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UNITED STATES DISTRICT COURT
IN AND FOR THE MIDDLE DISTRICT OF FLORIDA
CIVIL DIVISION, CASE NO. 96-1706-CIV-T-23B

GENEVA JENKINS, as Personal Representative of the Estate of ALBERT JENKINS, deceased,

Plaintiff,

vs.

PHILIP MORRIS INCORPORATED, a foreign corporation; LORILLARD TOBACCO COMPANY, a foreign corporation; and R.J. REYNOLDS TOBACCO CO., a foreign corporation,

Defendants.

PLAINTIFF'S PARTIAL EXPERT WITNESS DISCLOSURE

COMES NOW, Plaintiff, Geneva Jenkins, as Personal Representative of the Estate of Albert Jenkins, deceased, hereby submits her partial expert witness disclosure.

CERTIFICATE OF SERVICE

I HEREBY CERTIFY that true and correct copies of the foregoing have been provided by regular U.S. Mail to all persons listed on the attached service list this 11 day of September, 1997.

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PLAINTIFF JENKIN'S PARTIAL EXPERT WITNESS DISCLOSURE

A. I. ALLAN FEINGOLD, M.D.

I. Allan Feingold
South Miami Hospital
Division of Pulmonary Medicine
6200 S.W. 73rd Avenue
Third Floor
So. Miami, FL 33143

Dr. Feingold is expected to testify that cigarettes caused Albert Jenkin's chronic obstructive pulmonary disease (COPD) and lung cancer and associated symptoms and death. He is further expected to testify that Mr. Jenkins was addicted to the nicotine contained in cigarettes. He will further opine as to the health of Mr. Jenkins as identified in the medical records.

Dr. Feingold's reports (to be provided)

Affidavit of Dr. Feingold

CV

Testimony

B. ALLAN GOLDMAN, M.D.

Allan Goldman, M.D.
USF Medical Center
College of Medicine
Department of Internal Medicine
12901 Bruce B. Downs Boulevard
Suite 4127
Tampa, FL 33612

Dr. Goldman is expected to testify that cigarettes caused Albert Jenkin's chronic obstructive pulmonary disease (COPD) and lung cancer and associated symptoms and death. He is further expected to testify that Mr. Jenkins was addicted to the nicotine contained in cigarettes. He will further opine as to the health of Mr. Jenkins as identified in the medical records.

Dr. Goldman's reports (to be provided)

CV

Testimony

C. HERMAN BAER, M.D.

Herman Baer, M.D.
USF Medical Center
Health Center
1600 S.W. Archer Road
Ground Floor Room
Gainesville, FL 32610

Dr. Baer is expected to testify that cigarettes caused Albert Jenkin's chronic obstructive pulmonary disease (COPD) and lung cancer and associated symptoms and death. He is further expected to testify that Mr. Jenkins was addicted to the nicotine contained in cigarettes. He will further opine as to the health of Mr. Jenkins as identified in the medical records.

Dr. Baer's report (to be provided)

CV

Testimony

D. TREATING PHYSICIANS

Please see attachment for further information.

Affidavit of Allan Feingold M.D.

Deponent **Allan Feingold, M.D., F.R.C.P.(C)** being sworn in Dade County, Florida, deposes and says:

1 Identification

1.1 He is a physician licensed to practice medicine in the state of Florida, being duly board certified in internal medicine and pulmonary medicine.

1.2 Dr. Feingold received a B.A. degree from McGill University and an M.D. degree from McMaster University. He is currently Chief of Pulmonary Medicine at South Miami Hospital, 6200 SW 73 St., Miami, Florida. Dr. Allan Feingold trained in Internal Medicine and Pulmonary Medicine at the Royal Victoria Hospital, the Montreal Chest Hospital Center and other teaching hospitals of McGill University. Dr. Feingold has written numerous medical computer programs, including artificial intelligence diagnostic programs and statistical apportionment programs. He has written and lectured extensively on asbestos medicine and tobacco issues.

1.3 He has studied the medical literature on smoking, smoking cessation, diseases related to smoking, addiction and habituation, and also the history of medical knowledge related to smoking and smoking related diseases. Affiant's study and research has been ongoing since at least the middle 1970's.

1.4 Affiant has examined, interviewed, and treated smokers, and ex-smokers continually throughout his training and practice. Such examination and treatment has included treatment for various cigarette-related disease including lung cancer; chronic obstructive lung disease, cancer of various other organs including the bladder, kidney, larynx, esophagus, and other organs, plus treatment for addiction to nicotine.

1.5 In addition such examination and treatment has included medical and social histories on smoking, attitudes toward smoking, smoking cessation, addiction to nicotine, relapse to smoking after cessation, patient education and knowledge, and other similar issues.

1.6 Dr. Feingold has studied and considered the following sources of information and knowledge, among others, in preparation for this testimony:

- Reports of the U.S. Surgeon General from 1964 until 1994
- Testimony before Congress on False and Misleading Advertising and on the Cigarette Labeling Acts, for the years 1957, 1965, 1969, 1984 and others.
- Thousands of medical articles, textbooks, journals, and similar materials on the diseases caused by cigarettes and their mechanisms.

- Internal documents of the cigarette industry of a public health nature, revealed through publication in medical journals and through public disclosures in press and litigation.

- Testimony of cigarette industry officials taken in connection with public appearances, litigation, and congressional testimony.

- Public advertisements and advocacy statements authored by the cigarette industry and appearing in newspapers and magazines

- Original data from epidemiological studies

1.7 Dr. Feingold's opinions were accepted by the Court as expert testimony in *Carter v. Brown and Williamson Tobacco Company* (4th Judicial Circuit FL 1996)

2 Health effects of cigarette smoking

2.1 Cigarette smoking is "*the major* preventable cause of death and disability" in the developed world.[1] According to the U.S. Surgeon General[2], smoking is directly responsible for one out of every six deaths in the United States. In 1990, approximately 450,000 deaths were attributable to cigarette smoking, and the total is now higher.

2.2 In smokers, death rates for serious diseases of the heart, lung, and other organs exceed those of nonsmokers by factors of two to 30.[3] The impact of smoking on lung and other cancers, and on heart disease mortality, shows that tobacco-induced deaths make up *at least 35%* of all fatal conditions in males ages 35 to 69.[4]

2.3 Medical literature has shown that the death rate for cigarette smokers is almost twice that of nonsmokers. National Cancer Institute showed that *between 40% and 50% of regular cigarette smokers will eventually be killed* by their habit.

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[1]American Thoracic Society Cigarette Smoking and Health, OFFICIAL ATS STATEMENT ADOPTED BY THE ATS BOARD OF DIRECTORS, November 1984.

[2]U.S. Department of Health and Human Services, Public Health Service, U.S. *Report of the Surgeon General, The Health Consequences of Smoking*, 1982.

[3]Doll R, Peto R. *Mortality in relation to smoking: 20 years' observations on male British doctors* 1976 BRIT MED J 1525,1535 T. XII (1976)

[4]Peto R, Lopez AD, Boreham J, Thun M, Heath C, *Mortality from tobacco in developed countries: indirect estimation from national vital statistics*, LANCET 1992: 339:1268-78.

2.4 The following diseases are caused wholly or in large measure by cigarette smoking:

Cancer of lung
Cancer of esophagus
Cancer of other respiratory sites
Chronic bronchitis and emphysema
Pulmonary heart disease
Ischemic heart disease
Aortic aneurysm
Cerebral thrombosis [stroke]
Arteriosclerosis
Pneumonia
Peptic Ulcer
Hernia
Cancers of the bladder and kidney.

2.5 Lung cancer, has become the number one cancer death for both men and women. The Surgeon General estimated, "Eighty-five percent of all lung cancers are directly attributable to cigarette smoking."[5] Summarizing significant epidemiological studies on lung cancer risk to smokers, The Surgeon General reported that the relative risk for smokers can be as high as 23. For heavy smokers, the risk can be over 40 times the nonsmokers' risk.

2.6 Lung cancer is an especially dangerous risk of smoking, because it is generally incurable. Advances in survival rates for lung cancer have not generally been seen. Depending upon cell type, the number of lung cancer patients who survive for five years after diagnosis ranges from almost zero to 15 out of 100, with an average of 5 or 5%. Of special concern, lung cancer often metastasizes or spreads to other, remote organs, before diagnosis. These organs include the brain, bone, liver, pancreas, and other organs. Metastatic cancer is universally fatal, and the patient's demise is often excruciatingly painful and debilitating.

2.7 Cardiovascular diseases are leading killers in the United States. According to the Surgeon General 30% of all coronary heart disease deaths in the United States are attributable to cigarette smoking habit. Cigarette smoking is also significantly related to other types of cardiovascular disease, including peripheral vascular disease, aneurysm and stroke.

2.8 Oral cancer among heavy smokers is increased about 13 times, or 1300

[5]U.S. Department of Health and Human Services, Public Health Service. *Report of the Surgeon General, The Health Consequences of Smoking* (1982).

percent.[6] Esophageal cancer is increased 1000 percent among smokers. Tobacco smoking is responsible for about 50% of bladder cancers in males living in Western countries.[7] Laryngeal cancer is a well known result of heavy cigarette smoking. Between 80 and 90% of all laryngeal cancers are associated with cigarette smoking.[8]

2.9 Chronic obstructive pulmonary disease (COPD) collectively refers to chronic bronchitis and emphysema. Chronic bronchitis results in dyspnea, chronic cough and sputum production because of bronchospasm and excessive production of bronchial mucus. Emphysema produces very similar symptoms and results in the destruction of the architecture of the lung. The risk of COPD in smokers is thirty times the risk in nonsmokers. In addition, chronic bronchitis and emphysema are major sources of impairment among the smoking population. Thirty-five thousand Americans are totally disabled each year from COPD, the greatest frequency of activity limitation from any disease category.

2.10 Smoking causes a tremendous and unfortunate increase in risk for diseases of various types. These increases can be summarized in percent as follows:

Lung cancer	2240%
Esophagus cancer	1000%
Other Resp. Cancer	2700%
Chronic Bronchitis, Emphysema	2900%
Ischemic Heart Disease	163%
Aortic Aneurysm	900%
Stroke	152%
All deaths	186%

[6]Doll R, Peto R, *Mortality in relation to smoking: 20 years observations on male British doctors.* BRIT MED J 1976;2:1525-1536.

[7]Vineis P, Simonato L, *Proportion of lung and bladder cancers in males resulting from occupation: a systematic approach* [see comments]. ARCH ENVIRON HEALTH 1991 JAN-FEB;46(1):6-15.

[8]Fielding JE, *Smoking: Health Effects & Control,* NEW ENGL J MED 1985;313:8, 491-498.

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3 Latency and carcinogenesis

3.1 Lung cancer has an extended "latency" period following first exposure to the carcinogens in cigarette smoke. It is typical to see 20 to 40 years' elapse before lung cancer manifests itself clinically. During the latency period, unknown to the patient, microscopic cellular changes are occurring in response to the carcinogen. These changes are set in motion by the first exposure, and consist of cellular mutations and DNA damage. This irreversible chromosome damage silently perpetuates and accumulates over time until some unfortunate threshold is reached where a malignant transformation or transformations occur. Malignant cells surviving this transformation will in some number go on to replicate as cancer, and many years later will manifest as clinical lung cancer.

3.2 Similar latency periods are displayed in cigarette-induced cancers of the larynx, stomach, esophagus, aerodigestive tract, bladder, and some other organs.

3.3 Chronic obstructive pulmonary disease also demonstrates an extended latency period during which the disease progresses silently and often without symptoms. After a period of many years since first exposure, the disease manifests with structural and functional changes.

3.4 It is understood that many cancers are caused by somatic mutations induced by carcinogens.[9] Recently developed techniques, including DNA polymerase chain reactions,[10] have permitted closer examination of these environmentally induced genetic mutations. Cigarette smoke has been proven to cause certain mutations in both oncogenes (including k-ras) and tumor suppressor genes

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[9] Anderson M, You M, Belinsky S, Hegil M, Maronpot B, Wiseman R, Reynold, *Genetic alterations in lung tumors (meeting abstract).* PROC ANNU MEET AM ASSOC CANCER RES 1992;33:A601-2 (1992)

[10] Rodenhuis S, Slebos RJ, Boot AJ, Evers SG, Mooi WJ, Wagenaar SS, van B, *Incidence and possible clinical significance of K-ras oncogene activation in adenocarcinoma of the human lung..* CANCER RES 1988 OCT 15;48(20):5738-41 (1988)

(including p53) thought to be responsible for certain cancers.[11] Slebos (1992)[12] explained:

> The three ras genes code for proteins with a putative role in cellular signal transduction. They belong to a larger family of small guanosine-triphosphate (GTP)-binding proteins. The ras proteins acquire transforming activity when amino acids are substituted at one of a few specific sites, as a result of a point mutation in the gene...**K-ras mutations are very rare among nonsmokers, and it is reasonable to assume that carcinogens in tobacco smoke directly cause the mutation.**

3.5 Westra (1993)[13] tested for overexpression of the p53 tumor suppressor gene in a series of adenocarcinomas. Fifty-six percent of current smokers with adenocarcinoma had p53 overexpression, whereas overexpression was found in *zero* percent of nonsmokers with the same tumor. From this the authors concluded:

> These findings indicate that p53 protein is frequently overexpressed in primary lung adenocarcinomas. **Furthermore, the association of tobacco smoking with this overexpression suggests that the p53 gene is a target of specific mutagens in tobacco smoke.**

3.6 Overexpression of p53 was also found by Kondo (1992)[14] in 44% of smoking lung cancer patients. The authors concluded, "The p53 mutations showed a significant association with a history of smoking ... We suggest that the p53 mutations may be associated with smoking-induced lung carcinogenesis."

3.7 Another paper by Westra (1993)[15] found mutations in codon 12 of the k-ras

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[11].Reynolds SH, Hunnicutt CK, Beattie T, Pero R, Anderson MW, *Ras oncogenes in human lung tumors associated with exposure to cigarette smoke (meeting abstract).* PROC ANNU MEET AM ASSOC CANCER RES 1988;29:A543 (1988)

[12].Slebos RJ, Rodenhuis S, *The ras gene family in human non-small-cell lung cancer..* MONOGR NATL CANCER INST 1992(13):23-9 (1992)

[13].Westra WH, Offerhaus GJ, Goodman SN, Slebos RJ, Polak M, Baas IO, Rode, *Overexpression of the p53 tumor suppressor gene product in primary lung adenocarcinomas is associated with cigarette smoking..* AM J SURG PATHOL 1993 MAR;17(3):213-20 (1993)

[14].Kondo K, Umemoto A, Akimoto S, Uyama T, Hayashi K, Ohnishi Y, Monden Y, *Mutations in the P53 tumour suppressor gene in primary lung cancer in Japan..* BIOCHEM BIOPHYS RES COMMUN 1992 MAR 31;183(3):1139-46 (1992)

[15].Westra WH, Slebos RJ, Offerhaus GJ, Goodman SN, Evers SG, Kensler TW,, *K-ras oncogene activation in lung adenocarcinomas from former smokers. Evidence that K-ras mutations are an early and irreversible event in the development of adenocarcinoma of the lung.* CANCER 1993 JUL 15;72(2):432-8 (1993)

oncogene prevalent in current and ex-smokers with adenocarcinoma, and uncommon in never smoking adenocarcinoma patients. The mutation was a guanine-to-thymine transversion, "the specific type of mutation induced by benzo(a)pyrene, one of the chemical carcinogens found in tobacco smoke." The authors concluded that their findings "support previous findings that suggest that codon 12 of the K-ras oncogene may be a specific target of the mutagenic activity of tobacco smoke." Furthermore, because the frequency of the k-ras mutation was "independent of the duration of abstinence from smoking" the authors suggested that "DNA alterations at this site can occur *early and irreversibly* during the development of adenocarcinomas of the lung." This and other work is significant because it establishes a plausible molecular basis of cigarette-induced pathogenesis. Furthermore, such work explains why cancer risks from cigarettes remain substantial after cessation of smoking: early smoking causes irreversible damage to lung DNA, which mutations would be expected to persist through inheritance during cell divisions.

4 Carcinogens

4.1 Exempt from most Federal safety and health laws, tobacco products have never labeled their ingredients, which include many potentially harmful compounds.

4.2 *Nicotiana tabacum* is the cultivated species of the genus *Nicotiana*, named for Jean Nicot, French Ambassador to the Netherlands, who championed tobacco smoking in 1560.

4.3 The tobacco leaf contains an extremely complex mixture of compounds, including starches, proteins, sugars, alkaloids, hydrocarbons, phenols, fatty acids, sterols, and inorganic minerals. When subject to burning temperatures of 830 to 890 degrees centigrade, these compounds undergo extensive pyrolytic reactions, making the cigarette a tiny "chemical factory" that generates thousands of different compounds. Cigarette smoke is a concentrated aerosol consisting of an heterogenous mixture of gases, uncondensed vapors, and particulates, many in liquid phase and smaller than .5 microns. By the late 1950's many of the compounds in tobacco smoke had been identified; many were known carcinogens, identified earlier by Hartwell in 1951 and others.

4.4 Writing in 1964, the Surgeon General stated[16]:

A compilation ... prepared by J.L. Hartwell of the National Cancer Institute lists 2109 compounds

[16] U.S. Department of Health, Education, and Welfare, Public Health Service. *Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service.* P.H.S. Publication 1103 (1964)

of which 481 were reported to cause malignant tumors in animals. **All but one of the polycyclic hydrocarbons listed ... as having been identified in tobacco smoke have already been documented in the Hartwell report and can be assigned a rating as very potent,... potent,... moderately carcinogenic ..., or weakly carcinogenic.**

4.5 By the early 1960's, it was accepted by the Surgeon General and others that tobacco smoke contained at least seven, and probably many more, known carcinogens, of which benzo(a)pyrene, a polycyclic hydrocarbon, was "the most potent" and was present in the highest quantity in tobacco smoke. In addition, as remarked by the Surgeon General in 1964, other polycyclic hydrocarbons isolated from tobacco smoke were "not yet adequately tested for carcinogenicity." It is now well documented, by the Surgeon General in 1989[17] and by others, that at least 43 compounds in cigarette smoke are known carcinogens. These include compounds in these major groups:

- *Polyaromatic hydrocarbons* -- 11 various compounds with known animal carcinogenicity, including benzo(a)pyrene, a"probable" human carcinogen.
- *Aza-arenes* -- four known animal carcinogens
- *N-Nitrosamines* -- nine known animal carcinogens
- *Aromatic amines* -- three known carcinogens, including 2-Naphthylamine and 4-Aminobiphenyl, both known human carcinogens.
- *Aldehydes* -- three known carcinogens including formaldehyde, a suspected human carcinogen.
- *Miscellaneous organic compounds* -- six carcinogens, including benzene and vinyl chloride, both known human carcinogens.
- *Inorganic compounds* -- seven carcinogens, including arsenic, chromium, and polonium-210, all known or suspected human carcinogens.

4.6 Other compounds, such as HCN, zinc, cadmium, carbon monoxide, and others are not known to be carcinogenic but may be associated with other pathology, including acute toxicity, psychoactivity and addiction liability, cardiovascular disease, and ciliastasis. Commenting on the research to date, the Surgeon General in 1989 concluded:

[T]he estimated number of compounds in tobacco smoke exceeds 4,000, including some that are pharmacologically active, toxic, mutagenic, or carcinogenic. The diverse biological

[17] U.S. Department of Health and Human Services. *Reducing the Health Consequences of Smoking: 25 Years of Progress. A Report of the Surgeon General.* U.S. Department of Health and Human Services. Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. DHHS Publication No. (CDC) 89-8411 (1989).

effects of tobacco smoke constituents provide a framework for understanding the multiple adverse consequences of smoking.

4.7 When burned, nicotine yields certain carcinogenic tobacco specific nitrosamines (TSNA's) including compounds named NNN and NNK, which are delivered to lung and airway tissue in smoke and are absorbed and circulated in the bloodstream, where they cause cancer in other organs.

4.8 The medical literature fails to contain evidence of serious technological attempts by any U.S. manufacturer to reduce or eliminate TSNA's in the smoke from cigarettes. Nor have TSNA levels dropped significantly since the 1960's. Excessive levels of TSNA's, and of their nicotine precursors, constitutes a defect in those cigarettes.

5 Cessation

5.1 A cigarette user who stops consuming cigarettes will experience some reduction in hazard from lung cancer and other diseases. However, after many years of smoking, irreversible changes occur in lung and other tissue which can progress to cancer even without further smoke exposure. Ex-smokers are therefore at much greater risk of cancer than neversmokers, a finding confirmed by epidemiology.

6 Public awareness of health risks

6.1 In counseling my patients on cigarette smoking, I am struck by their lack of appreciation for the true risks of this deadly habit. Limited cautionary labeling has been in effect since 1966, but it has been singularly inadequate. Studies of public opinion, plus marketing studies involving the cautionary labels, show that the labels are inadequate to convey the real risks of smoking.

6.2 It is not generally appreciated by the public any of the following:

6.2.1 that the risks of smoking are as serious as they are, amounting to a risk of premature death of almost 50%. Nothing else commonly done in life carries such excess risk, and the public is unaware of the magnitude of the risk.

6.2.2 that the risks of serious diseases are as large as they are, for instance, few patients realize that the risk of lung cancer is over two thousand percent increased, or that the risk of stroke is elevated over 150 percent.

6.2.3 that in addition to lung cancer, there is a terrible risk of cancers of the

kidney, bladder, throat, esophagus, and other organs. Patients are almost universally unaware of these risks, as they are generally ignorant of the extent of risk to heart attack and cardiovascular disease.

6.2.4 that smoking in addition carries with it a substantial risk of addiction and habituation, so that it is very difficult to break the habit once it has begun

6.2.5 that smoking filter-tip or "low tar" cigarettes may not in fact make much of a difference to their serious life-threatening risks, especially if they are not aware of the phenomenon of "compensatory smoking", which means increasing the amount of smoke inhaled to compensate for the low tar and low nicotine smoke.

6.2.6 that breaking the habit does not return their risks of disease to what they were before they smoked, but that these risks stay elevated throughout their life.

6.3 My counseling and contact with patients convinces me that patients in 1995 do not know the real risks of smoking, and moreover that they did not know the risks before either. When many of these patients with serious diseases today began to smoke in the 1940's thru 1960's, they did not, according to my conversations with them and my knowledge of what information was available, understand the risks.

