also heard the argument that compensation is not complete?

A: Yes.

Q: Will you explain that argument to the Court?

A: That argument suggests that when smokers shift to lower delivery cigarettes they change their pattern of smoking but maintain only a fraction of their prior nicotine intake. It is based on studies where smokers smoked under laboratory conditions or studies where smokers were forced to switch brands as part of the study. In those settings smokers who switch to lower yield brands have lower nicotine intake.

Q: In your opinion, is the theory a valid one?

A: No.

Q: Why not?

A: Because the studies on which the theory is based are ones that introduce an artificial reason for changing brands and require the smoker to continue using that brand even if the smoker does not like the brand or is not satisfied by it. Those smokers might get less nicotine and they might also take the switching trial as an opportunity to voluntary reduce their smoking. In the real world, smokers who switch and do not like the new brand or are not satisfied by it don't continue to use it, they switch back to another brand. As a result, they only stay with the new

brand if it satisfies their nicotine need. This self selection explains why the levels of nicotine measured in smokers shows little relationship to the nicotine yield on machine testing.

Q: Do you consider the design of cigarettes that are marketed as light or low tar to be deceptive?

A: Absolutely. There is a dual deception. First, you would expect that in a low tar and nicotine cigarette you would start out with less tobacco and less nicotine in the tobacco rod. That turns out to be false. Low tar cigarettes have an amount of tobacco and nicotine in the rod similar to high-tar cigarettes. The cigarette manufacturers can simply take the same cigarette and change the size of the ventilation hole and call it a different yield of tar and nicotine. That's why a smoker can get the same amount of nicotine from it when it is smoked differently. The second part is that one would expect that because you have a long filter, it would remove all the bad stuff. And, indeed, it does take out some of it; but you're not smoking to get air, you're smoking to get nicotine. If the filter removes a little bit of the smoke as it comes through, you just draw harder and deeper until you get a full dose of tar and nicotine. So the perception of most smokers when they see a cigarette, and it's a low tar cigarette, is that they believe that when they smoke this cigarette, they're going to get less tar and nicotine. And that isn't true. They get the same amount of tar and nicotine as they would get if they were smoking a higher tar and nicotine cigarette.

Q: What, if anything, do the internal industry documents that you have reviewed reveal about these engineering design changes?

A: The actions of the tobacco companies in developing cigarettes that could be marketed as

lower risk products are well documented by internal industry documents. Those documents demonstrate that the companies not only knew that low tar cigarettes did not deliver lower smoke exposure to the smoker, but also that these engineering changes in cigarettes were intended to take advantage of the known compensatory changes among smokers such that the cigarettes yielded very low levels of tar and nicotine when smoked by machine, but much higher levels of tar when smoked by smokers. For instance, in a March 24, 1961 internal Philip Morris memorandum from Helmut Wakeham to Hugh Cullman (U.S. Exhibit 35,484) Dr. Wakeham wrote:

> As we know, all too often the smoker who switches to a hi-fi cigarette winds up smoking more units in order to provide himself with the delivery which he had before. In short, I don't believe the smoking pattern has changed much, even with cancer scares and filter cigarettes.

Q: Is this document cited in Chapter 4 of Monograph 13?

A: Yes.

Q: In your opinion, what does this document indicate regarding the defendants' understanding of compensation?

A: This observation by Dr. Wakeham indicates that the tobacco companies knew that smokers were smoking to derive a fixed amount of nicotine and would change their smoking behaviors (compensate) to preserve that intake of nicotine when cigarettes were modified to deliver a lower dose of nicotine under machine smoking protocols.

Q: How, if at all, does reassuring the public that smoking filtered or "low tar"

cigarettes was safe, or at least safer than unfiltered or high tar products, benefit the tobacco industry?

A: It keeps at least some smokers smoking; many smokers who were concerned about the risks of smoking responded by switching to low tar cigarettes instead of quitting.

Q: Are there smokers who switch to low tar cigarettes for health reassurance purposes?

A: Yes. Marketing of light cigarettes as delivering less tar, and by implication less risk, has resulted in many smokers who switched from higher yield cigarettes reporting that they did so in an attempt to reduce disease risk. This is discussed in NCI Monograph 7 at Chapter 4 (U.S. Exhibit 78,714), and in NCI Monograph 13 at Chapter 6 (U.S. Exhibit 58,700).

Q: Are smokers who switch to lights as part of a quitting strategy more likely to quit smoking than those who do not switch?

A: Many smokers switch to lower yield cigarettes as part of an effort to quit or substantially reduce their smoking, but existing evidence suggests that they are not more likely to quit successfully than those who do not switch.

Q: What is the significance of this?

A: Smokers who delay cessation by switching to lights face an increasing disease risk instead of the decreased disease risk that would have occurred through cessation. The reality that many smokers switched to light cigarettes rather than quit, as was the expressed intent of the tobacco industry, means that they have foregone the opportunity to quit, a change that would have produced real reductions in disease risks.

Q: To your knowledge, did the defendants in this case voluntarily provide the public

health agencies of the U.S. Government with any information about elasticity of delivery?

A: No. The tobacco companies failed to disclose to public health authorities their internal industry understanding of the deceptive nature of low tar cigarettes and the influence of compensatory behavior on exposure of smokers who used these cigarettes. The companies were benefiting from the lack of knowledge among smokers and among public health authorities.

