STATE OF CONNECTICUT

SITING COUNCIL

Re:	The Connecticut Light and Power Company and The United Illuminating Company Application for a Certificate of Environmental Compatibility and Public Need for the Construction of a New 345-kV Electric Transmission Line and Associated Facilities Between Scovill Rock Switching Station in Middletown and Norwalk Substation in Norwalk, Connecticut Including the Reconstruction of Portions of Existing 115-kV and 345-kV Electric Transmission Lines, the Construction of the Beseck Switching Station in Wallingford, East Devon Substation in Milford, and Singer Substation in Bridgeport, Modifications at Scovill Rock Switching Station and Norwalk Substation and the Reconfiguration of Certain Interconnections	)	Docket 272
		)	March 16, 2004

APPLICANTS' PRE-FILED DIRECT TESTIMONY
CONCERNING POWER-FREQUENCY ELECTRIC AND MAGNETIC FIELDS

The Applicants, The Connecticut Light and Power Company and The United Illuminating Company, submit herewith their pre-filed direct testimony, consisting of the following:

A. Testimony

1. Philip Cole, M.D., Ph.D. (Dr. Cole is a physician and an epidemiologist.)

2. Stuart Aaronson, M.D. (Dr. Aaronson is a physician and a cancer biologist.)

3. Kathleen Shanley, The United Illuminating Company
-and-
Robert E. Carberry, P.E., The Connecticut Light & Power Company

4. William H. Bailey, Ph.D., Exponent (Dr. Bailey is a research scientist)

B. Statements of Qualifications

In order to facilitate review of the testimony, the relevant biographical information for each of the witnesses is provided, in alphabetical order, in Part B of this filing, rather than appended to each witnesses' testimony. Part B also includes biographical information for Gary Johnson, P.E., of Exponent, to whom Dr. Bailey refers in his testimony.

Respectfully submitted,

APPLICANTS,

THE CONNECTICUT LIGHT AND POWER COMPANY,

BY: _____

ANTHONY M. FITZGERALD
BRIAN T. HENEBRY
Carmody & Torrance LLP
195 Church Street
New Haven, CT 06509-1950

THE UNITED ILLUMINATING COMPANY,

BY: _____

LINDA L. RANDELL
BRUCE L. MCDERMOTT
Wiggin & Dana LLP
265 Church Street
PO Box 1832
New Haven, CT 06508-1832

CERTIFICATE OF SERVICE

I hereby certify that a copy of the foregoing **Applicants' Pre-Filed Direct Testimony Concerning Power-Frequency Electric and Magnetic Fields** has been mailed, postage prepaid on this 16th day of March, 2004 to the following:

Julie Donaldson Kohler, Esq.
Hurwitz & Sagarin, LLC
147 North Broad Street
Milford, CT 06460
jdk@hurwitz-sagarin.com

Representative Al Adinolfi
103rd District
Legislative Office Building
Hartford, CT 06106-1591
Alfred.adinolfi@housegop.state.ct.us

Linda L. Randell, Esq.
Bruce L. McDermott, Esq.
Wiggin &Dana LLP
One Century Tower
New Haven, CT 06508-1832
lrandell@wiggin.com
bmcdermott@wiggin.com

Town of Middlefield
c/o Eric Knapp, Esq
Branse & Willis, LLC
41-C New London Turnpike
Glastonbury, CT 06033
eknapp@bransewillis.com

Peter G. Boucher, Esq.
Halloran & Sage, LLP
One Goodwin Square
225 Asylum Street
Hartford, CT 06103
boucher@halloran-sage.com

Louis S. Ciccarello, Esq.
Corporation Counsel
P.O. Box 798
Norwalk, CT 06856-0798
lciccarello@norwalkct.org

Representative Mary G. Fritz
90th District
43 Grove Street
Yalesville, CT 06492
mary.fritz@po.state.ct.us

Town of Westport
c/o Ira W. Bloom, Esq.
27 Imperial Avenue
Westport, CT 06880
ibloom@wsdb.com

Joanne D'Angelo, Esq.
Woodbridge Town Counsel
70 Beecher Road
Woodbridge, CT 06525
jdangelo@optonline.net

Lawrence J. Golden, Esq.
Pullman & Comley, LLC
90 State House Square
Hartford, CT 06103
lgolden@pullcom.com

Norwalk Association of Silvermine Homeowners
c/o Leigh Grant
99 Comstock Hill Road
Norwalk, CT 06850
cartellino@aol.com

Representative Robert W. Megna
97th District
40 Foxon Hill Road, #54
New Haven, CT 06513
Robert.Megna@po.state.ct.us

Deborah L. Moore, Esq.
Legal Department
City Hall
142 East Main Street
Meriden, CT 06450
dmoore@ci.meriden.ct.us

Michael C. Wertheimer
Assistant Attorney General
Attorney General's Office
10 Franklin Square
New Britain, CT 06051
Michael.wertheimer@po.state.ct.us

Representative Raymond Kalinowski
100th District
P.O. Box 391
Durham, CT 06422
Raymond.kalinowski@housegop.state.ct.us

Melanie J. Howlett
Associate City Attorney
City of Bridgeport
999 Broad Street
Bridgeport, CT 06604-4328
Howlem0@ci.bridgeport.ct.us

Bruce C. Johnson
Litigation Attorney
Office of Consumer Counsel
Ten Franklin Square
New Britain, CT 06051
bruce.johnson@po.state.ct.us

Representative Themis Klarides
114th District
23 East Court
Derby, CT 06418
Themis.klarides@housegop.state.ct.us

Anthony M. Macleod, Esq.
Whitman, Breed, Abbott & Morgan LLC
100 Field Point Road
Greenwich, CT 06830
amacleod@wbamct.com

Trish Bradley, President
Ed Schwartz, Treasurer
Communities for Responsible Energy, Phase II
45 Ironwood Lane
Durham, CT 06422
thebradco@aol.com

Arthur W. Gruhn, P.E.
Chief Engineer
Bureau of Engineering and Highway Operations
Department of Transportation
2800 Berlin Turnpike
P.O. Box 317546
Newington, CT 06131-7546
Arthur.gruhn@po.state.ct.us

Honorable Kenneth Flatto
First Selectman
Independence Hall
725 Old Post Road
Fairfield, CT 06824
firstselectmanflatto@town.fairfield.ct.us

Monte E. Frank, Esq.
Cohen and Wolf, P.C.
158 Deer Hill Avenue
Danbury, CT 06810
mfrank@cohenandwolf.com

Janis M. Small, Esq.
Town Attorney
Wallingford Town Hall
45 South Main Street
Wallingford, CT 06492
wlfdlaw@snet.net

Richard J. Buturla, Esq.
Berchem, Moses & Devlin, P.C.
75 Broad Street
Milford, CT 06460
rbuturla@bmdlaw.com
mmilone @ cheshirect.org

Timothy P. Lynch
Deputy City Attorney
City Attorney's Office
245 deKoven Drive
P.O. Box 1300
Middletown, CT 06457-1300
timothy.lynch@cityofmiddletown.com

Honorable William A. Aniskovich
12th District
15 Grove Avenue
Branford, CT 06405
William.A.Aniskovich@po.state.ct.us

Mr. Franco Chieffalo
General Supervisor
First District Water Department
P.O. Box 27
Norwalk, CT 06852
fchieffalo@norwalkfdwd.org

Harold W. Borden
Vice President and General Counsel
PSEG Power Connecticut LLC
80 Park Plaza
Newark, NJ 07102-4194
h.borden@pseg.com

David A. Ball, Esq.
Cohen and Wolf, P.C.
1115 Broad Street
Bridgeport, CT 06604
dball@cohenandwolf.com

Maryann Boord
First Selectwoman
Durham Town Hall
30 Townhouse Road
Durham, CT 06422
mboord@townofdurhamct.org

Joaquina Borges King
Assistant Town Attorney
Hamden Government Center
2750 Dixwell Avenue
Hamden, CT 06518
jborgesking@hamden.com

Honorable Derrylyn Gorski
First Selectman
Bethany Town Hall
40 Peck Road
Bethany, CT 06524-3378
Dgorski@Bethany-CT.com

David J. Monz
Updike, Kelly & Spellacy, P.C.
One Century Tower
265 Church Street
New Haven, CT 06510
dmonz@uks.com

Mitchell R. Goldblatt
First Selectman
Town of Orange
617 Orange Center Road
Orange, CT 06477-2499
Mitchgoldblatt@aol.com

Andrew W. Lord, Esq.
Murtha, Cullina LLP
CityPlace I, 29th Floor
185 Asylum Street
Hartford, CT 06103-3469

Robert E. Earley
Connecticut Business & Industry Assoc.
350 Church Street
Hartford, CT 06103-1106
earleyr@cbia.com

David A. Reif
Jane K. Warren
Joel B. Casey
McCarter & English, LLP
Cityplace I
Hartford, CT 06103
dreif@mccarter.com

William J. Kupinse, Jr.
First Selectman
Easton Town Hall
225 Center Road
P.O. Box 61
Easton, CT 06612
wkupinse@eastonct.org

David R. Schaefer, Esq.
Brenner Saltzman & Wallman LLP
271 Whitney Avenue
New Haven, CT 06511
dschaefer@bswlaw.com

Anthony M. Fitzgerald

DIRECT TESTIMONY OF PHILIP COLE, M.D., Ph.D

Q. What is your occupation, Dr. Cole?

A. I am a professor emeritus of Epidemiology at the University of Alabama at Birmingham (UAB). Although I am now retired, I continue to teach and to conduct research at UAB. Also, I am engaged in consulting activities of several types.

Q. What will your role be as a member of the panel of witnesses to be presented by the Companies with respect to electric and magnetic fields (EMF)?

A. I understand that my primary role will be to respond to questions that members of the Siting Council, parties, and intervenors may have with respect to the science of epidemiology and the suggestion that exposure to power line magnetic fields may cause or promote human cancers, particularly childhood leukemia.

Q. What qualifications do you have to provide expert testimony on that subject?

A. The curriculum vitae attached to this testimony describes my education, certifications and career. Briefly, I am a physician, and I hold a master's degree and a doctorate in Epidemiology from Harvard University and am Board Certified in Preventive Medicine. I regularly teach in the area of general epidemiology and cancer epidemiology (the study of the causes of cancer in man). I have published nearly 200 articles primarily relating to cancer epidemiology and in the peer-reviewed literature.

Q. What is the School of Public Health of the UAB?

A. It is one of 26 accredited schools of public health in the United States. It is solely a graduate school, which trains professionals in the public health field. The school awards masters' and doctoral degrees in various sciences that are related to public health.

Q. What positions did you hold at the University before your retirement?

A. I was Professor and Chairman of Epidemiology at the School of Public Health and Associate Director for Epidemiology at the University's Comprehensive Cancer Center.

Q. What were your responsibilities as Associate Director for Epidemiology at the University's Comprehensive Cancer Center?

A. To develop and oversee a program of research into the causes of cancer in human beings.

Q. Have you published any of your research on epidemiology in scientific journals?

A. Yes. I have published nearly 200 articles in major medical journals, such as the New England Journal of Medicine, the Journal of the American Medical Association, and the Lancet; and in virtually all of the epidemiology and public health journals.

Q. Has any of your work involved EMF ?

A. Yes. The health effects of electric and magnetic fields has been one of my research interests.

Q. When did you start investigating the question whether EMF may cause cancer in humans?

A. In 1986.

Q. Would you please briefly summarize your opinion, based on your education, training, and experience, particularly your research work and your review of the literature, with respect to the suggestion that exposure to the electric or magnetic fields (EMF) associated with electric power transmission may cause cancers in humans, particularly childhood leukemia?

A. My opinion is that the available evidence does not support the view that magnetic fields from power lines cause cancer, including childhood leukemia.

Q. What is the basis for that opinion?

A. The question of EMF as a possible cause of cancer in human beings has been investigated by epidemiologists in more than 150 studies, now spanning 25 years. There also have been hundreds of animal and molecular studies reported. In addition, innumerable reviews of the question have been prepared both by academic and regulatory bodies.

Despite this extensive research, EMF is not considered to be a human carcinogen. No scientific or regulatory body, including the International Agency for Research on Cancer (IARC), the cancer research arm of the World Health Organization, has categorized EMF as a carcinogen for human beings. There is no precedent for an agent that has received such intense investigation and that has failed to be recognized as a carcinogen – subsequently to become so recognized.

Q. What is epidemiology?

A. Epidemiology is a branch of science that seeks to identify the causes of diseases in human beings by studying human beings. Epidemiologic studies report results in the form of statistical associations. The term "statistical association" is used to describe the tendency of two things to be linked or to vary in the same way, such as higher level of exposure and increased occurrence of disease. If a statistical association between a factor under study and an increased occurrence of disease is documented, epidemiologists will then seek to determine if the association is one of cause and effect.

Q. What are the strengths of epidemiology as a means of identifying the causes of disease in humans?

A. The major strength is that it epidemiology is based on observations of people in their natural environments, as they go about their daily lives, so that you see~~m~~ them as they actually live with the many possible causes of disease interacting. It provides a means by which we can learn ~~something~~ about the causes of diseases of people, rather than of ~~to~~ animals.

Q. What are the limitations of epidemiological studies?

A. Because you are studying people in their natural circumstances, you have no control over the situation as an experimentalist does in the laboratory. You cannot monitor or control the exposure. You can not keep your subjects, i.e. people, from exposing themselves to possible causes of the disease of interest other than the suspect cause you are studying. Also, people are essentially infinitely variable. All this is in contrast to the experimental situation where all the animals are bred the same, they are all treated the same, and half get the suspect cause and half do not. In epidemiology, the investigator is not at all in control of the situation and so must work very hard to allow a pattern to emerge from the observations made.

Q. Can you give us an example of a statistical association that is strong, but not causal?

A. Yes. There is a very strong association between roosters crowing shortly before sunrise and the rise of the sun. But the rooster's crow does not cause the sun to rise. Similarly, there is a strong association between high alcohol consumption and lung cancer. But high alcohol consumption does not cause lung cancer; it just so happens that heavy drinkers are over-represented among smokers, as compared to those who drink lightly or not at all and, on the basis of their smoking, are at increased risk of cancer.

Q. How does an epidemiologist go about evaluating a body of epidemiological research?

A. The first step is to gather all the relevant studies and evaluate each study according to certain criteria. First, we look to see if the design of the study is sufficiently rigorous to rule out bias, by which we mean not prejudice, but a systematic error in the study.

The next step is to consider whether the study design is subject to "confounding" - that is, whether the investigator thought he was studying one thing, but was actually studying something else.

An investigator who thought he was studying the health effects of alcohol consumption-but was actually seeing the effects of smoking would be an example.

Finally, one has to consider the possibility that an association that is found in even a well designed study may be due simply to chance.

Unfortunately, it is much more difficult to detect these flaws in actual studies than in the examples I have given.

Q. How do epidemiologists determine whether associations that are documented by individual studies are causal?

A. These determinations are made by evaluating multiple studies of the same suspected risk factor, and evaluating the data by standard criteria. These criteria are called the Hill Criteria, named for their originator, Sir Austin Bradford Hill, who was one of the first scientists to determine that smoking causes lung cancer.

- *Strength of association.* The strength of association is described as a "risk ratio" or as an "SMR." For instance, the SMR for lung cancer among smokers is at least 1000. That means that people who smoke are ten times more likely to have lung cancer than people who do not. An SMR of 100-means that there is no difference in the risk of disease in the exposed versus the non-exposed group. An SMR in the range 100-200
- *Dose response.* Does the risk go up as the exposure increases? For instance, cigarette smoking shows a very significant dose response. The more a smoker smokes, the more likely he is to develop-lung cancer.
- *Consistency of Association.* If all of the investigators who investigate a particular question find essentially the same thing, there is very good consistency across the studies. Again, by way of example, ever since epidemiologists first started studying lung cancer

and smoking in the 1950's, there was very strong consistency in the studies. All the well conducted and well designed studies found essentially the same thing – a very strong association.

- *Specificity* – Does the exposure under investigation tend to produce a narrow spectrum of diseases? Cancer is not a single disease, but many different diseases, and different diseases tend to have different specific causes. For instance, cigarette smoking does not seem to cause skin cancer and exposure to sunlight does not seem to cause lung cancer.
- *Biological Plausibility* – Is there some biological mechanism that we would find plausible that could explain how the exposure could cause the disease under study. As applied to cancer, that generally means a plausible biological mechanism at the cellular or subcellular level.
- *Temporal relationship* - This simply means that the exposure under study came before the disease under study. This will almost always be the case.

Q. Do all of these criteria have to be satisfied in order to make a determination that an association is real and causal?

A. No, the evaluation is a matter of judgment. The only factor that absolutely has to be satisfied is temporal relationship. The effect can not precede the cause. But there can be no judgment of causality unless at least most of the criteria are satisfied very definitely. For instance, there was some question at first whether the causation of lung cancer by cigarette smoking was biologically plausible. On the one hand, the smoke was clearly directed to the lungs, and caused a variety of known physiological changes in the body. But on the other, the specific causal mechanism by which smoking causes cancer was not known. But the strength of the other evidence provided the basis for a judgment of causality even though a plausible causal mechanism was uncertain. The SMRs were among the

highest ever seen, the studies were extremely consistent, and the dose response remarkably powerful. In addition, the "ecological" evidence was strong. Before manufactured cigarettes were developed and mass marketed, lung cancer was an extremely rare disease. It became widespread about thirty years after the habit of cigarette smoking became widespread.

Q. How do the Hill criteria apply to the studies of exposure to EMF and cancer?

A. As all of the national and international multi-disciplinary scientific bodies who have evaluated the literature have concluded, the studies do not provide a basis for concluding that there is any causal association – and it is doubtful that they establish any real association at all. Where an association is found, it is always a very weak one. There is no indication of dose response. There is inconsistency in the results both across studies and within individual studies. The associations claimed for EMF are not specific; to the contrary, they include many different cancers and may other adverse outcomes. In spite of a great deal of work in recent years, no biologically plausible mechanism by which power line EMF could cause cancer, particularly leukemia, has been found. Another member of this panel, Dr. Stuart Aaronson, who is a cancer biologist, will answer questions that deal with that specific subject.

Q. It has been suggested that there might be a parallel between smoking and lung cancer and EMF and power lines, in that it took a long time before the suspected causal link between smoking and lung cancer was proven, and the same might be true for power lines and EMF. Do you believe that to be the case?

A. No, not at all. In the case of smoking and lung cancer, the epidemiological evidence was so convincing, even without an identified biological mechanism of causation, that all but a few scientists were convinced within five to ten years after the first studies were published that cigarette smoking caused lung cancer. There was no true scientific "controversy," but rather a strong scientific consensus.

By contrast, with respect to EMF and power line magnetic fields, there is a strong scientific consensus that no causal relationship has been found, in spite of hundreds of epidemiologic and possibly thousands of other types of biomedical studies have conducted over a 25 year period. Indeed, as I said earlier in this testimony, there is no precedent for an agent that has received such intense investigation and that has failed to be recognized as a carcinogen – subsequently to become so recognized.

Q. Does this conclude your testimony?

A. Yes.

DIRECT TESTIMONY OF STUART AARONSON, M.D.

Q. What is your occupation?

A. I am a physician employed by the Mount Sinai School of Medicine, where I am the Jane B. and Jack R. Aron Professor and Director of the Derald H. Ruttenberg Cancer Center.

Q. Please describe your duties and activities in those positions.

A. I am head of a research department focused on understanding of the causes of cancer with the goals of developing better approaches to prevention and treatment of this disease. I am responsible for hiring faculty members and am involved in strategic planning of Mount Sinai's cancer initiatives. In addition, I have my own research group, which studies molecular alterations and signaling pathways involved in cancer.

Q. Please describe your prior work experience?

A. My work history, education, publications, and other information are fully stated in the curriculum vitae submitted as part of this testimony. Briefly, I became Chief, Laboratory of Cellular and Molecular Biology in the National Cancer Institute in 1977. My laboratory at the NCI made critical discoveries concerning the molecular basis of cancer. Specifically, we were involved in the discovery of the first normal function of a cancer gene (oncogene), the identification of oncogenes of human cancers, and the discovery of important signaling molecules involved in normal cell proliferation and differentiation. We identified a number of molecular mechanisms, which activate cellular genes to become oncogenes.

I was recruited in 1993 to be Director of the Ruttenberg Cancer Center at Mount Sinai, where I have been involved in building a nationally recognized cancer program. I am responsible for hiring faculty members, developing disease focused multidisciplinary cancer research efforts, and serving as a

senior academic leader within the Mount Sinai School of Medicine. I have my own grant-supported research program as well. This program involves investigation of cancer genes and the signaling pathways in which they act as well as the multistep process of carcinogenesis. In the course of my work, I train graduate and medical school students as well as postdoctoral investigators in the area of cancer biology. I have published over 520 articles primarily related to cancer and have more than 50 patents or patent applications arising from my discoveries, one of which has led to an approved drug with others at different stages of clinical development.

Q. What affiliations do you have with professional organizations and associations?

A. I am presently a member of the American Association for Cancer Research, and serve as a member of its Public Relations and Communications Committee. I also serve as a Member of the National Neurofibromatosis Foundation Research Advisory Board. I am an Associate Editor or Editorial Board member of a large number of cancer focused scientific journals. These include Cancer Research, Oncogene, International Journal of Cancer, and Cancer and Metastasis Reviews. I serve on the Scientific Advisory Boards of the Kimmel Cancer Center, Thomas Jefferson University, and the Georgetown University Breast Cancer Specialized Program of Research Excellence (SPORE). I have previously served as organizer of a number of scientific meetings including the Princess Takamatsu Symposium. I have served as an elected officer of scientific societies including Councilor of the Society for Experimental Biology and Medicine and President of the Harvey Society.

Q. Are you familiar with the research on the question whether power frequency electric and magnetic fields (EMF) may cause cancer?

A. I have followed this field for a number of years, although I have not published in it. Specifically, I have reviewed the literature related to efforts to study whether power frequency EMF

may initiate and/or promote cancer by methods that have been utilized to test other environmental agents.

Q. Would you please summarize your opinion, based on your education, training, and experience, particularly your research work and your review of the literature, with respect to the suggestion that exposure to EMF associated with electric power transmission may cause cancers in humans?

A. There has been an extensive assessment of the possibility that exposure to power electric and magnetic frequency fields could be associated with an increased risk of cancer. From my review of this literature including the reports of nationally constituted scientific review groups, I conclude that there is no convincing or consistent evidence that power lines pose a cancer risk.

Q. What do you refer to as "power frequency"electric and magnetic fields?

A. The "frequency" of electromagnetic energy is expressed is hertz (Hz), which is a measure of the rapidity at which the field varies. Electric and magnetic fields do not vary at 0 Hz. The fields associated with alternating current (AC) electric power transmission are 60-Hz (50-Hz in Europe), which means that they oscillate 60 times per second. This frequency is in the Extremely Low Frequency portion of the electromagnetic spectrum. Toward the top of the spectrum, where we find ultraviolet radiation and X-rays, the frequencies are much higher. For instance, the frequency of X-rays is about 1 billion billion Hz.

Q. What is cancer?

A. Cancer is a term used to describe many different diseases, all of which involve uncontrolled cell growth.

Q. How is cancer caused?

A. Cancer is caused by alterations in DNA, the hereditary (genetic) structure of a cell. DNA is organized into units termed chromosomes, which contain double stranded helical coiled DNA with associated proteins. The human cell contains 46 chromosomes. Within our chromosomes is the information for specific units termed genes. Each gene directs the production of a messenger RNA, which encodes a single protein. Individual proteins are the building blocks responsible for carrying out all of the cell's normal functions.

Cells are constantly replicating themselves. In the course of this process, mutations can occur spontaneously, due to errors in normal DNA replication. Fortunately, in most cases, these alterations of DNA do not lead to cancer. However, mutations that alter the functions of certain genes can be sufficient to initiate a cancer. These genes are called *oncogenes* and *tumor suppressor genes.* Oncogenes greatly accelerate the rate of cell division. Tumor suppressor genes act as brakes on abnormal growth. But that brake function can be inactivated by mutation. In many cancer models, mutations of both oncogenes (creating an abnormal acceleration of cell growth) and of tumor suppressor genes (inactivating the brake on abnormal cell growth) are believed to be required.