7 Medical knowledge of health risks

7.1 In reviewing the medical literature, important medical articles were published in the 1920's through 1940's linking cigarette smoking to lung cancer. In particular, Dr. Alton Ochsner, founder of the Ochsner Clinic in New Orleans, and Dr. Michael DeBakey, one of America's preeminent thoracic surgeons published an article in 1941[18]. These doctors correctly observed that the increase in smoking after World War I caused the increase in lung cancer in the 1930's, after a latency period of about 20 years. The authors wrote:

> It is our definite conviction that the increase in the incidence of pulmonary carcinoma is due largely to the increase in smoking, particularly cigaret smoking, which is universally associated with inhalation. [T]here is an obvious parallelism between the increased production of tobacco and carcinoma of the lung.

7.2 The paper from Drs. Ochsner and DeBakey was published in an important medical journal, and was especially notable for having surveyed the world literature up to that time. The conclusions reached, implicating cigarette

[18] Ochsner A, DeBakey M, *Carcinoma of the lung.* ARCH SURG 42:209-258 (1941)

smoking with lung cancer, was extremely important because lung cancer was such a serious and largely incurable disease.

7.3 It is my opinion that any commercial enterprise manufacturing cigarettes would and should have been aware of this very significant medical article, and that it would have been a lack of reasonable care to have ignored it or failed to acquire it.

7.4 In 1950 Dr. Ernest Wynder and Dr. Evarts Graham published[19] a very significant paper in the Journal of the American Medical Association. This study found that smoking was strongly associated with lung cancer, which was extremely rare in nonsmokers. This paper proved conclusively that smoking caused lung cancer. Publication of this work in a prestigious journal carried special impact because one author Dr. Graham was one of the most famous pulmonary surgeons of the time, who had earlier done the first successful surgical lung removal.

7.5 The British Medical Journal realized the significance of Wynder and Graham's work in a 1950 editorial and stated:[20]

> "[M]ost researchers agree ... that the increase [in lung cancer] is likely to be real .... What environmental changes have taken place which can have accounted for the increase? The use of tobacco is one obvious explanation. [Wynder and Graham's] results are striking. Of 605 male patients with ... carcinoma only 1.3% were non-smokers [and] 51.2% had smoked the equivalent of more than 20 cigarettes a day. [I]n contrast...14.6% of [controls were] nonsmokers and 19.1% ... smoked more than 20 a day....[Interview bias] would not appear to have biased the results.
>
> These reports provide an important advance in knowledge. If they are confirmed it will still remain to trace the carcinogenic agent and **to apply the lesson.** Meanwhile strong support has been given to the existing financial reason for not smoking." [1477]

Confirmation would come quickly, but the tobacco industry would refuse, even to today, to "apply the lesson."

7.6 At almost the same time, Sir Richard Doll, the personal physician to the Royal Court, and Bradford Hill, perhaps England's most celebrated physician-statistician, published a large scale survey[21] of 709 lung cancer patients. Only two nonsmokers were found, leading the investigators to conclude that "smoking is a factor, and an important factor,in the production of carcinoma of the lungs."

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[19]Wynder EL, Graham EA, *Tobacco smoking as a possible etiologic factor in bronchogenic carcinoma.* JAMA 143:336-338 (1950)

[20]Editorial: *Smoking and cancer of the lung,* BMJ 1950 1:1477. (1950)

[21]Doll R, Hill AB, *Smoking and cancer of the lung.* BR MED J 1950;2:739-748 (1950)

7.7 The Doll and Hill study validated Wynder and Graham's work, creating virtual consensus in the medical literature. All the researchers who looked at the problem concurred. Schreck[22] studied 5003 hospital admission records for a relation between cancer and smoking and concluded:

> **"[The] positive correlation between the incidence of cigarette smoking and the incidence of cancer of the respiratory tract appeared to be both statistically and biologically significant.** There is strong circumstantial evidence that cigarette smoking was an etiologic factor in cancer of the respiratory tract." (p 57)

7.8 Mills and Porter[23] looked at 568 men dying of mouth and lung cancer matched against a population of controls who were nonsmokers. They found that the effect of cigarette smoke in causing cancer was undeniable:

> The percentage of cigar and pipe smokers is almost twice as high among white male victims of buccal cancer as among ... controls.
>
> **Cigarette smoking seems to bear a highly significant relationship to cancers of the respiratory tract...** (p.542)

7.9 In further accord was the independent study of Levin and Goldstein[24], reported in JAMA in 1950. This study of 1045 male cancer patients admitted to a hospital, versus 605 controls, showed that "in a hospital population, cancer of the lung occurs more than twice as frequently among those who have smoked cigarettes for 25 years than among other smokers or nonsmokers of comparable age."

7.10 The impact of the Doll/Hill work on the medical community was tremendous. The British Medical Journal ran an editorial[25] the same year, stating:

> "There can be little doubt that the incidence of cancer of the lung is really increasing, and tarred roads, exhaust gases, common respiratory infections, certain types of occupation, and tobacco smoking have all been blamed for the greater frequency of the disease. Of these possible causes smoking seems the most likely, for the rise in the death rate from cancer of the lung has followed a great increase in the consumption of tobacco and cigarettes." [767]
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> "[T]he proof that cancer of the lung is associated with smoking must obviously be based on statistical and not on clinical evidence."[767]

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[22] Schreck R, Baker LA, Ballard CP, et al, *Tobacco smoking as an etiologic factor in disease. I Cancer.*. CANCER RES 1950;10:49-58 (1950)

[23] Mills CA, Porter MM, *Tobacco smoking h. bits and cancer of the mouth and respiratory system.* CANCER RES 1950;10:539-542 (1950)

[24] Levin ML, Goldstein H, Gerbardt PR, *Cancer and tobacco smoking: a preliminary report.* JAMA 143: 336-338 (1950)

[25] Editorial: *Cigarettes and cancer*, BMJ 1950 2:767-68 (1950)

"Doll [and] Hill, ... have carried out a **meticulously conducted inquiry, the results of which have very serious implications**, for they conclude that 'smoking is a factor, and an important factor, in the production of carcinoma of the lung.' [767]

7.11 Doll further expanded his study in 1952[26], and then in 1953[27] summarized the world literature and stated "The results amount, I believe, to *proof that smoking is a cause of bronchial carcinoma*." Experimental proof was added by Wynder and Graham[28] in 1953, who in a famous experiment produced tumors in 44% of mice tarred with tobacco smoke condensate. The authors concluded that "tobacco contains specific carcinogen(s)."

7.12 Although the fact that cigarette smoke caused lung cancer could scarcely be doubted, the *mechanism* by which this occurred was unknown. However, the protection of public health cannot await complete knowledge. Enough was known about the cause of cancer, and how to prevent it by eliminating or reducing cigarette smoke, to save millions of lives.

7.13 To protect its financial interests, the cigarette industry refused to heed this advice, and continued to complain that there was no "proof" (as in laboratory proof) that cigarettes caused harm.

7.14 In part to silence critics, Wynder and Graham[29] in 1953, conducted a world-famous laboratory experiment that produced tumors in 44% of mice tarred with tobacco smoke condensate. This work, combined with earlier studies[30], demonstrated biologic plausibility. The authors concluded that "tobacco contains specific carcinogen(s)." Mouse cancers developed after a latency of one-half a mouse lifetime, similar to the human experience. Wynder and Graham recounted that 12 recent studies had by 1953 confirmed the relationship between lung cancer and cigarette smoking in humans:

7.15 So compelling was the evidence by 1953 (and so compelling as well was the absence of contrary evidence) that the New England Journal of Medicine

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[26]Doll R, Hill AB, *A study of the aetiology of cancer of the lung.* BR MED J 1952;2:1271-1286 (1952)

[27]Doll R, *Bronchial carcinoma: incidence and aetiology.* BRIT M J 2:521-590 (1953)

[28]Wynder EL, Graham EA, Croninger AB, *Experimental production of carcinoma with cigarette tar.* CAN RES 13,855 (1953).

[29]Wynder EL, Graham EA, Croninger AB, *Experimental production of carcinoma with cigarette tar.* CAN RES 13,855 (1953).

[30]Flory *Experimental Production of Carcinoma from Tobacco Tars* (1941)

published an editorial[31] stating:

"Males between the ages of 45 and 64 experience an incidence of lung cancer between four and thirty-four times as great as that of nonsmokers of the same age, and incidence increases proportionately to the average daily consumption of cigarettes."

If similar data had incriminated a food contaminant that was not habit forming and was not supported by the advertising of a financial empire, there is little doubt that effective countermeasures would have followed quickly. *It is not insufficiency of evidence that accounts for lack of such measures against tobacco tars...*" (p 466)

7.16 If the evidence was conclusive by 1950 and overwhelming by 1953, it was absolutely beyond question by 1954, as two large, independent epidemiological investigations reported their grim preliminary figures: that lung cancer was a smoker's disease, and other cancers and heart disease were related to cigarette smoke as well. The American Study of Hammond[32] was an analysis of 4854 deaths in a population of 187,766 men between 50 and 69. The study found that the total death rate among heavy smokers was 75 percent higher than nonsmokers. The authors concluded, reading their paper before at the AMA Convention for 1954:

"All of the evidence we have seen seems to be consistent with the hypothesis that the association between smoking habits and death rates from lung cancer and diseases of the coronary arteries results from a cause and effect relationship. We know of no alternative hypothesis that is consistent with all of the known facts." (p 1328)

7.17 From England again came confirmation in a major independent study. Reasoning that physicians were likely to be accurate in their reporting of their own smoking habits and diseases, Doll and Hill conducted a major epidemiological study of over 40,000 British physicians.[33] Their report revealed that mild smokers were seven times as likely to die from lung cancer as non-smokers; moderate smokers were 12 times as likely to die from lung cancer as non-smokers, and "immoderate" smokers were 24 times as likely to die from lung cancer as non-smokers:

"The [death] rates reveal a significant and steadily rising mortality from deaths due to cancer of the lung as the amount of tobacco smoked increases. There is also a rise in the mortality from deaths attributed to coronary thrombosis..." (p 1455)

7.18 So critical was the information developed in the early 1950's to the health of

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[31]Editorial: Cancer of the lung, *The New England J of Med 1953 Sept;249(11):465-466.* 1953

[32]Hammond EC, Horn D, *The relationship between human smoking habits and death rates.* JAMA 1954 Aug;155(15):1316-1328 (1954)

[33]Doll R, Hill AB, *Lung cancer and other causes of death in relation to smoking. A second report on the mortality of British doctors.* BR MED J 1956;2:1071-1081 (1956). Doll WR, Hill AB, *The mortality of doctors in relation to their smoking habits. A preliminary report.* BRIT MED. J. I,1451 (1954).

the consumers of cigarettes, that in 1954 Dr. Hammond stated that the "public should be informed" of the "very convincing case" that proved the dangers of cigarettes.[34]

All of the evidence taken together builds a very convincing case for the theory that cigarette smoking, and particularly heavy cigarette smoking, causes an increase in the incidence of lung cancer..."

The public should be informed as to the present state of our knowledge..." By far the happiest solution would be to make smoking safe."

7.19 Tragically, the public was not informed in the 1950's, and has never been completely informed of the true health hazards of the product.

7.20 A blue ribbon study group was convened in 1956, jointly with the U.S. Public Health Service, the National Cancer Institute, the National Heart Institute, the American Cancer Society, and the American Heart Association. The group reviewed 16 studies published over the last 18 years and concluded that the relationship between lung cancer and smoking was "causal."

7.21 In 1957 the U.S. Surgeon General declared that excessive smoking was a causal factor in lung cancer.[35]

The Public Health Service feels the weight of the evidence is increasingly pointing in one direction: **that excessive smoking is one of the causative factors in lung cancer."**

7.22 By 1957, the British Medical Research Council had studied all available literature on the subject and issued a report which was unequivocal, stating that cigarette smoke caused lung cancer by "direct cause and effect."[36]

"1. A very real increase has occurred during the past twenty-five years in the death-rate from lung cancer in Great Britain and other countries.

"2. A relatively small number of the total cases can be attributed to specific industrial hazards.

"3. A proportion of cases, the extent of which cannot yet be defined, may be due to atmospheric pollution.

"4. Evidence from many investigations in different countries indicates that a major part of the increase is associated with tobacco smoking, particularly in the form of cigarettes. **In the opinion of the Council, the most reasonable interpretation of this evidence is that the relationship is one of direct cause and effect.**

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[34]Hammond EC, *Epidemiologic studies on smoking relation to lung cancer.* THE PENN MED J 1954;57(11):1084-1087 (1954).

[35]Burney I.E. *Statement. July 12, 1957.* CA. BULLETIN OF CANCER PROGRESS. 8:44 (March-April 1958)

[36]Editorial: *Medical Research Council's statement on tobacco smoking and cancer of the lung,* LANCET 1957 JUNE 29; 1345-1346. (1957)

"5. The identification of several carcinogenic substances in tobacco smoke provides a rational basis for such a causal relationship." [1346-47]

7.23 Meanwhile, very compelling non-epidemiological proof was accumulating. Auerbach in 1957 reported on an extensive microscopic investigation of the lungs and larynxes of smokers and nonsmokers who had died in Veterans' hospitals.[37] This study, which was extended for many years with consistent results, was a review of lung sections by pathologists of 117 cases of smokers and nonsmokers. It demonstrated, in graphic detail, a "stepwise progression" from bronchial metaplasia to cancer in situ to carcinoma or true cancer, in the lungs of the smokers. Thirty-five of 47 patients who smoked more than 1 pack per day had carcinoma in situ, a pre-cancerous lesion very rare in nonsmokers. This research showed that, in smokers, lung cancer was not a rare "lightening-bolt" but an almost inevitable process.

7.24 In 1958 Dr. E. Cuyler Hammond, on behalf of the American Cancer Society published[38] the followup to the 1954 report. This was a large study following 187,783 men aged 50 to 69 for 44 months. Recruiting over 22,000 volunteers to acquire data, Hammond's study demonstrated not only large increases in lung cancer with cigarette smoking but also clear evidence of a dose-response relationship. Heavy smokers were found to have risks of lung cancer elevated over 2,000% (two thousand percent). Hammond also demonstrated an increased risk of other cancers and of coronary thrombosis, and a time-related decrease in risk following cessation of smoking.

7.25 Consistent evidence from large statistical studies continued to accumulate. Dorn (1959)[39] followed 200,000 veterans holding Government life insurance policies for a period of over two years. Dorn found that men smoking more than one pack per day were 16 times as likely to die of lung cancer as non-smokers. Hammond (1959), expanding the American Cancer Society study to include over million men and women, concluded as follows:

(1) "Lung cancer turns out to be closely associated with the inhaling of cigarette smoke."

(2) "No prospective studies have failed to show a relationship between cigarette smoking and lung cancer."

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[37]Auerbach O, Gere JB, Forman JB et al. *Changes in the Bronchial Epithelium in Relation to Smoking and Cancer of the Lung.* NEW ENG. J. MED. JAN 1957.

[38]Hammond EC, Horn D, *Smoking and Death Rates Report on Forty-Four Months of Follow-Up of 187,788 Men.*. JAMA, 166:10, 1159-1172. (1958)

[39]Dorn HF, *Tobacco consumption and mortality from cancer and other diseases.* PUBLIC HEALTH REP 1959;74:581-593 (1959)

7.26 Further irrefutable evidence continued to accumulate during the period. In 1959 the Surgeon General called smoking "the principal factor in the increased incidence of lung cancer."[40]

7.27 The urgent need to inform the public of the severe hazard from the product was evident. In 1960 an editorial in the New England Journal of Medicine demanded that "lives can be saved" if the facts about cigarettes are made known to the public:[41]

> Although the search should continue for a carcinogen in cigarette smoke, there is already sufficient evidence on hand to implicate smoking as 'the principal etiologic factor in the increased incidence of lung cancer,' and **lives can be saved if these facts are made known to the medical profession and the smoking public.**
>
> It is not necessary to have precise information on etiology to prevent disease. Vaccination protected against smallpox more than a hundred years before the virus was identified, and the cholera vibrio was unsuspected at the time that John Snow had the foresight to take the handle off the Broad Street pump. ... **Lives will continue to be lost if control measures must await definitive studies.**

7.28 Throughout this time, the cigarette industry had publicly denied that cigarettes were dangerous or indeed caused any disease at all. The industry committed scientific fraud on the public by inventing bogus arguments to create doubt in the public mind. Such bogus arguments included:

- Statistics can't prove causation
- The epidemiological studies are biased
- No one knows the cause of cancer
- Animal studies do not establish cancer hazard in man
- There is no "proof" that cigarettes cause disease

8 There were many variations on these arguments, but they all shared one important characteristic: they were false, and knowingly and deliberately false.

8.1 In 1961 Dr. Ernest Wynder wrote a reply to various cigarette company arguments that had been made by Clarence Cook Little. Dr. Wynder correctly characterized Little's protestations as "destructive criticisms" and "misrepresentations."

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[40] Burney LE: *Smoking and lung cancer. A statement of the Public Health Service*, JAMA 1959 171:1829-37 (1959)

[41] Editorial: *Smoking and lung cancer*, N ENG MED J 1960 262:417-18 (1960)

It is the task of the independent researcher, as well as that of the tobacco industry, to determine the facts as they are and then let the facts speak for themselves. There are fields of human endeavor in which facts can be suppressed and at times submerged forever. Not so, however, in science. Here, facts may be destructively criticized and misrepresented, but if indeed they are facts, they will eventually be accepted, as the history of scientific progress testifies. ...

Any scientist welcomes constructive criticism. **Destructive criticism, however, from whatever source does not aid scientific progress. If one negates the value of statistics as part of scientific proof, disregards animal evidence as at least aiding human data and sets a goal for acceptable proof that is based upon impossible conditions, the very aim to resolve a given issue is paralyzed.**

8.2 The Royal College of Physicians of London set up a committee in 1959 to report on cigarette smoking and lung cancer. The report was published in 1962, a short but detailed work entitled SMOKING AND HEALTH[42]. Analyzing 23 retrospective studies in nine countries and at least four prospective studies in three countries, the report found the association between lung cancer and cigarette smoking "confirmed," noting death rates "increase steeply" with increasing consumption to 30 times the rate of nonsmokers.

8.3 The report also found relationships between smoking and heart disease, cancers of the mouth and other organs, and chronic bronchitis, and suggested that nicotine was addictive.

8.4 At that time (1962) similar conclusions on the dangers of cigarettes had been reached by the British Ministry of Health, the British Medical Research Counsel, the National Cancer Institute of Canada, the International Union Against Cancer, the World Health Organization, the Netherlands Ministry of Social Affairs and Public Health, the United States Public Health Service, the American Public Health Association, the Public Health Cancer Association, the American Heart Association, the National Tuberculosis Association, and the American Cancer Society.

8.5 Although the evidence by the 1960's could not really be improved upon, the cigarette industry continued to deny the truth. There was a need for a national statement, so in 1962 President Kennedy appointed a blue-ribbon panel to prepare a comprehensive report. The panel was selected of physicians and scientists who had not in public expressed any opinions about cigarettes and disease. The cigarette industry, in fact, was given veto power over the membership on the committee. Nevertheless, a committee was appointed, and it sifted through thousands of medical reports for almost two years. The result was a detailed report on cigarette smoking risks, summarizing all the available literature. This included animal experiments, where known compounds in smoke induced cancer in laboratory animals, histopathology studies, where

[42] SMOKING AND HEALTH. SUMMARY AND REPORT OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON ON SMOKING IN RELATION TO CANCER OF THE LUNG AND OTHER DISEASES (Pitman Publishing, New York 1962)

lungs and airways from smokers were found to have precancerous changes whereas nonsmokers did not, and by epidemiological studies. To the chagrin of the industry, the Surgeon General concluded:

> Cigarette smoking is **causally related** to lung cancer in men; the magnitude of the effect of cigarette smoking **far outweighs all other factors**.... The risk of developing lung cancer increases with duration of smoking and the number of cigarettes smoked per day, and is diminished by discontinuing smoking.

8.6 Discussed in the 1964 report were 30 retrospective studies which all showed an association between lung cancer and smoking. **There were no studies performed which negated any such association.** In addition seven large prospective studies were combined in a "meta-analysis" of 1,123,000 men and 37,391 deaths. Lung cancer and other cancers, as well as cardiovascular diseases and obstructive airway diseases were significantly elevated in smokers:

> In the combined results from these seven studies, the mortality ratio of cigarette smokers was particularly high for a number of diseases: cancer of the lung (10.8), bronchitis and emphysema (6.1), cancer of the larynx (5.4), oral cancer (4.1), cancer of the esophagus (3.4), stomach and duodenal ulcers (2.8), and ...other circulatory diseases (2.6).[p. 113]

9 Reasonable care requirements of cigarette industry

9.1 From a human health perspective, reasonable care would have required any cigarette manufacturer to immediately respond to this overwhelming body of research linking cigarettes to very serious diseases of the lungs and body. The absolute minimum response required would have been, in my opinion:

9.1.1 to immediately as of 1941 launch research projects to evaluate whether humans who smoke cigarettes were becoming ill with lung cancer and other diseases, and to timely report on these projects to the medical community and the public.

9.1.2 to test cigarettes in every scientifically valid way to determine the carcinogenic properties of the cigarette, the contents of the cigarette smoke, and whether there existed any safe dose of cigarette smoke, and to timely report on these tests to the medical community and the public.

9.1.3 to warn and communicate to potential customers and health care providers of the seriousness of medical concerns attendant upon smoking; including cancer, nonmalignant disease, addiction and others. Such communications including the taking out of pubic interest statements, the conducting of seminars, and other such activities.

9.1.4 to cease the misleading and fraudulent public denial of the health hazards of the product

9.1.5 to design cigarettes to minimize the carcinogenic and other health risks.

9.1.6 to cease the suggestive, aggressive advertising and packaging of cigarettes

9.1.7 to cease the sale and advertising of cigarettes to minors

9.1.8 to deliver important information to consumers including: how to smoke to minimize carcinogenic dose; how to break the nicotine addiction; how to monitor how much smoke is being inhaled; how to properly use ventilated cigarettes, and other information.

9.2 The medical literature fails to contain evidence of good-faith investigation and reporting by the tobacco industry in the time period addressed above. Neither the general public nor the medical community were given knowledge about the health effects of cigarettes which originated from scientifically valid studies or tests performed by the tobacco industry. Thus I conclude that the industry failed to use reasonable care to address the important medical studies outlined above.