Q: Did you cite and quote from documents of the defendants in this case in the text of Chapters 1 and 4 of Monograph 13 that demonstrate their knowledge that you just discussed regarding the design and marketing of low tar cigarettes?

A: Yes.

Q: What conclusion(s), if any, have you reached as a result of your review of internal documents of the defendants?

A: The internal industry documents that I have reviewed establish that the cigarette companies were aware of nicotine compensation and design changes that could be employed to facilitate compensation by smokers. They show that the companies were aware that the FTC-machine measured yield was misleading to consumers and that the FTC yield it provided little to no information on how much tar and nicotine, whether in absolute or relative terms, were likely to be ingested into the smoker's body.

Q: What effect, if any, did your review of the internal industry documents have on the conclusions of Chapter 4 of Monograph 13?

A: It confirmed them.

Q: Did you feel any responsibility to tell the American people about what you learned

as a result of the work involved in Monograph 13?

A: Yes. In 1981, I was one of the editors of the Surgeon General's Report. Although it included several caveats, that Report concluded that if you couldn't quit, you were well-advised to switch to low tar and nicotine cigarettes. Over the ensuing twenty years, many people who were powerfully addicted, struggling with that addiction, latched onto that recommendation as a reason for not quitting. A review that we conducted with Monograph 13 made it clear that the recommendation was erroneous, particularly in light of the scientific information that was available to the tobacco industry but not to the public health community as of 1981. And, therefore, I felt a deep responsibility to try and correct that recommendation and that misinformation that had been provided by the Surgeon General's Report to the American public.

Q: Is it your opinion that the defendants in this case use low tar cigarettes to allay smokers' health concerns, but that defendants have long understood that these cigarettes would not actually be less harmful to smokers?

A: Yes. As the tobacco companies developed their understanding that cigarettes needed to deliver a sufficient dose of nicotine in order to satisfy the smoker and that smokers would change their smoking behavior in predictable ways to compensate for changes that reduced machine yield, the opportunity presented itself to design and develop cigarettes that could be marketed to reassure smokers that they were reducing their exposure based on low machine measurements, while actually providing a full dose of smoke when used as the companies knew they would be used.

Q: Are there internal documents by the defendants that discuss this?

A: Yes. For example, the alternatives in attempting to develop a cigarette that could be marketed as reducing risk are clearly articulated by S.J. Green in his minutes of a September 1968 British-American Tobacco Research Conference held at Hilton Head, South Carolina (U.S. Exhibit 85,044). In numbered paragraph 5 of Bates page 110075140 of that document, Green stated:

> Research staff should lay down guide lines against which alternative products can be chosen in everyday operations. Although there may, on occasions, be conflicts between saleability and minimal biological activity, two types of product should be clearly distinguished, viz:
>
> a) A Health-image (health reassurance) cigarette.
>
> b) A Health-oriented (minimal biological activity) cigarette,
>
> to be kept on the market for those consumers choosing it.

Q: What is the significance of this document?

A: It shows that BAT clearly understood the difference between the changes in cigarette design that would create a false reassurance and those that would actually reduce risk. BAT scientist S.J. Green distinguished between cigarettes that only "reassured" smokers as to the health benefit from those cigarettes that demonstrated an actual reduction in "biological activity."

Q: Is this document cited in Chapters 1 and 4 of Monograph 13?

A: Yes.

Q: What are some of the other important internal documents of defendants, considered

in the preparation of Monograph 13, that bear on the issue of the defendants' knowledge and use of cigarette design features that facilitate smoker compensation?

A: With respect to cigarette design, Colin Greig from BATCo wrote in U.S. Exhibit 20,230 – at Bates pages 100515901-5902 – that: "Given the design parameters of the cigarettes, it is possible to speculate that human compensation has, for a significant part of the smoking population, negated attempts to reduce tar deliveries." At Bates page 100515907, he then proposed the need for a "compensatable" cigarette that could be smoked more intensely to obtain more nicotine when needed. At Bates page 100515909, Greig specifically proposed a product that offered "'elasticity' of delivery" achieved through "non-obvious cigarette design features"; under the heading "Specific Proposal," he stated: "What would seem very much more sensible, is to produce a cigarette which can be smoked at a certain tar band, but which, in human hands, can exceed this tar banding."

Q: What is the title of this document?

A: It is an undated BATCo document with the heading "'Structured creativity group' Thoughts by C.C. Greig – R&D, Southampton Marketing scenario."

Q: What is the significance of Greig's statements in this document?

A: Clearly a tobacco industry goal was to develop products that could be promoted as delivering lower yields, but when smoked would deliver a full dose of nicotine in order to satisfy the smoker.

Q: Is this document cited in Chapter 4 of Monograph 13?

A: Yes; at page 70.

Q: What was the result of the defendants' use of cigarette design features that influenced smoker compensation?

A: As the tobacco companies used their understanding of compensatory smoking behavior and of the dilution of machine smoke measurements with filter ventilation to design cigarettes, the results were brands of cigarettes offered to the smoker as delivering less smoke while in actuality they delivered the same amount of smoke (and risk) as higher tar cigarettes.

Q: What are some of the other internal documents demonstrating that defendants knew their design changes were having this effect?