Another mechanism by which genetic alterations can initiate cancer is through the insertion of tumor viruses into the DNA. These viruses themselves encode proteins that inactivate the functions of specific tumor suppressor genes or act as oncogenes.

Q. What is a carcinogen?

A. The genetic changes that characterize cancer can be spontaneous, or can be induced by an agent. Agents that are capable of inducing genetic changes that can cause cancer are called "carcinogens." If the agent can cause changes that can lead to a cancer in the absence of any other exposure to carcinogens, it is called a "complete carcinogen."

There are also agents that are not complete carcinogens, but will act on cells that have already been genetically damaged to produce cancer. Such an agent is said to be a "promoter" of cancer. Promoters do not directly damage DNA, but instead indirectly bring about further genetic change by such means as causing increased cell proliferation (thus accelerating the occurrence of spontaneous mutations) or by inhibiting cell functions, such as those involved in the normal repair of DNA damage.

Q. Can exposures to power frequency EMF directly damage DNA?

A. It is generally accepted that the energy in power frequency EMF is insufficient to cause changes in the chemical structure of DNA.

Q. What experimental information is available about any potential link between EMF and cancer?

A. There have been many laboratory studies aimed at assessing whether power frequency EMF could cause or in some manner promote the development of cancer. The laboratory assessment of the carcinogenicity of EMF, as that for any other suspected carcinogen, has involved both long term studies in which whole animals are chronically exposed to EMF, and studies of cancer-related changes in genes or other cellular processes observed in isolated cells. The traditional term for the whole animal studies is *in vivo,* Latin for "in life, or alive." Since isolated cells are traditionally studied in a culture contained in a glass vessel, and the type of test is called *in vitro* ("in glass").

Q. What kind of whole animal studies have been performed to assess a possible link between EMF and cancer?

A. There have been several large, well conducted long term studies (called bioassays) in which laboratory mice and rats have been chronically exposed to very high doses of 60-Hz EMF for long periods, in some cases for almost their entire lifetimes. These types of studies have a proven record for predicting the carcinogenicity of chemicals, physical agents, and other suspected cancer-causing

agents. Typically, one group of animals is exposed to a controlled, high 60-Hz magnetic field and another group of the same size is not so exposed. Any animals that die during the experiment are autopsied, and at the conclusion of a predetermined time, all remaining animals are sacrificed and autopsied. Tumors are carefully noted by type and number and the tumor incidence between the two groups is compared.

Such experiments have been performed with animals that are in normal health at the beginning of the experiment; with animals that have been bred to be particularly susceptible to cancer; and with animals that have been administered a known carcinogen. Thus, the *in vivo* tests were designed both to assess the potential of EMF as a complete carcinogen and as a promoter of cancer. The controlled exposures were to fields ranging from 1 to over 1,000 microtesla; that is, from 10 to over 10,000 milligauss.

Q. What have the results of these whole animal experiments been?

A. The whole animal experiments are overwhelmingly negative. As a whole, they provide no consistent or convincing evidence of any relationship between EMF and cancer, including brain cancer, breast cancer and leukemia.

Q. What kinds of *in vitro* laboratory studies have been conducted to examine whether power frequency EMF might cause or promote cancer?

A. A great many studies of different types have been performed. some of these studies have looked for evidence that power frequency EMF is "genotoxic," that is, that it damages DNA directly; others have looked for evidence that EMF promotes the development of cancer.

Q. Please first describe the studies that have looked for evidence that power frequency EMF is genotoxic.

A. Because of the very substantial funding until recently available through the federal RAPID research program, this literature is massive. These studies involve controlled exposures normal cells to EMF, in various controlled environments, and then examining them for evidence of damage, such as cell transformations, chromosome aberrations, the breaking and rejoining of chromosomes, detached pieces of chromosomes, and DNA strand breaks. The field intensities used in these experiments ranged from less than 1 microtesla (or 10 milligauss) to more than 1,000 microtesla (or 10,000 milligauss).

Q. Please describe the results of these studies of the possible genotoxicity of power frequency EMF.

A. These assays are overwhelming negative. Of the few studies that do report evidence for genotoxicity, most contain a mixture of positive and negative results, or ambiguous results, and none of them have been replicated. They provide no basis for concluding that power frequency EMF is genotoxic.

Q. Please describe the *in vitro* studies that have investigated whether power frequency magnetic fields are cancer promoters?

A. There have been a great many laboratory experiments aimed at assessing possible biologic effects of power frequency fields that might conceivably cause them to act as cancer promoters or to enhance the effectiveness of genotoxic agents. Thus, experiments have been performed to test whether such fields inhibit programmed cell death or DNA repair, affect cell proliferation or differentiation, or affect gene expression or enzyme activity. Like the whole animal studies, the cell

studies have produced no consistent or convincing evidence that power frequency electric or magnetic fields promote the development of cancer.

Q. Please describe laboratory studies that have specifically power frequency magnetic fields and leukemia.

A. Numerous laboratory studies have examined the relationship of exposure to power frequency magnetic fields and the initiation or promotion of leukemia. Near life long exposure to magnetic fields does not increase the risk of leukemia or lymphoma in animals. Mice with a hereditary predisposition to leukemia and rats exposed to ionizing radiation or transplanted leukemia cells do not develop leukemia sooner or a more severe form of the disease when exposed to magnetic fields.

Q. Would you summarize the conclusions you have drawn from your review of the literature regarding the risk of cancer from power lines?

A. Based on my assessment of the published literature, including the reports of nationally constituted scientific review groups, there is no convincing or consistent evidence that power lines pose a cancer risk.

Q. Does this conclude your testimony?

A. Yes.

DIRECT TESTIMONY OF KATHLEEN SHANLEY AND ROBERT CARBERRY
CONCERNING POWER-FREQUENCY ELECTRIC AND MAGNETIC FIELDS

Q. Would you please identify yourself and the other members of the panel who will respond to cross examination?

A. We are Kathleen Shanley, employed by The United Illuminating Company ("UI") as Process Leader of Environmental, Safety and Real Estate and Robert Carberry from Northeast Utilities ("NU"), Project Director for the Bethel to Norwalk Transmission Project. Other UI employees and NU employees may also be called upon to respond to questions that may require knowledge of specific topics.

Q. Ms. Shanley and Mr. Carberry, what is the purpose of your testimony?

A. The purpose of our testimony is to summarize the activities, policies, and efforts of UI and The Connecticut Light & Power Company ("CL&P") (collectively, the "Companies") to address their customers' concerns regarding electric and magnetic fields ("EMF"). We are submitting joint testimony. Ms. Shanley is the witness with respect to UI-specific information and Mr. Carberry is the witness with respect to CL&P-specific issues. Both of us will testify with respect to general matters that are common to our experience.

Q. Please describe your responsibilities with respect to EMF.

A. (Ms. Shanley) I am the primary contact with UI's customers who have concerns regarding EMF. For example, if UI's Client Relations Center receives a call from a customer regarding EMF, the UI representative directs the customer's call to me. I will then speak with the customer and answer the customer's questions. Additionally, I have conducted periodic educational classes for the Client Relations Center representatives so they can properly identify a call concerning EMF and know that such calls should be directed to me. Periodically, I am asked to speak before business,

neighborhood or social organizations in order to explain electric and magnetic fields and their relationship with AC power lines or devices supplied with AC electricity. I have conducted many spot measurements of magnetic fields on customer property and have directed others within UI to conduct similar measurements for customers. Since 1990, I have represented UI on various task forces concerning EMF such as the Edison Electric Institute's ("EEI") EMF Task Force, advised the Electric Power Research Institute ("EPRI") on areas of research to solicit proposals from scientists and engineers and universities and research organizations, and attended numerous seminars and meetings sponsored by EEI, EPRI and other organizations to keep informed of developments. I have represented the United States on the EMF issue at international meetings of the electric utility industry. I have served as UI's expert on EMF, as a witness and measurements reporter for Connecticut Siting Council proceedings, as an advisor to the Connecticut Interagency EMF Task Force, and a communicator on EMF issues for UI with legislators, the media, customers, employees and external organizations.

A. (Mr. Carberry) Until I took my present position managing the Bethel-Norwalk Transmission Project, I led NU's EMF Task Force and the development and implementation of NU's EMF policies. In fulfilling this responsibility, I served on EEI's EMF Task Force, provided local assistance to EPRI contractors performing EMF research and attended numerous seminars and meetings sponsored by EEI, EPRI and other organizations to keep informed of developments. I have served since 1975 as NU's transmission engineering expert on EMF, as a witness and measurements reporter for Siting Council proceedings and for litigation, as an advisor to the Connecticut Interagency EMF Task Force, and a communicator on EMF issues for NU with legislators, the media, customers, employees and external organizations. Like Ms. Shanley, I have spoken with and provided information resources to many customers who have called CL&P with EMF questions, and I have conducted many spot

measurements of magnetic fields on customer properties. Members of NU's EMF Task Force have also responded to customer questions and have made measurements for customers under my direction.

Q. Do the Companies follow the Council's Electric and Magnetic Field Best Management Practices in designing new electric transmission lines?

A. Yes. Each Company's transmission and substation projects comply with the Connecticut Siting Council's Electric and Magnetic Field Best Management Practices and the prevailing standards regarding EMF in the electric industry.

Specifically, the Companies incorporate into the design of new construction practical ways to reduce exposure to EMF (provided that the measures are consistent with environmental, safety, and engineering factors). These measures might include the installation of taller poles, closer spacings between wires, optimal phasings of the conductors in closely parallel lines, and longer distances from equipment or increased buffer space in the case of substations. Design alternatives are modeled in order to optimize magnetic field reductions, while balancing the relevant environmental, safety and engineering factors. Measurements are taken prior to and after construction using a uniform measurement protocol.

Q. Is EMF a concern to the Companies?

A. Because EMF issues are a concern to some of our customers, it is a concern to us. Additionally, the safety of our employees who work near electric lines and equipment is very important to the Companies, and therefore we monitor developments on the issue. We address our customers' concerns and questions diligently. We keep aware of developments in the scientific literature, particularly major agency review efforts.

Q. What types of efforts do the Companies undertake to address the concerns of their customers about EMF?

A. Generally, since the early 1980's, the Companies have undertaken various activities regarding EMF, including distribution of information, the taking of magnetic field measurements, support of research, and project siting and design.

First, the Companies make certain that their customers have access to information concerning EMF. The Companies have asked a scientist with expertise in EMF to address specific questions regarding EMF and health at public meetings and open houses. The Companies believe these educational efforts will provide the foundation for informed decisions by our customers, and the Companies are committed to this effort.

In addition to our personal efforts to answer customer questions, the Companies refer customers to sources where additional information concerning EMF can be obtained. These sources include, among others, the National Institute of Environmental Health Sciences ("NIEHS"), the International Agency for Research on Cancer, the International EMF Project of the World Health Organization, Connecticut's Interagency Task Force on Electric and Magnetic Fields, and the State of Connecticut Department of Public Health. Both Companies make available specific, current information on EMF from many sources to customers and employees interested in furthering their understanding of the issue.

As noted above, UI and CL&P each have employees with responsibility for following the EMF issue. Both Companies have created internal EMF task forces that are responsible for monitoring developments, providing information, responding to customer and employee questions, and recommending actions to company management. Both Companies have employees who have served on national and state advisory boards on EMF, attended numerous seminars and other meetings sponsored

by EEI, EPRI and other organizations to keep informed on developments and have offered testimony on a national and state level.

Second, as part of the Companies' public information efforts, representatives from each Company take measurements of magnetic fields for customers and employees upon request. We have visited dozens of homes, businesses and other locations and have conducted numerous magnetic field readings. The Companies believe that customers should be provided with as much information concerning electric and magnetic fields in their environment as they wish to receive so they can understand the sources of EMF in their environment and, if they choose, help them to learn how to manage their exposure where possible.

Third, the Companies have supported, and continue to support, independent research on EMF. For example, we contributed to the national EMF RAPID program during the 1990's and have long supported the work of scientists at universities and other research institutions through contributions to EPRI's EMF research program. The topics of research have included field management, source characterization, exposure assessment, residential and occupational epidemiology, and laboratory studies.

Finally, the Companies have responded to concerns of their customers by incorporating into the design of new construction practical and prudent ways to reduce exposure to EMF (consistent with environmental, safety, and engineering factors). These measures might include the installation of taller poles, closer spacings between wires, optimal phasing of the conductors in closely parallel lines, and longer distances from equipment or increased buffer space in the case of substations. Each Company's new transmission and substation projects comply with the Connecticut Siting Council's Electric and Magnetic Field Best Management Practices and the prevailing standards regarding EMF in the electric industry.

UI and CL&P endeavor to deliver electricity to our customers safely, reliably, and in an environmentally sound and responsible manner at a reasonable cost. The question of the possibility of health effects from EMF exposure continues to be followed closely by the Companies. This enables us to respond to our customers and employees and to monitor design considerations as well.

Q. Have the Companies designed the Project in a way that manages EMF exposure?

A. Yes. As set forth in the testimony of Dr. William Bailey, the Companies have applied several design and siting measures to manage field levels and exposures associated with the Project. As Dr. Bailey notes, this includes the Companies' offer to work with organizations with property or buildings next to the proposed route where children or other special groups might congregate, to reduce field levels outside the right-of-way. Specifically, the Companies have listened to the concerns of community members at meetings and hearings and are actively studying the possibility of relocating several transmission poles to increase the distance from the transmission conductors to existing buildings and/or increasing the height of the poles or making other changes to the configurations of existing and proposed lines in order to mitigate EMF exposures.

Q. Do the Companies take similar actions with respect to requests from individual customers?

A. Yes. The Companies consider requests from customers for information as to how they could mitigate the EMF at their home or business. While the Companies do not frequently receive such requests, they endeavor to provide information that would be useful to the customer.

Q. Does this conclude your testimony?

A. Yes.

Exponent®

Pre-Filed Testimony of Dr. William H. Bailey
Middletown-Norwalk Transmission Project

Q. What is your name and business address?

A. My name is William H. Bailey. My business address is Exponent®, 420 Lexington Avenue, Suite 408, New York, NY 10170.

Project Role and Testimony

Q. What is Exponent's role in this project?

A. We are providing consulting services to Connecticut Light and Power and The United Illuminating Company (CL&P/UI) relating to 60-Hertz electric and magnetic fields (EMF). For the Application to the Connecticut Siting Council (CSC), we projected the effect of the operation of the proposed and existing transmission lines on EMF levels along proposed and alternative routes based on calculations (Exponent, 2003[1]). We recently updated these values based upon updated modeling of system load flows provided by the companies **Exhibit A**[2]. We also have provided an overview of current research on EMF and health and assisted CL&P/UI in complying with the CSC's Electric and Magnetic Fields Best Management Practices. In addition, we have assisted the Companies in responding to data requests relating to EMF.

Q. What is the purpose of your testimony?

A. The purpose of my testimony is to summarize Exponent's projections on the effect of the operation of the proposed Middletown-Norwalk transmission line on 60-Hz electric and magnetic field (EMF) levels. I will also summarize our evaluation and review those of multidisciplinary organizations that have evaluated current scientific research on EMF and health, which are contained in our report (Exponent, 2003).
I also will provide additional commentary on issues that have concerned some members of the public.

---

[1] "Electric and Magnetic Field Assessment: Middletown-Norwalk Transmission Reinforcement". Exponent, October 3, 2003 in Volume 6 of the Application to the Connecticut Siting Council for a Certificate of Environmental Compatibility and Public Need for a 345-kV Electric Transmission Facility and Associated Facilities.

[2] Excerpted from Letter from A. Bartosewicz and J. J. Prete to Pamela B. Katz dated March 15, 2004.

Training and Experience

Q. What is your position at Exponent?

A. I am a Principal Scientist and Director of Exponent's New York office.

Q. Please describe your current responsibilities and professional experience.

A. Exponent, Inc. is a research and consulting firm engaged in a broad spectrum of activities in science and technology. I work primarily in the practices that specialize in exposure assessment and health sciences. My work involves reviewing, analyzing, and conducting research. One of the areas in which I have done a great deal of work over the past 20 years relates to potential biological and health effects of electrical facilities, such as transmission lines, substations, and electrified railroad lines.

Q. Please summarize your academic appointments.

A. Since 1986, I have been a visiting research scientist at the Cornell University Medical College. I also have been a visiting lecturer at Rutgers University, the University of Texas (San Antonio), and the Harvard School of Public Health. From 1983-1987, I was head of the Laboratory of Neuropharmacology and Environmental Toxicology at the New York State Institute for Basic Research. For the nine previous years, I was an Assistant Professor and Postdoctoral Fellow in Neurochemistry at The Rockefeller University.

Q. Please outline your scientific and research experience concerning electric and magnetic fields.

A. I have studied and conducted research on EMF for the past 20 years. My research has included exposure assessment, laboratory studies, and epidemiological studies concerning alternating current (AC) electric and magnetic fields, studies on direct current (DC) electric fields, and air ions.

Q. What are electric and magnetic fields?

A. Electric and magnetic fields associated with the operation of AC power lines or devices supplied with AC electricity are often referred to as EMF. These fields may be imagined as invisible lines of force in space near their electrical source. The voltage, which is the "pressure," produces an electric field that moves the electricity through wires. The standard unit for measuring the strength of an electric field is "volts per meter," abbreviated as V/m. The current produces a magnetic field, which is a measure of how much electricity is flowing. The unit in which magnetic field levels are measured is "milligauss," abbreviated as mG. Electric and magnetic fields are characterized by the frequency at which their direction and magnitude oscillate each second. The fields produced by the use of electricity oscillate at a frequency of 60 cycles-per-second (60-Hertz).

Q. Have you served as a reviewer and scientific policy advisor on health-related issues for state and federal agencies or scientific organizations?

A. Yes. I have reviewed research for the National Institutes of Health, the National Science Foundation, and other government agencies. Concerning transmission lines in particular, I served on a Scientific Advisory Panel convened by the Minnesota Environmental Quality Board to review health aspects of a high voltage transmission line. I also served as a consultant to the Vermont Department of Public Service, the New York State Department of Environmental Conservation, and staffs of the Maryland Public Service Commission and the Maryland Department of Natural Resources on transmission line issues.

I have worked with the National Institute of Occupational Health and Safety, the Oak Ridge National Laboratories, the U.S. Department of Energy, and the Federal Railroad Administration to review and evaluate health issues related to electric and magnetic fields from other sources. I also assisted the U.S. EMF Research and Policy Information Dissemination Program (RAPID) to evaluate biological and exposure research as part of its overall risk assessment process.

Most recently, I worked with other scientists from 10 countries to evaluate possible hazards from exposures to static and extremely low frequency (ELF) electric and magnetic fields for the International Agency for Research in Cancer (IARC), a division of the World Health Organization in Lyon, France (IARC, 2002).

Q. Have you presented the results of your research in this and other areas to the scientific community?

A. Yes. I have published or presented more than 50 scientific papers on this and related subjects.

Q. Are you a member of any professional organizations?

A. I am a member of The Rockefeller University Chapter of Sigma Xi, a national scientific honor society; the Health Physics Society; the International Committee on Electromagnetic Safety, Subcommittees 3 and 4 - Safety Levels with Respect to Human Exposure to Fields; the Bioelectromagnetics Society; the IEEE Power Engineering Society - Field Effects Guide Design Task Force; the IEEE Engineering in Medicine and Biology Society; the American Association for the Advancement of Science; the New York Academy of Sciences; the Society for Neuroscience; the Air & Waste Management Association; and the Society for Risk Analysis.

Q. Are your educational and professional experience summarized elsewhere?

A. Yes. Additional details of my educational and professional experience are summarized in my curriculum vitae, which is filed with this testimony.

Q. Have you ever appeared as a witness before regulatory agencies?

A. Yes. I have testified before public utility commissions in Vermont and Maryland as an independent expert witness engaged by these commissions. I have also testified before public utility commissions and siting boards in Arizona, Arkansas, Connecticut, Illinois, Massachusetts, Rhode Island, and Pennsylvania as an independent expert witness engaged by a party.

Project EMF and their Relation to Safety Standards and Guidance for Siting Transmission Lines

Q. What are typical sources of 60-Hz electric and magnetic fields?

A. Typical sources of these fields include power lines (both transmission and distribution lines), home and office appliances, tools, building wiring, and electric currents flowing on water pipes. The importance of these sources to overall exposure varies considerably. For example, if a residence is very close to a transmission line, or even a distribution line (which runs near most everyone's residence), these could be the dominant, but not necessarily the only, sources of magnetic fields in the home.
Depending on the circumstances, other sources may be of equal or greater importance than power lines. For example, a random survey of 1,000 residences in the U.S. reported that electric currents flowing on water pipes and on other components of house grounding systems are twice as likely as outside power lines to be the source of the highest magnetic fields measured in homes (Zaffanella, 1993).

Q. How was the effect of the proposed 345-kV transmission line on EMF levels determined?

A. The expected electric and magnetic field levels produced by the proposed and existing transmission lines in the year 2007 were calculated by my associate, Dr. Gary Johnson. His curriculum vitae is also filed with this testimony.

The model he used to calculate electric and magnetic fields was developed by the U.S. Department of Energy, Bonneville Power Administration, and has been validated and used by engineers and scientists for many years. The inputs to the model are line voltage, load flow, and the physical dimensions of the line (conductor spacing and height). The field values were calculated at a reference height of one meter above ground. For modeling purposes, it was assumed that the maximum voltage of this circuit was 5% above nominal values. The magnetic field from existing transmission lines and the proposed transmission line along the proposed route was calculated for average loading (current flow) under normal operating conditions. The magnetic field was also calculated for peak load flow conditions for normal system operating conditions.

Dr. Johnson calculated electric and magnetic fields along transects perpendicular to the transmission lines at the point of minimum ground clearance for each of eight sections of

the proposed route and 14 sections of alternative routes between the Middletown and Norwalk substations. The updated values are given in **Exhibit A** attached to this testimony.

Q. In general, what would be the effect of the proposed 345-kV transmission line on EMF levels?

A. The proposed project Middletown-Norwalk right-of-way (ROW) will affect ambient levels of electric and magnetic fields, with the greatest effect within the boundaries of the right-of-way. Outside the boundaries of substation sites and the right-of-way, the effect of the project on EMF levels will be limited because of the design and the location of the substations; the proposal to expand the right-of-way in some sections; and, between the East Devon and Norwalk Substations, the proposed placement of the 345-kV line underground.

Despite the addition of a 345-kV overhead transmission line to the existing rights-of-way, the electric field will be lower along one or both right-of-way edges for five of the eight sections of the primary overhead route (Cross Sections 1-8, Table 4; Exponent, 2003). The changes in electric field level (increase or decrease) at the ROW edge would be less than 0.8 kV/m except for one route section. No change in the electric field will occur on sections where the line would be placed underground on the proposed route (Section 9), Alternative A (Section 9A) or Alternative B (Section 10).

Similarly, the magnetic field will be lower along one or both right-of-way edges for some of the overhead sections of the primary route and increased on others (Cross Sections 1-8, **Exhibit A**). The contribution of the HPFF underground lines and XLPE underground lines to magnetic fields on streets directly above the lines would be less than 3 mG or 40 mG, respectively. At 20 feet from theses lines the magnetic field would diminish to less than 1 mG and less than 4 mG, respectively.

The "supported changes" to the proposed route that arose from the public consultation process involve the relocation of a 115-kV line to an underground XLPE line. This may lower the electric field, particularly for Alternative A on Cross Section 8. The effects of the "supported changes" on magnetic field levels are mixed, producing increases and decreases on the edge of right-of-way values on Section 8 and increases on Section 7B.

On the Alternative Route A (Cross Sections 9A and 17-22) and Alternative Route B (Cross Sections 10-22), the addition of a 345-kV overhead line has no great effect on the electric field at the ROW edge of most Alternative cross sections. However, along the East/South edge of the ROW of Sections 20-22, closest to Norwalk on Alternative A and B, the electric field increases by just over 2 kV/m. The magnetic fields from existing overhead lines tend to be higher along the right-of-way edges of Alternative routes than those on the proposed overhead route but the addition of the 345-kV line reduces the magnetic field on one or both sides of the right-of-way on 8 of 12 alternative cross sections. The addition of the proposed line will result in increases and decreases in the magnetic field; on some sections the change will less and on some sections the changes will be greater than 20 or 30 mG at the right-of-way edge.