9.3 The medical and scientific literature demonstrates that the first cautionary labeling of any time was printed on cartons of cigarettes on January 1, 1966. The failure of the manufacturer to notify potential customers in any way from 1941 until 1966 was inexcusable and amounted to a failure to use reasonable care, and to a deliberate and reckless act. Millions of people began to smoke during that time and ultimately died of lung cancer, chronic obstructive lung disease, and other diseases.

10 Inadequate cautionary labeling or warning

10.1 Prior to 1966 no warnings of any kind appeared on cigarettes in the United States. In light of the extensive knowledge of danger available to the manufacturers at that time, such failure to warn cannot be justified and amounts to a willful and reckless disregard of the health of tobacco customers.

10.2 The cautionary label which appeared in 1966 through January 1, 1970 was inadequate. The label stated:

CAUTION: CIGARETTE SMOKING MAY BE HAZARDOUS TO YOUR HEALTH

10.3 In view of the tremendously significant health consequences of smoking known at that time, this relatively mild cautionary label was inadequate to inform

customers (many of whom would later appear as patients in the hospitals across the country.)

- The warning was much *too weak*. The term "may be hazardous" suggests that the issue is debatable and that, in fact, it may not be hazardous.

- The warning failed to give *consequences*. There is no indication that serious diseases, and death, could result from use of the product.

- There was no warning of the *magnitude or severity* of the risk, including the fact that 40 to 50% of regular smokers would die prematurely, the fact that lung cancer risks were elevated almost 3000%, and the fact that cancers of the aerodigestive tract and other organs, obstructive airway disease, and heart disease, were increased significantly.

- There was no warning of *addiction or habituation* as a likely consequence of continued use;

- The *persistence in risk* even after smoking cessation and the possibility of irreversible genetic damage from smoking was not addressed;

- The warning never advised minors or their guardians of the special risk for cancer, heart disease, and addiction experienced when smoking is begun at an *early age*.

- The warning failed to advise that *excessive use* brought vastly increased risks, and that persons who chose to smoke would be advised to moderate their consumption.

- The warning never advised of the *harmful ingredients* of tobacco smoke, the fact that many ingredients were known carcinogens, nor the fact that many ingredients had never been adequately tested for carcinogenic potential

- The warning was neutralized by the *commercial advertisements* and public statements of the tobacco industry, denying health risks, and even making claims of modest health benefits.[43]

10.4 Thus, prospective users were never offered adequate information on which to base their decisions about smoking.

10.5 Based on my years' of experience in counseling patients about health risks, the cautionary label failed to inform these patients because it lacked specification of the degree and nature of the hazard. When patients are counseled, the

[43]For example, a 1953 advertisement for Philip Morris stated, "Stop Worrying" about cigarette irritation. Philip Morris is "entirely free of irritation used in all other lading cigarettes." Other advertisements in the 1940-1960 era suggested that sports figures and even physicians preferred one brand of cigarettes over another.

communication must include a definite risk such as CANCER RISK – TWO THOUSAND PERCENT INCREASE or similar language. There must be similar warnings about addiction or habituation plus warnings about other diseases.

10.6 Attached to this affidavit is a sample package insert that should have been attached to or included with cigarette products as early as 1954.

10.7 In 1970 another Federal law changed the required text slightly, substituting "Warning" for "Caution" and "Is Dangerous" for "May Be Hazardous":

WARNING: THE SURGEON GENERAL HAS DETERMINED THAT CIGARETTE SMOKING IS DANGEROUS TO YOUR HEALTH

10.8 The 1970 warning failed, as before, to adequately advise prospective users of the known risks. Although the strength of the warning was increased somewhat, the specifity was not improved.

10.9 The required warnings were amended again in 1984, mandating the current four rotating warnings:

SURGEON GENERAL'S WARNING: Smoking Causes Lung Cancer, Heart *Disease, and Emphysema.*

SURGEON GENERAL'S WARNING: Quitting Smoking Now Greatly Reduces Serious Health Risks.

SURGEON GENERAL'S WARNING: Pregnant Women Who Smoke Risk Fetal Injury and Premature Birth.

SURGEON GENERAL'S WARNING: Cigarette Smoke Contains Carbon Monoxide.

10.10 Warnings concerning the magnitude of risk, the high risk to children, the liability for addiction, the dose-responsiveness of disease and of addiction, the harmfulness of various ingredients, and other risks, were omitted from the 1984 warning as well and have never been publicized.

10.11 Public surveys have shown that the 1965 and subsequent warnings did increase public awareness of the hazards of tobacco and did lead to reductions in the number of smokers. However, the same surveys have shown that the public, although aware of some risks in smoking, does not appreciate the magnitude and severity of the risk. Few smokers know that 40-50% of regular smokers will die prematurely, for example; few appreciate the significance of the addiction risk; and few realize that substantial risks are irreversible even if smoking is stopped.

11 Cigarette as unreasonably dangerous

11.1 Furthermore, I have considered the question of whether the cigarette (present or past) is or is not "reasonably safe" or "unreasonably dangerous" or "meets consumer expectations" as these terms have been explained to me. There is abundant medical literature on the design of cigarettes, the delivery of nicotine and other smoke elements to the smoker, the adequacy of filtration, the use of different papers, the use of ventilation and smoke dilution, the use of expanded or other types of tobacco, and so on.

11.2 From a medical point of view, the diseases associated with cigarette smoking obey the "dose-response" concept.[44] Thus it is theoretically true that the less nicotine, carcinogens, or other harmful ingredients inhaled, the less the risk for various diseases. Thus, a product that fails to incorporate those features that result in the lowest possible doses of harmful ingredients to the smoker is, in my opinion, "unreasonably dangerous," especially since there is no overriding socially beneficial goal or result from smoking.

11.3 I have reviewed the medical literature on various design alternatives to reduce the delivery of harmful ingredients. Among these design features are (1) changing the porosity of the paper; (2) changing the diameter of the cigarette; (3) changing the density or firmness of the tobacco; (4) improving the quality or quantity of filtration; (5) improving the ventilation with outside air; (6) changing the acidity of the tobacco; (7) other engineering and design features.

11.4 Tobacco manufacturers have published certain values for "tar" and nicotine from machine smoking of various brands. Although this information has some uses, it is not a reliable index of carcinogenicity and assumes smoking by machine in a defined way that is not practiced by smokers.[45]

11.5 The design of the cigarette is defective in that it fails to properly utilize the engineering features outlined above to reduce, minimize, or eliminate harmful ingredients from the smoke.

11.6 In addition to the above, certain other design features should have been included on cigarettes to minimize the amount of harmful substances smoked and inhaled. These include the following:

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[44] Vutuc C, Kunze M, *Tar yields of cigarettes and male lung cancer risk.*. J NATL CANCER INST 1983 SEP;71(3):435-7 (1983).

[45] Woodward M, Tunstall-Pedoe H, *Do smokers of lower tar cigarettes consume lower amounts of smoke components? Results from the Scottish Heart Health Study.*. BR J ADDICT 1992 JUN;87(6):921-8 (1992)

11.6.1 The amount of harmful substances inhaled depends upon the amount of tobacco smoked and the resulting length of the "butt." The published tar and nicotine figures assume a certain butt length, but smokers can and do smoke beyond this (to a shorter length). Marking the intended butt length (where the smoker should stop smoking) is a feasible design improvement.

11.6.2 To ventilate the smoke cigarette manufacturers punch holes in the filter. These holes are extremely small and cannot be seen. Nor do smokers know that they are there. Blocking the vent holes with one's fingers can and does increase the delivery of carcinogens to the smoker. Sometimes smokers unknowingly block the vent holes to get more smoke per puff, trying to maximize their intake of nicotine. The cigarette should include prominent marking of the filter ventilation holes so that they will not be intentionally or accidently obscured by smokers. This is also feasible.

11.6.3 The overriding defect in the cigarette is its delivery of addictive levels of nicotine, when such levels could feasily be removed. The removal of nicotine is commonly done by solvent during cigarette manufacture. Reconstituted tobacco is de-nicotinized and later re-nicotinized. The level of nicotine is deliberately kept at or near 1 milligram per cigarette, because this amount sustains addiction in a majority of smokers.

11.7 In addition, changes in smoke chemistry, including smoke pH, can affect the amount of nicotine absorbed by the user. Manufacturers have long capitalized on this fact to design cigarettes with high nicotine impact or absorption that was not reflected in the FDA tests for nicotine.

11.8 I believe and testify herein that cigarettes that deliver addictive levels of nicotine are "defective" in that regard, as that term has been explained to me.

12 Lack of directions for use

12.1 In addition, it is axiomatic that consumers should be given directions to properly use a product. My counseling with smokers convinces me that they do not know what types of smoking behavior is most associated with cancer and other diseases. The cigarette packages should contain a "direction sheet" specifying the least risky types of smoking behavior. In addition, directions could be given to smokers in the following ways:

- Directions printed on the outside of packs
- A direction sheet included within a pack or carton

- Directions for use published with advertisements
- Television or radio announcements with directions
- Directions published to physicians, authors, interest groups, government agencies, and others.

12.2 The directions referenced above should contain information on how to properly smoke or handle the product. Such directions could have take the following forms:

12.2.1 The directions should instruct users to *smoke fewer cigarettes.*

12.2.2 The directions should inform users that they should *reduce their puff volume, take fewer puffs, and decrease their tendency to inhale.*

12.2.3 In addition, smokers should be instructed *not to block the vent holes* of cigarettes.

12.2.4 Directions should be provided on how users could *quit smoking* and thereby reduce their risk of cancer and other serious illnesses. Specifically, users should be directed that quitting attempts may need to be accompanied by counseling or withdrawal drug therapy.

12.2.5 Compensatory smoking is an increase in puff volume, puff frequency and inhalation depth in response to smoking fewer cigarettes or smoking lower tar and/or nicotine cigarettes. Users should have been advised and instructed *not to engage in compensatory smoking.*

12.2.6 Directions should be provided to users that *low yield cigarettes may not significantly reduce health risks.*

12.2.7 Likewise users should be instructed to *avoid smoking the entire cigarette,* because tar condensate on the proximate end increases carcinogen delivery.

12.2.8 Directions should be affixed or included in the purchased package that if users smoked cigarettes they should *notify their physician* of this fact and seek regular physical examinations, including chest X-rays, to increase the probability that serious illnesses including cancer and chronic obstructive lung disease, would be detected early to improve her chances of survival.

12.3 To my knowledge, there never have been such directions, of any kind, published to users or affixed to products. Therefore, in addition to the other

opinions expressed above, it is my opinion that the cigarettes in question, lacking such directions, are unreasonably dangerous to consumers and users.

13 Defective delivery of addictive nicotine

13.1 In addition, one of the most dangerous qualities of the cigarette is the addictive nature of nicotine. I have witnessed the effect of nicotine addiction on my patients, who will often have great difficulty or impossibility in discontinuing the habit.

13.2 The medical literature has contained references some time that nicotine is an addictive substance.

13.2.1 As early as 1939, Head[46] stated:

> There is ample evidence apart from that supplied by experimental methods that tobacco contains drugs which are habit forming. ... The lungs are one of the largest and most important absorbing surfaces in the body and it has been observed experimentally that drugs injected into them are absorbed more rapidly than when induced into the body by any route save that of the blood stream. ... Further evidence in support of the contention that tobacco contains habit forming drugs is the fact that a tolerance can be acquired for it... p 283

13.2.2 In 1942, Dr. Lennox Johnston, conducting his own experiments on nicotine, [47] concluded: "It seems probable ... that satisfaction is caused by the stimulation of the sensory cells in the brain and that craving is the subjective manifestation of the depression which follows."

13.2.3 By 1945 researchers at the Medical College of Virginia, supported by the tobacco industry, concluded:[48]"Smokers show the same attitude to tobacco as addicts to their drug, and their judgment is therefore biased in giving an opinion of its effect on them." By 1963 Knapp and others[49] confirmed the findings of Lennox Johnston many years earlier, that cigarette smokers were "addicts."

> "Heavy cigarette smokers thus appear to be true addicts, showing not only social habituation but mild physiologic withdrawal effects." (p 971)

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[46]Head, JR. *The Effects of Smoking.* ILLINOIS MED. J. 1939.

[47]Johnston, L. *Tobacco Smoking and Nicotine.* LANCET Dec 19, 1942 p 742.

[48]Finnegan JK, Larson PS, Haag, HB. *The Role of Nicotine in the Cigarette Habit.* SCIENCE 102:2639:94 (1945)

[49]Knapp PH, Bliss CM, Wells H, *Addictive aspects in heavy cigarette smoking.* AM J PSYCHIATRY 1963 APRIL;119:966-72 (1963)

13.2.4 In 1962 the Royal College of Physicians of London noted the prevalence of withdrawal symptoms on those attempting to quit smoking and stated, "Smokers may be addicted to nicotine."[50]

13.2.5 Unbeknownst to the medical community at large, considerable secret research on addiction was occurring in the 1960's.

13.2.6 In 1964 the U.S. Surgeon General concluded that cigarettes were more "habit forming" than "addictive." The report remarked that the "habitual" use of tobacco was "primarily" due to social and psychological factors, but warned that these were "*reinforced and perpetuated by the pharmacological actions of nicotine.*"[51]

13.3 In 1988 the U.S. Surgeon General concluded:

- Cigarettes and other forms of tobacco are addictive
- Nicotine is the drug in tobacco that causes addiction
- The pharmacological and behavioral processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin and cocaine.

13.4 It is now understood that nicotine addiction is the primary reason people smoke.

- Two-thirds of adults who smoke say they wish they could quit;
- Only one in ten smokers who try to quit succeed;
- Three out of four adult smokers say they are addicted;
- Eight out of ten smokers say they wish they had not started;
- 70% of young people ages 12-18 who smoke say they are dependent or addicted;
- 40% of high school seniors who smoke regularly have tried to quit and failed;
- Over 50% of teenagers do not believe they will become addicted.

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[50] SMOKING AND HEALTH. SUMMARY AND REPORT OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON ON SMOKING IN RELATION TO CANCER OF THE LUNG AND OTHER DISEASES (Pitman Publishing, New York 1962)

[51] U.S. Department of Health, Education, and Welfare, Public Health Service. *Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service.* P.H.S. Publication 1103 (1964)

13.5 The FDA in August 1996 issued a Jurisdictional Determination: NICOTINE IN CIGARETTES AND SMOKELESS TOBACCO IS A DRUG AND THESE PRODUCTS ARE NICOTINE DELIVERY DEVICES UNDER THE FEDERAL FOOD, DRUG, AND COSMETIC ACT: JURISDICTIONAL DETERMINATION[52]. This Jurisdictional Determination concludes, among other things, that "Nicotine is widely recognized as addictive, and it is foreseeable that consumers will use cigarettes and smokeless tobacco to satisfy an addiction." The report also concludes that manufacturers manipulate nicotine in their products to keep smokers addicted.

13.5.1 According to the FDA[53], it is nicotine, not "pleasure" that makes smokers return to their habit:

> A number of top tobacco industry officials have stated that they do not believe that tobacco is addictive. They may tell you that smokers smoke for "pleasure," not to satisfy a nicotine craving. Experts tell us that their patients report that only a small minority of the cigarettes they smoke in a day are highly pleasurable. Experts believe that the remainder are smoked to primarily sustain nicotine blood levels and to avoid withdrawal symptoms. ... The industry couches nicotine's effects in euphemisms such as "satisfaction" or "impact" or "strength." ... But these terms only sidestep the fact that the companies are marketing a powerfully addictive agent. Despite the buzzwords used by industry, what smokers are addicted to is not "rich aroma" or "pleasure" or "satisfaction." **What they are addicted to is nicotine, pure and simple, because of its psychoactive effects and its drug dependence qualities.**

13.6 When inhaled, nicotine reaches the brain in seven to nine seconds, initiating a sequence of events that includes the production of dopamine. Dopamine, a substance connected with regulation of motivation and emotion, is commonly triggered by addictive substances.

13.7 There have never been any warning about addiction to nicotine in any of the cigarette warnings.

13.8 A threshold for addiction based on the biological concentration of nicotine was suggested in recent review by Benowitz (1994)[54]

13.8.1 Benowitz asserted "nicotine addiction sustains tobacco use for most smokers" and that approximately 90 percent of the smoking public is addicted to cigarettes. Five mg of serum nicotine is "a threshold level that can readily establish and sustain addiction." Nicotine amounts to 1.5-2.5 percent of tobacco weight; a typical cigarette contains 8-9 mg of

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[52]Fed Reg 61:168 Wednesday, August 28 1996.

[53]Statement on Nicotine Containing Cigarettes by David A. Kessler, M.D., Commissioner of Food and Drugs before the Subcommittee on Health and the Environment, U.S. House of Representatives, March 25, 1994.

[54]Benowitz NL, Henningfield JE, *Establishing a nicotine threshold for addiction. The implications for tobacco regulation.* N ENGL J MED 1994 JUL 14;331(2):123-5 (1994).

nicotine. Bioavailability ranges from 3 to 40 percent with an average of 12 percent, depending on smoking habits. The typical nicotine dose per cigarette thus averages 1 mg but can be several times that figure. These calculations suggest that persons who smoke more than five cigarettes per day are at risk of addiction from nicotine.

13.8.2 In a British review, Russell (1990)[55] concluded that smoking as little as four cigarettes may lead to lifetime addiction.

13.9 Tobacco Use Disorder/Tobacco Dependence now has been listed in the ninth revision of the International Classification of Diseases authored by the WHO[56], and Nicotine Withdrawal Syndrome is described in the Diagnostic and Statistical Manual, DSM IV. DSM IV contains criteria for substance dependence which apply to nicotine in the case of most regular users.

13.10 As stated by the FDA Commissioner[57], most people who become addicted to tobacco simply lose the ability to choose to quit:

> It is fair to argue that the decision to start smoking may be a matter of choice. But once they have started smoking regularly, most smokers are in effect deprived of the choice to stop smoking....Seventeen million Americans try to quit smoking each year. But more than 15,000,000 individuals are unable to exercise that choice because they cannot break their addiction to cigarettes. My concern is that the choice that they are making at a young age quickly becomes little or no choice at all and will be very difficult to undo for the rest of their lives.

13.11 The tobacco industry regulates and controls the amount of nicotine in its cigarette products, as witnessed by the published differences in nicotine content by brand. This regulation of the amount of addictive nicotine in the cigarette is accomplished by some or all of the following procedures:

13.11.1 by regulation of the type and processing of tobacco in the cigarette

13.11.2 by regulation of smoke ventilation, paper porosity, filtration, tobacco packing density, butt length, and cigarette size, among other factors

13.11.3 by chemical regulation of smoke pH including regulation of the levels of ammonium and other compounds, which increase the availability of

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[55]Russell MA., *The nicotine addiction trap: a 40-year sentence for four cigarettes.* BR J ADDICT 1990 FEB;85(2):293-300 (1990).

[56]Pollin W, Ravenholt RT, *Tobacco addiction and tobacco mortality. Implications for death certification.* JAMA 1984 Nov 23-30;252(20):2849-54 (1984).

[57]Statement on Nicotine Containing Cigarettes by David A. Kessler, M.D., Commissioner of Food and Drugs before the Subcommittee on Health and the Environment, U.S. House of Representatives, March 25, 1994.

nicotine to the smoker (even without changing the published nicotine yield.)

13.12 Cigarettes are defective and unreasonably dangerous because they utilize the above technology to deliver an addictive quantity of nicotine to foreseeable users and consumers.

13.13 Evidence suggests the tobacco industry manipulates and enhances nicotine levels in its cigarettes. In congressional hearings[58], the FDA asserted that the industry can

> precisely manipulate nicotine levels ... manipulate the rate at which the nicotine is delivered ...transfer nicotine from one material to another ... increase the amount of nicotine in cigarettes ... add nicotine to any part of the cigarette ... and use a variety of methods to add nicotine to tobacco.

14 Tobacco industry coverup

14.1 The tobacco industry knew at least from the early 1950's that its products were carcinogenic. In 1953 Claude Teague, a scientist working for R.J. Reynolds Tobacco Company, compiled a comprehensive summary of the problem of a cancer hazard to customers. The document proves that R.J. Reynolds had exact knowledge of what was being discovered by outside scientists, and that its own scientific advisors thought the problem was serious.[59]

> [S]everal recent, well controlled large scale, clinical studies on correlation of tobacco smoking with cancer of the lung have been made.
>
> The increased incidence of cancer of the lung in man which has occurred during the last half century is probably due to new or increased contact with carcinogenic stimuli. The closely parallel increase in cigarette smoking has led to the suspicion that tobacco smoking is an important etiologic factor in the induction of primary cancer of the lung. **Studies of clinical data tend to confirm the relationship between heavy and prolonged tobacco smoking and incidence of cancer of the lung.** Extensive though inconclusive testing of agents in those substances. One worker has identified known carcinogens in a tobacco pyrolysate. Compounds are present in tobacco which on pyrolysis could theoretically give rise to compounds similar to known carcinogens. The possible role of tobacco additives and cigarette paper in the production of carcinogens cannot be determined because of lack of access to information on substances use.
>
> In view of the facts presented in this report it is recommended that management take cognizance of the problem and its implications to our industry. . .

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[58]Statement on Nicotine Containing Cigarettes by David A. Kessler, M.D., Commissioner of Food and Drugs before the Subcommittee on Health and the Environment, U.S. House of Representatives, March 25, 1994.

[59]*No.: 9448.00000. Survey of Cancer Research with Emphasis Upon Possible Carcinogens from Tobacco.* From Teague. Date [1953/ 2/ 2].

14.2 By 1959 Reynolds had confirmed the presence of specific carcinogens in the smoke from its cigarettes. One potent carcinogen was a polyaromatic hydrocarbon known as benzo(a)pyrene, which in subsequent years has been confirmed as a cause of genetic damage leading to cancer. In a report by one Dr. Rodgman, a Reynolds scientist, the company confirmed that "it would be better for the consumer" if such carcinogens were removed:[60]

> In 1954 the first report of the presence of a carcinogenic polycyclic hydrocarbon 3,4-benzpyrene in cigarette smoke was published. Since then, approximately 60 similar compounds have been isolated from the smoke of cigarettes.
>
> There is a distinct possibility that these substances would have a carcinogenic effect on the human respiratory system. Medical experience has shown that man responds to various chemical substances in the same manner as experimental animals. It follows therefore that **it would be better for the consumer if cigarette smoke were devoid of such components.**
>
> Cigarette smoke should contain as little as possible (preferably at the zero level) of the polycyclic hydrocarbons, should possess satisfactory flavor to please the consumer and should contain sufficient nicotine to supply the necessary requirements of the smoker with respect to this compound."