A: For example, Philip Morris's awareness of the deception of smokers by the use of machine measured tar values and use of the term "light" is demonstrated in U.S. Exhibit 20,348; an internal report dated September 17, 1975, from Barbro Goodman to Leo Meyer. Goodman reported on "smoker profile data" obtained using a device called a "Smoker Simulator." Goodman's study compared data of Marlboro 85 smokers to data for the same smokers when switched to the lower yield product Marlboro Lights.

Q: What is significant about this document to you?

A: I believe that there are three things that are powerfully significant in this document. The first is the conclusion. The conclusion of this document, at Bates page 2021544488, reads, "In effect, the Marlboro 85 smokers in this study did not achieve any reduction in smoke intake by smoking a cigarette (Marlboro Lights) normally considered lower in delivery. Conversely, the Marlboro Lights smokers did not increase their smoke intake when they changed to the regular delivery cigarette." That information very clearly demonstrates that, in contrast to what we

believed six years later when we wrote the 1981 Surgeon General's Report, smokers who smoked brands of cigarettes on the market in 1975 were not getting different yields when they smoked those products. We believed they were. That leads to the second significant point, which is that this is dated 1975, six years prior to the time the Surgeon General's Report reached its conclusion. And we did not have access to this information or comparable information.

The document is also significant because this study was done on a machine that mimicked actual smoking behaviors, that actually matched the behavior of the individual when the machine smoked the cigarette. In 1981, one of the recommendations that we made as a research recommendation was that this type of machine should be developed so that we could develop a better understanding of the relationship between delivery of tar and nicotine of these cigarettes when they were actually smoked. So, again, six years prior to the time we were reviewing that evidence for the Surgeon General, this information was available to Philip Morris.

Q: Did Barbro Goodman indicate in the "Conclusions" section whether the data that formed the conclusions in the study were consistent with other studies?

A: Yes; the "Conclusions" section starts on Bates page 2021544487 with the statement: "The smoker data collected in this study are in agreement with results found in other project studies."

Q: Is this document cited in the chapters of Monograph 13 that you coauthored?

A: Yes; at pages 6 and 71.

Q: Are there other Philip Morris documents by Barbro Goodman that bear on the defendants' knowledge of the effect of cigarette design features on smoker compensation?

A: Yes. Barbro Goodman also examined “the question of what might happen to deliveries to

the smoker when he partially covers the dilution holes" in an October 21, 1982 study (JE-40704). At Bates page 1003415280, the "Conclusions" section starts by stating: "The decrease in dilution from covering a portion of the perforated area can result in an increased delivery to the smoker of highly diluted cigarettes even though the puff parameters decrease."

Q: What does this statement demonstrate about Philip Morris's knowledge?

A: This demonstrates an understanding that ventilation holes placed in filter locations likely to be occluded by the smoker's lips or fingers would lead to an increase in the tar delivery of the cigarette when smoked by smokers.

Q: Do you cite this document in Chapter 4 of Monograph 13?

A: Yes; at pages 70-71.

Q: What are some of the other internal documents of the defendants in this case that inform your opinions on this issue?

A: Defendants' clear understanding that they were deceiving both the smokers and the public health authorities is further demonstrated by Brown & Williamson general counsel Ernest Pepples in an internal memorandum dated February 4, 1976, titled "Industry Response to Cigarette/Health Controversy" (U.S. Exhibit 20,292). In the fourth full paragraph of Bates page 170042568 of this memorandum, Pepples summarized a number of industry initiatives, and wrote:

> The new brands vying for a piece of the growing filter market made extraordinary claims. There was an urgent effort to highlight and differentiate one brand from the others already on the market. It was important to have the most filter traps.

Some claimed to possess the least tars. In most cases, however, the smoker of a filter cigarette was getting as much or more nicotine and tar as he would have gotten from a regular cigarette. He had abandoned the regular cigarette, however, on the ground of reduced risk to health.

Q: Is this document cited in Chapters 1 and 4 of Monograph 13?

A: Yes; on pages 6 and 69-70.

Q: How do the documents we have discussed above inform your opinions with regard to defendants' design and marketing of low tar cigarettes?

A: The documents identified and discussed above, and many other industry documents I have reviewed, (1) confirm the conclusions in Chapter 4 of Monograph 13; (2) establish that the cigarette companies were aware of nicotine compensation and design changes that could be employed to facilitate compensation by smokers; (3) show that the companies were aware that the FTC-machine measured yield was misleading to consumers in that it gave little to no measure of how much tar and nicotine, whether in absolute or relative terms, were ingested into the smoker's body; and (4) demonstrate that the tobacco companies recognized that the smoking public was being misled by the tar values and by the use of terms such as low tar and light.

Q: If known to you, what effect, if any, would this industry science have had on the recommendation of the 1981 Surgeon General's Report to switch to low tar and nicotine cigarettes if you couldn't quit?

A: As one of the scientific editors of the 1981 Surgeon General's Report, it is my expert opinion that, had the information available to the tobacco industry and described above been

available to the scientists preparing the 1981 Surgeon General's Report, that Report would not have drawn the erroneous conclusion that lower tar cigarettes produced lower risk or have made the recommendation that smokers who could not quit were "well advised to switch to cigarettes yielding less 'tar' and nicotine."

Q: Why is that?

A: It is particularly worth noting that much of the tobacco industry evidence cited above was generated in the early and mid 1970s, often five or more years before the 1981 Surgeon General's Report on the changing cigarette was released. In the preface of that Report (U.S. Exhibit 74,603), the Surgeon General stated:

> Overall, our judgment is unchanged from that of 1966 and 1979: smokers who are unwilling or as yet unable to quit are well advised to switch to cigarettes yielding less "tar" and nicotine, provided they do not increase their smoking or change their smoking in other ways.