At distances greater than approximately 100 feet from edges of the proposed right-of-way, however, the differences between the levels of fields produced by the lines in existing and future configurations become smaller.

Q. In Exponent's calculations of the effect of the proposed Middletown-Norwalk transmission line on EMF levels, were any sources of electric or magnetic fields other than overhead or underground transmission lines considered?

A. No, only the electric and magnetic field levels produced by transmission lines were calculated. The levels produced by existing transmission lines were compared to those produced by both the proposed line and the existing (or modified) transmission lines in the same time period.

Q. Will the addition of the proposed Middletown-Norwalk transmission line cause the electric field levels to exceed any applicable safety standards?

A. No. The proposed line is designed by CL&P/UI to meet the requirements of the National Electric Safety Code regarding electric field induction to large vehicles under the conductors (NESC, 2000).

Q. Have any other safety or health-based standards been recommended to limit exposures to electric or magnetic fields?

A. There are no federal standards for either 60-Hz electric or magnetic fields. However, there are general recommendations from scientific organizations regarding exposures to EMF for the general public and workers.

For example, an international organization recommends that the exposure of the general public to electric and magnetic fields at 60 Hz be limited to 4.2 kV/m and 83.3 μT (833 mG), respectively (ICNIRP, 1998). The International Committee on Electromagnetic Safety, of which I am a member, has published recommendations for field exposure (ICES, 2002). The limits are 5 kV/m for electric field exposures of the general public and 0.9 mT (9,040 mG) for magnetic field at 60 Hz. The purpose of these guidelines is to prevent exposures to electric fields that could produce contact shocks or magnetic fields that could stimulate tissues by induced electric fields.

Q. Have CL&P/UI designed the Middletown-Norwalk line to minimize EMF exposure?

A. Yes. The line was designed to minimize field levels in several ways:

- The proposal to operate the new line at 345 kV will result in lower current flow and hence a lower magnetic field than would occur had the proposed line been planned to deliver the same amount of power at a lower voltage. This is due to the fact: Power = Voltage x Current.

- Additional land devoted to an EMF source is minimized by locating the overhead sections of new line almost entirely on existing transmission line right-of-ways.

- The existing and proposed overhead lines are configured so that the fields from each line tend to mutually 'cancel' one another. The addition of the proposed line reduces magnetic fields on one or both sides of the right-of-way at a number of such sections of the route, e.g., Sections 1, 3, 4, and 8A of revised Table 5 of Exponent's report, **Exhibit A hereto).**

- An underground cable system proposed for a portion of the route, where existing rights-of-way are too narrow for overhead construction, effectively reduces magnetic fields by placing the phase conductors closer together.

- Finally, the companies have offered to work with organizations next to the proposed route, where large numbers of children or other special groups might congregate, to minimize field levels outside the right-of-way. Specifically, they are looking for ways to adapt the generic design of the overhead line to a specific site to minimize field levels.

Q. Is the CL&P/UI approach to addressing EMF consistent with the Connecticut Siting Council's Electric and Magnetic Field Best Management Practices?

A. Yes. Our report completes these requirements of the Siting Council, including documenting the steps taken to minimize field levels and update interested parties on the status of EMF research.

Scientific Evaluation of Research on EMF and Health

Q. Overall, has a great deal of research been done to assess the potential health effects of EMF?

A. Yes, and a large part of this research has been performed in the last 30 years, which means that the investigations have had the benefit of the modern advances in many fields of science and medicine. It is important to note that by comparison, more effort has been devoted to the study of EMF than to most of the 50,000 or so chemicals that are in everyday use.

Q. How do scientists assess the potential risks of exposure to EMF from power lines and other sources?

A. We consider all of the studies of different types on this topic because no study is perfect or provides all the answers. Each study has its strengths and weaknesses. Repeating a study is important because a reproducible result increases confidence that the observation is correct and not the result of some sort of unforeseen error.
Both epidemiology studies and experimental studies have drawbacks, but taken together the strengths and limitations balance one another. For example, epidemiological studies

of humans include people of varying health and background, but cannot obtain precise measurements of exposure. Experimental studies of animals can produce precisely measured exposures to similar animals fed the same diet – but after all, laboratory animals are not humans. When we consider both of these types of studies, we get more information than if we relied on only one type of study.

Q. Why are these different approaches needed?

A. No single study or approach is able to address all questions about what can affect our health. Epidemiology and laboratory studies are also considered together because the strengths of one type tend to balance the limitations of the other type of study. Therefore, scientists consider different studies, and different types of studies, to get more information to develop sound conclusions.

EMF and Cancer

Q. What was the assessment of the epidemiologic research that focused on EMF and cancer?

A. The results of the latest epidemiologic studies of childhood cancer do not provide sufficient or convincing evidence to support the hypothesis that exposure to electric or magnetic fields or power lines near the home are a cause of leukemia or other cancers in children. The larger or more reliable residential and occupational studies do not support the idea that fields in the residence or workplace contribute to the risk of cancer in adults. As regard to appliances, there was no discernable pattern to indicate that children with leukemia or other cancers had higher or longer exposures to fields from these sources.

EMF and Reproduction/Development

Q. What was the assessment of epidemiologic research that focused on EMF and reproduction and development?

A. Overall, there is little evidence from epidemiologic studies for an association between sources of EMF, including electric bed heating and power lines, and problems of reproduction and development. Two recent studies have suggested an association with maximum or peak magnetic field exposures and miscarriage but the interpretation of the association as reflecting a possible causal influence has been questioned because of several sources of bias.

EMF and Neurobiological Effects and Neurological Diseases

Q. What was the assessment of epidemiologic research on neurobiological effects and diseases?

A. There has been little new research on this topic since the assessment of the literature by the National Institute of Health Sciences except for continuing studies of occupational exposure. These studies, however, are difficult to interpret because of limitations in the

assessment of exposure to EMF and the methods for excluding effects of other risk factors.

Support from Experimental Studies for Hypotheses Based upon Epidemiologic Studies

Q. Has experimental research in laboratories provided solid support for any hypothesis linking EMF to cancer, reproduction/development, or neurobiological effects based upon the epidemiologic studies just discussed?

A. No. One of the major goals of the $60 million national EMF research program known as RAPID[3], begun in 1992 and completed in 1998, was to assess the biological plausibility of hypotheses derived from some EMF epidemiological studies. The results of research from this and other research programs around the world have tested hypotheses deriving from both epidemiology and experimental studies.

Regarding cancer, multiple studies report that animals exposed to magnetic fields to a wide range of levels (up to 50,000 mG) for most of their lifespan show no increases in cancer or other adverse health effects.

Large studies of laboratory animals exposed to magnetic fields have shown no increase in birth defects, no multigenerational effects, and no changes that would indicate an increase in miscarriage or loss of fertility or adverse effects on the growth and development of the embryo.

Experimental studies of both human subjects and animals exposed to EMF at levels much greater that would be found on or outside the proposed right-of-way do not demonstrate adverse effects on neurobehavioral functions. Every year, experimental support for a mechanism that had been proposed to account for such effects involving the neurohormone, melatonin, has been waning.

Evaluations of the EMF Research Literature by National and International Scientific Agencies

Q. Did your report summarize the evaluations of the EMF research literature by multidisciplinary groups of scientists who have reviewed the literature for international and national scientific agencies, such as IARC?

A. Yes. These evaluations were summarized in our report and Table 5 from our report is appended as **Exhibit B**.

Q. Did the multidisciplinary groups of scientists follow a similar evaluation process as you did?

[3] The Electric and Magnetic Fields Research and Public Information Dissemination Program

A. Yes. As a member of the Working Group that was assembled by the International Agency for Research on Cancer (IARC), I am particularly familiar with their evaluation process.

Q. What overall conclusion did you and others on the IARC Working Group reach regarding childhood leukemia?

A. The Working Group concluded that the epidemiologic studies do not provide support for an association between childhood leukemia and residential magnetic fields at intensities less than 4 mG. Overall, magnetic fields were evaluated as "possibly carcinogenic to humans" (Group 2B), based on the statistical association of higher-level residential magnetic fields with childhood leukemia. IARC reviewers also evaluated the animal data and concluded that they were "inadequate" to support a risk for cancer. We stated that the EMF data does not merit the category "carcinogenic to humans" or the category "probably carcinogenic to humans," nor did we find that "the agent is probably not carcinogenic to humans." Many hypotheses have been suggested to explain possible carcinogenic effects of electric or magnetic fields; however, no scientific explanation for carcinogenicity of these fields has been established (IARC, 2002).

In the rating system used by IARC, the recognition of an association between exposure and cancer in epidemiology studies is considered "limited evidence" of carcinogenicity. A rating of "limited evidence" for epidemiology studies, even without any evidence from laboratory studies that an exposure might pose a cancer risk, requires that the exposure be categorized as a "possible carcinogen," even though chance, bias and confounding cannot be ruled out with reasonable confidence (IARC, 2002).

Q. Why did the IARC Working Group not regard the association between magnetic fields and childhood leukemia as reflecting a causal relationship?

A. Because there was neither sufficient evidence from epidemiology studies that magnetic fields caused cancer in humans, nor sufficient evidence that magnetic fields caused cancer in laboratory studies of animals (or evidence for a mechanism to predict cancer).

Relationship of Magnetic Field Levels under Lines or at the Edge of Rights-of-Way to Personal Magnetic Field Exposures

Q. Are you aware of a concern about magnetic fields based upon measured or calculated magnetic field values near the line and reports of a statistical association between magnetic fields and childhood leukemia?

A. Yes, I have attended public meetings and read transcripts of other meetings where this concern was of paramount importance. While the concern is genuine, I believe that it has been fueled by a fundamental misunderstanding of the difference between *measured* or *calculated field values* on one hand and estimates of *exposure* on the other.

Q. Please explain.

A. A magnetic field value calculated or measured at a particular location at an instant in time is not a measure of human exposure.

What we mean by exposure are the magnetic field levels encountered by a person as averaged over a specific period of time. For example, it takes about a second to measure a magnetic field. Over that second, my exposure and the measurement would be the same, i.e., the value displayed on the meter. However, if I took readings every second over an entire year wherever I went, the average of those 31,536,000 measurements would represent my average annual exposure to magnetic fields in milligauss.

Hence, my exposure to magnetic fields reflects the contribution from all of the magnetic field sources that I encounter in all the locations where I spend time. Because people typically spend most of their time at home, the sources here are frequently the major determinant of their time-weighted-average (TWA) exposures.

The misunderstanding arises because both field values and exposures are both expressed in units of milligauss, though they represent quite different concepts.

Q. Could you provide an example of how a time-weighted-average value differs from a measured value at a particular location?

A. Yes. I have attached Figure 2 from our report as **Exhibit C** to illustrate the difference between a single measured value and a time-weighted average. The figure shows the magnetic field recorded at 10-second intervals during a two-hour walk through a Connecticut town and its stores. The fields at these locations range from 0 mG to almost 100 mG. Yet the time-weighted average of these measurements is 4.6 mG (Table 1, Exponent, 2003). Thus, no single measured value at any of the locations visited provides an estimate of the total exposure of the person to magnetic fields during the measurement period, as reflected in the average value, except by chance.

Q. What do the calculated magnetic field values at the edge of right-of-way given in Table 5 of Exponent's 2003 report represent?

A. They are calculated values at a particular location and cannot be meaningfully compared to estimates of long-term exposure to magnetic fields, as I have explained above.

Q. Why are estimates of long-term exposure to magnetic fields of interest?

A. They are of interest because the strongest data supporting the existence of any health effect from magnetic fields are reports of associations between estimates of magnetic field exposure and childhood leukemia (See discussion of the reviews by NIEHS, IARC, NRPB and HCN in Exponent's report). Based primarily upon two published statistical evaluations of epidemiology studies of childhood leukemia and long-term exposures (Greenland et al 2000; Ahlbom et al, 2000), members of the IARC Working Group concluded that there was "limited" evidence for an association between childhood leukemia and magnetic field exposures above 4 mG.[4] The Working Group also noted that "[i]t cannot be excluded that a combination of selection bias, some degree of confounding and chance could explain the results."

The IARC report appears to be the primary source of public concern about magnetic field exposures above 4 mG. Note that 4 mG refers to an estimated long-term TWA exposure. The goal of EMF epidemiology studies has been to estimate the average exposure of an individual over a long time such as a year, not the fluctuating level at any single spot, whether it is in the playground, a school, or a place in the home.

The reason for our focus on long-term exposures is based upon the knowledge that chemicals and agents in the environment that are known to cause cancer, e.g., tobacco smoke, alcohol, and sunlight, require repeated exposures at elevated levels over long periods of time. Therefore, scientists do not expect an association with these diseases to exist for persons with infrequent or very low-level exposures to these cancer-causing agents. The same rationale has been applied by epidemiologists in the study of magnetic fields; while opportunities for short-term exposures to magnetic fields in excess of a few mG or even 100 mG abound in our environment, the exposure of interest is a person's long-term average exposure.

Thus, a calculated or measured magnetic field greater than 4 mG in a playground, school or even a small area of a residence would not necessarily indicate that a child would have a TWA exposure greater than 4 mG, i.e. in the range of exposure where IARC scientists indicated an association with childhood leukemia. Bear in mind that this statistical association has not been considered by IARC or other agencies as sufficient evidence to conclude that there is a cause and effect relationship between magnetic field exposure and childhood leukemia (or other cancer/diseases), including the fact that there is neither a plausible mechanism nor a biologic basis to support such an association.

---

[4] The IARC Working Group concluded that the EMF data do not merit the category "carcinogenic to humans" or the category "probably carcinogenic to humans," nor did it find that "the agent is probably not carcinogenic to humans."

Q. How would one know whether the fields from the existing or proposed transmission lines would increase a child's TWA exposure to magnetic fields above 4 mG?

A. With CL&P/UI, we have begun to develop information to answer this question in relation to locations adjacent to existing transmission lines.
We have analyzed records from a national survey of personal magnetic field exposure sponsored by the U.S. EMF RAPID program to identify the typical magnetic field levels encountered by children in a range of environments throughout a 24-hour period (Zaffanella, 1993). The typical (median) TWA daily exposure of the children in this survey under the age of 14 was 0.6 mG.

To estimate what effect the presence of the existing transmission lines near a school would have on a student's TWA exposure to magnetic fields, we assumed that the exposures of children at school in the national survey would be replaced by exposures from the existing line. For example, we calculated magnetic fields from hourly records of current flow from September 1, 2001 to September 30, 2002 at a school building 15 feet from the transmission line right-of-way. This is a very conservative modeling assumption since the children are not expected to spend all of their schooling time in only one location where the field level is highest. From our hourly-calculated values of magnetic field and the time spent by children in school from the national survey, we constructed an exposure model for a student's TWA exposure to magnetic fields that included both school and non-school exposures over a day. The TWA daily average exposure of children over a year as estimated above is 1.3 mG. These analyses indicate that even conservative estimates of the TWA exposures from the existing lines at the selected location would not exceed a TWA value of 4 mG. Hence the estimated exposure of these children at this adjacent school would be below the level of magnetic field exposure where an association with childhood leukemia has been identified by IARC.

As to the potential contribution to TWA magnetic field exposure from the new transmission line at this location, our analyses are continuing. The new line could be located further away from such a building than the existing transmission lines. We have also identified several possible modifications to the design and location of the lines on the right-of-way that have been estimated to reduce the average level of the magnetic field below the levels produced by the existing transmission lines. CL&P/UI is examining these modifications to assess their feasibility. Should these estimates be realized, the contribution of all the transmission lines (existing and proposed) to the annual TWA exposure of children at such a location would be reduced below the contribution of the existing lines to their TWA.

The above analyses were performed to illustrate the concept that a measured or calculated value of over 90 mG on the right-of-way of the proposed transmission line or a calculated value at the edge of the right-of-way of 9 mG does not mean that the proposed transmission line would necessarily cause a child's TWA exposure to magnetic fields to exceed 4 mG, even if the school is a short distance away.

Q. In thinking about the concept of TWA exposure you just discussed, it would seem that sources of magnetic fields that students encounter indoors could more frequently contribute to their TWA exposures than do transmission lines outside the school. Is there any evidence that indoor sources at schools are important sources of magnetic fields?

A. Yes. A comprehensive survey of classrooms in California (Zaffanella and Hooper, 2000) reports that 17% had magnetic field levels that exceeded 1 mG, the average of the magnetic field readings in a typical home (Zaffanella and Kalton, 1998). In these classrooms, 10 major sources were identified and transmission lines ranked as ninth in importance (**Exhibit D**). This survey data clearly shows that indoor sources of magnetic field can have a far greater effect on children's exposure at school than transmission lines. In addition, we should keep in mind that a child's magnetic field exposures at school are only a small fraction of their total exposure. A national survey reports that children in school spend only about 27% their time there. Exposures encountered in the other 73% of their time, particularly at home, are the dominant component of their overall exposure to magnetic fields.

Q. Based upon your experience in working regulatory agencies and the EMF issues that have been raised in this proceeding do you have any recommendations to the Connecticut Siting Council?

A. Yes. I have two recommendations. First, the Council should expand the Council's Electric and Magnetic Fields Best Management Practices to include more site-specific planning to minimize magnetic fields as a way of responding to public concern.

At present, the generic Electric and Magnetic Field Best Management Practices approach works best where there are few or no closely adjacent community facilities, such as schools. However, additional site-specific assessments are needed to determine how best to fine-tune the overall design and location of lines at a specific location where large populations may spend significant time so as to achieve the greatest field reduction.

Second, while the Electric and Magnetic Fields Best Management Practices provides sound guidance to utilities for addressing EMF issues during the siting process, it cannot substitute for proactive and coordinated long-range planning of major transmission projects so as to minimize real or perceived conflicts with residential and other land uses. It would be helpful for the Council to require and enable the utilities to plan for such projects years and even decades in advance so that land for future transmission lines and substations could be acquired today and set aside for the future. In this way, it would be easier to site transmission lines as well as other linear utilities along pre-determined and perhaps common corridors, even in densely populated areas. This concept has been implemented in several Canadian cities in Greenbelt plans that maintain access to developing urban areas for future electric and gas transmission facilities.

Q. Based upon your assessment of the scientific evidence described in your testimony, would the operation of the proposed Middletown-Norwalk 345-kV transmission line pose a hazard to humans?

A. No, it would not. The weight of the evidence I have reviewed does not support a conclusion that exposure to EMF at the levels associated with the proposed project would have adverse effects on human health, compromise normal function, or cause cancer. This conclusion is based on my knowledge of the relevant scientific literature, the results of expert scientific panels that have examined epidemiologic and laboratory research on 60-Hz electric and/or magnetic fields and health, and the field levels anticipated from the operation of this transmission line. Together, this evidence does not support the conclusion that 60-Hz electric or magnetic fields associated with the operation of the proposed transmission line would pose a hazard to human health.

Q. Does this conclude your testimony?

A. Yes.

References

Ahlbom A, Day N, Feychting M, et al. A pooled analysis of magnetic fields and childhood leukemia. Brit J Cancer, 83:692-698, 2000.

Exponent. Electric and magnetic field assessment: Middletown-Norwalk transmission reinforcement, In: Application to the Connecticut Siting Council for a Certificate of Environmental Compatibility and Public Need for a 345-kV Electric Transmission Line Facility and Associated Facilities. Volume 6, 2003.

Greenland S, Sheppard AR, Kelsh MA, Kaune WT. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Epidemiology, 11:624-634, 2000.

Health Council of the Netherlands (HCN). ELF electromagnetic fields committee. electromagnetic fields: annual update 2001. The Hague: Health Council of the Netherlands. Publication No. 2001/14, 2001.

International Committee on Electromagnetic Safety (ICES). IEEE standard for safety levels with respect to human exposure to electromagnetic fields 0 to 3 kHz C95. 6-2002.

International Commission on Non-Ionizing Radiation Protection (ICNIRP). Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Physics, 74: 494-522, 1998.

International Agency for Research on Cancer (IARC). IARC Monographs on the evaluation of carcinogenic risks to humans. Volume 80: Static and extremely low-frequency (ELF) electric and magnetic fields. IARC Press, Lyon, France, 2002.

National Electric Safety Code (NESC). IEEE Standard NESC C-2, 2000.

National Institute of Environmental Health Sciences (NIEHS). Assessment of health effects from exposure to power-line frequency electric and magnetic fields: working group report. NIH Publication No. 98-3981. Research Triangle Park, NC: National Institute of Environmental Health Sciences of the U.S. National Institutes of Health, 1998.

National Institute of Environmental Health (NIEHS). Health effects from exposure to power line frequency electric and magnetic fields. NIH Publication No. 99-4493. Research Triangle Park, NC: National Institute of Environmental Health Sciences of the U.S. National Institutes of Health, 1999.

National Radiological Protection Board (NRPB). Documents of the NRPB. Volume 12. ELF electromagnetic fields and the risk of cancer. Chilton, Didcot, Oxon OX11 0RQ, 2001.

Zaffanella LE. Survey of residential magnetic field sources. Volume I. Electric Power Research Institute, EPRI TR-102759-V1, Project 3335-02, Final Report, September, 1993.

Zaffanella LE and Kalton GW. Survey of personal magnetic field exposure: phase ii 1000-person survey – final report. RAPID Program, 1998.

Zaffanella LE and Hooper HC. Electric and magnetic field exposure assessment of powerline and non-powerline sources for california public school environments. Enertech Consultants. Campbell, CA, 2000. (See also: California Department of Health, EMF Program, http://www.dhs.cahwnet.gov /ehib/emf/school_exp_ass_exec.pdf.)

Exhibit A. Edge of right-of-way magnetic field values for existing, proposed, and alternative line configurations

2007 annual average loading (15 GW)

Cross Section	Existing Magnetic Field (mG)		Route	Proposed Magnetic Field (mG)	
	East/South* ROW	West/North# ROW		East/South ROW	West/North ROW
Proposed 345-kV Overhead Route					
1	32.6	33.8	Proposed	29.0	18.7
			Alternative A	22.9	18.1
			Alternative B	29.8	17.5
2	9.2	13.9	Proposed	30.4	17.1
			Alternative A	29.6	16.5
			Alternative B	29.8	16.6
3	12.2	4.7	Proposed	5.9	12.9
			Alternative A	6.0	14.2
			Alternative B	5.5	15.0
4	6.1	11.9	Proposed	5.3	11.5
			Alternative A	5.4	13.1
			Alternative B	5.4	14.2
5	5.2	24.7	Proposed	15.9	27.8
			Alternative A	14.3	27.1
			Alternative B	13.2	26.4
6	0.2	1.2	Proposed	5.4	14.3
			Alternative A	4.7	12.3
			Alternative B	4.1	10.9
7 and 7a	0.4	4.4	Proposed	11.9	10.2
			Alternative A	10.2	9.0
			Alternative B	9.1	8.4
8 and 8b	6.2	2.8	Proposed	8.7	15.7
			Alternative A	7.6	13.5
			Alternative B	6.8	12.0
"Supported Changes" – 345-kV Overhead and Relocation of 115-kV to Underground					
7b *(25')*▽	0.4	4.4	Proposed	6.2	17.9
8a *(-20')*Ψ	6.2	2.8	Proposed	5.0	16.0
*(-400')*Ψ	6.2	2.8	Proposed	5.0	16.0
Proposed and Alternative Underground Line Routes+					
9 (HPFF)	- na -	- na -	Proposed	0.2	0.2
(East Devon to Singer)				0.2	0.2
(Singer to Norwalk)					
9A (XLPE)	- na -	- na -	Alternative A	1.1	1.0
(East Devon to Singer)				3.6	3.3
(Singer to Hawthorne)					
10 (XLPE)	- na -	- na -	Alternative B	2.4	3.2
(Singer to Seaview Loop)					

Cross Section	Existing Magnetic Field (mG)		Route	Proposed Magnetic Field (mG)	
	East/South* ROW	West/North# ROW		East/South ROW	West/North ROW
Alternative 345-kV Overhead Line Routes					
11	2.3	8.5	Alternative B	3.2	7.4
12	7.1	30.9	Alternative B	8.0	25.8
13	2.8	1.5	Alternative B	4.9	8.3
14	48.4	5.2	Alternative B	22.5	9.6
15	62.2	59.9	Alternative B	22.5	16.4
16	55.7	51.2	Alternative B	12.5	22.6
17	40.8	40.9	Alternative A Alternative B	23.9 14.2	36.0 26.7
18	29.4	41.0	Alternative A Alternative B	31.0 27.5	39.9 34.7
19	57.1	8.7	Alternative A Alternative B	30.7 26.9	14.4 9.4
20	48.7	4.9	Alternative A Alternative B	75.9 67.0	13.1 9.0
21	13.1	5.9	Alternative A Alternative B	45.3 40.0	13.1 9.0
22	42.9	11.1	Alternative A Alternative B	75.9 67.0	13.1 9.0

* Identified in NU documentation as left ROW

\# Identified in NU documentation as right ROW

∇ Distance from edge of ROW. +25' indicates 25' outside of the right (West/North) ROW.