14.3 The Rodgman document is proof that Reynolds had actual knowledge that its products were extremely dangerous to the consumer when used as directed. The document also admits that the experimental proof of carcinogenesis in laboratory mice that had accumulated since 1953 was relevant to humans, a point denied by Reynolds even to today. Also, Reynolds failed to heed its scientist's advice that it would be "better for the consumer" if the carcinogens were removed.

14.4 In 1968 Claude Teague, prepared a memorandum on the objectives of Reynolds research, acknowledging the the tobacco and health "problem:"[61]

> a. smoking is detrimental to health and shortens life span.;
> b. smoking causes lung cancer and emphysema;
> c. smoking causes a variety of lesser known conditions.

The alternate strategies for coping with the "problem" were outlined:

> **a. Ignore the problem - hope it will go away or subside;**
> **b. Deny the allegations; prove them false;**
> c. Hope that a complete cure for cancer etc. arises;
> d. Eliminate offending substances from tobacco smoke;
> e. Accept the consequences to our business - maybe finally go out of business;
> f. Develop new products, away from conventional tobacco products, which pose no health

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[60] *No.: 7593.00000. The Optimum Composition of Tobacco & Its Smoke.* From Rodgman. Date [1959/11/ 2]. (to) = Kenneth Hoover

[61] *No.: 9935.00000. Behavior of Pre-Smokers: New Business Opportunity Arising from Long Range Research Planning Tobacco- Health Problem.* From Teague. Date [1968/11/12].

hazard but which give our customer all of the satisfactions he now derives from smoking.

14.5 Nicotine is itself a carcinogen, as was recognized early on by Reynolds and others. In a memorandum from 1976, a research scientist noted:[62]

> **Bock has reported (1976 AACR meeting) low to moderate concentrations of nicotine act as a cocarcinogen stimulus when applied to mouse skin.** During our discussion the accuracy of the results of Bock (and of all the NCI skin painting studies) was questioned. Possibly an explanation of nicotines promoter activity may be due to its ability to rapidly transport across the skin barrier, an effect not relevant in inhalation studies.

15 False and fraudulent public statements

15.1 In 1953, concerned that the publicity on the health effects of cigarettes were hurting sales, the entire tobacco industry met in secret to formulate a strategy. This strategy, which was followed for over 40 years, was in essence:

- To continue to publicly deny the evidence proving that cigarettes caused disease.
- To create a public relations front called the Tobacco Industry Research Committee, which could pose as a research institution engaged in finding out the "truth" about cigarette smoke.

15.2 To further this goal, an advertisement prepared by The Tobacco Industry Research Committee was published in over 50 newspapers across the country. The "Frank Statement to Cigarette Smokers"[63] was the beginning of a scheme of widespread pro-industry propaganda wherein the industry made promises to the American public. The Frank statement announced that the cigarette industry took "an interest in people's health as a basic responsibility, paramount to every other consideration in our business."

15.2.1 The Frank Statement stressed that "the products we make are not injurious to health," and promised the American public the industry "always will cooperate with those whose task it is to safeguard the public health."

15.2.2 The Statement made an obvious reference to the famous Wynder and Graham experimental study of 1953:

> Recent reports on experiments with mice have given wide publicity to a theory that cigarette

[62] No.: 9443.00000. *Nicotine Research. From Henley.* Date [1976/11/ 9]. (to)= D. Piehl

[63] *No.: 10245.00000. A Frank Statement to Cigarette Smokers.* From . Date [1954]

smoking is in some way linked with lung cancer in human beings.

15.2.3 The document's emphasis on "mice" was itself misleading in ignoring the conclusive human clinical and epidemiological proof that had been obtained through the work of Ochsner, Wynder and Graham, Doll and Hill, Breslow, and many others. The document went on to deny any relationship between cigarettes and disease, stating that there were "many possible causes of lung cancer," that there was "no agreement" among medical authorities about the cause, and there was "no proof that cigarette smoking is one of the causes."

15.2.4 All of these statements were knowingly false, and were "sold" to the public to conceal the true hazards and promote continued sales of the product.

15.2.5 "Many possible causes" of lung cancer had on occasion been discussed in the medical literature, but through the work of Kennaway and others, almost all causes except for tobacco had been discounted by 1950. Nor was there "no agreement" among physicians and researchers; by 1953 there was virtual unanimity, on both sides of the Atlantic, on the relationship between smoking and lung cancer. "Proof" that smoking was "one of the causes" had been ably furnished by human and animal studies; studies failing to find an association between smoking and lung cancer had not been published.

15.2.6 The promises made by the Industry in the Frank Statement were never repudiated and were never kept.

15.3 Liggett & Myers authored a public statement in 1954,[64] claiming that it had done important scientific research about its cigarettes. Hinting that it had proven the cigarettes safe, the company stated:

> From ... thousands of analyses, and other findings reported in the leading technical journals, our Research Department has found no reason to believe that the isolation and elimination of any element native to cigarette tobaccos today would improve smoking.

15.4 Liggett claimed to test its cigarettes using a "half-million dollar 30-ton machine, the world's most powerful source of high voltage electrons." How "high voltage electrons" tested cigarettes for safety or health was not explained. The company further claimed to have maintained "in the smokers interest an intensified larger scale diversified research program" which had already yielded "direct and significant information of benefit to the smoking public." Liggett never stated what this information was. The obvious implication was

[64] "Liggett & Myers Tobacco Co. says...", Florida Times-Union, January 18, 1954.

that the cigarettes were tested to be safe to consume.

15.5 By 1958, after further research of Hammond and Horn, Doll and Hill, and others, had further proven the connection between smoking and lung cancer, the industry continued to publicly deny the published science. speaking through the Tobacco Industry Research Committee's medical director[65], the industry claimed:

> No convincing clinical or experimental evidence has yet been brought forward that cigarette smoking is the positive cause of lung cancer.
>
> There have been and will continue to be speculations and opinions on the causes, but it is a matter of scientific fact that, in our present state of knowledge, no one knows the answers.

15.6 This denial changed little over the years, despite overwhelming scientific and clinical evidence.

15.7 In 1964 Bowman Gray, chairman of the board of directors of Reynolds stated before a congressional subcommittee that "[m]any distinguished scientists are of the opinion that it has not been established that smoking causes disease," and claimed a "lack of clinical and laboratory scientific evidence of the relationship between smoking and health." These statements were knowing attempts to mislead the public by misrepresenting the state of knowledge about cigarettes and human health.

15.8 In 1982 Edward Horrigan, CEO of R.J. Reynolds, stated "science to date after much research including over $100 million funded by our industry, indicates that no causal link [between smoking and human disease] has been shown," and that "there is absolutely no proof that cigarettes are addictive."

15.9 One manufacturer conducted a national campaign in 1984, challenging that the "studies which conclude that smoking causes disease have regularly ignored significant evidence to the contrary."[66] Of course there was never any "significant evidence to the contrary."

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[65]Brecher R, Brecher E, Herzog A, Goodman W, *The Consumers Union Report on Smoking and the Public Interest.* (Consumers Union 1963)

[66]R.J. Reynolds advertisement, *Can we have an open debate about smoking?* (1984)

16 Coverup of addiction research

16.1 Internal research on nicotine addiction has been performed since the 1950's. Much of this research has not been revealed to the medical community until recently.

16.2 In the early 1960's the British American Tobacco Company (BAT), the parent of U.S. manufacturer Brown and Williamson Tobacco Company, conducted extensive nicotine research in secret. (R.J. Reynolds and other U.S. manufacturers were privy to this research through the meetings of a committee of lawyers chaired by RJR's Henry Ramm and including Brown and Williamson's Addison Yeaman.)

16.3 Certain documents disclose the companies' understanding of nicotine addiction, long before it was well understood in the medical community.

16.3.1 In *Final Report On Project Hippo II*[67], the Batelle Laboratory, working for BAT, summarizes experiments on nicotine and reserpine on various hormones in both intact animals and isolated organs:

> "The aim of the whole research "HIPPO" was to understand some of the activities of nicotine – those activities that could explain why cigarette smokers are so fond of their habit. It was also our purpose to compare these effects with those of the new drugs called "tranquilizers" which might supersede tobacco habits in the near future." [p. 1]
>
> "Why does one smoke? It is certainly not because of nicotine's cardio-vascular activities.... The reasons for the "pleasure of smoking" must be found partly in the relief of anxiety that cigarette smoking brings so constantly and in such a very short time." [p 1]
>
> "A quantitative investigation of the relationships with time of nicotine and some possible brain mediators - on adreno - corticotrophin activity could give us the key to the explanation of both phenomena of tolerance and addiction, in showing the symptoms of withdrawal." [4]
>
> "Is it possible that the use of "tranquilizers" might supersede tobacco habits? To face this possibility, it seems necessary to understand more about the physiological activities of nicotine and of reserpine than can be related to their soothing effects." [p 6]

16.3.2 Page five also contains a chart summarizing what they viewed nicotine and reserpine's activities to be at the same which is an illustration of level of sophistication of their analysis.

16.3.3 In *The Fate Of Nicotine In The Body*[68] the researchers covered a variety of experiments on humans and animals to look at nicotine pharmacology

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[67] 1212.03. *Final Report On Project Hippo II.* (April 30, 1963) Haselbach, C.Libert, O.

[68] 1213.01. *The Fate Of Nicotine In The Body.* (May 1, 1963) Geissbuhler, H.Haselbach, C

in mechanisms of tolerance and addiction. The report shows that the absorbed nicotine is not related to the nominal levels in the smoke, and describes a whole variety of physiological experiments.

"There is increasing evidence that nicotine is the key factor in controlling, through the central nervous system, a number of beneficial effects of tobacco smoke, including its action in the presence of stress situations [cites.] In addition, the alkaloid appears to be intimately connected with the phenomena of tobacco habituation (tolerance) and/or addiction. [cites] Detailed knowledge of these effects of nicotine in the body of a smoker is therefore of vital importance to the tobacco industry, not only in connection with their present standard products, but also with regard to future potential uses of tobacco alkaloids." [p 1]

"The absolute quantity of nicotine absorbed upon smoking of a single cigarette depends (a) on the nicotine present in the tobacco, (b) on the amount of alkaloid transferred into the main stream smoke (or the smoke drawn into the mouth by the smoker), and (c) on the percentage of main stream nicotine absorbed by the smoker. These factors have been examined on a representative number of confirmed smokers by applying more refined and standardized techniques than were used by previous investigators (for technical details, see Appendix.)"[(p.5]

16.3.4 Of great importance, the researchers realized the connection of tolerance and addiction, a connection not made in the public medical journals until much later:

"We believe that both tolerance and addiction are intimately connected, and that it would be most useful to investigate the two phenomena with regard to cellular adaptation, especially in target organs of the central nervous system." [p 27]

16.3.5 Batelle scientists prepared an important memorandum, *A Tentative Hypothesis On Nicotine Addiction*[69] for BAT in which they concluded that chronic intake of nicotine "weakened" the nervous system, and that addicted smokers sought a return to the "normal" equilibrium:

The hypothalamo-pituitary stimulation of nicotine is the beneficial mechanism which makes people smoke; in other words, nicotine helps people to cope with stress. In the beginning of nicotine consumption, relatively small doses can perform the desired action. Chronic intake of nicotine tends to restore the normal physiological functioning of the endocrine system, so that ever-increasing dose levels of nicotine are necessary to maintain the desired action. Unlike other dopings, such as morphine, the demand for increasing dose levels is relatively slow for nicotine.

In a chronic smoker the normal equilibrium in the corticotropin releasing system can be maintained only by continuous nicotine intake. It seems that those individuals are slightly different in their aptitude into cope with stress in comparison with a non-smoker. If nicotine intake, however, is prohibited to chronic smokers, the corticotropin-releasing ability of the hypothalamus is greatly reduced, so that these individuals are left with an unbalanced endocrine system. **A body left in this unbalanced status craves for renewed drug intake in order to restore the physiological equilibrium. This unconscious desire explains the addiction of the individual to nicotine.**

[69] 1200.01. *A Tentative Hypothesis On Nicotine Addiction*. (May 30, 1963) Haselbach, C.Libert, O.

"In conclusion, a tentative hypothesis for the explanation of nicotine addiction ould be that of an unconscious desire to restore the normal physiological equilibrium of the corticotropin releasing system in a body in which the normal functioning of the system has been weakened by chronic intake of nicotine."

16.3.6 Addison Yeaman, Executive Vice President of Brown and Williamson Tobacco Company, President of CTR, and member of a committe of industry heads including Henry Ramm of RJR, was persuaded by the BAT research, and wrote in 1963 in a private memorandum:[70]

Strictly Private and Confidential

"Moreover, nicotine is addictive.

We are then in the business of selling nicotine, an addictive drug effective in the release of stress mechanisms. But cigarettes – we will assume the Surgeon General's Committee to say – despite the beneficent effect of nicotine, have certain unattractive side effects:

1) They cause, or predispose to, lung cancer.

2) They contribute to certain cardiovascular disorders.

3) They may well be truly causative in emphysema, etc. etc." [p. 4]

16.4 In 1963 the Surgeon General's committee was evaluating evidence to produce the first Surgeon General's report, and had requested all scientific evidence on smoking and disease, including addiction, possessed by the cigarette manufacturers. The important BAT reports were never disclosed.

16.4.1 A previously confidential memorandum shows the reluctance to forward this research: [71]

"PRIOR TO RECEIPT YOUR TELEX JULY 3 HOYT OF TIRC AGREED TO WITHHOLD DISCLOSURE BATTELLE REPORT TO TIRC MEMBERS OR SAB UNTIL FURTHER NOTICE FROM ME. FINCH AGREES SUBMISSION BATTELLE OR GRIFFITH DEVELOPMENTS TO SURGEON GENERAL UNDESIRABLE AND WE AGREE CONTINUANCE OF BATTELLE WORK USEFUL BUT DISTURBED AT ITS IMPLICATIONS RE CARDIOVASCULAR DISORDERS.

WE BELIEVE COMBINATION BATTELLE WORK AND GRIFFITH'S DEVELOPMENTS HAVE IMPLICATIONS WHICH INCREASE DESIRABILITY REEVALUATION TIRC AND REASSEMENT FUNDAMENTAL POLICY RE HEALTH. HOPE TO GET OFF COMPREHENSIVE NOTE NEXT WEEK.

16.4.2 In 1962 the Royal College of Physicians of London noted the prevalence of withdrawl symptoms on those attempting to quit smoking

[70] 1200.05. *Implications Of Battelle Hippo I & II And The Griffith Filter.* (July 17, 1963) Yeaman, A

[71] 1200.12. *Outgoing Cable [regarding disclosure of Surgeon General's committee].* (July 3, 1963) Yeaman, A.–McCormick

and stated, "Smokers may be addicted to nicotine."[72]

16.4.3 However, the 1964 Surgeon General's report[73] found that cigarette smoking was a "habit" as distinguished from and "addiction." Critical to the 1964 Report's finding was a lack of information on the physiological effects of nicotine. The Report characterized this as a "gap in knowledge."

16.4.4 Unbeknowst to the Surgeon General, the cigarette industry had filled this "gap in knowledge" secretly. Nicotine was a real physiological addiction.

16.4.5 Because the 1964 Surgeon General Report did not identify nicotine addiction (this was corrected when more public research was disclosed by 1988), the various cautionary labels on cigarette packages never disclosed that "NICOTINE IS ADDICTIVE." Nor was this voluntarily disclosed by any cigarette manufacturer. The net result was millions of customers dying of diseases caused by nicotine addiction.

16.5 Internal documents from other cigarette manufacturers also confirm that the industry was well aware, by virtue of secret research not revealed to the public, that nicotine in the product was addicting the customers:

16.5.1 Reynolds knew as early as 1959 that its addicted consumers had "physiological requirements" for nicotine. In a research memorandum, the company concluded that this requirement could be met by "application of the optimum amount nicotine" to the cigarette:[74]

> Cigarette smoke should contain as little as possible (preferably at the zero level) of the polycyclic hydrocarbons, should possess satisfactory flavor to please the consumer and should contain sufficient nicotine to supply the necessary requirements of the smoker with respect to this compound.
>
> The physiological requirements of the smoker with respect to nicotine can be met by the application of the optimum amount of nicotine to the extracted tobacco.

16.5.2 In a 1969 memorandum, Reynolds scientist Claude Teague analyzed what keeps people smoking and concluded that the first reason is the

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[72] SMOKING AND HEALTH. SUMMARY AND REPORT OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON ON SMOKING IN RELATION TO CANCER OF THE LUNG AND OTHER DISEASES (Pitman Publishing, New York 1962)

[73] U.S. Department of Health, Education, and Welfare, Public Health Service. *Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service.* P.H.S. Publication 1103 (1964)

[74] *No.: 7593.00000. The Optimum Composition of Tobacco & Its Smoke.* From Rodgman. Date [1959/11/ 2]. (to) = Kenneth Hoover

"nicotine habituation factor."[75]

A basic, simplistic equation relating the propensity of an individual to smoke (P), to a **nicotine habituation factor** (H), a gratification factor (G), a cost factor (C) and a government regulatory factor (R).

16.5.3 By 1972, Reynolds had long realized the need to maintain addictive levels of nicotine to keep its customers coming back. A research planning memorandum written by Reynolds research scientist, Claude Teague, is a recommendation that Reynolds market a high nicotine, low tar cigarette. Teague discusses tar and nicotine ratios in detail and concludes that the desired nicotine delivery is at least 1.3 mg. "In designing any cigarette product, the dominant specification should be nicotine delivery."[76] Teague demonstrated Reynolds understanding that nicotine is addictive:

I believe that for the typical smoker nicotine satisfaction is the dominant desire as opposed to flavor and other satisfactions. There are a variety of foods, beverages and confections from which a variety of flavors and other satisfactions may be obtained, if that were the dominant desire, but tobacco products alone provide nicotine satisfaction – therefore, that is the primary reason smokers smoke.

[A] given smoker on a given day has a rather fixed per hour and per day requirement for nicotine. Given a cigarette that delivers less nicotine than the desires, the smoker will subconsciously adjust his puff volume and frequency, and smoking frequency, so as to obtain and maintain his per hour and per day requirement for nicotine.

16.5.4 This memorandum proves to me that Reynolds possessed significant, substantial and secret knowledge of nicotine and addiction. and that despite important knowledge into the gravity of nicotine addiction, Reynolds intentionally concealed this knowledge from its consumers.

16.5.5 While publicly denying that nicotine was addictive, RJR internally, in secret memoranda, was well aware that nicotine addiction drove the "repeat business" and was essential for the profits of the industry.

16.5.6 RJR researcher Claude Teague wrote on April 14, 1972, a memo entitled *Research Planning Memorandum on the Nature of the Tobacco Business and Crucial Role of Nicotine Therein* that the tobacco industry is **"a specialized, highly ritualized and stylized segment of the pharmaceutical industry,"** and that tobacco products deliver nicotine

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[75] ***No.:*** *9580.00000. Proposal of a New Consumer- Oriented Business Strategy for RJR Tobacco Company Based Upon an Analysis of the Effects of the Smoking- Health Controversy and the 'Safer' Cigarette Strategy on Consumer Behavior.* From Teague. Date [1969/ 9/19]. (to) E. Vassallo, M. Senkus

[76] ***No.:*** *7290.00000. A New Type of cigarette Delivering a Satisfying Amount of Nicotine with a Reduced "Tar" - to Nicotine Ratio.* From Teague. Date [1972/ 3/28].

"a potent drug with a variety of physiological effects."

16.5.7 In this memo, which was not made public at the time, Teague admits the choice of product is primarily determined by *nicotine dosage* needs, and that other factors (i.e. taste) are secondary.

Tobacco product is in essence, a vehicle for delivery of nicotine." Teague referred to nicotine's physiological effects, stating tobacco competes with other products with "drug action". Teague called nicotine the *sine qua non* of tobacco, acknowledging that R&D must consider nicotine absorption, elimination, enhancement, and develop optimum means of *nicotine delivery*. Teague analyzed not only "dosage forms" but the factors which lead a smoker to become habituated.

16.5.8 RJR recognized that the smoker starts to emulate a valued image, to conform, experiment, defy, be daring -- and then the physiological "satisfaction" -- the addiction to nicotine -- becomes apparent. This memo additionally admits that nicotine tolerance is developed, another hallmark of addiction publicly disputed by RJR.

16.5.9 A 1973 memorandum from Reynolds employee Colby suggested that RJR develop a "youth-appeal brand" based on 1950's cigarettes which "delivered more flavor (tar)" and "delivered **more 'enjoyment' or 'kicks' (nicotine)**" because of design parameters including less porous paper and less efficient filters. Colby suggested a return to 1950's levels of tar and nicotine, but states that "for public relations reasons" (he was not concerned with health reasons) RJR could not "go all the way back to the 1955 type cigarettes." However, the levels of 1973 *nonfilter* cigarettes could be maintained, with "additional nicotine 'kick' ... obtained through ph regulation." (emphasis in original).[77]

16.5.10 This memorandum proves intent to regulate cigarette nicotine content to achieve success in the "youth" market; the feasibility of spiking nicotine by ph regulation and other methods; a lack of concern concern for the health and safety of industry customers in that the industry considered that a return to very high tar and nicotine cigarettes was impossible only for public relations reasons; and the marketing desirability of having addictive levels of nicotine and additional nicotine "kicks."

16.5.11 In 1982 Reynolds scientist Teague wrote a detailed memorandum on the status of the company's products. This memorandum admitted, "most of those who have smoked for any significant time would like to stop.

[77] *Colby Memorandum to Blevins. Cigarette Concept to Assure RJR a Larger Segment of the Youth Market* (12/4/73)

Many, but not most, of those who would like to stop smoking are able to do so.[78]

16.5.12 Teague further admitted, **"if the exit gate from our market should suddenly open, we could be out of business overnight."** This proves Reynolds knew that cigarettes are addictive and that it was crucial to keep smokers addicted and addict new smokers if the industry was to stay in business. These revelations are in sharp contrast to the public posture of the industry which has always been that its customers freely choose to smoke for taste and pleasure.