The Surgeon General clearly expressed a concern about reduced yield smoking leading to compensatory increases in smoking behaviors; but, at that time, the public health community was not aware of the role of nicotine addiction in altering puffing behavior, the elasticity of delivery designed into cigarettes then on the market which facilitated compensation on the part of the smoker, or the observations made by the industry that showed compensation was essentially complete for some "light" cigarettes. Had we known that, the recommendation would not have been made.

Q: How would you describe your knowledge of the design features of lower yield

cigarettes around the time of the 1981 Report?

A: We had a partial understanding of the mechanism by which cigarettes were designed to produce lower yield. We had a very limited understanding of the interaction between the smoker's addiction and those design features.

Q: What was missing from your understanding of compensatory behavior at that time?

A: What was missing was the precision and specificity of the link between the addiction and the need to preserve nicotine use and how that link altered the pattern of smoking for the individual. We thought at this time that individuals smoked to get nicotine; so, in general, they would smoke enough cigarettes to get a certain dose of nicotine. We did not appreciate the fact that when individuals draw on a cigarette, they perceive nicotine at sensors in the back of their throat. And, therefore, if they're not getting enough nicotine within that single puff, they will draw faster, harder and take a bigger puff in order to increase the amount of nicotine delivered to their lungs. That type of compensation is one that is much more specific and is much more likely to lead to complete compensation in terms of delivery of nicotine and tar. It wasn't until we got a much better understanding of both addiction and how the mechanical and design changes of the cigarette impacted the delivery with the different patterns of puffing, that we began to understand these issues better.

Q: Do current public health recommendations include the recommendation that smokers switch cigarette brands based on FTC machine-measured tar and nicotine yields as a means of reducing future disease risks?

A: No. The current recommendation is to quit. Monograph 13 concluded that "Existing

disease risk data do not support making a recommendation that smokers switch cigarette brands. The recommendation that individuals who cannot stop smoking should switch to low yield cigarettes can cause harm if it misleads smokers to postpone serious efforts at cessation."

Q: Let's briefly return to Monograph 13, Chapter 4. You're a co-author. Did Chapter 4 include any new analyses?

A: Yes, it did.

Q: Please explain to the Court the new analyses that were done in Chapter 4.

A: There were four sets of new analyses. We reexamined the American Cancer Society CPS1 Study to look at the relationship of tar to disease risk. We examined the relationship in that study of people who switched brands of cigarettes that had very different nicotine yields as to what happened to their number of cigarettes smoked per day. We did an analysis of the California Tobacco Surveys to examine the relationship between number of cigarettes smoked per day and the nicotine yield of those cigarettes by the FTC method, and we did an analysis that looked at smoking behaviors in the United States, the risks that occur with those behaviors, and the expected lung cancer risk that would occur in the population. So those were four types of analyses that we did for that volume.

Q: How significant a role did these analyses play in the conclusions of Chapter 4?

A: They didn't play any significant role in the conclusions. They were simply demonstrations on various issues that we had raised as concerns or as issues in the volume.

Q: Have any other agencies, either in the United States or abroad, reached conclusions similar to those reached in Monograph 13?

A: Yes.

Q: Please review U.S. Exhibit 20,919. What is this exhibit?

A: It is a 2001 report by the Institute of Medicine entitled "Clearing the Smoke: Assessing the Science Base for Tobacco Harm Reduction."

Q: Did this report by the Institute of Medicine address the issue of whether low tar cigarettes provide a health benefit to smokers?

A: Yes.

Q: What did the Institute of Medicine report conclude on this issue?

A: "The public health impact of PREPs is unknown." PREPs are potential reduced-exposure products.

Q: Please explain the significance of the Institute of Medicine report relative to the conclusions of Monograph 13.

A: This report agreed with the conclusions of Monograph 13.

Q: Has the World Health Organization also reached similar conclusions?

A: Yes, it has.

Q: Have you had any involvement in the Scientific Advisory Committee on Tobacco of the World Health Organization?

A: Yes.

Q: Describe your involvement in the Scientific Advisory Committee on Tobacco for the World Health Organization.

A: I was asked to participate on the committee as an expert during the drafting of this

particular position, and I am now a member of that committee.

Q: When was that committee formed?

A: The committee was formed, I believe, in 1998 or 1999. The purpose of the committee was to provide advice to the World Health Organization on issues related to regulation of tobacco products.

Q: What involvement did that committee have with regard to light cigarette issues?

A: One of the principal issues that it considered was the appropriate regulation of light cigarettes.

Q: Did the committee ultimately issue a recommendation?

A: Yes, it did.

Q: Please look over U.S. Exhibit 86,658. Is that a copy of the recommendations issued by that committee?

A: Yes, it is.

Q: Did this report reiterate the five main conclusions of NCI Monograph 13?

A: Yes; those conclusions are reiterated on the second page of the Report, which bears Bates number TLT101 0693.

Q: Please tell the Court what the conclusions of the World Health Organization Scientific Advisory Committee were.

A: There were four conclusions. Number one, "tar and nicotine and CO numerical ratings based upon current ISO/FTC methods and presented on cigarette packages and in advertising as single numerical values are misleading and should not be displayed."