Ψ Distance from edge of ROW. -20' *(or –400')* indicates 20' *(or 400')* outside of the left (East/South) ROW

\+ ROW edge taken as –20' left (East/South) ROW and +20' right (West/North) ROW

Exhibit B. Conclusions of international agencies and scientific groups

Agency or Scientific Group	Conclusions
National Institute of Environmental Health Sciences (NIEHS, 1998; NIEHS, 1999)	"The scientific evidence suggesting that ELF-EMF exposures pose any health risk is weak. The strongest evidence for health effects comes from associations observed in human populations with two forms of cancer: childhood leukemia and chronic lymphocytic leukemia in occupationally exposed adults . . . In contrast, the mechanistic studies and animal toxicology literature fail to demonstrate any consistent pattern . . . No indication of increased leukemias in experimental animals has been observed . . . The lack of consistent, positive findings in animal or mechanistic studies weakens the belief that this association is actually due to ELF-EMF, but it cannot completely discount the epidemiological findings . . . The NIEHS does not believe that other cancers or other non-cancer health outcomes provide sufficient evidence of a risk to currently warrant concern."
International Agency for Research on Cancer (IARC, 2002)	"Studies in experimental animals have not shown a consistent carcinogenic or co-carcinogenic effects of exposures to ELF [extremely low frequency] magnetic fields, and no scientific explanation has been established for the observed association of increased childhood leukaemia risk with increasing residential ELF magnetic field exposure." IARC categorized EMF as a "possible carcinogen" for exposures at high levels, based on the meta-analysis of studies of statistical links with childhood leukemia at levels above 3-4 mG.
National Radiological Protection Board of Great Britain (NRPB, 2001b)	"Laboratory experiments have provided no good evidence that extremely low frequency [ELF] electromagnetic fields are capable of producing cancer, nor do human epidemiological studies suggests that they cause cancer in general. There is, however, some epidemiological evidence that prolonged exposure to higher levels of power frequency magnetic fields is associated with a small risk of leukemia in children. In practice, such levels of exposure are seldom encountered by the general public in the UK [or in the US]."
Health Council of the Netherlands (HCN, 2001)	"Because the association is only weak and without a reasonable biological explanation, it is not unlikely that it [an association between ELF exposure and childhood leukemia] could also be explained by chance... The committee therefore sees no reason to modify its earlier conclusion that the association is not likely to be indicative of a causal relationship."

Exhibit C. Typical personal exposures to magnetic fields

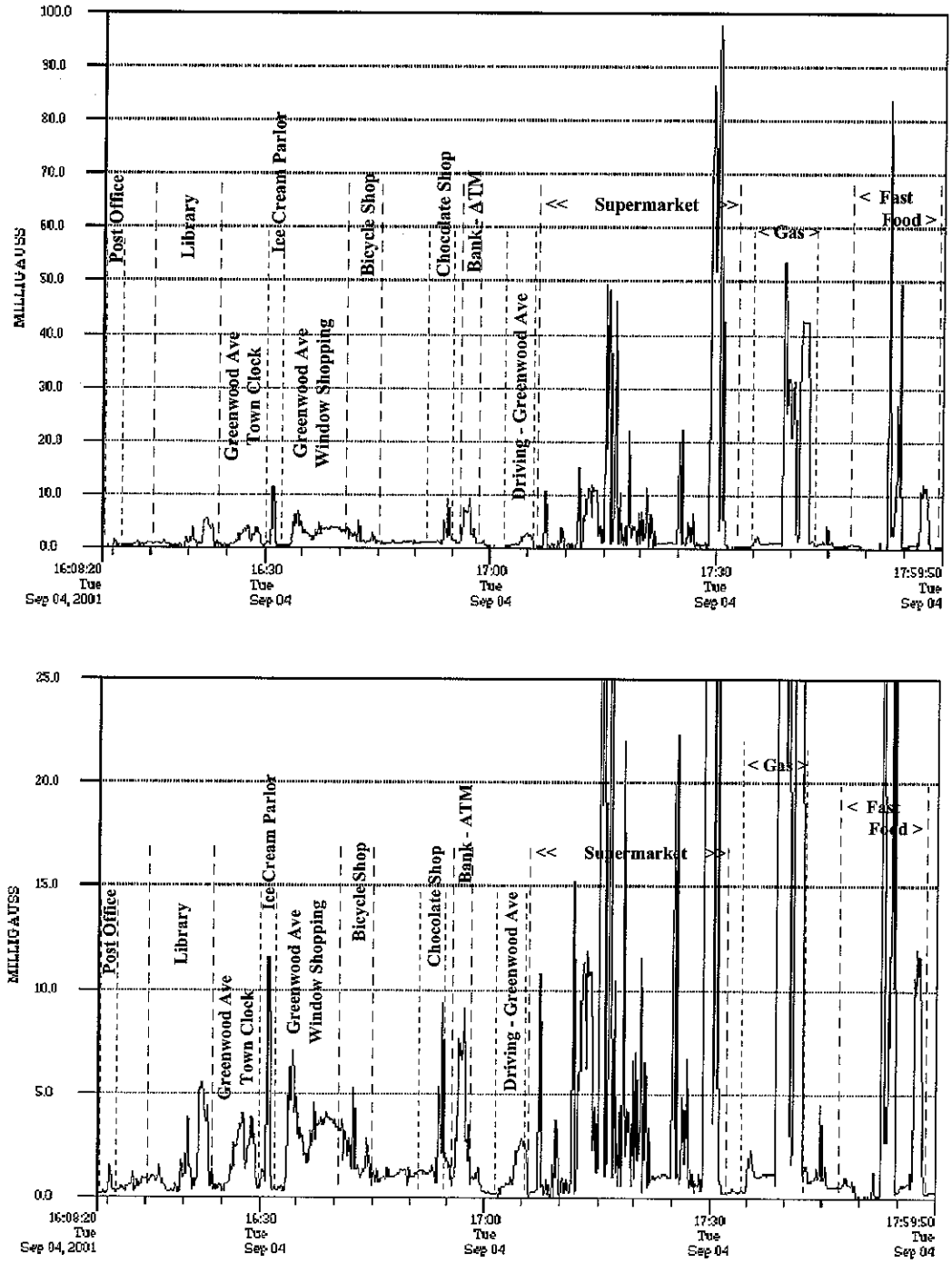

Exhibit D. Number of classrooms in which different sources cause a field greater than given value of 1 mg in more than 5% of the area

Field Source	>1 mG
Net Current	61,000
Electrical Panel	13,800
Fluorescent Lights	11,500
Distribution Line	6,100
Office Equipment	6,000
Power Transformer	2,200
Air Conditioners	2,200
Power Cable	1,700
Transmission Line	1,300
Current in Water Main	0

Adapted from: Enertech, Inc., Electric and Magnetic Field Exposure Assessment of Powerline and Non-Powerline Sources for California Public School Environments, Executive Summary. Accessed from California Department of Health, EMF Program, http://www.dhs.cahwnet.gov/ehib/emf/school_exp_ass_exec.pdf

CURRICULUM VITAE

Name: Stuart A. Aaronson

Date and Place of Birth: February 28, 1942, Mt. Clemens, Michigan

Citizenship: U.S.A.

Marital Status: Married, three children

Education and Training:

1959-1962	B.S. (Chemistry; summa cum laude), University of California, Berkeley
1962-1966	M.D., University of California Medical School, San Francisco
1965-1966	Fellowship, Dept. of Biochemistry, University of Cambridge, Cambridge, United Kingdom
1966-1967	Intern, Medicine, Moffitt Hospital, San Francisco

Brief Chronology of Employment:

1967-1969	Staff Associate, Viral Carcinogenesis Branch, National Cancer Institute, Bethesda, MD
1969-1970	Senior Staff Fellow, Viral Carcinogenesis Branch
1970-1977	Head, Molecular Biology Section, Viral Carcinogenesis Branch
1977-1993	Chief, Laboratory of Cellular and Molecular Biology, National Cancer Institute, Bethesda, Maryland
1993-present	Director, Ruttenberg Cancer Center, Mount Sinai Medical Center, New York, NY & Jane B. and Jack R. Aron, Professor of Neoplastic Diseases

Medical Licenses

New York
Virginia

Honors and Awards:

1962	Phi Beta Kappa
1966	Alpha Omega Alpha
1982	Rhoads Memorial Award
1982	PHS Meritorious Service Medal
1989	Paul Ehrlich Award
1989	PHS Distinguished Service Medal
1990	Milken Award
1991	Chirone Prize
1991	Harvey Lecture
1991	Wadsworth Memorial Foundation Award

Societies:

American Society for Microbiology
American Association for the Advancement of Science
Society for Experimental Biology and Medicine
American Association for Cancer Research, Inc.
American Society for Virology, Inc.

Memberships and Affiliations:

1975-1978	Member, Viral Cancer Program Coordinating Committee
1975-1976	Ad Hoc Member, Experimental Virology Study Section, NIH
1975-1978	Member, Viral Oncology Scientific Advisory Committee for FCRC
1976-1980	Member, Experimental Virology Study Section, NIH
1977-present	Member, Editorial Board, International Journal of Cancer
1977-1986	Associate Editor, Journal of the National Cancer Institute
1980-1985	Editorial Advisory Board, Biochimica et Biophysica Acta (BBA Reviews on Cancer)
1981-present	Associate Editor, Cancer Research
1983-1992	Executive Committee, Duke Comprehensive Center, Duke University Medical Center
1984	Mott Selection Committee, General Motors Cancer Research Foundation
1984-1990	Advisory Committee, Maimonides Conferences on Cancer Research
1984-1990	Editorial Board, Virus Research
1984-1987	Scientific Advisory Committee, American Cancer Society
1985-1987	External Scientific Review Committee, Comprehensive Center, The University of Alabama in Birmingham
1985-present	Editorial Advisory Board, Cancer and Metastasis Reviews
1985-present	Editorial Board, Cancer Reviews
1985-1989	Councillor, Society for Experimental Biology and Medicine
1985-1990	Extramural Advisory Board, Cancer Center, The University of Arizona
1986	Program Chairman, American Association of Cancer Research
1986	Co-organizer, Princess Takamatsu Symposium
1986-present	Guest Editor, Japanese Journal of Cancer Research (Gann)
1986-present	Editorial Board, Environmental and Occupational Health Sciences
1986-1987	Member, Advisory Committee, American Type Culture Collection
1987-1989	Editorial Advisory Board, Molecular Endocrinology
1987-present	Editorial Board, Oncogene
1988-1989	Advisory Editorial Board, ISI Atlas of Science: Biochemistry
1988-1994	Member, Blood Services Scientific Council, American Red Cross
1989-1991	Editorial Board, Cancer Communications
1989-1992	Editorial Board, The New Biologist
1989	Visiting Professor, University of Texas, San Antonio
1990-2002	Advisory Board, BBA Reviews on Cancer, Biochimica et Biophysica Acta

1990	General Motors Visiting Professor, University of Wisconsin-Madison Medical School
1990	Visiting Professor, Jonsson Comprehensive Cancer Center, University of California, Los Angeles
1992-present	Editorial Board, Intl. Journal of Oncology
1991-2003	Editorial Board, Oncology Research
1992-present	Scientific Advisory Board, - Georgetown Univ – Breast Ca SPORE
1993-1995	Editorial Advisory Board, Molecular Aspects of Medicine
1994-present	International Advisory Board, Tumori
1995-1996	Vice President, Harvey Society
1996-1997	President, Harvey Society
1997-1998	Counselor, Harvey Society
1998-present	Member, Public Relations and Communications Committee, AACR
1998-present	Member, The National Neurofibromatosis Foundation Research Advisory Board
1998-present	Member, External Scientific Avisory Committee, Kimmel Cancer Center, Thomas Jefferson University
2003-present	Editorial Board, Cancer Genomics and Proteomics (CGP)

Research Interests:

Molecular genetics of cancer; retrovirology; cellular growth regulation by growth factors and their receptors.

Patents:

More than 50 patent applications issued or pending.

Social Security Number:

571-58-5069

Present Address:

40 East 94th Street, Apt. 23B
New York, NY 10128

Research Support

Active

R01 CA71672-07 Aaronson (PI) 07/01/1997-06/30/2005
NIH/NCI $225,000
Cloning and Analysis of Wnt Receptors in Breast Cancer

Aims of this include exploration of novel functions uncovered by us for a prototype Wnt receptor, Hfz1, structure/function analysis for this and other fzs, and immunoaffinity purification and identification of proteins in the receptor complex. Our goals would be to use this knowledge to investigate novel mechanisms of Wnt activation in tumor cells. A second major aim would be to elucidate mechanisms of transformation by Wnt and/or increase β-catenin levels through investigation of alterations induced in normal cells. The functions of Wnt target genes in inducing aspects of the transformed phenotype in vitro and in vivo would be explored, and we would search for novel effectors by application of expression array analysis. The final aim would be to characterize new mechanisms of Wnt signaling activation in breast and other human tumor cells. This aim is supported by our identification of human tumor cell lines with elevated uncomplexed β-catenin levels in the absence of genetic lesions affecting APC or β-catenin, Wnt inhibitors, also characterized by us, would be utilized in efforts to establish evidence of autocrine transforming or other novel mechanisms of Wnt activation.

T32 CA78207-05 Aaronson (PI) 07/20/1999-04/30/2004
NIH/NCI $165,604
Training Program in Cancer Biology

This training program combines research in the biology of cancer with a curriculum that challenges trainees to consider how their research may be translated into improvements in the diagnosis and treatment of cancer. The trainees will work closely with faculty who will be drawn from throughout Mount Sinai ensuring that this research is both comprehensive in scope and related to practical issues faced by physicians in preventing and treating cancer.

P01 CA80058-04 Aaronson (PI) 02/16/2000-01/31/2005
NIH/NCI $160,111
P53 Regulators and Effectors

Subproject: Project 1

This grant is specifically directed at elucidating the role of MAPK activation in p53 growth arrest/senescence, the mechanisms responsible and the effector pathways involved. The long-term goals of this project are to understand how MAPK activation contributes to p53 induced permanent grouth arrest/senescence as a means of developing nove approaches to therapy by targeting this terminal differentiation program in tumor cells.

Subproject: Administrative Core

The Administrative Core provides centralized services that support the Principal Invesitgator, Project Leaders, and Core Resource Directors of the program projec, and is responsible for continuity, coordination and oversight of the projects and support cores of the Program project. The primary functions of the Core include the following: (a0 provide direction and overall management of the Program; (b) foster communication and integration of the research projects and the cores by conducting regular scientific meetings of PPG investigators; (c) oversee and coordinate the efforts of the support cores by oversight committees and monitoring efficient usage; (d) monitor and track all expenditures of the PPG and ensure accurate allocation of funds; (e) provide logistics support for other apects including personnel actions, IRB documents, record keeping, progress reports; (f) arrange and coordinate meetings of the PPG internal and external scientific advisory boards, including travel.

N/A Aaronson (PI) 07/01/1997-06/30/2005
Sharp Foundation $666,667
Melanoma Gene Discovery and Translation Research

This is a consortium grant involving multiple laboratory and clinical investigators within Mount Sinai. The aims of the consortium are to translate laboratory advances in the molecular understanding of the invasive, metastatic phenotype of melanoma and resistance to therapy of melanoma to the clinic. Studies include melanoma gene discovery, mechanisms involved in p53 functional inactivation, studies of the molecular basis for invasion and metatisis, and efforts to untilize peptide inhibitors and antisense approaches to convert melanoma for a therapy resistant to sensitive phenotype. The P.I. serves as the administrative leader of this program and participate in the melanoma gene discovery effort.

R01 CA85214-05 (PI) 08/04/1999-05/31/2004
NIH/NCI $310,032
Cellular Senescene in Aging and Cancer

The aims of this project are to characterize the molecular pathways of senescence in normal cells, as well as induced by various stimuli in tumor cells in an effort to develop better understanding of this important cell aiging program.

N/A Aaronson (PI) 10/01/2001-09/30/2004
Breast Cancer Research Foundation $217,391
Mammaglobin in the Circularion as a Marker of Breast Cancer

The major goal of this proposal is to examine the pattern of mammaglobin expression in women with and without breast cancer in order to establish its clinical utility as a blood marker for early detection of breast cancer and response to treatment.

R24 CA95834-02 Aaronson (PI) 04/01/2002-03/31/2007
NIH/NCI $96,233
Cancer Resource – Pathology, Registry and Biorepositories

The overall aim of this study is to establish the infrastructure for conduction cancer research with human specimens consisting of a tissue repository, biorepository and data bank. It will build upon our existing infrastructure of colon and breast cancer familial registries, the Prostate Cancer Database and Tissue Bank, the Mount Sinai Tumor Registry, a pathology tissue bank, and a specimen bank used in specific studies. The Cancer Resource will consist of several components and will facilitate research ranging from basic science, translational research, and large-scale molecular epidemiologic studies.

N/A Aaronson (PI) — 12/11/2003-12/10/2004
Becton Dickinson and Company — $107,143
Collaborative Research Project

The overall aim of this study is to identify and optimize cellular environments for isolation and expansion of human tumor cells in vitro by using robotic screening methods in a systematic and comprehensive way.

Pending

T32 CA78207 Aaronson (PI) — 05/01/2004-04/30/2009
NIH/NCI
Training Program in Cancer Biology

R01 CA85214 Aaronson (PI) — 07/01/2004-06/30/2009
NIH/NCI
Cellular Senescence in Aging and Cancer

P01 CA80058 Aaronson (PI) — 02/01/2005-1/31/2010
NIH/NCI
P53 Regulators and Effectors

BIBLIOGRAPHY

Stuart A. Aaronson

1. Aaronson, S. A., Natori, Y., and Tarver, H.: Effect of estrogen on uterine ATP levels. Proc. Soc. Exp. Biol. Med. 120: pp. 9-13, 1965.

2. Aaronson, S. A., Korner, A., and Munro, A. J.: Inhibition of soluble ribonucleic acid of stimulatory effect of liver template ribonucleic acid. Biochem. J. 101: pp. 448-453, 1966.

3. Aaronson, S. A. and Todaro, G. J.: SV40 T antigen induction and transformation in human fibroblast cell strains. Virology 36: pp. 254-261, 1968.

4. Aaronson, S. A. and Todaro, G. J.: Development of 3T3-1ike lines from BALB/c mouse embryo cultures: transformation susceptibility to SV40. J. Cell Physiol. 72: pp. 141-148, 1968.

5. Todaro, G. J. and Aaronson, S. A.: Human cell strains susceptible to focus formation by human adenovirus type 12. Proc. Natl. Acad. Sci. USA 61: pp. 1272-1278, 1968.

6. Aaronson, S. A. and Todaro, G. J.: Basis for the acquisition of malignant potential by mouse cells cultivated *in vitro*. Science 162: pp. 1024-1026, 1968.

7. Todaro, G. J. and Aaronson, S. A.: Properties of clonal lines of murine sarcoma virus transformed BALB/3T3 cells. Virology 38: pp. 174-179, 1969.

8. Aaronson, S. A. and Todaro, G. J.: Human diploid cell transformation by DNA extracted from the tumor virus, SV40. Science 166: pp. 390-391, 1969.

9. Aaronson, S. A., Hartley, J. A., and Todaro, G. J.: Mouse leukemia virus "spontaneous" release by mouse embryo cells after long-term *in vitro* cultivation. Proc. Natl. Acad. Sci. USA 65: pp. 87-94, 1969.

10. Jainchill, J. L., Aaronson, S. A., and Todaro, G. J.: Murine sarcoma and leukemia viruses: assay using clonal lines of contact-inhibited mouse cells. J. Virol. 4: pp. 549-553, 1969.

11. Pollock, E. J., Aaronson, S. A., and Todaro, G. J.: X-irradiation of BALB/3T3: sarcoma forming ability and virus induction. Int. J. Radiat. Biol. 17: pp. 97-100, 1970.

12. Aaronson, S. A. and Todaro, G. J.: Transformation and virus growth by murine sarcoma viruses and human cells. Nature 225: pp. 458-459, 1970.

13. Aaronson, S. A. and Todaro, G. J.: Infectious SV40 and SV40 DNA: rapid fluorescent focus assay. Proc. Soc. Exp. Biol. Med. 134: pp. 103-106, 1970.

14. Todaro, G. J., Zeve, V., and Aaronson, S. A.: Virus in cell cultures derived from human tumor patients. Nature 226: pp. 1047-1048, 1970.

15. Aaronson, S. A., Todaro, G. J., and Freeman, A. E.: Human sarcoma cells in culture: identification by colony-forming ability on monolayers of normal cells. Exp. Cell Res. 61: pp. 1-5, 1970.

16. Aaronson, S. A. and Rowe, W. P.: Nonproducer clones of murine sarcoma virus transformed BALB/3T3 cells. Virology 42: pp. 9-19, 1970.

17. Aaronson, S. A., Jainchill, J. L., and Todaro, G. J.: Murine sarcoma virus transformation of BALB/3T3 cells: lack of dependence on murine leukemia virus. Proc. Natl. Acad. Sci. USA 66: pp. 1236-1243, 1970.

18. Aaronson, S. A.: Effect of ultraviolet irradiation on the survival of SV40 functions in human and mouse cells. J. Virol. 6: pp. 393-399, 1970.

19. Aaronson, S. A. and Lytle, C.: Decreased host cell reactivation of irradiated SV40 virus in Xeroderma pigmentosum. Nature 228: pp. 359-361, 1970.

20. Scolnick, E. M., Aaronson, S. A., and Todaro, G. J.: DNA synthesis by RNA-containing tumor viruses. Proc. Natl. Acad. Sci. USA 67: pp. 1034-1041, 1970.

21. Aaronson, S. A. and Martin, M. A.: Transformation of human cells with different forms of SV40 DNA. Virology 42: pp. 848-856, 1970.

22. Aaronson, S. A.: Susceptibility of human cell strains to transformation by SV40 and SV40 DNA. J. Virol. 6: pp. 470-475, 1970.

23. Kelloff, G., Aaronson, S. A., and Gilden, R.V.: Inactivation of murine sarcoma and leukemia viruses by ultraviolet irradiation. Virology 42: pp. 1133-1135, 1970.

24. Gerwin, B. I., Todaro, G. J., Zeve, V., Scolnick, E. M., and Aaronson, S. A.: Separation of RNA-dependent DNA polymerase activity from the murine leukemia virion. Nature 228: pp. 435-438, 1970.

25. Scolnick, E. M., Rands, E., Aaronson, S. A., and Todaro, G. J.: RNA-dependent DNA polymerase activity in five RNA viruses: divalent cation requirements. Proc. Natl. Acad. Sci. USA 67: pp. 1789-1796, 1970.

26. Aaronson, S. A.: Human cell transformation by SV40 and SV40 DNA. *In* Lapedes, D. N. (Ed): McGraw-Hill Yearbook of Science and Technology. New York, McGraw-Hill, Inc., 1971, pp. 421-423.

27. Aaronson, S .A., Todaro, G. J., and Huebner, R. J.: Transformation by murine sarcoma virus. In Silvestri, L. G. (Ed.): The Biology of Oncogenic Viruses. Amsterdam, North Holland Publishing Co., 1971, pp. 138-144.