Further affiant sayeth naught

Sworn and subscribed this 20th day of February 1997

Allan Feingold, M.D., F.R.C.P.(C), FCCP

Personally known to me

Notary signature

EARNESTINE J. THOMPSON
MY COMMISSION # CC 256112
EXPIRES: June 18, 1997
Bonded Thru Notary Public Underwriters

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[78] *No.: 9508.00000. Nordine Study.* From Teague. Date [1982/12/ 1]. (to) G.R. DiMarco

CURRICULUM VITAE

Dr. Ilan Allan Feingold

Born: 04-11-50
SSN [redacted]

CV revised September 1994

Academic:

1967-71	Undergraduate studies, McGill University, Montreal. Graduated June 1971, B.A., Major in Sociology, with distinction.
1972-76	McMaster University Faculty of Medicine, Hamilton, Ontario. Graduated May 1976, M.D.

Postgraduate Training:

1975-76	Straight Intern, Internal Medicine, Royal Victoria Hospital, McGill University, Montreal.
1976-77	Resident II, Internal Medicine, Royal Victoria Hospital.
1977-78	Resident III, Internal Medicine, Royal Victoria Hospital.
1978-79	Resident IV, Division of Pulmonary Medicine, Royal Victoria Hospital and Montreal Chest Hospital Center.
1979-80	Research Fellow, Meakins-Christie Laboratories, McGill University Clinic, McGill University.

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Hospital Appointments:

1977-78	Physician, Division of Critical Care, Reddy Memorial Hospital, Montreal.
1978-80	Physician, Department of Internal Medicine, Intensive Care Unit and Pulmonary Consultations, Centre Hospitalier de Valleyfield, Valleyfield, Quebec.
1979-80	Physician, Emergency Department, Jewish General Hospital Montreal.
1980-82	Physician, Montreal Chest Hospital Center, Montreal.
1980-82	Physiologist, Pulmonary Function and Pulmonary Exercise Laboratories, Mt. Sinai Hospital, Montreal and St. Agathe (sanitorium) Divisions.
1982-87	Assistant Chief, Division of Pulmonary and Hyperbaric Medicine, South Miami Hospital.
1987-	Chief, Division of Pulmonary and Hyperbaric Medicine, South Miami Hospital.
1990-92	Chairman, Department of Medicine, South Miami Hospital.
1990-	Chairman, Medical Ethics Committee, South Miami Hospital.

Other Positions:

1967-77	Director, Montreal Health Press Inc. - a non-profit health education organization.
1976-78	Lecturer, Dawson College, Montreal, Emergency Medical Technician Training Program.
1981-82	Health Education Committee, Canadian Lung Association, Ottawa.
1984-85	Secretary-Treasurer, Florida Chapter, American College of

	Chest Physicians.
1985-86	President-Elect, Florida Chapter, American College of Chest Physicians.
1987-88	President, Florida Chapter, American College of Chest Physicians.
1989-	President, MedLogic Inc., a medical software company incorporated in the State of Florida.
1990-	Volunteer Expert Witness Program, Florida Board of Medicine, Department of Professional Regulation, Division of Regulation, State of Florida.

South Miami Hospital Committee Appointments

Peer Review Committee (chairman 1987)
Nutritional Support Committee (chairman 1986)
Intensive Care Committee
Education Committee (chairman 1988)
Utilization Review Committee
Pharmacy and Therapeutics Committee (chairman 1988-1990)
Medical Ethics Committee (founding chairman 1990-)

License to Practice Medicine:

1976	Quebec, General License (176523)
1982	Florida (039452)
1983	California (C-4127)
1984	New York (157718-1)

All above licenses active and in good standing

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Board Examinations:

1975	L.M.C.C. Canada (41479)
1979	F.R.C.P. Royal College of Physicians and Surgeons of Canada, Internal Medicine (3795)
1979	C.S.P.Q. Corporation Professionnelle des Medecins du Quebec, Internal Medicine (8611)
1979	American Board of Internal Medicine, Diplomate, Internal Medicine (73847)
1982	C.S.P.Q. Corporation Professionnelle des Medecins du Quebec, Pulmonary Medicine (9403)
1983	American Board of Internal Medicine, Subspecialty of Pulmonary Disease (73847)

Other Specialty Training

1983	Diplomate, U.S. Department of Commerce, National Oceanic and Atmospheric Administration (NOAA), Diving and Hyperbaric Medicine, Physicians Program IX.
1985	"B" Reader - Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, Appalachian Laboratory for Occupational Safety and Health.
1989	B" Reader re-certification, NIOSH.
1992	"B" Reader re-certification, NIOSH

Current Association Memberships:

American College of Physicians
American College of Chest Physicians
American Lung Association
American Medical Association
American Thoracic Society
Dade County Medical Association
Canadian Medical Association
Golden Circle Society (South Miami Hospital)
Quebec Medical Association
Royal College of Physicians and Surgeons (Canada)

Publications:

Birth Control Handbook. 1st edition 1968, 12th edition 1974, - 7 million copies in print

VD Handbook. 1st edition 1972, 3rd edition 1977 - 5 million copies in print

The Independent Medical Examination in Occupational Pulmonary Medicine, *in* The Eye of the Storm, Andrews Publications, 1992.

The Physiological Consequences of Pleural Plaques *in* On a Course to a New World: Asbestos Litigation After the Future Class Actions, Andrews Publications, 1994.

Asbestos Medicine on Trial, *with* Norwood Wilner. *in press*, pulication date April 1995.

Computer Programs

Coronary Artery Disease - Conditional Probability Analysis. Bayes Theorem application. Copyright Medlogic Inc., 1989.

EXCAL: An Artificial Intelligence Program for Differential Diagnosis in Cardiopulmonary Exercise Testing. (Commercial application), 1985.

COCAL: A Program for the Non-invasive Determination of Cardiac Output Using the SVO2

94918130

Swan-Ganz Catheter. Presented at the annual meeting of the Florida Chapter of the American College of Chest Physicians, 1986 and now in the public domain.

VO2ERR: A Program for the Determination of Errors in VO2 Measurements Using Gas Collection, 1986.

PFT-PREDICT: A Program for the Prediction of Normal Values in Pulmonary Function Testing, 1987.

Apportionment of Risk of Lung Cancer in Asbestos Workers. Computer model and statistical analysis. Copyright Medlogic Inc., 1990.

Blood Gas Data Base: An integrated blood gas-cardiac profile program for hospital pulmonary laboratories. Copyright Medlogic Inc., 1990.

Analysis of the Baltimore Cohort: Statistical analysis of database, 8500 plaintiffs. Analytical program Copyright Medlogic Inc., 1992.

DR. ALLAN FEINGOLD DEPOSITIONS 1993-1997					
	Date	Case or reference	Attorney	Subject	Jurisdiction
1	01/21/93	Bellows, Lawrence	Bunnell, Woulfe		Ft. Lauderdale
2	03/11/93	Hancock, Alfred	Adams & Reese	Asbestos	New Orleans
3	03/19/93	Blue, Harry	Ferraro	Asbestos	Miami
4	03/25/93	Similien, Samson	Allison, Robertson & Smith	Toluene diisocyanate	Miami
5	09/02/93	Hessler v. T&N	Lipman	Mesothelioma	Miami
6	11/12/93	Fink, Roy	Ferraro	Mesothelioma	Miami
7	11/19/93	Levine v. Rosenblum	Rossman & Baumberger	MedMal	Miami
8	11/23/93	Hall, Arthur	Walton, Lantaff, Schroeder	Perchlorethylene	Miami
9	12/01/93	Dikun, John	Baron & Budd	Asbestos	Dallas
10	12/11/93	Watson, Sterling	Brown, Terrell, Hogan	Asbestos	Jacksonville
11	12/13/93	Levine v. Rosenblum	Rossman & Baumberger	MedMal	Miami
12	03/02/94	Cabrera v. AAR Landing	Liberty Mutual Insurance	Spray painting	Ft. Lauderdale
13	03/16/94	Calvo v. Kelly Tractor	Walton Lantaff Schroeder	Workers' Comp	Miami
14	03/23/94	Maheuron, Harry	Marcos & Rothman		Miami
15	03/04/94	Westberry, Charles O.	Brown, Terrell, Hogan	Asbestos	Jacksonville

DR. ALLAN FEINGOLD DEPOSITIONS 1993-1997					
	Date	Case or reference	Attorney	Subject	Jurisdiction
16	05/04/94	Mendez v. KingCole	Miller, Kagan & Cait	Perchlorethylene	Miami
17	05/10/94	Ruibal v. OPI	Deutsch & Blumberg	Methacrylate	Atlanta
18	05/23/94	Lindbloom, Allen	Ratiner, Reyes & O'Shea	Asbestos	Miami
19	06/04/94	Galotti, Robert Stephen	Ferraro	Asbestos	Miami
20	08/03/94	Osborne v. Florida East Coast Railway	Brown & Gavin	Asbestos	Miami
21	08/18/94	Alsene v. Armstrong World	Heyl, Royster, Voelker	Asbestos	Peoria, IL
22	09/13/94	Tito v. Rodriguez, M.D.	Daryl L. Merl	MedMal	Miami
23	11/17/94	Chase Manhattan Bank v. T&N	Chase Manhattan Bank	Asbestos in buildings	NYC Federal
24	11/18/94	Chase Manhattan Bank v. T&N	Chase Manhattan Bank	Asbestos in buildings	NYC Federal
25	11/28/94	Loveland v. Telectronic Pacing	Miller, Kagan & Chait	Chemical exposure	Miami
26	01/09/95	Mickow v. Waste Management Pest Control	Walton, Lantaff Schroeder	Chemical exposure	Miami
27	01/12/95	Mickow v. Waste Management Pest Control	Flaster & Bates	Chemical exposure	Plantation
28	01/19/95	Lodati, Paul	Abrams, Anton, Robbins	Explosion	Hollywood

DR. ALLAN FEINGOLD DEPOSITIONS 1993-1997					
	Date	Case or reference	Attorney	Subject	Jurisdiction
29	03/21/95	Hernandez v. Rinker Materials	Levine, Busch, Schnepper	Chemical exposure	Miami
30	03/22/95	Redd, James	Lipman	Mesothelioma	Miami
31	04/04/95	Sorenson, Richard	Brayton, Gisvold & Harley	Asbestos	Superior Court of California
32	04/18/95	Valente v. Dade County School Board	Peters, Robertson, Lax	Occupational asthma	Miami
33	05/02/95	Haugen v. T&N et al	Schroeter, Goldmark	Asbestos	Seattle
34	05/20/95	Perez, Jorge	Marcos & Rothman		Miami
35	06/12/95	Jackson vs Jones Chemicals	Marcos & Rothman	Chlorine	Miami
36	07/28/95	Bates v. Mobil Oil	Marlow, Connell, Valerius	Chemical exposure	Miami
37	08/15/95	Laboy v. St. Mary's Hospital	Montgomery & Larmoyeux	MedMal	West Palm Beach
38	10/11/95	Perez v. Alberti-Flor	Stephens, Lynn, Klein	MedMal	Miami
39	11/14/95	Bull, John v. Holy Cross	Billing, Cochran, Heath	MedMal	Ft. Lauderdale
40	03/12/96	Linero v. ABF Freight	Pyszka, Kessler, Massey	Chemical exposure	Ft. Lauderdale
41	03/16/96	Osborne v. Florida East Coast Railway	Solms & Price	Asbestos	Miami

DR. ALLAN FEINGOLD DEPOSITIONS 1993-1997					
	Date	Case or reference	Attorney	Subject	Jurisdiction
42	04/12/96	Vermilion v. ABF Freight System	Arkansas Best Corporation	Chemical exposure	Fort Smith, Ak
43	06/25/96	Carter v. Brown and Williamson	Chadbourne & Parke	Tobacco Lung Cancer	Jacksonville
44	06/26/96	Carter v. Brown and Williamson	Chadbourne & Parke	Tobacco Lung Cancer	Jacksonville
45	08/21/96	Clark v. Liggett Group	Latham & Watkins	Tobacco Lung Cancer	Jacksonville
46	08/22/96	Clark v. Liggett Group	Latham & Watkins	Tobacco Lung Cancer	Jacksonville
47	08/29/96	Baron,Walter	Donna L. Harvey	Asbestos	Miami
48	10/03/96	Clark v. Liggett Group	Latham & Watkins	Tobacco Lung Cancer	Jacksonville
49	10/06/96	Urroz, Raul	Florida Workers' Comp	Workers' Comp	Daytona Beach
50	11/02/96	Francois, J v. DPC General Contractors	Conroy, Simberg & Ganon	Asbestos abatement	Hollywood
51	11/12/96	Prior v. Ferguson Electric	Zimmerman, Shuffield, Kiser	Asbestos	Orlando
52	12/20/96	Dearsman, Herbert L.	Brown, Terrell, Hogan, Ellis	Asbestos	Jacksonville
53	10/10/96	Connor v. RJRTC	Jones, Day, Reavis & Pogue	Tobacco Lung Cancer	Jacksonville

DR. ALLAN FEINGOLD DEPOSITIONS 1993-1997					
	Date	Case or reference	Attorney	Subject	Jurisdiction
54	10/11/96	Connor v. RJRTC	Jones, Day, Reavis & Pogue	Tobacco Lung Cancer	Jacksonville
55	01/30/97	Port Authority of NY & NJ v. W.R. Grace	Ness Motley	Asbestos in buildings	NYC Federal
56	01/31/97	Port Authority of NY & NJ v. W.R. Grace	Ness Motley	Asbestos in buildings	NYC Federal
57	02/13/97	Chutz v. Ligget & B&W	Latham & Watkins	Tobacco Lung Cancer	Jacksonville

Updated: January 21, 1997

ALLAN L. GOLDMAN, M.D.
CURRICULUM VITAE

ADDRESS: 12901 Bruce B. Downs Blvd., MDC Box 19
Tampa, Florida 33612

DATE OF BIRTH: June 3, 1943

PLACE OF BIRTH: Minneapolis, Minnesota

TELEPHONE NUMBERS: Residence: (813) 286-2979
Work: (813) 974-2271

MARITAL STATUS: Married: Wife - Barbara
Children - Lisa, Carrie, Jennifer, Lindsey

EDUCATION:

University of Minnesota, 1961 - 1964, B.A., B.S.

University of Minnesota Medical School, 1964 - 1968, M.D.

POSTGRADUATE TRAINING

Internship: Straight Medicine, Wadsworth VA Hospital - UCLA, Los Angeles, California, 1968 - 1969

Residency: Internal Medicine, Brooke General Hospital, San Antonio, Texas, 1969 - 1970

Fellowship: Pulmonary Disease, Walter Reed Army Medical Center, Washington, D.C., 1970 - 1972

LICENSURE:

State of Florida

CERTIFICATIONS:

Board Certified, American Board of Internal Medicine, October 1972, #33670

Board Certified, American Board of Internal Medicine, Subspecialty Board of Pulmonary Disease, October 1972, #33670

Certificate of Added Qualifications, Critical Care Medicine, November 1987

MILITARY STATUS:

Honorable Discharge, June 1974, Major, Medical Corps

HONORS AND AWARDS:

Phi Beta Kappa

Alpha Omega Alpha

ACADEMIC APPOINTMENTS:

Clinical Instructor of Medicine, University of Texas at San Antonio, Texas County Hospital, 1970

Clinical Instructor of Medicine, Georgetown University School of Medicine, 1972 - 1974

Assistant Professor of Medicine, Chief, Pulmonary Disease Section, Department of Internal Medicine, University of South Florida, College of Medicine, June 1974 - July 1976

Associate Professor of Medicine, Director, Division of Pulmonary Disease, Department of Internal Medicine, University of South Florida College of Medicine, July 1976 - August 1981

Chief, Pulmonary Disease Section, Medical Service, James A. Haley Veterans Hospital, 1974 - 1979

Professor of Medicine, Director, Division of Pulmonary Disease, Department of Internal Medicine, University of South Florida College of Medicine, September 1981 - 1982

Professor of Medicine, Director, Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, University of South Florida, College of Medicine, 1982 - 1987

CURRENT TITLES:

Professor of Medicine, Director, Division of Pulmonary, Critical Care and Occupational Medicine, Department of Internal Medicine, University of South Florida College of Medicine, 1987 - Present

Program Director, Pulmonary Disease Residency

Program Director, Critical Care Medicine Residency

Professor, Department of Environmental and Occupational Health, College of Public Health, University of South Florida, 1987 - Present

Acting Chairman, Department of Internal Medicine, December 1, 1994 - August 1996

Chairman, Department of Internal Medicine, August 1996 - Present

Chairman, Governing Body USF Dialysis Center, 1996 - Present

Chairman, Governing Body USF Endoscopy and Surgery Center. 1997 - Present

COMMITTEES AND ORGANIZATIONS:

1. National Program Committee, American Thoracic Society - American Lung Association, Scientific Assembly on Clinical Problems, 1975 - 1976

2. Medical Advisor, Respiratory Therapy Program, St. Petersburg Junior College, 1974 - 1979

3. Institutional Review Board (IRB), University of South Florida, 1974 - 1980; Chairman, 1980 - 1990

4. Chairman, Health Services Review Organization (HSRO), James A. Haley Veterans Hospital, Tampa, Florida, 1975 -1983

5. Hillsborough County Health Planning Commission, Task Force on Health Care of the Indigent, 1975 -1976

6. Chairman, Florida Lung Association Committee on Quality Care for Tuberculosis Patients (Advisory Committee to the State of Florida), 1975 - 1977

7. Committee on Respiratory Disease Consultation Services, Florida Lung Association, Florida Regional Medical Program, 1974 - 1977; Chairman, 1977 - 1979

8. Florida Thoracic Society, Committee on Respiratory Services, 1975 - 1977. Chairman, Subcommittee on Legislation, 1976

9. Medical Advisory Committee, Florida Regional Medical Program Project #314 Rehabilitation of Patients with Chronic Obstructive Pulmonary Disease, 1974 - 1976

10. Florida Lung Association, Department of Health and Rehabilitative Services Medical Advisory Committee to Task Force on the Care of COPD Patient, 1976 - 1979

11. Director-at-Large, Florida Lung Association, 1976 - 1989

12. Secretary-Treasurer, Florida Thoracic Society, 1976 - 1977

13. Vice President, Florida Thoracic Society, 1977 -1978

14. Steering Committee, VA Pulmonary Physicians Association, 1977 - 1978

15. Program Committee, Hillsborough County Medical Association, 1977 - 1979

16. Pharmacology, Ph.D. Dissertation Committee, University of South Florida College of Medicine, 1976

17. Chairman, Program Committee, VA Pulmonary Physicians Association, 1977

18. President, VA Pulmonary Physicians Association, 1978 - 1979

19. President-Elect, Florida Thoracic Society, 1978 - 1979

20. President-Elect, Florida Chapter, American College of Chest Physicians, 1978 - 1979

21. Florida Lung Association Program and Budget Committee, 1978 - 1982

22. Quality Assurance Committee, University Community Hospital, 1978 - 1981, 1984 - 1986

23. Chairman, Florida Lung Association Committee on Adult Lung Disease, 1978 - 1980, 1985 - 1989

24. Co-Chairman, Planning Committee, VA Cooperative Study on Outpatient Treatment of COPD, 1978

25. Chairman, University of South Florida College of Medicine, Biomedical Research Support Grant Committee, 1978 - 1985

26. President, Florida Thoracic Society, 1979 - 1980

27. President, Florida Chapter, American College of Chest Physicians, 1979 - 1980

28. VA Respiratory Diseases Central Office Advisory Committee, 1979 - 1982

29. Pulmonary Consultation Panel - Will Rogers Institute, 1979 - Present

30. Advisory Director, American Lung Association of Florida, 1979 - 1990

31. Chairman, Florida Lung Association Professional Education and Research Committee, 1980 - 1982, Member 1982 - Present

32. Credentials and Nominating Committee Representative, Florida Region, American College of Physicians, 1980 - 1988

33. Steering Committee, Section on Environmental Health, American College of Chest Physicians, 1980 -1984, Secretary 1984 - 1985

34. University of South Florida College of Medicine Research Committee, 1980 - 1982

35. Steering Committee - Tri-State Consecutive Case Conference, 1981 - 1982; Chairman, 1982 - 1983

36. Medical Director, ICU and Chairman, ICU Committee, Tampa General Hospital, 1982-Present

37. University of South Florida College of Medicine Space Committee, 1983 - 1985

38. Tampa General Hospital Monitoring and Infusion Pump Committees, 1983 - 1985

39. Subspecialty Prereviewer, Accreditation Council for Graduate Medical Education, 1984-Present

40. USF Cancer Hospital Development Planning Committee, 1984 - 1985

41. American Lung Association/American Thoracic Society Research Review Committee, 1984 - 1987

42. Chairman, USF Medical Clinics Code Committee, 1984 - 1988

43. Tampa General Hospital Practice Development Committee, 1985 - 1986

44. Chairman, ICU Committee, H. Lee Moffitt Cancer Center and Research Institute, 1985 - 1988

45. Board of Directors, University Medical Service Association (UMSA), 1985 - 1988, 1989 - 1990, 1993 - Present

46. Chairman, Urology Search Committee, 1985 - 1987

47. Bylaws Committee, Tampa General Hospital, 1985 - Present

48. Chairman, Referral Market Task Force, USF College of Medicine, 1987 - 1988

49. Chairman, Research Committee, H. Lee Moffitt Cancer Center and Research Institute, 1987 - 1989

50. Chairman, UMSA Investment Committee, 1987 - 1989; Finance Committee, 1989 - 1994

51. Chairman, UMSA Benefits Committee, 1987 - Present

52. ALAF Nominating Committee, 1987 - 1988

53. ALAF Governmental Affairs Committee, 1987 - 1988

54. H. Lee Moffitt Cancer Center and Research Institute, Public Affairs Committee, 1987 - 1988

55. American College of Chest Physicians Postgraduate Education Committee, 1987 - 1990

56. American College of Chest Physicians Bylaws Committee 1987 - 1989

57. American Thoracic Society Membership Committee, 1988 - 1990

58. USF Self-Study Committee C - Clinical Facilities, 1989

59. USF - Suzhou Medical College Joint Working Group, 1988 - 1990

60. Chairman, Search Committee, Hugh Culverhouse Chair in Swallowing Disorders, 1989 -1990

61. President, USF College of Medicine Faculty, 1990 - 1991

62. USF College of Medicine Executive Council, 1990 - 1991

63. Chairman, USF College of Medicine Dean Search Committee, 1990 - 1991

64. LCME Advisory Committee, USF College of Medicine 1990 - 1992

65. USF College of Medicine Appointment, Promotion and Tenure Committee - 1993 - Present

66. ACGME Board of Appeals, Pulmonary Disease, 1994 - Present

67. USF College of Medicine Strategic Planning Work group 1995 - Present

68. USF Area Health Education Center Advisory Committee, 1995 - Present

69. University Community Hospital Emeritus Staff, 1996 - Present

PROFESSIONAL MEMBERSHIPS:

Florida Thoracic Society

Fellow, American College of Physicians

Phi Delta Epsilon

Hillsborough County and Florida Medical Association

American Federation for Clinical Research

Fellow, American College of Chest Physicians

Florida Chapter, American College of Chest Physicians

American Medical Association

Society of Critical Care Medicine

Association of Professors of Medicine

MISCELLANEOUS:

Consulting Editor, The Journal of the Florida Medical Association, 1979 - 1981

Consultant Reviewer - JAMA

Consultant Reviewer - Chest

Book Reviewer, Archives of Internal Medicine

Consultant Review, Archives of Internal Medicine

Consultant Reviewer, Journal of the American Medical Association

Book Reviewer, Critical Care Medicine

Editor, Problems in Pulmonary Disease, 1986 - 1988

Contributor, American Board of Internal Medicine Recertifying Examinations in Pulmonary Disease and Critical Care Medicine

Updated September 16, 1996

ALLAN L. GOLDMAN, M.D.
BIBLIOGRAPHY

JOURNAL ARTICLES:

1. Goldman AL, Braman SS. Isoniazid: a review with emphasis on adverse effects. Chest 1972;62:71-7.

2. Schwarz MI, Goldman AL, Roycraft DW, Hunt KK. Vascular invasion by chondrosarcoma simulating pulmonary emboli. Am Rev Respir Dis 1972;106:109-13.