Number two, "[a]ll misleading health and exposure claims should be banned."

Number three, "[t]he ban should apply to packaging, brand names, advertising and other promotional activities."

Number four, "[b]anned terms should include light, ultra-light, mild and low tar, and may be extended to other misleading terms. The ban should include not only misleading terms and claims but also names, trademarks, imagery and other means to conveying the impression that the product provides a health benefit."

Q: Is this also a document that you reviewed and relied upon for your opinions in this case?

A: Yes, it is.

Q: Have you also previously given expert testimony about the Scientific Advisory Committee report in cases where one or more of the defendants in this case were parties?

A: Yes.

Q: Now, you've given your opinion that low tar cigarettes are no better for you, correct?

A: Yes.

Q: Is there really any harm for people to choose to smoke low tar cigarettes anyway?

A: Yes. There is profound harm. The vast majority of people who smoke are addicted. They're interested in quitting but are unable to do so. In that setting, we need to provide as much encouragement and support for cessation as possible. To provide smokers an alternative that says you don't have to quit, you can use this other type of cigarette, to intercept them on the way

to quitting smoking is a profound harm because they continue to smoke longer than they might have otherwise. Some of those people who switched might have quit, might have been successful in quitting, and when they did that, they would have in actuality reduced their disease risks. And those individuals have been profoundly harmed.

Q: To your knowledge, do all of the defendant cigarette companies continue to manufacture and market light and low tar cigarettes?

A: Yes, they do.

Q: I would like to switch topics now and ask you some questions about secondhand smoke, also referred to as environmental tobacco smoke ("ETS"). First off, what is environmental tobacco smoke?

A: Environmental tobacco smoke is the combination of tobacco smoke exhaled by smokers and the combustion products of tobacco released into the environment between puffs by the smoker or without being inhaled by the smoker. This smoke contains the same toxic and carcinogenic constituents found in the smoke inhaled by smokers, but the constituents are present in lower concentrations, because they are diluted by the volume of air into which they are released.

Q: What terms are commonly used to describe inhalation of ETS by nonsmokers?

A: The inhalation of secondhand smoke by nonsmokers is referred to as secondhand smoking, passive smoking, involuntary smoking or environmental tobacco smoke exposure.

Q: Does ETS pose a health risk to nonsmokers?

A: Yes. Tobacco smoke contains carcinogens, toxins, and irritants that cause disease risks

proportionate to the intensity and duration of exposure. Exposure of nonsmokers to tobacco smoke in the general environment – particularly enclosed indoors environments – results in irritation and annoyance, causes lung cancer and heart disease in adults, and causes asthma, bronchitis, pneumonia, and chronic ear problems in children.

Q: When was it first suspected that ETS might pose a health risk to nonsmokers?

A: Early efforts to restrict exposure of nonsmokers to ETS were driven by the irritation and annoyance created by exposure to cigarette smoke. However, by the 1970s it was increasingly clear that ETS exposure also placed vulnerable individuals at increased risk. Infants and children of smoking parents were shown to be at increased health risk of respiratory problems, and individuals with pre-existing heart and lung disease had functional impairment following exposure to ETS.

Q: Were these health risks of ETS exposure documented in any Reports of the Surgeon General during that time?

A: Yes; the Surgeon General's Reports in 1975 (U.S. Exhibit 34,340) and 1979 (U.S. Exhibit 64,071) examined the health effects of ETS exposure.

Q: How did the scientific understanding of the health effects of ETS exposure develop in the early 1980s?

A: In the early 1980s, a concern was raised that ETS exposure could also cause serious illness in otherwise healthy adults. In 1981, three independent epidemiological studies on lung cancer in nonsmoking wives who lived with smoking husbands were published. Two of the studies observed an increased risk for lung cancers and demonstrated an increased risk with

increased levels of smoking by the husband. However, a review of these three studies in the 1982 Surgeon General's Report (U.S. Exhibit 60,598) found that the evidence was not yet sufficiently compelling to conclude that a causal connection existed, but the Report did warn that involuntary smoking might indeed pose a carcinogenic risk to the nonsmoker and that individuals should avoid exposure to ambient tobacco smoke to the greatest extent possible.

Q: What did later studies find about the health risks posed to nonsmokers by ETS?

A: Any genuine controversy over whether ETS could cause disease in nonsmokers ended in 1986 with the publication of the Surgeon General's Report on the health consequences of involuntary smoking (U.S. Exhibit 63,709). This Report examined 13 epidemiological studies of ETS exposure and lung cancer and concluded that involuntary smoking was a cause of lung cancer.

Q: Did the 1986 Surgeon General's Report reach any specific conclusions about the health risks of ETS?

A: Yes. The 1986 Surgeon General's Report concluded that: (1) "Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers"; (2) The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures"; and (3) "The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke." These conclusions are stated on page 7 (Bates page VXA211 0690) of U.S. Exhibit 63,709.

Q: What was the result, if any, from the publication of this Report?