28. Todaro, G. J., Aaronson, S. A., Scolnick, E. M., and Parks, W. P.: RNA-dependent DNA polymerase in viruses and in cells. In Silvestri, L. G. (Ed.): The Biology of Oncogenic Viruses. Amsterdam, North Holland Publishing Co., 1971, pp. 206-209.

29. Stone, L. B., Scolnick, E. M., Takemoto, K. K., and Aaronson, S. A.: Visna virus: a slow virus with an RNA dependent DNA polymerase. Nature 229: pp. 257-258, 1971.

30. Aaronson, S. A.: Isolation of a rat-tropic helper virus from M-MSV-O stocks. Virology 44: pp. 29-36, 1971.

31. Todaro, G. J., Aaronson, S. A., and Rands, E.: Rapid detection of mycoplasma infected cell cultures. Exp. Cell Res. 65: pp. 256-258, 1971.

32. Todaro, G. J., Zeve, V., and Aaronson, S. A.: Cell culture techniques in the search for cancer viruses in man. In Vitro 6: pp. 355-361, 1971.

33. Parks, W. P., Scolnick, E. M., Todaro, G. J., and Aaronson, S. A.: RNA-dependent DNA polymerase in primate syncytium-forming ("foamy") viruses. Nature 229: pp. 258-260, 1971.

34. Scolnick, E. M., Aaronson, S. A., Todaro, G. J., and Parks, W. P.: RNA-dependent DNA polymerase activity in mammalian cells. Nature 235: pp. 318-321, 1971.

35. Worthington, M. and Aaronson, S. A.: Interferon system in cells from human tumors and from persons predisposed to cancer. Infect. Immun. 3: pp. 424-428, 1971.

36. Aaronson, S. A., Parks, W. P., Scolnick, E. M., and Todaro, G. J.: Antibody to the RNA-dependent DNA polymerase of mammalian C-type RNA tumor viruses. Proc. Natl. Acad. Sci. USA 68: pp. 920-924, 1971.

37. Aaronson, S. A.: Common genetic alterations of RNA tumor viruses grown in human cells. Nature 230: pp. 445-447, 1971.

38. Aaronson, S. A. and Weaver, C. A.: Characterization of murine sarcoma virus (Kirsten) transformation of mouse and human cells. J. Gen. Virol. 13: pp. 245-252, 1971.

39. Gelb, L. D., Aaronson, S. A., and Martin, M. A.: Heterogeneity of murine leukemia virus in *in vitro* DNA: detection of viral DNA in mammalian cells. Science 172: pp. 1353-1355, 1971.

40. Stephenson, J. R. and Aaronson, S. A.: Murine sarcoma and leukemia viruses: genetic differences determined by RNA-DNA hybridization. Virology 46: pp. 480-484, 1971.

41. Ross, J., Scolnick, E. M., Todaro, G. J., and Aaronson, S. A.: Separation of murine cellular and murine leukemia virus DNA polymerases. Nature 231: pp. 153-167, 1971.

42. Aaronson, S. A., Todaro, G. J., and Scolnick, E. M.: Induction of murine C-type viruses from clonal lines of virus-free BALB/3T3 cells. Science 174: pp. 157-159, 1971.

43. Aaronson, S. A.: Chemical induction of focus-forming virus from nonproducer cells transformed by murine sarcoma virus. Proc. Natl. Acad. Sci. USA 68: pp. 3069-3072, 1971.

44. Parks, W. P., Scolnick, E. M., Ross, J., Todaro, G. J., and Aaronson, S. A.: Immunologic relationships of reverse transcriptases from ribonucleic acid tumor viruses. J. Virol. 9: pp. 110-115, 1972.

45. Stephenson, J. R. and Aaronson, S. A.: Antigenic properties of murine sarcoma virus transformed BALB/3T3 nonproducer cells. J. Exp. Med. 135: pp. 503-513, 1972.

46. Scolnick, E. M., Parks, W. P., Todaro, G. J., and Aaronson, S. A.: Immunological characterization of primate C-type virus reverse transcriptases. Nature New Biol. 235: pp. 35-40, 1972.

47. Lytle, C. D., Aaronson, S. A., and Harvey, E.: Host cell reactivation in mammalian cells. II. Survival of herpes simplex virus and vaccinia virus in normal human and Xeroderma pigmentosum cells. Int. J. Radiat. Biol. 22: pp. 159-165, 1972.

48. Stephenson, J. R., Reynolds, R. K., and Aaronson, S. A.: Isolation of temperature-sensitive mutants of murine leukemia virus. Virology 48: pp. 749-756, 1972.

49. Stephenson, J. R., Scolnick, E. M., and Aaronson, S. A.: Genetic stability of the sarcoma viruses in murine and avian sarcoma virus transformed nonproducer cells. Int. J. Cancer 9: pp. 577-583, 1972.

50. Aaronson, S. A., Bassin, R. H., and Weaver, C. A.: Comparison of murine sarcoma viruses in nonproducer and $S^{+}L^{-}$ transformed cells. J. Virol. 9: pp. 701-704, 1972.

51. Kersey, J. H., Gatti, R. A., Good, R. A., Aaronson, S. A., and Todaro, G. J.: Susceptibility of cells from patients with primary immunodeficiency diseases to transformation by simian virus 40. Proc. Natl. Acad. Sci. USA 69: pp. 980-982, 1972.

52. Stephenson, J. R. and Aaronson, S. A.: Genetic factors influencing C-type RNA virus induction. J. Exp. Med. 136: pp. 175-184, 1972.

53. Aaronson, S. A.: Immunologic detection of C-type RNA viral reverse transcriptases. Natl. Cancer Inst. Monogr. 35: pp. 83-87, 1972.

54. Stephenson, J. R. and Aaronson, S. A.: A genetic locus for inducibility of C-type virus in BALB/c cells: the effect of a nonlinked regulatory gene on detection of virus after chemical activation. Proc. Natl. Acad. Sci. USA 69: pp. 2798-2801, 1972.

55. Aaronson, S. A. and Stephenson, J. R.: Genetic factors involved in C-type RNA virus expression. *In* Day, S. B. and Good, R. A. (Eds.): Membranes and Viruses in Immunopathology. New York, Academic Press, 1972, pp. 355-366.

56. Scolnick, E. M., Stephenson, J. R., and Aaronson, S. A.: Isolation of temperature-sensitive mutants of murine sarcoma virus. J. Virol. 10: pp. 653-657, 1972.

57. Todaro, G. J., Aaronson, S. A., Scolnick, E. M., Ross, J., and Parks, W. P.: Reverse transcriptase of RNA tumor viruses: immunological relationships. *In* Dutcher, R. M., and Chieco-Bianchi, L. (Eds.): Unifying Concepts of Leukemia. Basel, Karger, 1973, pp. 269-271.

58. Aoki, T., Stephenson, J. R., and Aaronson, S. A.: Demonstration of a cell-surface antigen associated with murine sarcoma virus by immunoelectron microscopy. Proc. Natl. Acad. Sci. USA 70: pp. 742-746, 1973.

59. Stephenson, J. R., Reynolds, R. K., and Aaronson, S. A.: Characterization of morphologic revertants of murine and avian sarcoma virus-transformed cells. J. Virol. 11: pp. 218-222, 1973.

60. Aaronson, S. A. and Stephenson, J. R.: Endogenous RNA C-type viruses of mammalian cells. *In* Silvestri, L.G. (Ed.): Possible Episomes in Eukaryotes. Amsterdam, North Holland Publishing Co., 1973,pp. 42-49.

61. Aaronson, S. A.: Biologic characterization of mammalian cells transformed by a primate sarcoma virus. Virology 52: pp. 562-567, 1973.

62. Kersey, J. H., Yunis, E. J., Todaro, G. J., and Aaronson, S. A.: HL-A antigens of human tumor derived cell lines and viral-transformed fibroblasts in culture. Proc. Soc. Exp. Biol. Med. 143: pp. 453-456, 1973.

63. Stephenson, J. R. and Aaronson, S. A.: Segregation of loci for C-type virus induction in strains of mice with high and low incidence of leukemia incidence. Science 180: pp. 865-866, 1973.

64. Stephenson, J. R. and Aaronson, S. A.: Characterization of temperature sensitive mutants of murine leukemia virus. Virology 54: pp. 53-59, 1973.

65. Aaronson, S. A. and Stephenson, J. R.: Independent segregation of loci for activation of biologically distinguishable RNA C-type viruses in mouse cells. Proc. Natl. Acad. Sci. USA 70: pp. 2055-2058, 1973.

66. Tronick, S. R., Stephenson, J. R., and Aaronson, S. A.: Immunological characterization of a low molecular weight polypeptide of murine leukemia virus. Virology 54: pp. 199-206, 1973.

67. Gelb, L. D., Milstien, J. B., Martin, M. A., and Aaronson, S. A.: Characterization of murine leukemia virus-specific DNA present in normal mouse cells. Nature New Biol. 244: pp. 76-79, 1973.

68. Stephenson, J. R., Wilsnack, R. E., and Aaronson, S. A.: Radioimmunoassay for avian C-type virus group specific antigen: detection in normal and virus transformed cells. J. Virol. 11: pp. 893-899, 1973.

69. Stephenson, J. R. and Aaronson, S. A.: Expression of endogenous RNA C-type virus group specific antigens in mammalian cells. J. Virol. 12: pp. 564-569, 1973.

70. Giard, D. J., Aaronson, S. A., Todaro, G. J., Arnstein, P., Kersey, J. H., Dosik, H., and Parks, W. P.: *In vitro* cultivation of human tumors: Establishment of cell lines derived from a series of solid tumors. J. Natl. Cancer Inst. 51: pp. 1417-1423, 1973.

71. Greenberger, J. S. and Aaronson, S. A.: *In vivo* inoculation of RNA C-type viruses inducing regression of experimental solid tumors. J. Natl. Cancer Inst. 51: pp. 1935-1938, 1973.

72. Fishman, P. H., Brady, R. 0., Bradley, R. M., Aaronson, S. A., and Todaro, G. J.: Absence of a specific gaglioside galactosyl-transferase in mouse cells transformed by murine sarcoma virus. Proc. Natl. Acad. Sci. USA 71: pp. 298-301, 1974.

73. Stephenson, J. R., Greenberger, J. S., and Aaronson, S. A.: Oncogenicity of an endogenous C-type virus chemically activated from mouse cells in culture. J. Virol. 13: pp. 237-240, 1974.

74. Stephenson, J. R., Tronick, S. R., and Aaronson, S. A.: Analysis of type-specific antigenic determinants of two structural polypeptides of mouse RNA C-type viruses. Virology 58: pp. 1-8, 1974.

75. Greenberger, J. S. and Aaronson, S. A.: Morphologic revertants of murine sarcoma virus transformed nonproducer BALB/3T3: selective techniques for isolation and biologic properties *in vitro* and *in vivo*. Virology 57: pp. 339-346, 1974.

76. Aaronson, S. A. and Dunn, C. Y.: Endogenous C-type viruses of BALB/C cells: frequencies of spontaneous and chemical induction. J. Virol. 13: pp. 181-185, 1974.

77. Aaronson, S. A. and Dunn, C. Y.: High frequency C-type virus induction by inhibitors of protein synthesis. Science 183: pp. 422-423, 1974.

78. Stephenson, J. R. and Aaronson, S. A.: Temperature-sensitive mutants of murine leukemia virus. III. Mutants defective in helper functions for sarcoma virus fixation. Virology 58: pp. 294-297, 1974.

79. Tronick, S. R., Stephenson, J. R., and Aaronson, S. A.: Comparative immunological studies of RNA C-type viruses: Radioimmunoassay for a low molecular weight polypeptide of woolly monkey leukemia virus. Virology 57: pp. 347-356, 1974.

80. Stephenson, J. R., Tronick, S. R., Reynolds, R. K., and Aaronson, S. A.: Isolation and characterization of C-type viral gene products of virus-negative mouse cells. J. Exp. Med. 139: pp. 427-438, 1974.

81. Aaronson, S. A. and Stephenson, J. R.: Widespread natural occurrence of high titers of neutralizing antibodies to a specific class of endogenous mouse type C virus. Proc. Natl. Acad. Sci. USA 71: pp. 1957-1961, 1974.

82. Stephenson, J. R., Aaronson, S. A., Arnstein, P., Huebner, R. J., and Tronick, S. R.: Demonstration of two immunologically distinct xenotropic type C RNA viruses of mouse cells. Virology 61: pp. 56-63, 1974.

83. Aaronson, S. A., Stephenson, J. R., and Greenberger, J. S.: Cellular replication and the persistence of inducible RNA type C viruses. J. Virol. 13: pp. 1404-1407, 1974.

84. Stephenson, J. R., Crow, J. D., and Aaronson, S. A.: Differential activation of biologically distinguishable endogenous mouse type C RNA viruses: interactions with host cell regulatory factors. Virology 61: pp. 411-419, 1974.

85. Tronick, S. R., Stephenson, J. R., and Aaronson, S. A.: Immunological properties of two polypeptides of Mason-Pfizer monkey virus. J. Virol. 14: pp. 125-132, 1974.

86. Stephenson, J. R., Anderson, G. R., Tronick, S. R., and Aaronson, S. A.: Evidence for genetic recombination between endogenous and exogenous mouse RNA type C viruses. Cell 2: pp. 87-94, 1974.

87. Greenberger, J. S., Stephenson, J. R., Aoki, T., and Aaronson, S. A.: Cell surface antigens of murine sarcoma virus transformed nonproducer cells: further evidence for lack of transplantation immunity. Int. J. Cancer 14: pp. 145-152, 1974.

88. Greenberger, J. S., Anderson, G. R., and Aaronson, S. A.: Transformation-defective virus mutants in a class of morphologic revertants of sarcoma virus-transformed nonproducer cells. Cell 2: pp. 279-286, 1974.

89. Aaronson, S. A. and Stephenson, J. R.: Cellular regulation of biologically distinguishable endogenous mouse type C RNA viruses. *In* Robinson, W. S. and Fox, C. F. (Eds.): Mechanisms of Virus Disease. Menlo Park, Calif., W.A. Benjamin, Inc., 1974, pp. 49-67.

90. Lee, J. C., Hanna, M. G., Jr., Ihle, J. N., and Aaronson, S. A.: Autogenous immunity to endogenous RNA tumor virus: differential reactivities of immunoglobins M and G to virus envelope antigens. J. Virol. 14: pp. 773-781, 1974.

91. Stephenson, J. R., Tronick, S. R., and Aaronson, S. A.: Temperature-sensitive mutants of murine leukemia virus. IV. Further physiologic characterization and evidence for genetic recombination. J. Virol. 14: pp. 918-923, 1974.

92. Aaronson, S. A., Anderson, G. R., Dunn, C. Y., and Robbins, K. C.: Induction of type C RNA virus by cycloheximide: increased expression of virus specific RNA. Proc. Natl. Acad. Sci. USA 71: pp. 3941-3945, 1974.

93. Aoki, T., Stephenson, J. R., Aaronson, S. A., and Hsu, K. C.: Surface antigens of mammalian sarcoma virus-transformed nonproducer cells. Proc. Natl. Acad. Sci. USA 71: pp. 3445-3449, 1974.

94. Stephenson, J. R., Tronick, S. R., and Aaronson, S. A.: Isolation from BALB/C mouse cells of a structural polypeptide of a third endogenous type C virus. Cell 3: pp. 347-353, 1974.

95. Stephenson, J. R. and Aaronson, S. A.: Demonstration of a genetic factor influencing spontaneous release of a xenotropic virus of mouse cells. Proc. Natl. Acad. Sci. USA 71: pp. 4925-4929, 1974.

96. Aaronson, S. A. and Stephenson, J. R.: Differential cellular regulation of three distinct classes of type C RNA viruses endogenous to mouse cells. Cold Spring Harbor Symp. Quant. Biol. 39: pp. 1129-1137, 1975.

97. Stephenson, J. R. and Aaronson, S. A.: Genetics of endogenous mouse C-type viruses. *In* Ito, Y. and Dutcher, R. M. (Eds.): Comparative Leukemia Research, Leukemogenesis, 1973. Tokyo, Univ. Tokyo Press, 1975, pp. 657-658.

98. Greenberger, J. S. and Aaronson, S. A.: Cycloheximide induction of xenotropic type C virus from synchronized mouse cells: metabolic requirements for virus activation. J. Virol. 15: pp. 64-70, 1975.

99. Greenberger, J. S., Stephenson, J. R., Moloney, W. C., and Aaronson, S. A.: Different hematologic diseases induced by type C viruses chemically activated from embryo cells of different mouse strains. Cancer Research 35: pp. 245-252, 1975.

100. Aaronson, S. A. and Schlom, J.: The search for RNA tumor viruses in human cancer. *In* Airel, 1. M. (Ed.): Progress in Clinical Cancer. New York, Grune and Stratton, 1975, pp. 51-63.

101. Tronick, S. R., Stephenson, J. R., Aaronson, S. A., and Kawakami, T. G.: Antigenic characterization of type C RNA virus isolates of gibbon apes. J. Virol. 15: pp. 115-120, 1975.

102. Greenberger, J. S., Rosenthal, D. S., Aaronson, S. A., and Moloney, W. C.: Acute myelogenous leukemia of the Wistar-Furth rat: establishment of a continuous tissue culture line producing lysozyme *in vitro and in vivo*. Blood 46: pp. 27-38, 1975.

103. Greenberger, J. S., Phillips, S. M., Stephenson, J. R., and Aaronson, S. A.: Induction of mouse type C RNA virus by lipopolysaccharide A. J. Immunol. 115: pp. 317-320, 1975.

104. Stephenson, J. R., Reynolds, R. K., Tronick, S. R., and Aaronson, S. A.: Distribution of three classes of endogenous type C RNA viruses among inbred strains of mice. Virology 67: pp. 404-414, 1975.

105. Dunn, C. Y., Aaronson, S. A., and Stephenson, J. R.: Interactions of chemical inducers and steroid enhancers of endogenous mouse type C RNA viruses. Virology 66: pp. 579-588, 1975.

106. Greenberger, J. S., Aaronson, S. A., Rosenthal, D. S., and Moloney, W. C.: Continuous production of peroxidase, esterase, alkaline phosphatase and lysozyme by clones of promyelocytes. Nature 257: pp. 143-144, 1975.

107. Greenberger, J. S., Stephenson, J. R., and Aaronson, S. A.: Temperature-sensitive mutants of murine leukemia virus. V. Impaired leukemogenic activity *in vivo*. Int. J. Cancer 15: pp. 1009-1015, 1975.

108. Stephenson, J. R., Smith, E. J., Crittenden, L. B., and Aaronson, S. A.: Analysis of antigenic determinants of structural polypeptides of avian type C RNA tumor viruses. J. Virol. 16: pp. 27-33, 1975.

109. Hino, S., Stephenson, J. R., and Aaronson, S. A.: Antigenic determinants of the 70,000 molecular weight glycoprotein of woolly monkey type C RNA virus. J. Immunol. 115: pp. 922-927, 1975.

110. Tronick, S. R., Stephenson, J. R., Verma, I. M., and Aaronson, S. A.: Thermolabile reverse transcriptase of a mammalian leukemia virus mutant temperature-sensitive in its replication and sarcoma virus helper functions. J. Virol. 16: pp. 1476-1482, 1975.

111. Tronick, S. R., Stephenson, J. R., Verma, I. M., and Aaronson, S. A.: A thermolabile reverse transcriptase from a temperature-sensitive mutant of murine leukemia virus. *In* Goulian, M. and Hanawalt, P. (Eds.): DNA Synthesis and its Regulation. Menlo Park, W. A. Benjamin, Inc., 1975, pp. 1-9.

112. Stephenson, J. R., Tronick, S. R., and Aaronson, S. A.: Murine leukemia virus mutants with temperature-sensitive defects in precursor polypeptide cleavage. Cell 6: pp. 543-548, 1975.

113. Aaronson, S. A., Stephenson, J. R., Hino, S., and Tronick, S. R.: Differential expression of helper viral structural polypeptides in cells transformed by clonal isolates of woolly monkey sarcoma virus. J. Virol. 16: pp. 1117-1123, 1975.

114. Aaronson, S. A. and Stephenson, J. R.: Viruses and the etiology of cancer. In Drew, W. L. (Ed.): Clinical Concepts in Virology. Philadelphia, F. A. Davis, 1976, pp. 251-292.

115. Gonda, M. A., Aaronson, S. A., Ellmore, N., Zeve, V. H., and Hagashima, K.: Ultrastructural studies of surface features of human normal and tumor cells in tissue culture by scanning and transmission electron microscopy. J. Natl. Cancer Inst. 56: pp. 245-263, 1976.

116. Stephenson, J. R., Reynolds, R. K., and Aaronson, S. A.: Comparisons of the immunological properties of two structural polypeptides of type C RNA viruses endogenous to Old World monkeys. J. Virol. 17: pp. 374-384, 1976.

117. Kelloff, G. J., Peters, R. L., Donahoe, R. M., Stephenson, J. R., and Aaronson, S. A.: Natural occurrence of tumors in mouse strains with different xenotropic and ecotropic endogenus viruses. J. Natl. Cancer Inst. 57: pp. 85-89, 1976.

118. Phillips, S. M., Stephenson, J. R., Greenberger, J. S., Lane, P. E., and Aaronson, S. A.: Release of xenotropic type C RNA virus in response to lipopolysaccharide: activity of lipid-A portion upon B lymphocytes. J. Immunol. 116: pp. 1123-1128, 1976.

119. Fishman, P. H., Brady, R., and Aaronson, S. A.: A comparison of membrane glycoconjugates from mouse cells transformed by murine and primate RNA sarcoma viruses. Biochemistry 15: pp. 201-208, 1976.

120. Smith, E. J., Stephenson, J. R., Crittenden, L. B., and Aaronson, S. A.: Avian leukosis-sarcoma virus gene expression: noncoordinate control of group specific antigens in virus-negative avian cells. Virology 70: pp. 493-501, 1976.

121. Stephenson, J. R. and Aaronson, S. A.: Induction of an endogenous B-tropic type C RNA virus from SWR/J mouse embryo cells in tissue culture. Virology 70: pp. 352-359, 1976.

122. Aaronson, S. A., Stephenson, J. R., Tronick, S. R., and Hino, S.: Immunological analysis of structural polypeptides of woolly monkey-gibbon ape type C viruses: expression of wolly monkey helper viral antigens in cells transformed by woolly monkey sarcoma virus. *In* Clemmesen, J. and Yohn, D. S. (Eds.): Comparative Leukemia Research, Basel, Karger, 1976, pp. 102-109.

123. Okabe, H., Gilden, R. V., Hatanaka, M., Stephenson, J. R., Gallagher, R. E., Gallo, R. C., and Aaronson, S. A.: Immunological and biochemical characterization of type C viruses isolated from cultured human AML cells. Nature 260: pp. 264-266, 1976.

124. Hampar, B., Aaronson, S. A., Derbe, J. G., Chakrabarty, M., Showalter, S. A., and Dunn, C. Y.: Activation of an endogenous mouse type C virus by ultraviolet-irradiated herpes simplex virus types 1 and 2. Proc. Natl. Acad. Sci. USA 73: pp. 646-650, 1976.

125. Greenberger, J. S., Bensinger, W. I., and Aaronson, S. A.: Selective techniques for the isolation of morphologic revertants of sarcoma virus transformed cells. *In* Prescott, D. (Ed.): Methods in Cell Biology. New York, Academic Press, 1976, pp. 237-248.

126. Aaronson, S. A., Stephenson, J. R., Hino, S., and Cabradilla, C.: Endogenous type C RNA viruses of mouse cells: a model for the study of gene regulation in eukaryotes. *In* Criss, W. E., Ono, T., and Sabine, J. R. (Eds.): Control Mechanisms in Cancer. New York, Raven Press, 1976, pp. 279-294.

127. Stephenson, J. R. and Aaronson, S. A.: Search for antigens and antibodies crossreactive with type C viruses of the woolly monkey and gibbon ape in animal models and in humans. Proc. Natl. Acad. Sci. USA 73: pp. 1725-1729, 1976.