3. Goldman AL, Enquist RW. Methadone pulmonary edema. Chest 1973;63:275-6.

4. Goldman AL. Lung cancer in Bowen's disease. Am Rev Respir Dis 1973;108:1205-7.

5. Braman SS, Goldman AL, Schwarz MI. Steroid responsive hypercalcemia in disseminated bone tuberculosis. Arch Intern Med 1973;132:269-71.

6. Schwarz MI, Whitcomb ME, Goldman AL. The spectrum of diffuse pulmonary infiltrates in malignancy. Chest 1973;64:88-93.

7. Goldman AL, Enquist R. Hyperacute radiation pneumonitis. Chest 1975;67:613-5.

8. Goldman AL, Light LR. Anterior cervical infections as a complication of transtracheal aspiration. Am Rev Respir Dis 1975;111:707-8 (Letter to editor).

9. Goldman AL. Abnormalities of the respiratory and oxygen transport systems: hypothyroidism as a model. Comprehensive Therapy (by invitation) 1975;3:170-24.

10. Goldman AL, Halkias DG. Diagnostic smear of acid-fast bacilli. Ann Intern Med 1975;282 (Letter to editor).

11. Goldman AL. Cigar inhaling. Am Rev Respir Dis 1976;113:87-9.

12. Goldman AL. Smoking, carboxyhemoglobin levels, and oxygen therapy. Chest 1976;69:570 (Letter to editor).

13. Goldman AL. A new era for tuberculosis. J Fl Med Assoc 1976;63:278-80.

14. Whitcomb ME, Barham E, Goldman AL, Green DC. Indications for mediastinoscopy in bronchogenic carcinoma. Am Rev Respir Dis 1976;113:189-95.

15. Goldman AL, George J. Postural hypoxemia in quadriplegic patients. Neurology 1976;26:815-7.

16. Goldman AL. Carboxyhemoglobin levels in primary and secondary cigar and pipe smokers. Chest 1977;72:33-5.

17. Goldman AL. Rational use of bronchodilators. Hospital Formulary 1977;12:681-9.

18. Goldman AL. Respiratory distress: differential diagnosis and emergency management. Emergency Medical Services 1977;6:49-54.

19. Robbins H, Goldman AL. Failure of a prophylactic antimicrobial to prevent sepsis following fiberoptic bronchoscopy. Am Rev Respir Dis 1977;116:325-6.

20. Foster L, Corrigan K, Goldman AL. Effectiveness of oxygen therapy in hypoxic polycythemic smokers. Chest 1978;73:572-6.

21. Morrison DA, Goldman AL. Pulmonary function testing in Florida - what's available and how to use it. J Fl Med Assoc 1978;65:429-32.

22. Polson J, Krzanowski J, Goldman A, Szentivanyi A. Inhibition of human pulmonary phosphodiesterase activity by therapeutic levels of theophylline. Clin Exp Pharmacol Physiol 1978:5:535-9.

23. Behnke RH, Goldman AL, Solomon DA. What to do when you suspect thrombosis and embolism. Modern Medicine 1978;46:40-8.

24. Krzanowski J, Polson J, Goldman A, Ebel T, Szentivanyi A. Reduced adenosine 3'5' cyclic monophosphate levels in patients with reversible obstructive airways disease. Clin Exp Pharmacol Physiol 1979:6:111-5.

25. Robbins HM, Morrison DA, Sweet ME, Solomon DA, Goldman AL. Biopsy of the main carina: staging lung cancer with fiberoptic bronchoscopy. Chest 1979:75:484-6.

26. Goldman AL. Standardization of spirometry. CVP 1979;7:35-9.

27. Morrison D, Goldman AL. Roentgenographic patterns of drug induced lung disease. Radiology 1979;131:299-304.

28. Foster L, Goldman A, Trudeau W. Bronchodilator effects on gastric acid secretion. JAMA 1979;24:2613-15.

ALLAN L. GOLDMAN, M.D.
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Page Three

29. Goldman AL. Diagnosis and management of noncardiac pulmonary edema. Practical Cardiology 1980;6:55-64.

30. Ebel TA, Goldman AL, Molina JE. Postpneumonectomy shock. Chest 1980;77:787-8.

31. Rosser R, Goldman AL. Rational use of bronchodilators: an update. Hospital Formulary 1980;15:756-61.

32. Robbins HM, Sweet ME, Jefferson SE, Solomon DA, Goldman AL. The determination of resectability of lung cancer by fiberoptic bronchoscopy. Arch Intern Med 1981;141:649-50.

33. Rosser R, Solomon D, Goldman AL. Respiratory failure: when therapy is the problem. J Respir Dis 1980;1:35-41.

34. Goldman AL, Solomon DA, Behnke RH. Pulmonary embolism: pathogenesis, diagnosis, therapy. Primary Cardiology 1980;6:56-71.

35. Solomon DA, Goldman AL. Use of the lateral position test and perfusion lung scan in predicting mediastinal metastases. Chest 1981;79:406-9.

36. Lyman GH, Williams SC, Preston D, Goldman AL, et al. Lithium carbonate in patients with small cell lung cancer receiving combination chemotherapy. Am J Med 1981;70:1222-9.

37. Goldman AL, Morrison D, Foster L. Oral progesterone therapy: oxygen in a pill. Arch Intern Med 1981;141:574.

38. Polson JB, Krzanowski JJ, Goldman AL, Szentivanyi A. Cyclic nucleotide phosphodiesterase activity in patients with obstructive airways disease. Allergologia et Immunopathologia 1982;10(2):101-104.

39. Goldman AL. Emphysema - a study in flexibility and persistence. Therapaeia November 1981:32-42.

40. Rozas CJ, Goldman AL. Responses to bacterial pneumonia. Geriatrics 1982;37:61-66.

41. Rozas CJ, Goldman AL. Daily spirometric variability: normal subjects and subjects with chronic bronchitis with and without airflow obstruction. Arch Intern Med 1982;142:1287-91.

ALLAN L. GOLDMAN, M.D.
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42. Chandler KW, Rozas CJ, Kory RC, Goldman AL. Bilateral diaphragmatic paralysis complicating local cardiac hypothermia during open heart surgery. Am J Med 1984;77:243-249.

43. Karampelas D, Goldman AL. "Heparin resistance" in a patient with pulmonary embolus. Primary Cardiology 1984;10:124-27.

44. Goldman AL. Pulmonary exercise testing. In: Cardiology Clinics Vol 2, No 3, August 1984.

45. Ratliff WW, Goldman AL. Evaluation of disease exacerbation in COPD. Practical Cardiology 1984;11:109-129.

46. Peterson RT, Goldman AL. Symposium on pulmonary medicine: pulmonary embolism. Primary Care 1985;12:383.

47. Vaaler M, Goldman AL. Advances in diagnostic techniques in pulmonary medicine. Comprehensive Therapy 1986;12(7):28-35.

48. Murphy RL, Becklake MR, Brooks SM, Gaensler EA, Gee BL, Goldman AL, Kleinerman JL, Lewinsohn HC, Mitchell RS, Weill H. The diagnosis of nonmalignant diseases related to asbestos. Official statement of the American Thoracic Society. Am Rev Respir Dis 1986;134:363-368.

49. Morrison DA, Goldman AL. Oral progesterone treatment in chronic obstructive lung disease: failure of voluntary hyperventilation to predict response. Thorax 1986;41:616-19.

50. Goldman AL, McMeekin JO. Enigmatic fever in a young woman. Hosp Physician 1986;22(12):53-60.

51. Chandler KW, Goldman AL, Connar R. Laser therapy of primary lung cancer. J Fl Med Assoc 1986;73(1):25-28.

52. Alberts WM, Chandler KW, Solomon DA, Goldman AL. Pulmonary disease due to mycobacterium malmoense. Am Rev Respir Dis 1987;135(6):1375-1378.

53. Alberts WM, Goldman AL. The clinical use of methacholine bronchial challenge testing. South Med J 1987;80:827-830.

54. Goldman AL. Laser and radiation therapy to obstructed airways in lung cancer. Innovations in Oncology 1987;3:9-10.

55. Alberts WM, Goldman AL. Echocardiography in planned vena caval interruption. South Med J 1989;82(6):772-774.

56. Narain S, Smith DB, Goldman AL. High output heart failure - A case report. Clinical Interface; June 1989.

57. Walsh FW, Alberts WM, Solomon DA, Goldman AL. Malignant pleural effusions: pleurodesis using a small-bore percutaneous catheter. South Med J 1989;76:1019-1022.

58. Mitchell ME, Alberts WM, Chandler KW, Goldman AL. Intrapleural streptokinase in management of parapneumonic effusions: report of series and review of literature. J Fl Med Assoc 1989:76:1019-1022.

59. Goldman AL. Asbestos pleural disease. Pulmonary and Critical Care Update, 1990;5(22):1-8. Published by American College of Chest Physicians.

60. Alberts WM, Hammond MD, Colice GC, Goldman AL. The effect of mild hypoxia on bronchial responsiveness. Ann Allergy, 1990;65:189-193.

61. Alberts WM, Goldman AL, Leaverton P. Bronchodilator testing "confidence intervals" based on the level of bronchial responsiveness. Chest 1992;102:737-741.

62. Stockwell HG, Goldman AL, Lyman GH, et. al. Environmental tobacco smoke and lung cancer risk in nonsmoking women. J Natl Cancer Inst 1992;84:1417-1422.

63. Alberts WM, Ferris MC, Brooks SM, Goldman AL. The FEF 25-75% and the clinical diagnosis of asthma: Annals of Allergy 1994;73:221-225.

64. Rumbak MJ, Graves AE, Scott MP, Sporn GK, Walsh FW, Anderson WM, Goldman AL. Tracheostomy tube occlusion protocol predicts significant tracheal obstruction to airflow in patients requiring prolonged mechanical ventilation. Crit Care Med (in press)

65. Szentivanyi A, Goldman AL: Vagotonia and Bronchial Asthma. An editorial. Chest (in press)

BOOKS, AUDIOVISUAL PROGRAMS, ETC:

1. Goldman AL. Pulmonary evaluation series, Part I: spirometry. Medcom Famous Teachings in Modern Medicine, 1982.

2. Goldman AL. Pulmonary evaluation series, Part II: arterial blood gases. Medcom Famous Teachings in Modern Medicine, 1982.

3. Goldman AL. Pulmonary embolism. Audio Digest (Family Practice), 1990;38-43.

JOURNAL ABSTRACTS:

1. Goldman AL, Joyner LR. Reversible abnormalities of oxygen exchange in hypothyroidism. Clin Res 1975;12:33A.

2. Goldman AL. Carboxyhemoglobinemia and oxygen desaturation in cigar smokers. Am Rev Respir Dis 1975;111:942.

3. Goldman AL, Foster L, Shannon R. Smoking and hypoxemic ventilatory response. Am Rev Respir Dis 1976;113:120.

4. Goldman AL. carboxyhemoglobin levels in primary and secondary cigar and pipe smokers. Am Rev Respir Dis 1976;113:141.

5. Foster L, Goldman AL, Singer P, Shires D. Hypoxemic ventilatory response in uremic patients with neuropathy. Am Rev Respir Dis 1976;113:196.

6. Foster L, Corrigan K, Goldman AL. Oxygen therapy in secondary polycythemia. Chest 1976;70:426.

7. Corrigan K, Goldman AL, Pollett R, Garms P. Hemoglobin affinity for oxygen in hypothyroidism. Clin Res 1977;26:12A.

8. Goldman AL, Ebel TA, Krzanowski JJ, Polson JB. Szentivanyi A. Lung cyclic nucleotides in bronchospastic and nonreversible obstructive lung disease. Am Rev Respir Dis 1977;115(Part 2):330.

9. Ebel TA, Foster L, Goldman AL. Inhaled terbutaline, isoproterenol, freon and cardiac ectopy. Am Rev Respir Dis 1977;115(Part 2):103.

10. Krzanowski JJ, Polson JB, Goldman AL, Ebel TA, Szentivanyi A. Cyclic nucleotide levels in human lung from patients with bronchospastic airway disease. Pharmacologist 1977;19:203.

11. Polson JB, Krzanowski JJ, Goldman AL, Ebel TA, Szentivanyi A. A cyclic nucleotide phosphodiesterase (PDE) activity in patients with reversible and nonreversible airways obstruction. Adv Cyclic Nucleotide Research 1978;9.

12. Polson JB, Krzanowski JJ, Goldman AL, Ebel TA, Szentivanyi A. Inhibition of human pulmonary phosphodiesterase by therapeutic levels of theophylline. Pharmacologist 1977;19:203.

13. Foster L, Trudeau W, Goldman AL. Bronchodilator effects on gastric acid secretion. Chest 1977;72:399.

14. Robbins H, Morrison D, Jefferson S, Ebel T, Sweet M, Foster L, Solomon D, Goldman A. Main carinal biopsy through the fiberoptic bronchoscope for staging lung cancer. Am Rev Respir Dis 1978;117(4)(Part 2):169.

15. Morrison D, Kory R, Goldman A. The step exercise test in interstitial lung disease. Chest 1978;74:345.

16. Foster L, Goldman A, Trudeau W. Oral bronchodilator effects on gastric acid secretion. Chest 1978;74:337.

17. Phillips JW, Goldman AL, Lockey RF. Bahia grass pollen - an important aeroallergen. J Allergy Clin Immunol 1979;63:192-3.

18. Jefferson S, Lee E, Goldman AL. Diagnosis of sarcoidosis by sputum cytology. Am Rev Respir Dis 1979;119:131.

19. Morrison DA, Goldman AL. Oral progesterone therapy in COPD. Am Rev Respir Dis 1979;119:154.

20. Goldman AL. Influence of tracing selection technique on daily variation of FEV_1 and FVC. Am Rev Respir Dis 1979;119:218.

21. Morrison D, Solomon DA, Goldman AL. P_VO_2 and wedge pressure: predicting survival in acute respiratory failure. Chest 1979;76:361.

22. Solomon DA, Foster LJ, Goldman AL. The lateral position test and perfusion lung scan in detecting mediastinal metastases. Chest 1979;76:367.

23. Ebel TA, Goldman AL. Ectopic arrhythmias in patients with COPD and acute respiratory failure. Am Rev Respir Dis 1980;121:129.

24. Solomon DA, Goldman AL, Sokol J, Gordy L, Starr A. Accuracy of cytology in diagnosing small cell lung cancer. Am Rev Respir Dis 1980;121:193.

25. Engle WK, Prockop LD, Askansas V, Engle G, Hutchison R, Galdi AP, Williams JW, Goldman A, Foster L. Nearly-fatal lipid laden myopathy with myoglobinuria and myodeficiency of carnitine, and cultured muscle dependence on carnitine. Neurology 1980;30:368.

26. Howard TP, Solomon DA, Germain B, Goldman AL. Clinically silent pulmonary hypertension in the CREST syndrome. Chest 1980;78:522.

27. Solomon DA, Goldman AL, Gordy L, Ruffolo E, Sokol J, Starr A. Accuracy of cytology in diagnosing lung cancer. Chest 1980;78:541.

28. Kirkman L, Goldman AL, Krzanowski J, et al. Enhanced in vitro sensitivity to propranolol induced beta-adrenergic blockade in COPD patients. Am Rev Respir Dis 1981;123(2):79.

29. Rozas CJ, Goldman AL. Daily spirometric variability in normal subjects and subjects with chronic bronchitis. Am Rev Respir Dis 1982;125(2):95.

30. Chandler KW, Rozas CJ, Kory RC, Goldman AL. Bilateral diaphragmatic paralysis complicating local cardiac hypothermia during open heart surgery. Am Rev Respir Dis 1982;125(2):98.

31. Corrigan KC, Brady P, Foster LJ, Goldman AL. Effect of various theophylline preparations on gastric acid secretion. Am Rev Respir Dis 1982;125(2):123

32. Karampelas DT, Goldman AL. Diagnostic utility of fiberoptic bronchoscopy in "lung abscess." Am Rev Respir Dis 1984;129(2):55.

33. Mitchell ME, Avery JA, Alberts WM, Goldman AL. Drainage of pleural effusions with a percutaneous pneumothorax catheter. Chest 1986;89S;482S.

34. Alberts WM, Vaaler MA, Goldman AL. Slope of the dose response curve to inhaled methacholine in normal and asthmatic subjects. Chest 1986;89S:499S.

35. Alberts WM, Wolfe KW, Schwab KS, Goldman AL. The threshold concentration and the slope of the dose response curve in the interpretation of bronchial provocation tests. Am Rev Respir Dis 1987;135:A44.

36. Ferris MC, Holman Ferris S, Alberts WM, Walsh FW, Goldman AL. Small-bore chest tube drainage and pleurodesis using tetracycline, bleomycin or doxycycline in pleural effusions associated with malignancy. Am Rev Respir Dis 1994;A169.

37. Rumbak MJ, Graves AE, Scott MP, Sporn GK, Walsh FW, Anderson WM, Goldman AL. Tracheostomy tube occlusion protocol predicts succesful tracheal decanulation following long term mechanical ventilation. Crt Care Med 1995; 23:A11B (supplement)

PRESENTATIONS:

1. Goldman AL. Prevention of Transmission of Tuberculosis Infection, Modern Clinical Management of Tuberculosis. Orlando, Florida, 1974, Sponsored by Florida Bureau of TB Control.

2. Goldman AL, Joyner LR. Reversible Abnormalities of Oxygen Exchange in Uncomplicated Hypothyroidism. Southern Section of American Federation for Clinical Research, New Orleans, 1975.

3. Goldman AL, Course Director, AMA Regional Course, Pulmonary Function and Blood Gases. March 1975, Host Airport Hotel, Tampa, Florida.

4. Goldman AL, Carboxyhemoglobinemia and Oxygen Desaturation in Cigar Smokers. American and Canadian Thoracic Society Joint Meeting, Montreal, May 21, 1975.

5. Goldman AL. Use of Corticosteroids in the Adult Respiratory Distress Syndrome. Florida Medical Association Annual Meeting, Section on Chest Medicine, April 24, 1975.

6. Goldman AL. Modern Treatment of Tuberculosis. Florida Lung Association Physician Seminar on Respiratory Disease, May 9-11, 1975, Jacksonville, Florida.

7. Goldman AL. Moderator on "Respiratory Problems in Hospitalized Patients." Florida Lung Association Physician Seminar on Respiratory Disease, May 9-11, 1975, Jacksonville, Florida.

8. Goldman AL. American Medical Association Special Pulmonary Function Panel. AMA Annual Meeting, Atlantic City, New Jersey, June 6-18, 1975.

9. Goldman AL. Transmission of Tuberculosis--Fact and Fantasy. Dade-Monroe Lung Association Seminar on "Tuberculosis and the Community Hospital," Miami, Florida, May 6, 1975. Published in April 1976 J Fl Med Assoc.

10. Goldman AL. Blood Gas and Acid Base Changes in Acute Respiratory Diseases. American Association of Physicians Assistant's Annual Meeting, July 26, 1975, Breckenridge Hotel, St. Petersburg, Florida.

11. Goldman AL. Transmission and Epidemiology of Tuberculosis. Workshop for Health Professionals on Modern Treatment of Tuberculosis in the Community. American Lung Association of Southeast Florida, West Palm Beach, Florida, September 17, 1975.

12. Goldman AL. Pulmonary Embolism. Orange Memorial Hospital Grand Rounds, Orlando, Florida, October 2, 1975.

13. Goldman AL. Pulmonary Embolism. Halifax Medical Center Grand Rounds, Daytona Beach, Florida, November 7, 1975.

14. Goldman AL. Transmission and Epidemiologic Aspects of Tuberculosis and Hospital Management of Tuberculosis. November 18, 1975 at Understanding TB Care, Cocoa Beach, Florida, sponsored by the American Lung Association - Spaceport Area.

15. Goldman AL. Respiratory Aspects of Neurologic Emergencies at Emergency Neurology II. University of South Florida College of Medicine, November 19, 1975, Tampa, Florida.

16. Goldman AL. Grand Rounds on Carboxyhemoglobin. Visiting Professor, University of Florida, Gainsville, Florida, December 11, 1975.

17. Goldman AL. Panel discussion, Pulmonary Problems in Cardiovascular Disease. 18th Annual Cardiovascular Seminar, Hillsborough County and Suncoast Heart Association, Sheraton-Sand key Hotel, Clearwater Beach, Florida, January 31, 1976.