A: The Report contributed to a surge in efforts to protect nonsmokers from the health effects of ETS through legislation and policy changes. By far the most significant trend was toward the passage of local clean indoor air ordinances. By the fall of 1989, 397 ordinances limiting smoking in workplaces, restaurants, or other places had been enacted. These events are described in NCI Monograph 11 (U.S. Exhibit 78,717). With the growing recognition that tobacco smoke poses a significant health threat to individuals other than the smoker, Congress and federal health and regulatory agencies were increasingly asked to take action to protect the public. The General Services Administration issued new rules protecting workers and visitors in all federal buildings, and in 1988 Congress banned smoking on all domestic airline flights with a duration of 6 hours of less, resulting in virtually smoke-free air travel for all but a fraction of 1 percent of all flights within the United States. As the evidence linking ETS to adverse outcomes in nonsmokers increased, many businesses and employee groups began implementing smoking restrictions at their work sites.

Q: Are you aware of any significant later studies regarding the health risks of ETS exposure?

A: Yes. One of the more significant actions by a federal agency occurred in 1990, when the U.S. Environmental Protection Agency ("EPA") began a formal risk assessment to determine whether ETS met the Carcinogen Risk Assessment guidelines for classifying a compound as carcinogenic. By the time the EPA issued its report, Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders in January 1993, findings from 31 epidemiological

studies from 8 different countries comprised the available scientific evidence on ETS and lung cancer. Many of these studies showed a dose-response effect; that is, the greater level of exposure, the greater the lung cancer risk. The overwhelming weight of the evidence permitted the EPA to conclude that ETS belongs in the category of compounds classified as Group A (known human) carcinogens, a category reserved for only the most toxic of compounds regulated by the EPA, such as radon, asbestos, and benzene.

Q: Did the EPA report reach any other conclusions regarding the health effects of ETS exposure?

A: Yes. In addition to lung cancer, the EPA report examined the issue of ETS and respiratory diseases and disorders in children and concluded that ETS exposure was causally associated with (1) increased risk of lower respiratory tract infections such as bronchitis and pneumonia; (2) increased prevalence of middle-ear effusion; (3) a small but statistically significant reduction in lung function; and (4) decreased rate of lung growth. The report further estimated that ETS contributes between 150,000 and 300,000 lower respiratory infections annually among infants less than 18 months of age, resulting in between 7,000 and 15,000 hospitalizations annually. Of equal importance, the EPA estimates that ETS is causally related to additional episodes and increased severity of pre-existing asthma in children and that it exacerbates symptoms in approximately 20 percent of the estimated 2 to 5 million asthmatic children annually.

Q: The Court has heard testimony suggesting that the EPA used a 90% confidence interval for its estimate of the increased risk of lung cancer caused by passive exposure,

and that the use of such a confidence interval invalidated or otherwise minimized the significance of its conclusions. Do you agree with such testimony?

A: No.

Q: Please explain the basis of your opinion for the Court.

A: The EPA did not use a 90% confidence interval. They used a traditional 95% confidence interval, but they tested for that interval only in one direction. That is, rather than testing for both the possibility that exposure to ETS increased risk and the possibility that it decreased risk, the EPA only tested for the possibility that it increased the risk. It tested for that possibility using the traditional 5% chance or a P value of 0.05. It did not test for the possibility that ETS protected those exposed from developing lung cancer at the direction of the advisory panel which made that decision based on its prior decision that the evidence established that ETS was a carcinogen. What was being tested was whether the exposure was sufficient to increase lung cancer risk, not whether the agent it self, that is cigarette smoke, had the capacity to cause lung cancer with sufficient exposure. The statement that a 90% confidence interval was used comes from the observation that if you test for a 5% probability in one direction the boundary is the same as testing for a 10% probability in two directions.

Q: Are there any other studies regarding the health effects of ETS exposure that inform your opinion?

A: Yes. In September 1997, the California Environmental Protection Agency released a comprehensive review of the total range of health effects associated with ETS. The report concluded that: "The epidemiological data, from prospective and case-controlled studies

conducted in diverse populations, in males and females in western and eastern countries, are supportive of a causal association between ETS exposure from spousal smoking and coronary heart disease (CHD) mortality in nonsmokers." In addition, the Australian National Health and Medical Research Council also reviewed the evidence on the health effects of passive smoking in 1997 and concluded: "The scientific evidence shows that passive smoking causes lower respiratory illness in children and lung cancer in adults and contributes to the symptoms of asthma in children. Passive smoking may also cause coronary heart disease in adults." (U.S. Exhibit 22,092 – at 2065192431).

Q: Was the California EPA Report that you just mentioned reproduced in any of the NCI Monographs?

A: Yes; it was reproduced in NCI Monograph 10 (U.S. Exhibit 78,716).

Q: Do you have an opinion, to a reasonable degree of medical and scientific certainty, as to whether or not ETS causes disease?

A: Yes.

Q: What is that opinion?

A: Multiple reviews conducted by medical and governmental organizations over the 20 years leave no doubt that environmental tobacco causes disease in nonsmokers and is particularly dangerous for children. Regulation of smoking in indoor environments clearly stands on a strong foundation of scientific support.

Q: Do you believe there is consensus in the scientific community on the health risks of ETS?

A: Yes. The existence of a causal link between environmental tobacco smoke and lung cancer was demonstrated in the mid 1980's.

Q: Looking at cigarette smoking as a cause of disease as a whole, what effect, if any, has the defendants' conduct had on the number of smokers – historically and currently – in the U.S.?