128. Aaronson, S. A. and Stephenson, J. R.: Endogenous type C RNA viruses of mammalian cells. Biochim. Biophys. Acta 458: pp. 323-354, 1976.

129. Stephenson, J. R., Cabradilla, C. D., and Aaronson, S. A.: Genetic factors influencing endogenous type C RNA viruses of mouse cells: control of viral polypeptide expression in the C57BL/l0 strain. Intervirology 6: pp. 258-269, 1976.

130. Hino, S., Stephenson, J. R., and Aaronson, S. A.: Radio-immunoassays for the 70,000 molecular weight glycoprotein of endogenous mouse type C viruses: viral antigen expression in normal mouse tissues and sera. J. Virol. 18: pp. 933-941, 1976.

131. Barbacid, M., Stephenson, J. R., and Aaronson, S. A.: Structural polypeptides of mammalian type C RNA viruses: isolation and immunologic characterization of a low molecular weight polypeptide, pl0. J. Biol. Chem. 251: pp. 4859-4866, 1976.

132. Stephenson, J. R., Hino, S., Garrett, E. W., and Aaronson, S. A.: Immunological cross-reactivity of Mason-Pfizer monkey virus with type C RNA viruses endogenous to primates. Nature 261: pp. 609-611, 1976.

133. Barbacid, M., Stephenson, J. R., and Aaronson, S. A.: *gag* gene of mammalian type C RNA tumor viruses. Nature 262: pp. 554-559, 1976.

134. Barbacid, M., Stephenson, J. R., and Aaronson, S. A.: Endogenous type C RNA virus of Odocoileus hemionus, a mammalian species of New World origin. Cell 9: pp. 489-494, 1976.

135. Stephenson, J. R., Hino, S., Peters, R. L., Donahoe, R. M., Long, L. K., Aaronson, S. A., and Kelloff, G. J.: Natural immunity in mice to structural polypeptides of endogenous type C RNA viruses. J. Virol. 19: pp. 890-898, 1976.

136. Cabradilla, C. D., Robbins, K. C., and Aaronson, S. A.: Induction of mouse type C virus by translational inhibitors: evidence for transcriptional derepression of a specific class of endogenous virus. Proc. Natl. Acad. Sci. USA 73: pp. 4541-4545, 1976.

137. Devare, S. G., Stephenson, J. R., Chander, S., Sarma, P. S., and Aaronson, S. A.: Bovine lymphosarcoma: development of a radioimmunologic technique for detection of the etiologic agent. Science 194: pp. 1428-1430, 1976.

138. Aaronson, S. A. and Stephenson, J. R.: Intracellular and systemic regulation of biologically distinguishable endogenous type C RNA viruses of mouse cells. *In* Hanna, M. G. and Rapp, F. (Eds.): Contemporary Topics in Immunobiology. New York, Plenum Publishing Corp., 1977, pp. 107-123.

139. Essex, M., Cotter, S. M., Sliski, A. H., Hardy, W. D., Jr., Stephenson, J. R., Aaronson, S. A., and Jarrett, O.: Horizontal transmission of feline leukemia virus under natural conditions in a feline leukemia cluster household. Int. J. Cancer 19: pp. 90-96, 1977.

140. Stephenson, J. R., Barbacid, M., Tronick, S. R., Hino, S., and Aaronson, S. A.: Proteins of type C RNA tumor viruses. *In* Gallo, R. (Ed.): Cancer Research: Cell Biology, Molecular Biology and Tumor Virology. Cleveland, CRC Press, 1977, pp. 37-50.

141. Stephenson, J. R. and Aaronson, S. A.: Differential regulation of endogenous type C viruses of mouse cells. In Schlessinger, D. (Ed.): Microbiology 1977. Baltimore, Waverly Press, 1977, pp. 542-547.

142. Phillips, S. M., Stephenson, J. R., and Aaronson, S. A.: Genetic factors influencing mouse type C RNA virus induction by naturally occurring B-cell mitogens. J. Immunol. 118: pp. 662-666, 1977.

143. Bensinger, W. I., Robbins, K. C., Greenberger, J. S., and Aaronson, S. A.: Different mechanisms for morphologic reversion of a clonal population of murine sarcoma virus-transformed nonproducer cells. Virology 77: pp. 750-761, 1977.

144. Stephenson, J. R., Essex, M., Hino, S., Hardy, W. D., Jr., and Aaronson, S. A.: Feline oncornavirus-associated cell membrane antigen (FOCMA). VII. Distinction between FOCMA and the major virion glycoprotein. Proc. Natl. Acad. Sci. USA 74: pp. 1219-1223, 1977.

145. Essex, M., Stephenson, J. R., Hardy, W. D., Jr., Cotter, S. M., and Aaronson, S. A.: Leukemia, lymphoma and fibrosarcoma of cats as models for similar diseases of man. *In*

Hiatt, H., Watson, J. D., and Winsten, J. A. (Eds.): Origins of Human Cancer. New York, Cold Spring Harbor Laboratory, 1977,pp. 1197-1209.

146. Stephenson, J. R. and Aaronson, S. A.: Endogenous type C viral expression in primates. Nature 266: pp. 469-472, 1977.

147. Barbacid, M., Stephenson, J. R., and Aaronson, S. A.: Evolutionary relationships between *gag* gene-coded proteins of murine and primate endogenous type C RNA viruses. Cell 10: pp. 641-648, 1977.

148. Peters, R. L., Sass, B., Stephenson, J. R., Al-Ghazzouli, I. K., Hino, S., Donahoe, R. M., Kende, M., Aaronson, S. A., and Kelloff, G. J.: Immunoprevention of X-ray induced leukemias in the C57BL mouse. Proc. Natl. Acad. Sci. USA 74: pp. 1697-1701, 1977.

149. Krakower, J. M., Barbacid, M., and Aaronson, S. A.: Radio-immunoassay for mammalian type C viral reverse transcriptase. J. Virol. 22: pp. 331-339, 1977.

150. Robbins, K. C., Cabradilla, C. D., Stephenson, J. R., and Aaronson, S. A.: Segregation of genetic information for a B-tropic leukemia virus with the structural locus for BALB: virus-l. Proc. Natl. Acad. Sci. USA 74: pp. 2953-2957, 1977.

151. Tronick, S. R., Golub, M. M., Stephenson, J. R., and Aaronson, S. A.: Distribution and expression in mammals of genes related to an endogenous type C RNA virus of Odocoileus hemionus. J. Virol. 23: pp. 1-9, 1977.

152. Reynolds, F. R., Jr., Hanson, C. A., Aaronson, S. A., and Stephenson, J. R.: Type C viral *gag* gene expression in chicken embryo fibroblasts and avian sarcoma virus transformed mammalian cells. J. Virol. 23: pp. 74-79, 1977.

153. Krakower, J. M., Barbacid, M., and Aaronson, S. A.: Differential synthesis of mammalian type C viral gene products in infected cells. J. Virol. 24: pp. 1-7, 1977.

154. Robbins, K. C., Stephenson, J. R., Cabradilla, C. D., and Aaronson, S. A.: Endogenous mouse type C RNA virus of SWR cells: inducibility locus containing structural information for a distinct new virus class. Virology 82: pp. 392-400, 1977.

155. Hino, S., Tronick, S. R., Heberling, R. L., Kalter, S. S., Hellman, A., and Aaronson, S. A.: Endogenous New World primate retrovirus: interspecies antigenic determinants shared with the major structural protein of type D RNA viruses of Old World monkeys. Proc. Natl. Acad. Sci. USA 74: pp. 5734-5738, 1977.

156. Barbacid, M., Tronick, S. R., and Aaronson, S. A.: Isolation and characterization of an endogenous type C RNA virus of mink (MvlLu) cells. J. Virol. 25: pp. 129-137, 1978.

157. Robbins, K. C., Okabe, H., Tronick, S. R., Gilden, R. V., and Aaronson, S. A.: Molecular mechanisms involved in the differential expression of *gag* gene products by clonal isolates of a primate sarcoma virus. J. Virol. 26: pp. 471-478, 1978.

158. Barbacid, M., Robbins, K. C., Hino, S., and Aaronson, S. A.: Genetic recombination between mouse type C RNA viruses: a mechanism for endogenous viral gene amplification in mammalian cells. Proc. Natl. Acad. Sci. USA 75: pp. 923-927, 1978.

159. Barbacid, M. and Aaronson, S. A.: Membrane properties of the *gag* gene-coded p15 proteins of mouse type C RNA tumor viruses. J. Biol. Chem. 253: pp. 1408-1414, 1978.

160. Krakower, J. M. and Aaronson, S. A.: Radioimmunologic characterization of RD-114 reverse transcriptase: evolutionary relatedness of mammalian type C viral *pol* gene products. Virology 86: pp. 127-137, 1978.

161. Aaronson, S. A., Krakower, J. M., Tronick, S. R., and Stephenson, J. R.: Immunologic approaches toward detection of type C viral expression in man. Arthritis and Rheumatism 21: pp. S27-S45, 1978.

162. Tronick, S. R., Cabradilla, C. D., Aaronson, S. A., and Haseltine, W. A.: 5'-terminal nucleotide sequences of mammalian type C helper viruses are conserved in the genomes of replication-defective mammalian transforming viruses. J. Virol. 26: pp. 570-576, 1978.

163. Krakower, J. M. and Aaronson, S. A.: Seroepidemiologic assessment of feline leukemia virus infection risk for man. Nature 273: pp. 463-464, 1978.

164. Aaronson, S. A. and Barbacid, M.: Origin and biological properties of a new BALB/c mouse sarcoma virus (BALB-MSV). J. Virol. 27: pp. 366-373, 1978.

165. Lai, M-H .T., Verma, I. M., Tronick, S. R., and Aaronson, S. A.: Mammalian retrovirus-associated RNAse H is virus-coded. J. Virol. 27: pp. 823-825, 1978.

166. Barbacid, M., Troxler, D. H., Scolnick, E. M., and Aaronson, S. A.: Analysis of translational products of Friend strain of spleen focus-forming virus. J. Virol. 27: pp. 826-830, 1978.

167. Israel, M. A., Martin, M. A., Takemoto, K. K., Howley, P. M., Aaronson, S. A., Solomon, D., and Khoury, G.: Evaluation of normal and neoplastic human tissue for BK virus. Virology 90: pp. 187-196, 1978.

168. Aaronson, S. A., Barbacid, M., Hino, S., Tronick, S. R., and Krakower, J. M.: Common progenitors in the evolution of mammalian retroviruses: implications in the search for RNA tumor virus expression in man. *In* Bentvelzen, P., Hilgers, J., and Yohn, D. S. (Eds.): Advances in Comparative Leukemia Research, 1977. Amsterdam/New York, Elsevier/North Holland Biomedical Press, 1978, pp. 127-134.

169. Takemoto, K. K., Solomon, D., Israel, M., Howley, P. M., Khoury, G., Aaronson, S. A., and Martin, M. A.: Search for evidence of papovavirus involvement in human cancer. *In* Bentvelzen, P., Hilgers, J., and Yohn, D. S. (Eds.): Advances in Comparative Leukemia Research, 1977. Amsterdam/ New York, Elsevier/North Holland Biomedical Press, 1978, pp. 260-263.

170. Krakower, J. M., Tronick, S. R., Gallagher, R. E., Gallo, R. C., and Aaronson, S. A.: Antigenic characterization of a new gibbon ape leukemia virus isolate: seroepidemiologic assessment of an outbreak of gibbon leukemia. Int. J. Cancer 22: pp. 715-720, 1978.

171. Barbacid, M., Robbins, K. C., and Aaronson, S. A.: Wild mouse RNA tumor viruses: a nongenetically transmitted virus group closely related to exogenous leukemia viruses of laboratory mouse strains. J. Exp. Med. 149: pp. 254-266, 1979.

172. Porzig, K. J., Barbacid, M., and Aaronson, S. A.: Biological properties and translational products of three independent isolates of feline sarcoma virus. Virology 92: pp. 91-107, 1979.

173. Andersen, P., Barbacid, M., Tronick, S. R., Clark, H. F., and Aaronson, S. A.: Evolutionary relatedness of viper and primate endogenous retroviruses. Science 204: pp. 318-321, 1979.

174. Porzig, K. J., Robbins, K. C., and Aaronson, S. A.: Cellular regulation of mammalian sarcoma virus expression: A gene regulation model for oncogenesis. Cell 16: pp. 875-884, 1979.

175. Canaani, E. and Aaronson, S. A.: Restriction enzyme analysis of mouse cellular type C viral DNA: emergence of new viral sequences in spontaneous AKR/J lymphomas. Proc. Natl. Acad. Sci. USA 76: pp. 1677-1681, 1979.

176. Barbacid, M., Hunter, E., and Aaronson, S. A.: Avian reticuloendotheliosis viruses: evolutionary linkage with mammalian type C retroviruses. J. Virol. 30: pp. 508-514, 1979.

177. Frankel, A., Gilbert, J., Larsen, D., Porzig, K., Scolnick, E. M., Fischinger, P., and Aaronson, S. A.: Nature and distribution of feline sarcoma virus nucleotide sequences. J. Virol. 30: pp. 821-827, 1979.

178. Robbins, K. C., Barbacid, M., Porzig, K. J., and Aaronson, S. A.: Involvement of different exogenous feline leukemia virus subgroups in the generation of independent feline sarcoma virus isolates. Virology 97: pp. 1-11, 1979.

179. Rosen, S. W., Kaminska, J., Calvert, I. S., and Aaronson, S. A.: Human fibroblasts produce "pregnancy-specific" beta-l glycoprotein *in vitro*. Am. J. Obstet. Gynecol. 134: pp. 734-738, 1979.

180. Canaani, E., Robbins, K. C., and Aaronson, S. A.: The transforming gene of Moloney murine sarcoma virus. Nature 282: pp. 378-383, 1979.

181. Aaronson, S. A., Porzig, K. J., and Greenberger, J. S.: Mechanisms of reversion of mammalian sarcoma virus transformed cells. *In* Fox, M. (Ed.): Advances in Medical Oncology, Research and Education. Oxford/New York, Pergamon Press, 1979, pp. 75-85.

182. Barbacid, M., Steel, J., Long, L. K., and Aaronson, S. A.: Isolation and genetic analysis of mammalian type C RNA recombinant viruses. *In* Margison, G. P. (Ed.): Advances in Medical Oncology, Research and Education. Oxford/New York, Pergamon Press, 1979, pp. 43-49.

183. Tronick, S. R., Robbins, K. C., Canaani, E., Devare, S., Andersen, P. R., and Aaronson, S. A.: Molecular cloning of Moloney murine sarcoma virus: arrangement of virus-related sequences within the normal mouse genome. Proc. Natl. Acad. Sci. USA 76: pp. 6314-6318, 1979.

184. Barbacid, M., Long, L. K., and Aaronson S. A.: Major structural proteins of type B, type C, and type D oncoviruses share interspecies antigenic determinants. Proc. Natl. Acad. Sci. USA 77: pp. 72-76, 1980.

185. Aaronson, S. A. and Barbacid, M.: Viral genes involved in leukemogenesis. I. Generation of recombinants between oncogenic and non-oncogenic mouse type C viruses in tissue culture. J. Exp. Med. 151: pp. 467-480, 1980.

186. Dahlberg, J. R., Tronick, S. R., and Aaronson, S. A.: Immunological relationships of an endogenous guinea pig retrovirus with prototype mammalian type B and type D retroviruses. J. Virol. 33: pp. 522-530, 1980.

187. Barbacid, M., Daniel, M. D., and Aaronson, S. A.: Immunological relationships of OMC-I, an endogenous virus of owl monkeys, with mammalian and avian type C viruses. J. Virol. 33: pp. 561-566, 1980.

188. Reddy, P., Dunn, C. Y., and Aaronson, S. A.: Different lymphoid cell targets for transformation by replication-competent Moloney and Rauscher mouse leukemia viruses. Cell 19: pp. 663-669, 1980.

189. Barbacid, M., Bolognesi, D. P., and Aaronson, S. A.: Humans have antibodies capable of recognizing oncoviral glycoproteins: demonstration that these antibodies are formed in response to cellular modification of glycoproteins rather than as consequence of exposure to virus. Proc. Natl. Acad. Sci. USA 77: pp. 1617-1621, 1980.

190. Rosen, S. W., Weintraub, B. D., and Aaronson, S. A.: Nonrandom ectopic protein production by malignant cells: direct evidence *in vitro*. J. Clin. Endocrinol. Metab. 50: pp. 834-841, 1980.

191. Canaani, E., Tronick, S. R., Robbins, K. C., Andersen, P. R., Dunn, C. Y., and Aaronson, S. A.: Cellular origin of the transforming gene of Moloney murine sarcoma virus. Cold Spring Harbor Symp. Quant. Biol. 44: pp. 727-734, 1980.

192. Rosen, S. W., Kaminska, J., Calvert, I. S., Ellmore, N., and Aaronson, S. A.: Ectopic production of "pregnancy-specific" beta-l glycoprotein *in vitro*: discordance with three other placental proteins. Amer. J. Obstet. Gynecol. 137: pp. 525-529, 1980.

193. Canaani, E., and Aaronson, S. A.: Isolation and characterization of naturally occurring deletion mutants of Moloney murine sarcoma virus. Virology 105: pp. 456-466, 1980.

194. Reddy, E. P., Smith, M. J., Canaani, E., Robbins, K. C., Tronick, S. R., Zain, S., and Aaronson, S. A.: Nucleotide sequence analysis of the transforming region and large terminal redundancies of Moloney-murine sarcoma virus. Proc. Natl. Acad. Sci. USA 77: pp. 5234-5238, 1980.

195. Barbacid, M., Krakower, J., and Aaronson, S. A.: Search for evidence of humoral immunity to oncoviruses in man. *In* Todaro, G. J. and Essex, M. (Eds.): Viruses in Naturally Occurring Cancers. New York, Cold Spring Harbor Laboratory Press, 1980, pp. 869-883.

196. Gardner, M. B., Barbacid, M., Rasheed, S., Grant, C., and Aaronson, S. A.: Humoral immunity in natural FeLV-exposed and experimental FeSV-inoculated house cats. *In* Hardy, W. D., Essex, M., and McClelland, A. J. (Eds): Feline Leukemia Virus. New York, Elsevier/North Holland Press, 1980, pp. 159-169.

197. Nagao, K., Yokoro, K., and Aaronson, S. A.: Continuous lines of basophil/mast cells derived from normal mouse bone marrow. Science 212: pp. 333-335, 1981.

198. Oroszlan, S., Barbacid, M., Copeland, T. D., and Aaronson, S. A.: Chemical and immunological characterization of the major structural protein (p28) of MMC-1, a Rhesus monkey endogenous type C virus: homology with the major structural protein of avian reticuloendotheliosis virus. J. Virol. 39: pp. 845-854, 1981.

199. Srinivasan, A., Reddy, E. P., and Aaronson, S. A.: Abelson murine leukemia virus: molecular cloning of infectious integrated proviral DNA. Proc. Natl. Acad. Sci. USA 78: pp. 2077-2081, 1981.

200. Cremer, K., Reddy, E. P., and Aaronson, S. A.: Translational products of Moloney murine sarcoma virus RNA: identification of proteins encoded by the murine sarcoma virus *src* gene. J. Virol. 38: pp. 704-711, 1981.

201. Robbins, K. C., Devare, S. G., and Aaronson, S. A.: Molecular cloning of integrated simian sarcoma virus: Genome organization of infectious DNA clones. Proc. Natl. Acad. Sci. USA 78: pp. 2918-2922, 1981.

202. Aaronson, S. A., Barbacid, M., Dunn, C. Y., and Reddy, E. P.: Genetic approaches toward elucidating the mechanisms of type C virus induced leukemia. *In* Neth, R. (Ed.): Modern Trends in Human Leukemia. Berlin/Heidelberg, Springer-Verlag, 1981, pp. 455-459.

203. Merregaert, J., Barbacid, M., and Aaronson, S. A.: Recombinants between temperature-sensitive mutants of Rauscher murine leukemia virus and BALB:virus-2: genetic mapping of the Rauscher murine leukemia virus genome. J. Virol. 39: pp. 219-228, 1981.

204. Andersen, P. R., Devare, S. G., Tronick, S. R., Ellis, R. W., Aaronson, S. A., and Scolnick, E. M.: Generation of BALB-MuSV and Ha-MuSV by type C virus transduction of homologous transforming genes from different species. Cell 26: pp. 129-134, 1981.

205. Reddy, E. P., Smith, M. J., and Aaronson, S. A.: Complete nucleotide sequence and organization of the Moloney murine sarcoma virus genome. Science 214: pp. 445-450, 1981.

206. Andersen, P. R., Tronick, S. R., and Aaronson, S. A.: Structural organization and biological activity of molecular clones of the integrated genome of a BALB/c mouse sarcoma virus (BALB-MSV). J. Virol. 40: pp. 431-439, 1981.

207. Eva, A., Robbins, K. C., Andersen, P. R., Srinivasan, A., Tronick, S. R., Reddy, E. P., Ellmore, N. W., Galen, A. T., Lautenberger, J. A., Papas, T. S., Westin, E. W., Wong-Staal, F., Gallo, R. C., and Aaronson, S. A.: Transcription of retrovirus *onc* gene and analogues in human solid tumor cells. *In* Yohn, D. S., and Blakeslee, J. R. (Eds.): Advances in Comparative Leukemia Research 1981. New York/Amsterdam, Elsevier Biomedical, 1981, pp. 381-382.

208. Westin, E., Wong-Staal, F., Gelmann, E., Baluda, M., Papas, T., Eva, A., Reddy, E. P., Tronick, S. R., Aaronson, S. A., and Gallo, R. C.: Expression of the *abl, myc,* and *amv* genes in human hematopoietic cells. *In* Yohn, D. S. and Blakeslee, J. R. (Eds.): Advances in Comparative Leukemia Research 1981. New York/Amsterdam, Elsevier Biomedical, 1981, pp. 405-406.

209. Eva, A., Robbins, K. C., Andersen, P. R., Srinivasan, A., Tronick, S. R., Reddy, E. P., Ellmore, N. W., Galen, A. T., Lautenberger, J. A., Papas, T. S., Westin, E. H., Wong-Staal, F., Gallo, R. C., and Aaronson, S. A.: Cellular genes analogous to retroviral *onc* genes are transcribed in human tumor cells. Nature 295: pp. 116-119, 1982.

210. Robbins, K. C., Hill, R. L., and Aaronson, S. A.: Primate origin of the cell-derived sequences of simian sarcoma virus. J. Virol. 41: pp. 721-725, 1982.

211. Rasheed, S., Barbacid, M., Aaronson, S. A., and Gardner, M. B.: Origin and biological properties of a new feline sarcoma virus. Virology 117: pp. 238-244, 1982.

212. Westin, E. H., Gallo, R. C., Arya, S. K., Eva, A., Souza, L. M., Baluda, M. A., Aaronson, S. A., and Wong-Staal, F.: Differential expression of the *amv* gene in human hematopoietic cells. Proc. Natl. Acad. Sci. USA 79: pp. 2194-2198, 1982.

213. Westin, E. H., Wong-Staal, F., Gelmann, E. P., Dalla-Favera, R. C., Papas, T. S., Lautenberger, J. A., Eva, A., Reddy, E. P., Tronick, S. R., Aaronson, S. A., and Gallo, R. C.: Expression of cellular homologs of retroviral *onc* genes in human hematopoietic cells. Proc. Natl. Acad. Sci. USA 79: pp. 2490-2494, 1982.

214. Devare, S. G., Reddy, E. P., Robbins, K. C., Andersen, P. R., Tronick, S. R., and Aaronson, S. A.: Nucleotide sequence of the transforming gene of simian sarcoma virus. Proc. Natl. Acad. Sci. USA 79: pp. 3179-3182, 1982.

215. Devare, S. G., Reddy, E. P., Law, J. D., and Aaronson, S. A.: Nucleotide sequence analysis of the long terminal repeat (LTR) of integrated simian sarcoma virus: evolutionary relationship with other mammalian retroviral LTRs. J. Virol. 42: pp. 1108-1113, 1982.