18. Goldman AL. Physical Diagnosis of Respiratory Patients. 2nd Annual Florida Society of Respiratory Therapy Seminar on Clinical Applications of Respiratory Therapy, International Inn, Tampa, Florida, April 10, 1976.

19. Goldman AL. Management of Drowning. American College of Surgeons Annual Suncoast Trauma Seminar, Holiday Inn Central, Tampa, FL March 20, 1976.

20. Goldman AL. Course Director, University of South Florida Postgraduate Course on "Diagnosis and Management of Airways Disease," USF College of Medicine including talk on Bronchodilators;" Moderator of Sessions on "Diagnosis of Airways Disease" and "Outpatient Management of Airways Disease," March 28-29, 1976.

21. Goldman AL. Respiratory Distress: Differential Diagnosis and Emergency Management. The 1976 Southeast Emergency Medicine Congress, Fairmont Colony Square Hotel, Atlanta, Georgia, May 3, 1976.

22. Goldman AL. Co-Chairman, ATS Scientific Assembly on Clinical Problems, Sugarloaf Follow-up Session. American Thoracic Society Annual Meeting, New Orleans, Louisiana, May 18, 1976.

23. Goldman AL. Chairman, Symposium - ATS Scientific Assembly on Clinical Problems, American Thoracic Society Annual Meeting, "Newer Techniques in the Diagnosis of Pulmonary Embolism." New Orleans, Louisiana, May 19, 1976.

24. Goldman AL. Carboxyhemoglobin Levels in Primary and Secondary Cigar and Pipe Smokers. American Thoracic Society Annual Meeting, New Orleans, May 17, 1976.

25. Goldman AL. Metabolic Acidosis in Acid Base Problems in Pulmonary Disease (panel). American College of Chest Physicians Annual Meeting, October 26, 1976, Atlanta, Georgia.

26. Goldman AL. Moderator, Scientific Papers Selected Reports. American College of Chest Physicians Annual Meeting, October 28, 1976, Atlanta, Georgia.

27. Foster L, Corrigan K, Goldman AL. Oxygen Therapy in Secondary Polycythemia. American College of Chest Physician's Annual Meeting, October 28, 1986, Atlanta, Georgia.

28. Goldman AL. Rational Use of Bronchodilators at Internal Medicine Update 1977. Orlando, Florida, January 19, 1977.

29. Goldman AL. Course Director, AMA Regional Course, Pulmonary Function and Blood Gases. March 6, 1977, Innisbrook, Florida.

30. Goldman AL. Management of Drowning. American College of Surgeons Annual Trauma Seminar, Tampa, Florida, March 11, 1977.

31. Goldman AL. Course Director, USF Postgraduate Course on Pulmonary Infection and Pulmonary Infarction. USF, March 26-27, 1977.

32. Goldman AL. Newer Techniques in the Early Diagnosis of Lung Cancer. Mueller Surgical Society, USF, May 13, 1977.

33. Goldman AL. Bronchodilators - New Concepts. Pediatrics Grand Rounds, All Children's Hospital, St. Petersburg, Florida, July 8, 1977.

34. Krzanowski JJ, Polson JB, Goldman AL, Ebel TA, Szentivanyi A. Cyclic Nucleotide Levels in Human Lung from Patients with Bronchospastic Airway Disease. Presented Third International Conference on Cyclic Nucleotides, New Orleans, Louisiana, July 1977.

35. Polson JB, Krzanowski JJ, Goldman AL, Ebel TA, Szentivanyi A. Cyclic Nucleotide Phosphodiesterase (PDE) Activity in Patients with Reversible and Nonreversible Airways Obstruction. Presented Third International Conference on Cyclic Nucleotides, New Orleans, Louisiana, July 1977.

36. Foster L, Trudeau W, Goldman AL. Bronchodilator Effects on Gastric Acid Secretion. American College of Chest Physicians. Las Vegas, Nevada, November 2, 1977.

37. Goldman AL. Treatment of Pulmonary Emboli (Course presentation). American Medical Association Midwinter Meeting, Miami, Florida, December 11, 1977.

38. Goldman AL. Pulmonary Embolism - Treatment Update. USF Internal Medicine Update 1978, Orlando, Florida, February 15, 1978.

39. Goldman AL. Near Drowning. American College of Surgeons Annual Trauma Seminar, March 10, 1978.

40. Goldman AL. Early Detection of Lung Cancer. University of South Florida Postgraduate Course on Lung Cancer - Update, January 1978.

41. Robbins H, Morrison D, Jefferson S, Ebel T, Sweet M, Foster L, Solomon D, Goldman AL. Main Carinal Biopsy through the Fiberoptic Bronchoscope for Staging Lung Cancer. Presented at American Thoracic Society Annual Meeting, Boston, Massachusetts, May 17, 1978.

42. Goldman AL. Standardization of Spirometric Techniques. 1978 Annual Meeting, Florida Cardiopulmonary Association, Ft. Lauderdale, Florida, May 26, 1978.

43. Goldman AL. Physiology of the Upper and Lower Airways. Geigy Symposium. Asthma: A Practical Clinical Update. St. Petersburg, Florida, June 9, 1978.

44. Goldman AL. Ten Consecutive Cases of Clinically Significant Pulmonary Embolus Missed by Conventional Methods of Diagnosis. 24th Tri-State Consecutive Case Conference. Sea Island, Georgia, September 8, 1978.

45. Goldman AL. Early Detection of Lung Cancer and Near Drowning. 8th Annual Cardiopulmonary Seminar, Sun Coast Hospital, Largo, Florida, October 7-8, 1978.

46. Foster L, Goldman AL, Trudeau W. Oral Bronchodilator Effects on Gastric Acid Secretion. American College of Chest Physicians, Annual Meeting, Washington, D.C., October 31, 1978.

47. Morrison D, Kory R, Goldman AL. The Step Exercise Test in Interstitial Lung Disease. American College of Chest Physicians, Annual Meeting, Washington, D.C., November 1, 1978

48. Goldman AL. *Health Effects of Air Pollution. Hillsborough Community College* Air Quality Workshop, November 16, 1978.

49. Goldman AL. Standardization of Spirometry. National Society of Cardiopulmonary Technologists, 4th Annual Fall Seminar, St. Petersburg Beach, Florida, December 8, 1978.

50. Goldman AL. Control of Breathing. 1st Annual Walt Disney World Florida Thoracic Society Pulmonary Wintercourse, Orlando, Florida, January 15, 1979.

51. Goldman AL. Interpretation of Arterial Blood Gases. 1st Annual Walt Disney World Florida Thoracic Society Pulmonary Wintercourse, Orlando, Florida, January 15, 1979.

52. Goldman AL. Course Director, American Medical Association Regional Course on "Blood Gases and Pulmonary Function Clinical Evaluation." Innisbrook, Florida, February 23, 1979.

53. Goldman AL. Acute Respiratory Failure in Neuromuscular Disease. USF Postgraduate Course on "Respiratory Emergencies", Tampa, Florida, March 24, 1979.

54. Goldman AL. Inhalation of Noxious Gases, Mists and Fumes. USF Postgraduate Course on "Respiratory Emergencies", Tampa, Florida, March 25, 1979.

55. Phillips JW, Goldman AL, Lockey RF. Bahia Grass - An Important Aeroallergen. American Academy of Allergy Annual Meeting, New Orleans, Louisiana, March 1979.

56. Goldman AL. Lung Function Changes in Asthma. Astra Asthma Symposium. Miami Beach, Florida, April 23, 1979.

57. Goldman AL. Influence of Tracing Selection Technique on Daily Variation of FEV1 and FVC. American Thoracic Society Annual Meeting, Las Vegas, Nevada, May 14, 1979.

58. Jefferson S, Lee E, Goldman AL. Diagnosis of Sarcoidosis by Sputum Cytology. American Thoracic Society Annual Meeting, Las Vegas, Nevada, May 16, 1979.

59. Morrison DA, Goldman AL. Oral Progesterone Therapy in COPD. American Thoracic Society Annual Meeting, Las Vegas, Nevada, May 16, 1979.

60. Goldman AL. Pathophysiology of Asthma. FMA Section on Allergy, FMA Annual Meeting, Miami, Florida, May 26, 1979.

61. Goldman AL. Respiratory Insufficiency. FMA Section on Allergy, FMA Annual Meeting, Miami, Florida, May 26, 1979.

62. Goldman AL. Moderator FMA Section on Chest Medicine, FMA Annual Meeting, Miami, Florida, May 27, 1979.

63. Goldman AL. Grand Rounds on Asthma: Update and Visiting Professor. University of Florida, Gainsville, Florida, June 7-8, 1979.

64. Goldman AL. Grand Rounds on Asthma and Visiting Professor. Southwest Michigan Area Education Council, Borgess Hospital, Kalamazoo, Michigan, October 24, 1979.

65. Goldman AL. Respiratory Failure. Florida Academy of Physicians Assistants Fifth Annual Conference, Orlando, Florida, November 2, 1979.

66. Goldman AL. Laboratory Diagnosis of the Patient with Pneumonia at Algorithms in the Laboratory Diagnosis of Infectious Disease. Association for Practitioners in Infection Control, Tampa, Florida, November 9, 1979.

67. Morrison D, Solomon DA, Goldman AL. P_vO_2 and Wedge Pressure: Predicting Survival in Acute Respiratory Failure. American College of Chest Physicians 45th Annual Scientific Assembly, Houston, Texas, November 5, 1979.

68. Solomon DA, Foster LJ, Goldman AL. The Lateral Position Test and Perfusion Lung Scan in Detecting Mediastinal Metastases. American College of Chest Physicians 45th Annual Scientific Assembly, Houston, Texas, November 7, 1979.

69. Goldman AL. Pulmonary Embolus, Respiratory Failure. University of South Florida College of Medicine Family Medicine Review Course, Tampa, Florida, November 29, 1979.

70. Goldman AL. Oxygen Transport and Acid Base Balance. 2nd Annual Florida Thoracic Society Wintercourse. Disney World, Orlando, Florida, January 1980.

71. Goldman AL. Asthma. Bay Pines VA Medical Grand Rounds, St. Petersburg, Florida, January 29, 1980.

72. Goldman AL. Update on COPD. The Florida Academy of Family Physicians "Family Practice Weekend", Tampa, Florida, February 9, 1980.

73. Goldman AL. Arterial Blood Gases. AMA Regional Course, Tarpon Springs, Florida, March 1, 1980.

74. Goldman AL. Arterial Blood Gases: Basic Physiology and Clinical Interpretation. USF Postgraduate Course on Critical Pulmonary Care, Tampa, Florida, March 8, 1980.

75. Goldman AL. Drowning. American College of Surgeons Annual Trauma Seminar, Tampa, Florida, March 13, 1980.

76. Goldman AL. Pneumonia. Medical Grand Rounds. Sarasota Memorial Hospital, Sarasota, Florida, March 14, 1980.

77. Goldman AL. Chairman of Seminar, "Search for Early Lung Cancer". The Third World Congress on Bronchoesophagology. Palm Beach, Florida, April 10, 1980.

78. Goldman AL. Co-Chairman of Seminar, "Hemoptysis". The Third World Congress on Bronchoesophagology. Palm Beach, Florida, April 11, 1980.

79. Goldman AL. Moderator. "Current Concepts: Pulmonary Hypertension and Pulmonary Edema". Florida Medical Association Annual Meeting - Subspecialty Session on Chest Medicine. Hollywood, Florida, May 8, 1980.

80. Goldman AL. Grand Rounds on Smoking and Visiting Professor. Maricopa County Hospital and St. Lukes Medical Center. Phoenix, Arizona, May 13, 1980.

81. Goldman AL. Grand Rounds and Visiting Professor. Loma Linda, California, May 14-15, 1980.

82. Goldman AL. Visiting Professor. Los Angeles VA Hospital (Wadsworth). Los Angeles, California, May 16, 1980.

83. Goldman AL. Lung Function Changes in Asthma. Astra Pulmonary Symposium, 8th Annual Physician Assistants' Conference. New Orleans, Louisiana, May 26, 1980.

84. Ebel TA, Goldman AL. Ectopic Arrhythmias in Patients With COPD and Acute Respiratory Failure. American Thoracic Society Annual Meeting, May 1980, Washington, D.C.

85. Goldman AL. Arterial Blood Gases: Basic Physiology, Clinical Patterns and Interpretations. Third Annual Acute Pulmonary Care Seminar, June 14, 1980, Sandpiper Bay Resort Convention Center, Port St. Lucie, Florida

86. Goldman AL. Pulmonary Aspects in the Care of Elderly Psychiatric Patient, Psychiatry Grand Rounds. James A. Haley VA Hospital, Tampa, Florida, June 18, 1980.

87. Goldman AL. Co-Director, Pulmonary Disease Section, MKSAP V Review Course, American College of Physicians, June 20, 1980, Orlando, Florida.

88. Engle WK, Prockup LD, Askansas V, Engle G, Hutchinson R, Galdi AP, Williams JW, Goldman AL, Foster L. Nearly-Fatal Lip Laden Myopathy with Myoglobinuria and

Myodeficiency of Carnitine: Prednisone-Failure but Dramatic Improvement with Carnitine, and Cultured Muscle Dependence on Carnitine. American Academy of Neurology 32nd Annual Meeting, May 1, 1980, New Orleans, Louisiana.

89. Goldman AL. Near Drowning. Focus on Trauma III. Memorial Hospital of Jacksonville, September 27, 1980, Jacksonville, Florida.

90. Goldman AL. Bronchodilator Therapy. Twelfth Annual Joint Meeting, American College of Physicians Regional Meeting and Florida Society of Internal Medicine, October 12, 1980, Orlando, Florida.

91. Solomon DA, Goldman AL, Gordy L, Ruffolo E, Sokol J, Starr A. Accuracy of Cytology in Diagnosing Lung Cancer. American College of Chest Physicians 46th Annual Scientific Assembly, October 27, 1980, Boston, Massachusetts.

92. Howard TP, Solomon DA, Germain B, Goldman AL. Clinically Silent Pulmonary Hypertension in the Crest Syndrome. American College of Chest Physicians 46th Annual Scientific Assembly, October 28, 1980, Boston, Massachusetts.

93. Goldman AL. Consultant (Visiting Professor), Scott Air Force Base for "Seminar in the Practical Management of Pulmonary Disease". Includes Presentations on "Carboxyhemoglobin", "Bronchodilators", "Pulmonary Edema", "Pleural Effusion", and "Interstitial Lung Disease", November 17-19, 1980, Scott Air Force Base, Illinois.

94. Goldman AL. Blood Gas Interpretation. 3rd Annual Florida Thoracic Society Wintercourse, Disney World, Orlando, Florida, January 15, 1981.

95. Goldman AL. Moderator on session in "Environmental Lung Disease" and presentation on "Occupational Lung Disease", 3rd Annual Florida Thoracic Society Wintercourse, Disney World, Orlando, Florida, January 18, 1981.

96. Goldman AL. Pulmonary Function Testing. Grand Rounds, Orlando Regional Medical Center, January 22, 1981.

97. Goldman AL. Visiting Professor, Cambria-Somerset Council for Education of Health Professionals, Inc. University of Pittsburgh at Johnstown, PA, February 23-25, 1981. Included lectures on: Noncardiac Pulmonary Edema, Occupational Lung Disease Pulmonary Function Testing, Pulmonary Embolism, Asthma Update.

98. Goldman AL. University of South Florida College of Medicine Postgraduate Course on "Controversies in Critical Pulmonary Care", moderator on "Mechanical Ventilation/Respiratory Therapy" and "Pulmonary Infiltrates in the Immunosuppressed Host"; discusser on "Pneumonia: Diagnosis and Management" and "Pulmonary Thromboembolic Disease". Don Cesar Beach Resort, St. Petersburg, Florida, March 21-22, 1981.

99. Goldman AL. Update - The Mechanical Management of the Patient with Pulmonary Trauma. Section on Thoracic and Cardiovascular Surgery, Florida Medical Association's 107th Annual Meeting, May 1, 1981, Hollywood, Florida.

100. Goldman AL. Pulmonary Complications in the Traumatized Patient. Section on Surgery, Florida Medical Association's 107th Annual Meeting, May 2, 1981, Hollywood, Florida.

101. Kirkman L, Goldman AL, Krzanowski J, et al. Enhanced In Vitro Sensitivity to Propranolol Induced Beta-Adrenergic Blockade in COPD Patients. American Thoracic Society Annual Meeting, Detroit, Michigan, May 12, 1981.

102. Goldman AL. Occupational Lung Disease, Asthma, Topics in Pulmonary Pathology. Indian Path Hospital, May 19, 1981, Kingsport, Tennessee.

103. Goldman AL. Management of Acute Respiratory and Near Drowning. Sun Coast Hospital Emergency Medicine Seminar, June 5, 1981, Clearwater Beach, Florida.

104. Goldman AL. Pneumonia: Diagnosis and Management, Arterial Blood Gases Simplified. Lawnwood Medical Center 4th Annual Review of Medicine and Surgery, July 3, 1981, Ft. Pierce, Florida.

105. Goldman AL. Course Director, Energy, Air Pollution and Health. University of South Florida College of Medicine, October 10, 1981, Tampa, Florida.

106. Goldman AL. Course Director, Pulmonary Function And Blood Gases. AMA Clinical Update Meeting, November 1, 1981, Sarasota, Florida.

107. Goldman AL. A Practical Approach to Blood Gases and Acid Base Balance. Florida Thoracic Society 4th Annual Pulmonary Wintercourse, January 21, 1982, Orlando, Florida.

108. Goldman AL. Pulmonary Thromboembolism and Pulmonary Rehabilitation. Second Annual Samson Symposium, Gateway Community Hospital, February 20, 1982, St. Petersburg, Florida.

109. Goldman AL. Pulmonary Function Testing and the Assessment of Impairment. University of South Florida College of Medicine 1982 Physicians' Postgraduate Course on Occupational and Environmental Lung Disease, March 21, 1982, Tampa, Florida.

110. Goldman AL. Respiratory Abnormalities in Cystic Fibrosis. Cystic Fibrosis Seminar, All Children's Hospital, April 10, 1982, St. Petersburg, Florida.

111. Rozas CJ, Goldman AL. Daily Spirometric Variability in Normal Subjects and Subjects with Chronic Bronchitis. American Thoracic Society Annual Meeting, May 18, 1982, Los Angeles, California.

112. Chandler KW, Rozas CJ, Kory RC, Goldman AL. Bilateral Diaphragmatic Paralysis Complicating Local Cardiac Hypothermia During Open Heart Surgery. American Thoracic Society Annual Meeting, May 18, 1982, Los Angeles, California.

113. Goldman AL. Co-Moderator - Clinical Focus on Bronchodilators. October 22, 1982, Atlanta, Georgia.

114. Goldman AL. Job Placement of persons with Chronic Respiratory Disorders. 42nd Annual AMA Congress on Occupational Health, November 18, 1982, Tampa, Florida.

115. Goldman AL. Visiting Professor, University of South Carolina School of Medicine, December 16-17, 1982, Columbia, South Carolina.

116. Goldman AL. Assessment of Disability. Fifth Annual Florida Thoracic Society Wintercourse, January 23, 1983, Orlando, Florida.

117. Goldman AL. Preoperative Pulmonary Evaluation for Lung Cancer. Third Annual Treasure Coast Medical-Surgical Review, February 5, 1983, Dodgertown Conference Center, Vero Beach, Florida.

118. Goldman AL. Evaluating Pulmonary Fitness. American Medical Tennis Association Scientific Assembly, February 28, 1983, Saddlebrook-Wesley Chapel, Florida.

119. Goldman AL. Ventilation Perfusion Scans and Office Pulmonary Function Testing. 1983 Physicians Postgraduate Course: Diagnostic Techniques in Pulmonary Medicine, March 26-27, 1983, University of South Florida College of Medicine, Tampa, Florida.

120. Goldman AL. Occupational Lung Disease. Third Annual Osteopathic Occupational Medicine Symposium, April 6, 1983, Bay Harbor Inn, Tampa, Florida.

121. Goldman AL. Pharmacologic Therapy of COPD and Pulmonary Function Testing in COPD. Fourth Annual Borgess Medical Center Pulmonary Rehabilitation Symposia, April 7, 1983, Kalamazoo, Michigan.

122. Goldman AL. COPD, Medical Grand Rounds, Bay Pines VA Medical Center, April 12, 1983, St. Petersburg, Florida.

123. Goldman AL. Physical Assessment vs. High-Technology Respiratory Monitoring. Florida Society for Respiratory Therapy Spring Seminar, April 29, 1983, Tampa, Florida.

124. Goldman AL. Management of Respiratory Failure, Florida Society of Critical Care Medicine First Annual Meeting, June 11, 1983, Grenelefe, Florida.

125. Goldman AL. Office Spirometry in Chronic Obstructive Pulmonary Disease, 68th Scientific Assembly of Interstate Postgraduate Medical Association, November 2, 1983, Miami Beach, Florida.

126. Goldman AL. Pharmacologic Therapy of COPD and Occupational Lung Disease. Sun Coast Hospital 12th Annual Cardiopulmonary Seminar, November 4, 1983, Clearwater Beach, Florida.

127. Goldman AL. ACP MKSAP VI. Review Course - Pulmonary Section, November 7, 1983, Orlando, Florida.

128. Goldman AL. Hemodynamic Data and Primary Pleuropulmonary Causes of Pulmonary Hypertension. Pulmonary Hypertension Course Sponsored by American Heart Association of Central Florida, November 11, 1983, Lake Buena Vista, Florida.

129. Goldman AL. Moderator of Session on Pulmonary Infections at 6th Annual Florida Thoracic Society Pulmonary Wintercourse, January 26, 1984, Lake Buena Vista, Florida.

130. Goldman AL. Early Detection of Lung Cancer at Diagnostic Techniques in Pulmonary Medicine, Part II, April 8, 1984, University of South Florida College of Medicine, Tampa, Florida.

131. Goldman AL. Respiratory Disease Management. Section on Internal Medicine, FMA Annual Meeting, May 2, 1984, Lake Buena Vista, Florida.

132. Goldman AL. Detection, Diagnosis and Clinical Staging at Lung Cancer Update. Section on Chest Medicine, FMA Annual Meeting, May 3, 1984, Lake Buena Vista, Florida.