A: The tobacco industry's response to the scientific evidence of smoking as a cause of lung cancer in the 1950s was creation of the Council for Tobacco Research which was intended to provide the appearance of scientific legitimacy to the tobacco industry's media campaign to confuse the public about the strength of the scientific evidence linking cigarette smoking and disease. At the same time, cigarette companies introduced and marketed filtered cigarettes and "low tar and nicotine" cigarettes as an effort to prevent smokers from quitting based on growing health concerns among smokers. One result of these two actions was that the decline in smoking following demonstration of smoking-related lung cancer risks reversed; and subsequently, per-capita consumption of cigarettes rose to new heights.

A second decline in per-capita consumption followed release of the first Surgeon General's Report in 1964 and the widespread publication of the information it contained. This information was once again disputed by the tobacco industry as they conducted an extensive public relations campaign to convince the public that there was still substantial scientific uncertainty as to whether cigarette smoking actually caused lung cancer. Once again the impact of this misinformation campaign by the tobacco industry was to reverse the decline in smoking that resulted from the publication of the Surgeon General's Report and its statement that the

scientific community was certain that cigarette smoking caused lung cancer in men.

On June 2, 1967, the Federal Communication Commission ruled that significant amounts of free time be made available for anti-smoking commercials to balance the cigarette advertisements on television and radio. As a result, during the period 1967-70 a large number of anti-smoking television spots were broadcast free by the major television networks. Effectiveness of this anti-tobacco advertising is supported by changes in US per-capita consumption and smoking cessation during the period of intense broadcast activity occurring between 1967-70. When cigarette advertising was banned from broadcast media after 1970, anti-smoking spots were also removed; and per-capita consumption increased and cessation rates declined. This evidence demonstrates the powerful effect of a substantial media-led effort to inform the public about the dangers of smoking and to encourage smoking cessation. Internal tobacco industry documents demonstrate industry awareness of the ability of this type of public health effort to alter smoking behavior and that the tobacco industry conducted extensive lobbying, public relations and media efforts to prevent the development of similar public health efforts nationally and at the state level (particularly in California and Massachusetts) or to hinder their effectiveness once they were implemented.

Concerns about exposure to environmental tobacco smoke and the social acceptability of smoking surfaced around 1970, and grew rapidly in the 1970's and early 1980's. These concerns were reinforced with the demonstration of a causal link between environmental tobacco smoke and lung cancer in the mid 1980's. These concerns among nonsmokers resulted in a wave of state and local ordinances restricting where smokers could smoke in public and at work. Internal

tobacco industry documents demonstrate that the tobacco industry studied the response of smokers when these ordinances were implemented and understood that smokers who worked in environments where smoking was restricted smoked fewer cigarettes per day and were more likely to be successful when they quit smoking (two changes in smoking behavior that will clearly reduce the risk of smoking related disease). In response, the tobacco industry lobbied aggressively against these ordinances wherever proposed, conducted media and public relations campaigns to dispute the scientific evidence, set up front groups to oppose these ordinances at the local level, funded research to confuse the science on this issue and threatened financial retribution for companies who voluntarily implemented these regulations. One result of these actions is a substantial delay in the implementation of protections for nonsmokers and a corresponding higher cigarette consumption and lower rate of successful cessation among smokers.

Based on my training, experience and review of changes in cigarette smoking based on the actions of the tobacco companies, it is clear that the misconduct of the tobacco industry in denying the disease risks caused by smoking, conspiring not to do biologic testing or to compete by developing safer cigarettes, blocking appropriate public health efforts to reduce tobacco related disease, and deceptively marketing filtered and "lower yield" cigarettes as safer cigarettes has led to substantially higher rates of cigarette consumption than would have occurred absent these actions. This excess consumption has resulted in substantial excess rates of disease, a result which would not have occurred absent these actions by the tobacco industry.

Q: I want to conclude by talking about the subject of reducing initiation and increasing

cessation rates. What are some of the important scientific publications that provide guidance on reducing smoking initiation and increasing smoking cessation?

A: There is a substantial body of scientific evidence defining what can be done to assist smokers who want to quit and to prevent children from starting to smoke. This evidence is summarized in the various Reports of the Surgeon General, Public Health Service and Tobacco Control Monographs.

The current scientific understanding of how to best assist smokers in their cessation attempts presented in the Public Health Service guidelines establishes that there is no single approach that works best for all smokers, and therefore an effective approach to providing cessation assistance must include a menu of different approaches offered through different channels in a continuously available mode over a prolonged time period. Nicotine replacement therapy, telephone counseling, physician advice and clinic based cessation programs are all proven methods of increasing cessation success. Smokers need to be able to select from a menu of these services and have those services readily available at the time that they are motivated to quit. In addition, those cessation approaches that are most effective (i.e. a 6-8 week cessation clinic approach) are also the most time intensive; and only a small percentage of smokers will access them. As a result, an effective program must include both programs with a range of intensities and a promotional campaign to make smokers aware of the program and to stimulate interest in making a cessation attempt.

Q: You mentioned that the 2000 Surgeon General's Report on "Reducing Tobacco Use." In your opinion, does the Report represent the scientific consensus on the subject of

cessation?

A: Yes.

Q: We also discussed your participation in NCI Monograph 12 (U.S. Ex. 78,718), titled Population-Based Smoking Cessation, published in 2000. What were your specific contributions to Monograph 12?

A: I was the senior scientific editor, authored the introduction, and served as a co-author on four additional chapters.

Q: Please describe for the Court what the focus of Monograph 12 was.