216. Swan, D. C., McBride, O. W., Robbins, K. C., Keithley, D. A., Reddy, E. P., and Aaronson, S. A.: Chromosomal mapping of the simian sarcoma virus *onc* gene analogue in human cells. Proc. Natl. Acad. Sci. USA 79: pp. 4691-4695, 1982.

217. Aaronson, S. A., Storch, T. G., Balachandra, R., and Reddy, E. P.: Different hematopoietic target cells for transformation by replication-competent murine leukemia viruses. *In* Marchesi, V.T. and Gallo, R. C. (Eds.): Progress in Clinical and Biological Research: Differentiation and Function of Hematopoietic Cell Surfaces. New York, Allen R. Liss, Inc., 1982, pp. 251-261.

218. Santos, E., Tronick, S. R., Aaronson, S. A., Pulciani, S., and Barbacid, M.: T24 human bladder carcinoma oncogene is an activated form of the normal human homologue of BALB- and Harvey-MSV transforming genes. Nature 298: pp. 343-347, 1982.

219. Pierce, J. and Aaronson, S. A.: BALB- and Harvey-MSV transformation of a novel lymphoid progenitor cell. J. Exp. Med. 156: pp. 873-887, 1982.

220. Prakash, K., McBride, O. W., Swan, D. C., Devare, S. G., Tronick, S. R., and Aaronson, S. A.: Molecular cloning and chromosomal mapping of a human locus related to the transforming gene of moloney murine sarcoma virus. Proc. Natl. Acad. Sci. USA 79: pp. 5210-5214, 1982.

221. Srinivasan, A., Dunn, C. Y., Yuasa, Y., Devare, S. G., Reddy, E. P., and Aaronson, S. A.: Abelson murine leukemia virus: structural requirements for transforming gene function. Proc. Natl. Acad. Sci. USA 79: pp. 5508-5512, 1982.

222. Habara, A., Reddy, E. P., and Aaronson, S. A.: Rauscher-murine leukemia virus: molecular cloning of infectious integrated proviral DNA. J. Virol. 44: pp. 731-735, 1982.

223. Merregaert, J. and Aaronson, S. A.: Characterization of inducible type C RNA viruses of mouse strains from different geographic areas. Virology 123: pp. 165-17, 1982.

224. Robbins, K. C., Devare, S. G., Reddy, E. P., and Aaronson, S. A.: *In vivo* identification of the transforming gene product of simian sarcoma virus. Science 218: pp. 1131-1133, 1982.

225. Pulciani, S., Santos, E., Lauver, A. V., Long, L. K., Aaronson, S. A., and Barbacid, M.: Oncogenes in solid human tumors. Nature 300: pp. 539-542, 1982.

226. McBride, O. W., Swan, D. C., Santos, E., Barbacid, M., Tronick, S. R., and Aaronson, S. A.: Localization of the normal allele of T24 human bladder carcinoma oncogene to chromosome 11. Nature 300: pp. 773-774, 1982.

227. Taub, R., Kirsch, I., Morton, C., Lenoir, G., Swan, D., Tronick, S. R., Aaronson, S. A., and Leder, P.: Translocation of the c-*myc* gene into the immunoglobulin heavy chain locus in human Burkitt's lymphoma and murine plasmacytoma cells. Proc. Natl. Acad. Sci. USA 79: pp. 7837-7841, 1982.

228. Aaronson, S. A.: Unique aspects of the interactions of retroviruses with vertebrate cells: C. P. Rhoads Memorial Lecture. Cancer Research 43: pp. 1-5, 1983.

229. Devare, S. G., Reddy, E. P., Law, D. J., Robbins, K. C., and Aaronson, S. A.: Nucleotide sequence of the simian sarcoma virus genome: demonstration that its acquired cellular sequences encodes the transforming gene product $p28^{sis}$. Proc. Natl. Acad. Sci. USA 80: pp. 731-735, 1983.

230. Weissman, B. E. and Aaronson, S. A.: BALB and Kirsten murine sarcoma viruses alter growth and differentiation of EGF-dependent BALB/c mouse epidermal keratinocyte lines. Cell 32: pp. 599-606, 1983.

231. Aaronson, S. A., Dunn, C. Y., Ellmore, N. W., and Eva, A.: Retroviral *onc* genes in human neoplasia. *In* O'Connor, T.E. and Rauscher, F.J. (Eds.): Oncogenes and Retroviruses: Evaluation of Basic Findings and Clinical Potential. New York, Alan R. Liss, Inc., 1983, pp. 207-222.

232. Pierce, J. H. and Aaronson, S. A.: *In vitro* transformation of murine pre-B lymphoid cells by Snyder-Theilen feline sarcoma virus. J. Virol. 46: pp. 993-1002, 1983.

233. Eva, A. and Aaronson, S. A.: Frequent activation of c-*kis* as a transforming gene in fibrosarcomas induced by methylcholanthrene. Science 220: pp. 955-956, 1983.

234. Yuasa, Y., Srivastava, S. K., Dunn, C. Y., Rhim, J. S., Reddy, E. P., and Aaronson, S. A.: Acquisition of transforming properties by alternative point mutations within c-*bas/has* human proto-oncogene. Nature 303: pp. 775-779, 1983.

235. Doolittle, R. F., Hunkapiller, M. W., Hood, L. E., Devare, S. G., Robbins, K. C., Aaronson, S. A., and Antoniades, H. N.: Simian sarcoma *onc* gene, v-*sis*, is derived from the gene (or genes) encoding a platelet-derived growth factor. Science 221: pp. 275-277, 1983.

236. Eva, A., Tronick, S. R., Gol, R. A., Pierce, J. H., and Aaronson, S. A.: Transforming genes of human hematopoietic tumors: frequent detection of *ras*-related oncogenes whose activation appears to be independent of tumor phenotype. Proc. Natl. Acad. Sci. USA 80: pp. 4926-4930, 1983.

237. Naharro, G., Tronick, S. R., Rasheed, S., Gardner, M. B., Aaronson, S. A., and Robbins, K. C.: Molecular cloning of integrated Gardner-Rasheed feline sarcoma virus: genetic structure of its cell-derived sequence differs from that of other tyrosine kinase coding *onc* genes. J. Virol. 47: pp. 611-619, 1983.

238. Needleman, S., Yuasa, Y., Srivastava, S., and Aaronson, S. A.: Normal cells of patients with high cancer risk syndromes lack transforming activity in the NIH/3T3 transfection assay. Science 222: pp. 173-175, 1983.

239. Robbins, K. C., Antoniades, H. N., Devare, S. G., Hunkapiller, M. W., and Aaronson, S. A.: Structural and immunological similarities between simian sarcoma virus gene product(s) and human platelet-derived growth factor. Nature 305: pp. 605-608, 1983.

240. Chiu, I. M., Andersen, P. R., Aaronson, S. A., and Tronick, S. R.: Molecular cloning of the unintegrated squirrel monkey retrovirus genome: organization and distribution of related sequences in primate DNAs. J. Virol. 47: pp. 434-411, 1983.

241. Aaronson, S. A., Reddy, E. P., Robbins, K. C., Devare, S. G., Swan, D. C., Pierce, J. H., and Tronick, S. R.: Retroviruses, *onc* genes and human cancer. *In* Harris, C. C. and Autrup, H. N. (Eds.): Human Carcinogenesis. New York, Academic Press, 1983, pp. 609-630.

242. McBride, O. W., Swan, D. C., Tronick, S. R., Gol, R., Klimanis, D., Moore, D. E., and Aaronson, S. A.: Regional chromosomal localization of N-*ras*, K-*ras*-1, K-*ras*-2 and *myb* oncogenes in human cells. Nucleic Acids Res. 11: pp. 8221-8236, 1983.

243. Devare, S. G., Shatzman, A., Robbins, K. C., Rosenberg, M., and Aaronson, S. A.: Expression of the PDGF-related transforming protein of simian sarcoma virus in *E. coli*. Cell 36: pp. 43-49, 1984.

244. Chiu, I. M., Callahan, R., Tronick, S. R., Schlom, J., and Aaronson, S. A.: Major *pol* gene progenitors in the evolution of oncoviruses. Science 223: pp. 364-370, 1984.

245. Srinivasan, A., Reddy, E. P., Dunn, C. Y., and Aaronson. S. A.: Molecular dissection of transcriptional control elements within the long terminal repeat of the retrovirus. Science 223: pp. 286-289, 1984.

246. Chiu, I.-M., Reddy, E. P., Givol, D., Robbins, K. C., Tronick, S. R., and Aaronson, S. A.: Nucleotide sequence analysis identifies the human c-*sis* proto-oncogene as a structural gene for platelet-derived growth factor. Cell 37: pp. 123-129, 1984.

247. Tronick, S. R. and Aaronson, S. A.: Unique interactions of retroviruses with eukaryotic cells. *In* Notkins, A. L. and Oldstone, M. B. A. (Eds.): Concepts in Viral Pathogenesis. New York, Springer-Verlag, 1984, pp. 165-171.

248. McBride, O. W., Swan, D. S., Robbins, K. C., Prakash, K., and Aaronson, S. A.: Chromosomal mapping of tumor virus tranforming gene analogs in human cells. *In* Pearson, M. L. and Sternberg, N. L. (Eds.): Gene Transfer and Cancer 1982. New York, Raven Press, 1984, pp. 197-205.

249. Pierce, J. H., Aaronson, S. A., and Anderson, S. M.: Hematopoietic cell transformation by a murine recombinant retrovirus containing the *src* gene of Rous sarcoma virus. Proc. Natl. Acad. Sci. USA 81: pp. 2374-2378, 1984.

250. Fujita, J., Yoshida, O., Yuasa, Y., Rhim, J. S., Hatanaka, M., and Aaronson, S. A.: Ha-*ras* oncogenes are activated by somatic alterations in human urinary tract tumours. Nature 309: pp. 464-466, 1984.

251. Yuasa, Y., Gol, R. A., Chang, A., Chiu, I.-M., Reddy, E. P., Tronick, S. R., and Aaronson, S. A.: Mechanism of activation of an N-*ras* oncogene of SW-1271 human lung carcinoma cells. Proc. Natl. Acad. Sci. USA 81: pp. 3670-3674, 1984.

252. Robbins, K. C., Antoniades, H. N., Devare, S. G., Hunkapiller, M. W., and Aaronson, S. A.: Close similarities between the transforming gene product of simian sarcoma virus and human platelet-derived growth factor. *In* Arnold, J. L., Vande Woude, G. F., Topp, W. C. and Watson, J. D. (Eds.): Cancer Cells 1/The Transformed Phenotype. New York, Cold Spring Harbor Laboratory, 1984, pp. 35-42.

253. Yuasa, Y., Eva, A., Kraus, M. H., Srivastava, S. K., Needleman, S. W., Pierce, J. H., Rhim, J. S., Gol, R., Reddy, E. P., Tronick, S. R., and Aaronson, S. A: *ras*-related oncogenes of human tumors. *In* Vande Woude, G. F. Levine, A. J., Topp, W. C. and Watson, J. D. (Eds.): Cancer Cell 2/Oncogenes and Viral Genes. New York, Cold Spring Harbor Laboratory, 1984, pp. 433-439.

254. Narayanan, R., Srinivasan, A., and Aaronson, S. A.: Sequences in the long terminal repeats of the Moloney murine sarcoma virus-124 genome which control transforming gene function. Virology 137: pp. 32-40, 1984.

255. Kraus, M., Yuasa, Y., and Aaronson, S. A.: A position 12-activated H-*ras* oncogene in all HS578T mammary carcinosarcoma cells but not normal mammary cells of the same patient. Proc. Natl. Acad. Sci. USA 81: pp. 5384-5388, 1984.

256. Aaronson, S. A., Yuasa, Y., Robbins, K. C., Eva, A., Gol, R., and Tronick, S. R.: Oncogenes and the neoplastic process. *In* Aaronson, S. A., Frati, L. and Verna, R. (Eds.): Genetic and Phenotypic Markers of Tumors. New York, Plenum Press, 1984, pp. 261-278.

257. Pierce, J. H. and Aaronson, S. A.: Interaction of acute transforming retroviruses with murine hematopoietic cells. *In* Potter, M., Melchers, F. and Weigert, M. (Eds.): Oncogenes and B-Cell Neoplasia. Current Topics in Microbiology and Immunology. Berlin/Heidelberg, Springer-Verlag, 1984, pp. 258-264.

258. Eva, A. and Aaronson, S. A.: Identification and preliminary characterization of a new transforming gene from a human lymphoma. *In* Bishop, J. M., Rowley, J. D. and Greaves, M. (Eds.): Genes and Cancer. UCLA Symposia on Molecular and Cellular Biology. New York, Alan R. Liss, Inc., 1984, pp. 373-382.

259. Aaronson, S. A.: Transforming genes of retroviruses and human cancer cells. *In* Fortner, J. G. and Rhoads, J. E. (Eds.): Accomplishments in Cancer Research, 1983. Philadelphia, J. B. Lippincott Company, 1984, pp. 139-150.

260. Gazit, A., Igarashi, H., Chiu, I.-M., Srinivasan, A., Yaniv, A., Tronick, S. R., Robbins, K. C., and Aaronson, S. A.: Expression of the normal human *sis*/PDGF-2 coding sequence induces cellular transformation. Cell 39: pp. 89-97, 1984.

261. Pierce, J. H., Eva, A., and Aaronson, S. A.: Hematopoietic cell targets for transformation by retroviral oncogenes. *In* Peschle, C. and Rizzoli, C. (Ed.): New Trends in Experimental Hematology. Rome, Italy, Ares Serono Symposia, 1984, pp. 1-18.

262. Aaronson, S. A., Frati, L., and Verna, R. (Eds.): Genetic and Phenotypic Markers of Tumors. New York, Plenum Press, 1984, pp. 379.

263. Balachandran, R., Reddy, E. P., Dunn, C. Y., Aaronson, S. A., and Swan, D: Immunoglobulin synthesis and gene rearrangements in lymphoid cells transformed by replication-competent Rauscher murine leukemia virus: transformation of B cells at various stages of differentiation. EMBO J. 3: pp. 3199-3207, 1984.

264. Tronick, S. R., Yuasa, Y., Robbins, K. C., Eva, A., Gol, R., and Aaronson, S. A.: Oncogene research: closing in on a better understanding of cancer causation. *In* Selikoff, I. J., Teirstein, A. S. and Hirschman, S. Z. (Eds.): Acquired Immune Deficiency Syndrome. New York, The New York Academy of Sciences, 1984, pp. 150-160.

265. Aaronson, S. A., Robbins, K. C., and Tronick, S. R.: Human proto-oncogenes, growth factors, and cancer. *In* Ford, R. J. and Maizel, A. L. (Eds.): Mediators in Cell Growth and Differentiation. New York, Raven Press, 1985, pp. 241-255.

266. Aaronson, S. A. and Tronick, S. R.: The role of oncogenes in human neoplasia. *In* Devita, V. T., Hellman, S., and Rosenberg, S. A. (Eds.): Important Advances in Oncology 1985. Philadelphia, J. B. Lippincott Co., 1985, pp. 3-15.

267. Srivastava, S. K., Yuasa, Y., Reynolds, S. H., and Aaronson, S. A.: Effects of two major activating lesions on the structure and conformation of human *ras* oncogene products. Proc. Natl. Acad. Sci. USA. 82: pp. 38-42, 1985.

268. Rhim, J. S., Jay, G., Arnstein, P., Price, F. M., Sanford, K. K., and Aaronson, S. A.: Neoplastic transformation of human epidermal keratinocytes by AD12-SV40 and Kirsten sarcoma viruses. Science 227: pp. 1250-1252, 1985.

269. Aaronson, S. A. and Robbins, K. C.: Activation of a gene coding for a normal human growth factor to one with transforming properties. *In* Furmanski, P., Hager, J. C. and Rich, M. A. (Eds): RNA Tumor Viruses, Oncogenes Human Cancer and AIDS: On the Frontiers of Understanding. Boston, Martinus Nijhoff Publishing, 1985, pp. 54-66.

270. Reddy, E. P., Lipman, D., Andersen, P. R., Tronick, S. R., and Aaronson, S. A.: Nucleotide sequence analysis of the BALB/c murine sarcoma virus transforming gene. J. Virol. 53: pp. 984-987, 1985.

271. Igarashi, H, Gazit, A., Chiu, I.-M., Srinivasan, A., Yaniv, A., Tronick, S. R., Robbins, K. C., and Aaronson, S. A.: Expression of a normal growth factor gene causes morphologic transformation. *In* Giraldo, E., Beth, E., Castello, G., Giordano, G. G., and Zarrilli, D. (Eds.): From Oncogenes to Tumor Antigens. Amsterdam, Elsevier, 1985, pp. 3-15.

272. Aaronson, S. A., Tronick, S. R., and Robbins, K. C.: Oncogenes and pathways to malignancy. *In* Boynton, A. L. and Leffert, H. L. (Eds): Control of Animal Cell Proliferation. New York, Academic Press, 1985, pp. 3-24.

273. Pierce, J. H. and Aaronson, S. A.: Myeloid cell transformation by *ras*-containing murine sarcoma viruses. Mol. Cell. Biol. 5: pp. 667-674, 1985.

274. Callahan, R., Chiu, I.-M., Horn T., Ali, I., Robbins, J.,. Aaronson, S. A., and Schlom, J.: A new class of human endogenous retroviral genes. *In* Rich, M. (Ed.): RNA Tumor Viruses in Human Cancer. Denver, Martinus Nijhoff Publishing, 1985, pp. 76-92.

275. Callahan, R., Chiu, I.-M., Wong, J. F. H., Tronick, S. R., Roe, B. A., Aaronson, S. A., and Schlom, J.: A new class of endogenous human retroviral genomes. Science 228: pp. 1208-1211, 1985.

276. Fujita, J., Srivastava, S. K., Kraus, M. H., Rhim, J. S., Tronick, S. R., and Aaronson, S. A.: Frequency of molecular alterations affecting *ras* proto-oncogenes in human urinary tract tumors. Proc. Natl. Acad. Sci. USA 82: pp. 3849-3853, 1985.

277. Igarashi, H., Gazit, A., Chiu, I.-M., Srinivasan, A., Yaniv, A., Tronick, S. R., Robbins, K. C., and Aaronson, S. A.: Normal human *sis*/PDGF-2 gene expression induces cellular transformation. *In* Feramisco, J., Ozanne, B., and Stiles, C. (Eds.): Cancer Cells 3/Growth Factors and Transformation. New York, Cold Spring Harbor Laboratories, 1985, pp. 159-166.

278. Eva, A. and Aaronson. S. A.: Isolation of a new human oncogene from a diffuse B-cell lymphoma. Nature 316: pp. 273-275, 1985.

279. Pierce, J. H., Di Fiore, P. P., Aaronson, S. A., Potter, M., Pumphrey, J., Scott, A., and Ihle, J. N.: Neoplastic transformation of mast cells by Abelson-MuLV: Abrogation of IL-3 dependence by a nonautocrine mechanism. Cell 41: pp. 685-693, 1985.

280. Chiu, I.-M., Huang, R.-C. C., and Aaronson, S. A.: Genetic relatedness between intracisternal A particles and other major oncovirus genera. Virus Res. 3: pp. 1-11, 1985.

281. Popescu, N. C., Amsbaugh, S. C., DiPaolo, J. A., Tronick, S. R., Aaronson, S. A., and Swan, D. C.: Chromosomal localization of three human *ras* genes by *in situ* molecular hybridization. Somatic Cell and Mol. Genet. 11: pp. 149-155, 1985.

282. Robbins, K. C., Leal, F., Pierce, J. H., and Aaronson, S. A.: The v-*sis*/PDGF-2 transforming gene product localizes to cell membranes but is not a secretory protein. EMBO J. 4: pp. 1783-1792, 1985.

283. Yaniv, A., Dahlberg, J. E., Tronick, S. R., Chiu, I.-M., and Aaronson, S. A.: Molecular cloning of integrated caprine arthritis-encephalitis virus. Virology 145: pp. 340-345, 1985.

284. King, C. R., Giese, N. A., Robbins, K. C. and Aaronson, S. A.: *In vitro* mutagenesis of the v-*sis* transforming gene defines functional domains of its growth factor-related product. Proc. Natl. Acad. Sci. USA 82: pp. 5295-5299, 1985.

285. Aaronson, S. A. and Tronick, S. R.: Transforming genes of human malignancies. *In* Huberman, E. and Barr, S. H. (Eds.): The Role of Chemicals and Radiation in the Etiology of Cancer. Carcinogenesis: A Comprehensive Survey. New York, Raven Press, 1985, pp. 35-49.

286. King, C. R., Kraus, M. H., and Aaronson, S. A.: Amplification of a novel v-*erb*B-related gene in a human mammary carcinoma. Science 229: pp. 974-976, 1985.

287. Rhim, J. S., Sanford, K. K., Arnstein, P., Fujita, J., Jay, G., and Aaronson, S. A.: Human epithelial cell carcinogenesis: combined action of DNA and RNA tumor viruses produces malignant transformation of primary human epidermal keratinocytes. *In* Barrett, J. C. and Tennant, R. W. (Eds.): Carcinogenesis. New York, Raven Press, 1985, pp. 57-66.

288. Chiu, I.-M., Yaniv, A., Dahlberg, J. E., Gazit, A., Skuntz, S., Tronick, S. R., and Aaronson, S. A.: Nucleotide sequence evidence for relationship of AIDS retrovirus to lentiviruses. Nature 317: pp. 366-368, 1985.

289. Leal, F., William, L. T., Robbins, K. C., and Aaronson, S. A.: Evidence that the v-*sis* gene product transforms by interaction with the receptor for platelet-derived growth factor. Science 230: pp. 327-330, 1985.

290. Srivastava, S. K., Lacal, J. C., Reynolds, S. H., and Aaronson, S. A.: Antibody of predetermined specificity to a carboxy-terminal region of H-*ras* gene products inhibits their guanine nucleotide-binding function. Mol. Cell. Biol. 5: pp. 3316-3319, 1985.

291. Robbins, K. C., Igarashi, H., Gazit, A., Chiu, I.-M., Tronick, S. R., and Aaronson, S. A.: Establishing the link between human proto-oncogenes and growth factors. *In* Castellani, A. (Ed.): Epidemiology and Quantitation of Environmental Risk in Humans from Radiation and Other Agents. New York, Plenum Press, 1985, pp. 459-470.

292. Weissman, B. and Aaronson, S. A.: Members of the *src* and *ras* oncogene families supplant the epidermal growth factor requirement of BALB/MK-2 keratinocytes and induce distinct alterations in their terminal differentiation program. Mol. Cell. Biol. 5: pp. 3386-3396, 1985.

293. King, C. R., Giese, N. A., Kraus, M. H., Robbins, K. C., and Aaronson, S. A.: Oncogenes as growth factors and growth factor receptors: genetic studies of v-*sis* and a novel *erb*B-related gene. *In* Galeotti, T., Cittadini, A., Neri, G., Papa, S., and Smets, L. A. (Eds.): Cell Membranes and Cancer. Amsterdam, Elsevier Science Publishers, 1985, pp. 411-416.

294. Eva, A., Pierce, J. H., and Aaronson, S. A.: Interactions of oncogenes with hematopoietic cells. *In* Gale, R. P. and Golde, D. W. (Eds): Leukemia: Recent Advances in Biology and Treatment. New York, Alan R. Liss, Inc., 1985, pp. 3-15.

295. King, C. R., Kraus, M. H., Williams, L. T., Merlino, G. T., Pastan, I. H., and Aaronson, S. A.: Human tumor cell lines with EGF receptor gene amplification in the absence of aberrant sized mRNAs. Nucleic Acids Res. 13: pp. 8477-8486, 1985.

296. Eva, A., Aaronson, S. A., and Tronick, S. R.: Transforming genes of human malignancies. *In* Pullman, B. et al. (Eds.): Interrelationship Among Aging, Cancer and Differentiation. Holland, D. Reidel Publishing Co., 1985, pp. 43-57.

297. Tronick, S. R., Eva, A., Srivastava, S. K, Kraus, M. H., Yuasa, Y., and Aaronson, S. A.: The role of human *ras* proto-oncogenes in cancer. *In* Castellani, A. (Ed.): Epidemiology and Quantitation of Environmental Risk in Humans from Radiation and Other Agents. New York, Plenum Press, 1985, pp. 225-233.