133. Karampelas DT, Goldman AL. Diagnostic Utility of Fiberoptic Bronchoscopy in "Lung Abscess". American Thoracic Society Annual Meeting, May 22, 1984, Miami Beach, Florida.

134. Goldman AL. Moderator. 7 Consecutive Cases of AIDS with Long-Term Follow-up. Tri-State Consecutive Conference, Ponte Vedra, Florida, September 8, 1984.

135. Goldman AL. An Update in Asthma Therapy. 15th Annual Suncoast Cardiopulmonary Seminar. October 5, 1984. Clearwater Beach, Florida.

136. Goldman AL. Adult Respiratory Distress Syndrome. Medical Grand Rounds. Bay Pines VA Hospital, October 24, 1984, Bay Pines, Florida.

137. Goldman AL. What's New in Pulmonary Disease. USF College of Medicine Grand Rounds, November 1, 1984.

138. Goldman AL. Occupational Asthma, Asbestos-Related Diseases and Airway Obstruction - Laser Therapy. 7th Annual Florida Thoracic Society Pulmonary Wintercourse. January 24-27, 1985, Orlando, Florida.

139. Goldman AL. Laser Therapy of Bronchopulmonary Lesions. Section in Chest Medicine at Florida Medical Association Annual Meeting, May 2, 1985, Hollywood, Florida.

140. Goldman AL. Airway Obstruction - Laser Therapy. Mississippi Society for Respiratory Therapy Annual Meeting, May 22, 1985, Jackson, Mississippi.

141. Goldman AL. Occupational Lung Disease. Orlando Regional Medical Grand Rounds, August 22, 1985.

142. Goldman AL. Ventilatory Management. Surgical Grand Rounds, University of South Florida College of Medicine, October 7, 1985.

143. Goldman AL. Pneumonia. Grand Rounds, Bay Pines Medical Center, November 27, 1985.

144. Goldman AL. Laser Therapy, Pulmonary Exercise Testing, Physiologic Consequences of Pulmonary Embolus, Anticoagulation. 85th Annual Florida Thoracic Society Pulmonary Wintercourse, January 9-10, 1986, Orlando, Florida.

145. Goldman AL. Pulmonary Rehabilitation Comprehensive Care for the COPD Patient. Medical Grand Rounds, USF College of Medicine, February 27, 1986.

146. Goldman AL. Does Pulmonary Rehabilitation Help the COPD Patient. "Difficult Questions for Pulmonary Medicine". March 1, 1986, Rusty Pelican, Sponsored by USF College of Medicine.

147. Goldman AL. Occupational Lung Disease and Update on Asthma Therapy. Orlando Regional Medical Center Internal Medicine Update 86, March 7, 1986.

148. Goldman AL. Diagnosis and Treatment of Complex Community and Hospital Acquired Pneumonias, Moderator. October 22, 1986, Tampa, Florida.

149. Goldman AL. Laser Therapy. Florida Thoracic Society Wintercourse. February 6, 1987, Orlando, Florida.

150. Goldman AL. Moderator, Catastrophic Illnesses, Critical Care Dialogues. March 14, 1987, Tampa, Florida.

151. Goldman AL. Endoscopic Laser and Brachytherapy, Lung Cancer Update. USF College of Medicine, March 18, 1987.

152. Goldman AL. Pulmonary Disease Update, MKSAP VII Review Course. April 9, 1987, Orlando, Florida.

153. Goldman AL. Evaluation of Dyspnea and Wheezing: Update in COPD, Primary Care and Preventive Medicine. Orlando Regional Medical Center, July 11, 1987.

154. Goldman AL. Occupational Lung Disease. Medical Grand Rounds, University of South Florida College of Medicine, July 16, 1987.

155. Goldman AL. Laser and Brachytherapy. Suncoast Cardiovascular Seminar, Sheraton Sand Key, November 19, 1987.

156. Goldman AL. Interstitial Lung Disease. Orlando Regional Medical Center Grand Rounds, February 4, 1988.

157. Goldman AL. Asbestos Related Respiratory Disorders. Florida Thoracic Society Wintercourse, Orlando, Florida, February 19, 1988.

158. Goldman AL. Pulmonary Medicine Update. Hernando County Medical Society, March 17, 1988.

159. Goldman AL. Lung Cancer and COPD. University of South Florida Geriatric Board Review Course, Orlando, Florida, March 31, 1988.

160. Goldman AL. Moderator, Cystic Fibrosis in Adults. Tri-State Thoracic Society Meeting, Ponte Vedra, Florida, September 10, 1988.

161. Goldman AL. Pneumonia. Bay Pines VA Medical Center Grand Rounds, October 11, 1988.

162. Goldman AL. Lung Cancer and Occupational Lung Disease. Suzhou Medical College, People's Republic of China, October 25, 1988.

163. Goldman AL. Asthma. Bay Pines VA Medical Center Grand Rounds, January 13, 1989.

164. Goldman AL. Hospital Acquired Pneumonia. Florida Thoracic Society Wintercourse, Orlando, Florida, February 17, 1989.

165. Goldman AL. Office Pulmonary Function Testing. Florida Society of Internal Medicine Annual Meeting, Tampa, Florida, March 4, 1989.

166. Goldman AL. Moderator, Home Oxygen Therapy. USF Physicians' Postgraduate Course, Tampa, Florida, March 11, 1989.

167. Goldman AL. Pneumonia and Diagnostic Techniques. Orlando Regional Medical Center Internal Medicine Review Course. Orlando, Florida, March 13, 1989.

168. Goldman AL. Hospital Acquired Pneumonia, FMA Annual Meeting, ACCP/FTS Section, Orlando, Florida, August 31, 1989.

169. Goldman AL. Occupational Lung Disease. American College of Chest Physicians Interactive Session, Boston, November 2, 1989.

170. Goldman AL. Occupational Lung Disease. Medical Grand Rounds, USF College of Medicine, Tampa, Florida, February 1, 1990

171. Goldman AL. Indoor Air Pollution. Sarasota Memorial Hospital Medical Grand Rounds, Sarasota, Florida, February 9, 1990.

172. Goldman AL. Asbestos Pleural Disease. Florida Thoracic Society, Pulmonary Wintercourse, Orlando, Florida, February 10, 1990.

173. Goldman AL. ARDS and Pulmonary Emboli. Suncoast Emergency '90 Seminar, Sheraton Sand Key, April 19, 1990.

174. Goldman AL. 1) Approach to the Patient Suspected of Having an Occupational Lung Disease; 2) Asbestos and Asbestosis; 3) Asbestos Related Pleural Disease. American College of Chest Physicians Postgraduate Course, Wesley Chapel, Florida, June 8, 1990.

175. Goldman AL. Lung Cancer. USF Health Sciences Teachers Institute, Tampa, Florida, June 20, 1990.

176. Goldman AL. Occupational Lung Disease. The Orlando Family Medicine Review Course, Orlando, Florida, July 2, 1990.

177. Goldman AL. MKSAP. Orlando, Florida, August 15, 1990.

178. Goldman AL. Diagnosis of Lung Cancer. American Cancer Society Conference, Tampa, Florida, October 20, 1990.

179 Goldman AL. Asbestos Diseases. State of Florida DHRS Seminar on Environmental and Occupational Medicine, Tampa, Florida, November 8, 1990.

180. Goldman AL. Pulmonary Embolism Prophylaxis. Florida Thoracic Society Wintercourse, Orlando, Florida, February 17, 1991.

181. Goldman AL. How Significant Are Pleural Changes. Andrews Publications Asbestos Seminar, Miami Beach, Florida, February 26, 1991.

182. Goldman AL. Visiting Professor, Georgia Thoracic Society, Pine Mountain, Georgia, March 1-2, 1991.

183. Goldman AL. Approach to Respiratory Surveillance. USF College of Public Health, Tampa, Florida, April 17, 1991.

184. Goldman AL. Management of Acute Asthma. Emergency '91 Seminar, Clearwater Beach, Florida, April 25, 1991.

185. Goldman AL. Pulmonary Embolism. TGH Emergency Medicine Grand Rounds, Tampa, Florida, May 3, 1991.

186. Goldman AL. Benign Asbestos Pleural Disease. ACCP Postgraduate Course Occupational Lung Disease: Asthma and Asbestos, Wesley Chapel, Florida, June 13, 1991.

187. Goldman AL. Asbestos and Cancer. ACCP Postgraduate Course Occupational Lung Disease: Asthma and Asbestos, Wesley Chapel, Florida, June 13, 1991.

188. Goldman AL. Atypical Pneumonia. Orlando Regional Medical Center Grand Rounds, Orlando Regional Medical Center Grand Rounds, Orlando, Florida, July 25, 1991.

189. Goldman AL. Blood Gases and Pulmonary Functions; COPD and Ventilator Management. Pulmonary, Critical Care & Occupational Medicine, American College of Physicians Internal Medicine Review Course, Orlando, Florida, August 7, 1991.

190. Goldman AL. Evaluation and Assessment of the Patient with a Lung Mass. Lung Cancer: Trends and Treatment. H. Lee Moffitt Cancer Center and Research Institute, Hyatt Regency Westshore, Tampa, Florida, September 21, 1991.

191. Goldman AL. Asbestos Related Respiratory Disorders. USF College of Public Health, Department of Environmental and Occupational Health, Occupational Health for Health Professionals, Tampa, Florida, February 5, 1992

192. Goldman AL. Lung Cancer Clinical Controversies. Florida Thoracic Society Wintercourse, Orlando, Florida, February 13, 1992.

193. Goldman AL. Ventilator Management. Medical Grand Rounds, USF College of Medicine, Tampa, Florida, March 12, 1992.

194. Goldman AL. Trends in Treatment of Asthma; Deep Venous Thrombosis/Pulmonary Embolus Prophylaxis; Newer Ventilatory Techniques. Lloyd Noland Hospital Course, Orlando, Florida, March 17-18, 1992.

195. Goldman AL. Occupational and Environmental Asthma: Natural History, Prognosis, Management and Prevention. Michigan Thoracic Society and Michigan Chapter of the American College of Chest Physicians Annual Meeting, Detroit, Michigan, May 1, 1992.

196. Goldman AL. Asbestos Related Respiratory Disease. Florida Thoracic Society and Florida Chapter of the American College of Chest Physicians Annual Meeting, Orlando, Florida, May 30, 1992.

197. Goldman AL. Acute Effects and Management of Toxic Inhalation Exposures. American College of Chest Physicians Meeting on Occupational Lung Disease, Wesley Chapel, Florida, June 6, 1992.

198. Goldman AL. Pulmonary Assessment of Marginally Resectable Patients. Lung Cancer Trends in Treatment II, Don Cesar, St. Petersburg, Florida, September 19, 1992.

199. Goldman AL. Acute Toxic Inhalation: Pathophysiology and Management Annual American Toxicology Meeting, Tampa, Florida, September 21, 1992.

200. Goldman AL. Metals and Pulmonary Disorders in Interactive Symposium. American College of Chest Physicians Annual Meeting, Chicago, Illinois, October 28, 1992.

201. Goldman AL. Moderator, Indoor Air Quality and It's Effect on Health at the Problem of the "Sick Building" - Facts and Implications. Sponsored by the American Academy of Allergy and Immunology, Orlando, Florida, December 4, 1992.

202. Goldman AL. Moderator, Non Pulmonary Critical Care, Florida Thoracic Society 15th Annual Pulmonary Wintercourse, Orlando, Florida, February 11, 1993.

203. Goldman AL. Acute Toxic Inhalations. Florida Thoracic Society 15th Annual Pulmonary Wintercourse, Orlando, Florida, February 12, 1993.

204. Goldman AL. Management of Asthma and Management of Pulmonary Embolus. Suncoast Emergency Medicine Seminar, Clearwater Beach, Florida, March 18, 1993.

205. Goldman AL. *COPD. MKSAP Review Course, Orlando, Florida, April 27, 1993.*

206. Goldman AL. Literature Review: Asbestos Related Lung Disease. American College of Chest Physicians Postgraduate Course, Wesley Chapel, Florida, June 26, 1993.

207. Goldman AL. Tuberculosis. An Old Friend Returns. University Community Hospital 25 Year Anniversary CME Course, Tampa, July 10, 1993.

208. Goldman AL. COPD Update. Bay Pines VA Medical Center Grand Rounds, St. Petersburg, Florida, October 6, 1993.

209. Goldman AL. COPD - Literature Review: Postgraduate Course, American College of Chest Physicians 59th Annual Scientific Assembly, Orlando, Florida, October 24, 1993.

210. Goldman AL. Laser and Brachytherapy. Florida Thoracic Society 16th Annual Pulmonary Wintercourse, Orlando, Florida, February 17, 1994.

211. Goldman AL. Pneumoconiosis. USF College of Public Health Course, Tampa, Florida, February 23, 1994.

212. Goldman AL. Toxic Inhalation Injuries. Florida Bar Association - Toxic Injuries in the Workplace. Tampa, Florida, May 20, 1994.

213. Goldman AL. Moderator. Seven Consecutive Cases of Complicated Pulmonary Infection Requiring Surgical Intervention. Tri-State Consecutive Case Conference, Ponte Vedra Beach, Florida, September 9, 1994.

214. Goldman AL. Management of the Patient With Asthma. Amelia Island, Florida, October 20, 1994.

215. Goldman AL. Pulmonary Emboli: Overview. Florida Thoracic Society Annual Wintercourse, Orlando, Florida, February 16, 1995.

216. Goldman AL. Managing Upper Respiratory Infections. Midwinter Seminar in Obstetrics and Gynecology. St. Petersburg Beach, Florida, March 3, 1995.

217. Goldman AL: Pulmonary Emboli. Florida Thoracic Society Annual Wintercourse, Orlando, Florida, February 17, 1996.

DR. ALLAN GOLDMAN'S TESTIMONY
Reconstructed from available calendars
Page 1

DATE	CASE OR REFERENCE	ATTORNEY	SUBJECT	JURISDICTION
5/13/94	Depo - James, Marie, Shockey and Teegarten	unknown	Workers' Compensation*	unknown
6/7/94	Depo - Danny Strutt	unknown	Workers' Compensation*	unknown
6/8/94	Depo - Barbara Newman	unknown	Workers' Compensation*	unknown
7/15/94	Depo - Laurie Frederick	unknown	Workers' Compensation*	unknown
8/12/94	Depo - Donald Sasser	unknown	Workers' Compensation*	unknown
9/16/94	Depo - Cowsky	unknown	Medical Malpractice	unknown
9/26/94	Depo - Leeann Peel	unknown	Medical Malpractice	unknown
9/28/94	Depo - F. Diaz	unknown	Workers' Compensation*	unknown
9/28/94	Depo - Keener	unknown	Medical Malpractice	unknown
9/30/94	Depo - Sandra Brown	unknown	Workers' Compensation*	unknown
10/25/94	Depo - Fleury	unknown	Medical Malpractice	unknown
11/2/94	Depo - Kevin Ward	unknown	Workers' Compensation*	unknown
11/9/94	Depo - DeLeo	unknown	Medical Malpractice	unknown

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DR. ALLAN GOLDMAN'S TESTIMONY
Reconstructed from available calendars
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DATE	CASE OR REFERENCE	ATTORNEY	SUBJECT	JURISDICTION
11/29/94	Depo - Hayes	unknown	Workers' Compensation*	unknown
12/14/94	Depo - Sandlin	unknown	Workers' Compensation*	unknown
1/25/95	Depo - Parker	unknown	Workers' Compensation*	unknown
2/3/95	Depo - Jackie Wells	unknown	Workers' Compensation*	unknown
2/15/95	Depo - Ray Jackson	unknown	Workers' Compensation*	unknown
3/22/95	Depo - Tadlock	unknown	Medical Malpractice	unknown
4/5/95	Depo - Mary Jane Griffin	unknown	Workers' Compensation*	unknown
4/11/95	Depo - Durrant Spencer	unknown	Workers' Compensation*	unknown
4/26/95	Depo - W. Bailey	unknown	Workers' Compensation*	unknown
5/17/95	Depo - Miller	unknown	Medical Malpractice	unknown
5/30/95	Depo - Hollis & Martin	unknown	Workers' Compensation*	unknown
5/31/95	Depo - J. Cockayne	unknown	Workers' Compensation*	unknown
6/12/95	Depo - Mary Jo Larkin	unknown	Medical Malpractice	unknown

DR. ALLAN GOLDMAN'S TESTIMONY
Reconstructed from available calendars
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DATE	CASE OR REFERENCE	ATTORNEY	SUBJECT	JURISDICTION
7/1/95 - 12/31/95	CANNOT LOCATE CALENDAR			
1/10/96	Depo - Nixon	unknown	Medical Malpractice	unknown
1/12/96	Depo - Maldonado	unknown	Workers' Compensation*	unknown
1/30/96	Depo - Holzendorf	unknown	Workers' Compensation*	unknown
2/2/96	Depo - Regina Glass	unknown	Workers' Compensation*	unknown
2/6/96	Depo - Daniel Beck	unknown	Workers' Compensation*	unknown
2/21/96	Depo - John Arroyo	Melanie Cease	Workers' Compensation	unknown
3/13/96	Depo - Willingham	unknown	Medical Malpractice	Orlando
3/15/96	Depo - Diane Smith	unknown	Workers' Compensation*	unknown
4/15/96	Depo - Hochberger	unknown	Workers' Compensation*	unknown
5/14/96	Depo - Edith Cancel	unknown	Workers' Compensation*	unknown
6/26/96	Depo - Griffin	unknown	Workers' Compensation*	unknown
7/8/96	Depo - Jeff Odom	unknown	Workers' Compensation *	unknown

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DR. ALLAN GOLDMAN'S TESTIMONY Reconstructed from available calendars Page 4				
DATE	**CASE OR REFERENCE**	**ATTORNEY**	**SUBJECT**	**JURISDICTION**
7/11/96	Depo - Harold Nelson	unknown	Workers' Compensation*	unknown
8/13/96	Depo - Barry Brown	unknown	Workers' Compensation*	unknown
8/22/96	Depo - Vivian Richards	unknown	Workers' Compensation*	unknown
8/27/96	Depo - Debra Mayo	unknown	Workers' Compensation*	unknown
10/15/96	Depo - Linda Walker	unknown	Medical Malpractice	unknown
11/4/96	Depo - cannot read name	unknown	Medical Malpractice	unknown
11/6/96	Depo - Joyce Carter	unknown	Workers' Compensation*	unknown
12/3/96	Depo - Michelle Dayton	unknown	Workers' Compensation*	unknown
12/27/96	Depo - John Fritaccia	unknown	Workers' Compensation*	unknown

* almost always patients referred into the USF Medical Clinic as Workers' Compensation cases. These patients are seen in the USF Clinic and the bill is submitted by the University and Dr. Goldman generally does not receive any payment for these depositions since these are Clinic patients.

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CURRICULUM VITAE

NAME	Herman Baer, M.D.	
TITLE	Professor and Director Autopsy Services Department of Pathology School of Medicine University of Florida Gainesville FL 32610	
BIRTH	September 11, 1933 - Uetikon, ZH, Switzerland	
MARITAL STATUS	Married - Eleonore 3 children – September 2, 1965 – July 10, 1967 – May 28, 1969	
CITIZENSHIP	U.S.A. – December, 1969	
EDUCATION	Grade School in Uetikon, Switzerland	1940 – 1946
	Gymnasium Kantonsschule, Switzerland, Baccalaureat	1946 – 1952
	University of Zürich, Switzerland, School of Medicine	1952 – 1958
	Research Fellow at the Department of Biochemistry, University of Basle, Switzerland	1958 – 1960
	M.D. – University of Basle, Switzerland	1960
	Postdoctoral Fellow, Institute for Medical Microbiology, University of Zürich, Switzerland, School of Medicine	1960 – 1962
	Postdoctoral Fellow in the Division of Infectious Diseases, Department of Medicine, University of Pittsburgh Medical School, under Dr. Braude	1963 – 1965
	Postdoctoral Fellow in the Department of Bacteriology, Division of Natural Sciences, University of Pittsburgh, under Dr. Brinton	1965 – 1966

APPOINTMENTS	Head of Diagnostic Unit, Institute for Medical Microbiology, University of Zürich, School of Medicine, Switzerland	1962 – 1963
	Assistant Professor and Director of Clinical Microbiology, University of Florida	1966 – 1970
	Associate Professor and Director of Clinical Microbiology, University of Florida	1970 – 1975
	Associate Professor of Pathology	1970 – 1983
	Professor of Pathology	1983 – Date
	Resident in Anatomic and Clinical Pathology	1978 – 1981
	Director, Autopsy Services	1981 – 1995
AWARDS	Diplomate in Medical Bacteriology, American Academy of Medical Microbiology	1969
MEMBERSHIPS	American Society for Microbiology	
BOARD CERTIFICATION	Anatomic Pathology	

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Dr. Baer's Testimony (from Dr. Baer's deposition transcript, Chutz case)				
Year	Case	Atty	Type	Jurisdiction
1984 - 1994	Approx. 500	Spohrer, Wilner firm	Asbestos	
		Bob Hannah	Asbestos	Orlando
		Tomlin	Asbestos	Miami
		firm in Mobile, AL	Asbestos	
1/2 dozen/ year			Non-asbestos cases	
1993	LaFever	Erickson	Asbestos	Ft. Myers
1993	Taggert			Jacksonville
1996			Asbestos	
1996	Renicke	Hollingshead	Asbestos	Jacksonville
1996	Nail	Knopnik	Medical Malpractice	Tampa
1996	Threm	Delaparte, Gilbert	Medical Malpractice	Tampa
1996	Flores		Medical Malpractice	Miami
?	Spellman		Medical Malpractice	Daytona
?	Button	Metzger		Ft. Myers

TREATING PHYSICIANS - ALBERT JENKINS

It is anticipated that those physicians who treated Albert Jenkins will testify that cigarettes caused his chronic obstructive pulmonary disease (COPD) and lung cancer and associated symptoms and death. They are further expected to testify in accordance with their records:

Abib T. Conteh, M.D.
508 W. MLK Blvd.
Tampa, Florida

Alison Calkins, M.D.
Fred Woods Radiation Therapy Dept.
3001 W. MLK Blvd.
Tampa, Florida

Ramesh K. Shah, M.D.
4910 No. Armenia Avenue
Tampa, Florida

N. Mandaliya, M.D.
Modh & Mandaliya, M.D.
2810 W. Waters Avenue
Tampa, Florida