A: As I wrote in the introduction to the Monograph, "This volume examines cessation at the population level. By population level, we mean that all segments of society form the denominator for evaluation of the effectiveness of tobacco control interventions. Therefore, this volume relies heavily on representative surveys of smoking behaviors in state and national populations. By doing so, it defines measures of cessation that can be used to assess the effects of tobacco control programs or public policy changes on smoking behavior. It then uses those measures to identify who is quitting, who is being successful, who is being exposed to various tobacco control interventions, and which tobacco control interventions are proving effective." We also looked at the fact that both physician advice and pharmacological treatment have been established in controlled clinical trials to have a substantive effect on long-term smoking cessation, and in Monograph 12 we therefore addressed the evidence for an effect at the population level.

Q: What conclusions did the review in Monograph 12 reach?

A: We concluded, among other things, that data on cessation rates over time suggest that

public health efforts to change smoking behavior can have an effect above and beyond the effect of information on risk alone. The temporal association of change in cessation rates with tobacco control events strongly suggests that deliberate programmatic efforts *can* alter smoking behavior at the population level and provides one cornerstone of the foundation for current comprehensive tobacco control campaigns.

Q: Did you reach conclusions that were specific to population-based cessation programs, as distinct from the broader issue of tobacco control?

A: Yes, as set out in the overview to the Monograph, "examination of population-based data on gum and patch use suggest that they are a part of a large number of cessation attempts and are likely to make a substantive difference in the success rate of those attempts. However, the rates of success in the California population are well below those demonstrated in clinical trials, which suggests that there is substantial potential to increase both utilization of nicotine replacement products and the impact of these products on the success rate of smokers trying to quit.

The gap between the effect achieved in clinical trials and the population data defines the potential that can be achieved if these modalities are delivered in a more comprehensive and organized manner and integrated with the other available cessation resources. If physician advice achieves the effectiveness demonstrated in clinical trials, it could result in as many as 750,000 additional quits among the 35 million smokers who visit their physicians each year. If the success rate of pharmacological interventions matched that in the clinical trials, as many as 500,000 additional quits each year could be achieved, and an even greater number could be expected if larger numbers of smokers who are trying to quit could be persuaded to use pharmacological

methods."

Q: Did the Monograph look at the best way to deliver cessation therapy "in a more comprehensive and organized manner"?

A: Yes, it did. We concluded that "[t]he answer to improving the effectiveness of these interventions may not lie in providing additional resources into the health care system to change physician behavior or additional promotional activity for pharmaceutical assistance with cessation. The answer may be to try to supplement these interventions by linking them with other components of comprehensive tobacco control interventions to improve their effectiveness. For example, linking physician advice with telephone hotline counseling, providing information on how to effectively utilize over-the-counter medications at community cessation events, and encouraging healthcare systems to view cessation as a population-based intervention delivered across all interactions with the system rather than as a process initiated exclusively by physicians.

If other components of a comprehensive tobacco control program can be linked to physician advice and pharmacological assistance, it may be possible to provide the enhanced level of support and follow-up that characterized the delivery of these interventions in the clinical trial setting as these interventions are delivered to large segments of the population. When this was done within a large HMO setting (Curry *et al.*, 1998), and when the barriers to accessing these modalities were reduced by lowering or eliminating the cost to smokers, cessation results were consistent with those achieved in clinical trials. This experience suggests that the limited population effects of physician advice and pharmacological assistance represent limitations in the integration of the support provided to smokers who are trying to quit rather than absolute

limitations of these approaches when they are utilized in the general population. The frequency with which physician advice is provided to smokers as well as the frequency with which smokers are using pharmacological assistance are both increasing, and these increases should be supported and encouraged. To obtain the maximal benefit from these effective interventions, we need to integrate them into health care delivery systems, link them to community cessation resources, and create an environment that encourages their access. Once these steps have been taken, dramatic improvements in population-based rates of cessation are possible (Curry *et al.*, 1998). Moreover, it is reasonable to expect that the experience could be replicated in other settings."

We also addressed the importance of media for effective cessation programs, concluding, "Evidence reviewed and presented in this volume supports the effectiveness of tobacco control programs that are media led and media intensive."

Q: Did you look at the effects of broader tobacco control messages on the success of a cessation program?

A: We did, specifically concluding that "individual components of a comprehensive tobacco control program may affect the process of cessation at different stages. For example, mass-media campaigns may get smokers to think about the need to quit, physician advice may trigger a cessation attempt, and working in a smoke-free environment may facilitate cessation once a cessation attempt is made. An additional advantage of the formulation is that it facilitates identification of potential synergistic interactions among different program components."

More specifically, we concluded that "A variety of environmental and interventional

influences have substantial impacts on successful cessation." We further determined that evidence "supports an effect of changes in cost and environmental restrictions on smoking in the workplace on long-term success. Nicotine replacement therapy is shown to be associated with improved cessation success at the population level, confirming its demonstrated effect in clinical trials. Telephone counseling and clinic-based cessation efforts have been established as effective interventions for those who receive them, but there is little evidence that they are reaching a sufficient proportion of the smoking population to affect cessation at the population level."

Q: Do you agree with the conclusions in Monograph 12?

A: Yes.

Q: In your opinion, would the impact of cigarette smoking on the health of the American public be reduced if a comprehensive, national cessation effort were funded and undertaken containing the programmatic elements identified in Monograph 12?

A: Yes.

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