298. Needleman, S. W., Kraus, M. H., Srivastava, S. K., Levine, P. H., and Aaronson, S. A.: High frequency of N-*ras* activation in acute myelogenous leukemia. Blood 67: 753-757, 1986.

299. Lacal, J. C., Srivastava, S. K., Anderson, P. S., and Aaronson, S. A.: *Ras* p21 proteins with high or low GTPase activity can efficiently transform NIH/3T3 cells. Cell 44: pp. 609-617, 1986.

300. Lacal, J. C., Anderson, P. S., and Aaronson, S. A.: Deletion mutants of Harvey *ras* p21 protein reveal the absolute requirement of at least two distant regions for GTP-binding and transforming activities. EMBO J. 5: pp. 679-687, 1986.

301. Rhim, J. S., Fujita, J., Arnstein, P., and Aaronson, S. A.: Neoplastic conversion of human keratinocytes by adenovirus 12-SV40 virus and chemical carcinogens. Science 232: pp. 385-388, 1986.

302. Rao, C. D., Igarashi, H., Chiu, I.-M., Robbins, K. C., and Aaronson, S. A.: Structure and sequence of the human c-*sis*/platelet-derived growth factor 2 (sis/PDGF2) transcriptional unit. Proc. Natl. Acad. Sci. USA 83: pp. 2392-2396, 1986.

303. Lacal, J. C. and Aaronson, S. A.: Monoclonal antibody Y13-259 recognizes an epitope of the p21 *ras* molecule not directly involved in the GTP-binding activity of the protein. Mol. Cell. Biol. 6: pp. 1002-1009, 1986.

304. Lacal, J. C. and Aaronson, S. A.: *ras* p21 deletion mutants and monoclonal antibodies as tools for localization of regions relevant to p21 function. Proc. Natl. Acad. Sci. USA 83: pp. 5400-5404, 1986.

305. Yuasa, Y., Reddy, E. P., Rhim, J. S., Tronick, S. R., and Aaronson, S. A.: Activated N-*ras* in a human rectal carcinoma cell line associated with clonal homozygosity in *myb* locus-restriction fragment polymorphism. Jpn. J. of Cancer Res. 77: pp. 639-647, 1986.

306. Leal, F., Igarashi, H., Gazit, A., Williams, L. T., Notario, V., Tronick, S. R., Robbins, K. C., and Aaronson, S. A.: Mechanism of transformation by an oncogene coding for a normal growth factor. *In* Harris, C. C. (Ed.): Biochemical and Molecular Epidemiology of Cancer. UCLA Symposia on Molecular and Cellular Biology. New York, Alan R. Liss, Inc., 1986, pp. 155-165.

307. Vecchio, G., Di Fiore, P. P., Fusco, A., Coletta, G., Weissman, B. E., and Aaronson, S. A.: *In vitro* transformation of epithelial cells by acute retroviruses. *In* Blasi, F. (Ed.): Human Genes and Diseases. Horizons in Biochemistry and Biophysics. Chichester, England, John Wiley & Sons Ltd., 1986, pp. 415-470.

308. Yaniv, A., Dahlberg, J., Gazit, A., Sherman, L., Chiu, I.-M., Tronick, S. R., and Aaronson, S. A.: Molecular cloning and physical characterization of integrated equine infectious

anemia virus: molecular and immunologic evidence of its close relationship to ovine and caprine lentiviruses. Virology 154: pp. 1-8, 1986.

309. Robbins, K. C., King, C. R., Giese, N. A., Leal, F., Igarashi, H. and Aaronson, S. A.: Involvement of oncogene-coded growth factors in the neoplastic process. *In* Papas, T. S. and Vande Woude, G. F. (Eds.): Gene Amplification and Analysis. Oncogenes. New York, Elsevier, 1986, pp. 161-176.

310. Robbins, K. C. and Aaronson, S. A.: Elucidation of a normal function for a human proto-oncogene. *In* Luderer, A. A. and Weetall, H. H. (Eds.): The Human Oncogenic Viruses. Molecular Analysis and Diagnosis. New Jersey, Humana Press, Inc., 1986, pp. 89-112.

311. Gazit, A., Pierce, J. H., Kraus, M. H., Di Fiore, P. P., Pennington, C. Y., and Aaronson, S. A.: Mammalian cell transformation by a murine retrovirus vector containing the avian erythroblastosis virus *erb*B gene. J. Virol. 60: pp. 19-28, 1986.

312. Pierce, J. H., Gazit, A., Di Fiore, P. P., Kraus, M. H., Pennington, C. Y., Holmes, K. L., Davidson, W. F., Morse III, H. C., and Aaronson, S. A.: Mammalian cell transformation by a recombinant murine retrovirus containing the avian erythroblastosis virus *erb*B gene. *In* Potter, M. (Ed.): Current Topics in Microbiology and Immunology. Berlin/Heidelberg, Springer-Verlag, 1986, pp. 55-61.

313. Pierce, J. H., Eva, A., and Aaronson, S. A.: Interactions of oncogenes with hematopoietic cells. *In* Gale, R. P., and Hoffbrand, A. V. (Eds): Clinics in Haematology. Acute Leukaemia. London/Philadelphia/Toronto, W. B. Saunders Co., 1986, pp. 573-596.

314. Srivastava, S. K., Wheelock, R. H. P., Aaronson, S. A., and Eva, A.: Identification of the protein encoded by the human diffuse B-cell lymphoma (*dbl*) oncogene. Proc. Natl. Acad. Sci. USA 83: pp. 8868-8872, 1986.

315. Tronick, S. R. and Aaronson, S. A.: Oncogenes, growth factors, and receptors. *In* Notkins, A. and Oldstone, M. (Eds.): Concepts in Viral Pathogenesis 11. New York, Springer-Verlag, 1986, pp. 98-109.

316. Lacal, J. C. and Aaronson, S. A.: Activation of *ras* p21 transforming properties associated with an increase in the release rate of bound guanine nucleotide. Mol. Cell Biol. 6: pp. 4214-4220, 1986.

317. Pasti, G., Lacal, J. C., Warren, B. S., Aaronson, S. A., and Blumberg, P. M.: Loss of mouse fibroblast cell response to phorbol esters restored by microinjected protein kinase C. Nature 324: pp. 375-377, 1986.

318. Kruh, G. D., King, C. R., Kraus, M. H., Popescu, N. C., Amsbaugh, S. C., McBride, W. O, and Aaronson, S. A.: A novel human gene closely related to the *abl* proto-oncogene. Science 234: pp. 1545-1548, 1986.

319. Reynolds, S. H., Stowers, S. J., Maronpot, R. R., Anderson, M. W., and Aaronson, S. A.: Detection and identification of activated oncogenes in spontaneously occurring benign and malignant hepatocellular tumors of the B6C3F1 mouse. Proc. Natl. Acad. Sci. USA 83: pp. 33-37, 1986.

320. Igarashi, H., Rao, C. D., Siroff, M., Leal, F., Robbins, K. C., and Aaronson, S. A.: Detection of PDGF-2 homodimers in human tumor cells. Oncogene 1: pp. 79-85, 1987.

321. Rao, C. D., Igarashi, H., Pech, M. W., Robbins, K. C., and Aaronson, S. A.: Oncogenic potential of the human platelet-derived growth factor transcriptional unit. Cold Spring Harbor Symp. Quant. Biol. 51: pp. 959-966, 1987.

322. Dahlberg, J. E., Mitsuya, H., Blam, S. B., Broder, S., and Aaronson, S. A.: Broad spectrum antiretroviral activity of 2', 3'-dideoxynucleosides. Proc. Natl. Acad. Sci. USA 84: pp. 2469-2473, 1987.

323. Hagag, N., Lacal, J. C., Graber, M., Aaronson, S. A., and Viola, M.: Microinjection of *ras* p21 induces a rapid rise in intracellular pH. Mol. Cell. Biol. 7: pp. 1984-1988, 1987.

324. Giese, N. A., Robbins, K. C., and Aaronson, S. A.: The role of individual cysteine residues in the structure and function of the v-*sis* gene product. Science 236: pp. 1315-1318, 1987.

325. Albini, A., Iwamoto, Y., Kleinman, H. K., Martin, G. R., Aaronson, S. A., Kozlowski, J. M., and McEwan, R. N.: A rapid *in vitro* assay for quantitating the invasive potential of tumor cells. Cancer Research 47: pp. 3239-3245, 1987.

326. Kawakami, T., Sherman, L., Dahlberg, J., Gazit, A., Yaniv, A., Tronick, S. R., and Aaronson, S. A.: Nucleotide sequence analysis of equine infectious anemia virus proviral DNA. Virology 158: pp. 300-312, 1987.

327. Finzi, E., Fleming, T., Segatto, O., Pennington, C. Y., Bringman, T. S., Derynck, R., and Aaronson, S. A.: The human transforming growth factor type αcoding sequence is not a direct-acting oncogene when overexpressed in NIH 3T3 cells. Proc. Natl. Acad. Sci. USA 84: pp. 3733-3737, 1987.

328. Di Fiore, P. P., Pierce, J. H., Kraus, M. H., Segatto, O., King. C. R., and Aaronson, S. A.: *erb*B-2 is a potent oncogene when overexpressed in NIH/3T3 cells. Science 237: pp. 178-182, 1987.

329. Aaronson, S. A., Igarashi, H., Rao, C. D., Finzi, E., Fleming, T. P., Segatto, O., and Robbins, K. C.: Role of genes for normal growth factors in human malignancy. *In* Aaronson, S. A., Bishop, J. M., Sugimura, T., Terada, M., Toyoshima, K., and Vogt, P. K. (Eds.): Oncogenes and Cancer. Tokyo, Japan, Japan Scientific Societies Press and Utrecht The Netherlands, VNU Science Press BV, 1987, pp. 95-108.

330. Aaronson, S. A., Bishop, J. M., Sugimura, T., Terada, M., Toyoshima, K., and Vogt, P. K. (Eds.): Oncogenes and Cancer. Tokyo, Japan Scientific Societies Press and Utrecht The Netherlands, VNU Science Press BV, 1987, pp. 309.

331. Matyas, G. R., Aaronson, S. A., Brady, R. O., and Fishman, P. H.: Alteration of glycolipids in *ras*-transfected NIH/3T3 cells. Proc. Natl. Acad. Sci. USA 84: pp. 6065-6068, 1987.

332. Eva, A., Srivastava, S. K., Vecchio, G., Ron, D., Tronick, S. R., and Aaronson, S. A.: *dbl*: A new transforming gene isolated from a human diffuse B-cell lymphoma. *In* Cimino, F., Birkmayer, G. D., Klavins, J. V., Pimentel, E., and Salvatore, F. (Eds.): Human Tumor Markers. Berlin/New York, Walter De Gruyter & Co., 1987, pp. 83-92.

333. Reynolds, S. H., Stowers, S. J., Patterson, R. M., Maronpot, R. R., Aaronson, S. A., and Anderson, M. W.: Activated oncogenes in B6C3F1 mouse liver tumors: implications for risk assessment. Science 237: pp. 1309-1316, 1987.

334. Lacal, J. C., de La Pena, P., Moscat, J., Garcia-Barreno, P., Anderson, P. S., and Aaronson, S. A.: Rapid stimulation of diacylglycerol production in Xenopus oocytes by microinjection of H-*ras* p21. Science 238: pp. 533-536, 1987.

335. Lacal, J. C., Fleming, T. P., Warren, B. S., Blumberg, P. M., and Aaronson, S. A.: Involvement of functional protein kinase C in the mitogenic response to the H-*ras* oncogene product. Mol. Cell. Biol. 7: pp. 4146-4149, 1987.

336. Lacal, J. C., Moscat, J., and Aaronson, S. A.: Novel source of 1,2-diacylglycerol elevated in cells transformed by Ha-*ras* oncogene. Nature 330: pp. 269-272, 1987.

337. Eva, A., Vecchio, G., Diamond, M., Tronick, S. R., Ron, D., Cooper, G. M., and Aaronson, S. A.: Independently activated *dbl* oncogenes exhibit similar yet distinct structural alterations. Oncogene 1: pp. 355-360, 1987.

338. Di Fiore, P. P., Pierce, J. H., Fleming, T. P., Hazan, R., Ullrick, A., King, C. R., Schlessinger, J., and Aaronson, S. A.: Overexpression of the human EGF receptor confers an EGF-dependent transformed phenotype to NIH/3T3 cells. Cell 51: pp. 1063-1070, 1987.

339. Eva, A., Pierce, J. H., and Aaronson, S. A.: Interactions of retroviral and cellular transforming genes with hematopoietic cells. *In* Peschle, C. (Ed.): Normal and Neoplastic Blood Cells: From Genes to Therapy. New York, The New York Academy of Sciences, 1987, pp. 148-170.

340. Rao, C. D., Pech, M., Robbins, K. C., and Aaronson, S. A.: The 5' untranslated sequence of the c-*sis*/PDGF-2 transcript is a potent translational inhibitor. Mol. Cell. Biol. 8: pp. 284-292, 1988.

341. Di Fiore, P. P., Falco, J., Borrello, I., Weissman, B., and Aaronson, S. A.: The calcium signal for BALB/MK keratinocyte terminal differentiation counteracts epidermal growth factor (EGF) very early in the EGF-induced proliferative pathway. Mol. Cell. Biol. 8: pp. 557-563, 1988.

342. Pierce, J. H., Ruggiero, M., Fleming, T. P., Di Fiore, P. P., Greenberger, J. S., Varticovski, L., Schlessinger, J., Rovera, G., and Aaronson, S. A.: Signal transduction through the EGF receptor transfected into interleukin-3-dependent hematopoietic cells. Science 239: pp. 628-631, 1988.

343. Mitsuya, H., Dahlberg, J. E., Spigelman, Z., Matsushita, S., Jarrett, R. F., Matsukura, M., Currens, M. J., Aaronson, S. A., Reitz, M. S., McCaffrey, R. S., and Broder, S.: 2',3'-dideoxynucleosides: Broad spectrum antiretroviral activity and mechanisms of action. *In* Bolognesi, D. (Ed.): Human Retroviruses, Cancer, and AIDS: Approaches to Prevention and Therapy. Alan R. Liss, Inc., New York, 1988, pp. 407-421.

344. Eva, A., Vecchio, G., Rao, C. D., Tronick, S. R., and Aaronson, S. A.: The predicted *dbl* oncogene product defines a distinct class of transforming proteins. Proc. Natl. Acad. Sci. USA 85: pp. 2061-2065, 1988.

345. Tronick, S. R. and Aaronson, S. A.: Oncogenes, growth regulation and cancer. *In* Adelstein, R., Klee, C. and Rodbell, M. (Eds.): Advances in Second Messenger and Phosphorphoprotein Research. New York, Raven Press, Ltd., 1988, pp. 201-215.

346. Kawakami, T., Kawakami, Y., Aaronson, S. A., and Robbins, K. C.: Acquisition of transforming properties by *fyn*, a normal *src*-related human gene. Proc. Natl. Acad. Sci. USA 85: pp. 3870-3874, 1988.

347. Falco, J. P., Taylor, W. G., Di Fiore, P. P., Weissman, B. E., and Aaronson, S. A.: Interactions of growth factors and retroviral oncogenes with mitogenic signal transduction pathways of BALB/MK keratinocytes. Oncogene 2: pp. 573-578, 1988.

348. King, C. R., Di Fiore, P. P., Pierce, J. H., Segatto, O., Kraus, M. H., and Aaronson, S. A.: Oncogenic potential of the *erb*B-2 gene: frequent overexpression in human mammary adenocarcinomas and induction of transformation *in vitro*. *In*: Lippman, M. E. (Ed.): Growth Regulation of Cancer. Proceedings of UCLA Symposium. New York, Alan R. Liss, Inc., 1988, pp. 189-199.

349. Ron, D., Tronick, S. R., Aaronson, S. A., and Eva, A.: Molecular cloning and characterization of the human *dbl* proto-oncogene: evidence that its overexpression is sufficient to transform NIH/3T3 cells. EMBO J. 7: pp. 2465-2473, 1988.

350. Eva, A., Srivastava, S., Vecchio, G., Ron, D., Tronick, S. R., and Aaronson, S. A.: Biochemical characterization of dbl oncogene and its product. *In* Tonini, G. P., Massimo, L., and Cornaglia-Ferraris, P., (Eds.): Oncogenes in Pediatric Tumors.

London/New York, Harwood Academic Publishers, Life Science Series, 1988, vol. 4, pp. 215-231.

351. Moscat, J., Molloy, C. J., Fleming, T. P., and Aaronson, S. A.: Epidermal growth factor activates phospho-inositide turnover and protein kinase C in BALB/MK keratinocytes. Mol. Endocrinol. 9: pp. 799-805, 1988.

352. Blam, S.B., Mitchell, R., Tischer, E., Rubin, J. S., Silva, M., Silver, S., Fiddes, J. C., Abraham, J. A., and Aaronson, S. A.: Addition of growth hormone secretion signal to basic fibroblast growth factor results in cell transformation and secretion of aberrant forms of the protein. Oncogene 3: pp. 129-136, 1988.

353. Finzi, E., Kilkenny, A., Strickland, J. E., Balaschak, M., Bringman, T., Derynck, R., Aaronson, S. A., and Yuspa, S. H.: TGF® stimulates growth of skin papillomas by autocrine and paracrine mechanisms but does not cause neoplastic progression. Mol. Carcinogen 1: pp 7-12, 1988.

354. Beckmann, M. P., Betsholtz, C., Heldin, C.-H., Westermark, B., Di Fiore, P. P., Robbins, K. C., and Aaronson, S. A.: Comparison of biological properties and transforming potential of human PDGF-A and PDGF-B chains. Science 241: pp. 1346-1349, 1988.

355. Kraus, M. H., Di Fiore, P. P., Pierce, J. H., and Aaronson, S. A.: Different mechanisms are responsible for oncogene activation in human mammary neoplasia. *In* Lippman, M. E., and Dickson, B. (Eds.): Breast Cancer: Cellular and Molecular Biology. Boston, Martinus Nijhoff Publishing, 1988, pp. 49-66.

356. Robbins, K. C. and Aaronson, S. A.: The *sis* oncogene. *In* Reddy, E. P., Curran, T., and Skalka, A. (Eds.): The Oncogene Handbook. Elsevier, 1988, pp. 427-452.

357. Kraus, M. H., Pierce, J. H., Fleming, T. P., Robbins, K. C., DiFiore, P. P., and Aaronson, S. A.: Mechanisms by which genes encoding growth factors and growth factor receptors contribute to malignant transformation. *In* Galeotti, T., Cittadini, A., Neri, G., and Scarpa, A. (Eds.): Membrane in Cancer Cells. New York, Annual of the New York Academy of Sciences, 1988, pp. 320-336.

358. Di Fiore, P. P., Pierce, J. H., Kraus, M. H., Fleming, T. P., Robbins, K. C., and Aaronson, S. A.: The role of growth factors and growth factor receptors in neoplastic cell transformation. *In* Sorenson, G.D. (Ed.): Lung Cancer, International Conference on Hormones, Growth Factors and Oncogenes in Pulmonary Carcinoma. Elsevier Science Publishers, 1988, pp. 135-154.

359. Segatto, O., King, C. R., Pierce, J. H., Di Fiore, P. P., and Aaronson, S. A.: Different structural alterations upregulate *in vitro* tyrosine kinase activity and transforming potency of the *erb*B-2 gene. Mol. Cell. Biol. 8: pp. 5570-5574, 1988.

360. Pech, M., Rao, C. D., Robbins, K. C., and Aaronson, S. A.: Functional identification of regulatory elements within the promoter region of platelet-derived growth factor 2. Mol. Cell. Biol. 9: pp. 396-405, 1989.

361. Rubin, J. S., Osada, H., Finch, P. W., Taylor, W. G., Rudikoff, S., and Aaronson, S. A.: Purification and characterization of a newly identified growth factor specific for epithelial cells. Proc. Natl. Acad. Sci. USA 86: pp. 802-806, 1989.

362. Matsui, T., Heidaran, M., Miki, T., Popescu, N., La Rochelle, W. J., Kraus, M. H., Pierce, J. H., and Aaronson, S. A.: Isolation of a novel receptor cDNA establishes the existence of two PDGF receptor genes. Science 243: pp. 800-804, 1989.

363. Pech, M., Gazit, A., Arnstein, P., and Aaronson, S. A.: Generation of fibrosarcomas *in vivo* by a retrovirus that expresses the normal B chain of platelet-derived growth factor and mimics the alternative splice pattern of the v-*sis* oncogene. Proc. Natl. Acad. Sci. USA 86: pp. 2693-2697, 1989.

364. Giese, N., May-Siroff, M., LaRochelle, W. J., Van Wyke Coelingh, K., and Aaronson, S. A.: Expression and purification of biologically active v-*sis*/PDGF-B protein by using a baculovirus vector system. J. Virology 63: pp. 3080-3086, 1989.

365. Di Marco, E., Pierce, J. H., Fleming, T. P., Kraus, M. H., Molloy, C. J., Aaronson, S. A., and Di Fiore, P. P.: Autocrine interaction between TGF® and the EGF-receptor: quantitative requirements for induction of the malignant phenotype. Oncogene 4: pp. 831-838, 1989.

366. Moscat, J., Fleming, T. P., Molloy, C. J., Lopez-Barahona, M., and Aaronson, S. A.: The calcium signal for BALB/MK keratinocyte terminal differentiation induces sustained alterations in phospho-inositide metabolism without detectable protein kinase C activation. J. Biol. Chem. 264: pp. 11228-11235, 1989.

367. LaRochelle, W. J., Robbins, K. C., and Aaronson, S. A.: Immunochemical localization of the epitope for a monoclonal antibody that neutralizes human platelet-derived growth factor mitogenic activity. Mol. Cell. Biol. 9: pp. 3538-3542, 1989.

368. Finch, P. W., Rubin, J. S., Miki, T., Ron, D., and Aaronson, S. A.: Human KGF is FGF-related with properties of a paracrine effector of epithelial cell. Science 245: pp. 752-755, 1989.

369. Ron, D., Graziani, G., Aaronson, S. A., and Eva, A.: The N-terminal region of proto-*dbl* down regulates its transforming activity. Oncogene 4: pp. 1067-1072, 1989.

370. Aaronson, S. A., Falco, J. P., Taylor, W. G., Cech, A. C., Marchese, C., Finch, P. W., Rubin, J. S., Weissman, B. E., and Di Fiore, P.P.: Pathways in which growth factors and oncogenes interact in epithelial cell mitogenic signal transduction. Ann. NY Acad. Sci. 567: pp. 122-129, 1989.

371. Kraus, M. H., Di Fiore, P. P., Pierce, J. H., Robbins, K. C., and Aaronson, S. A.: Overexpression of proto-oncogenes encoding growth factor or growth factor receptor proteins in malignancy. *In* Furth, M., and Greaves, M. (Eds.): Cancer Cells 7/Molecular Diagnostics of Human Cancer. New York, Cold Spring Harbor Laboratory, 1989, pp. 303-309.

372. Fleming, T. P., Matsui, T., Molloy, C. J., Robbins, K. C., and Aaronson, S. A.: Autocrine mechanism for v-*sis* transformation requires cell surface localization of internally activated growth factor receptors. Proc. Natl. Acad. Sci. USA 86: pp. 8063-8067, 1989.

373. Matsui, T., Pierce, J. H., Fleming, T. P., Greenberger, J. S., LaRochelle, W. J., Ruggiero, M., and Aaronson, S. A.: Independent expression of human ⊛ or ⊛ platelet-derived growth factor receptor cDNAs in a naive hematopoietic cell leads to functional coupling with mitogenic and chemotactic signaling pathways. Proc. Natl. Acad. Sci. USA 86: pp. 8314-8318, 1989.

374. Miki, T., Matsui, T., Heidaran, M. A., and Aaronson, S. A.: An efficient directional cloning system to construct cDNA libraries containing full-length inserts at high frequency. Gene 83: pp. 137-146, 1989.

375. Molloy, C. J., Bottaro, D. P., Fleming, T. P., Marshall, M. S., Gibbs, J. B., and Aaronson, S. A.: PDGF induction of tyrosine phosphorylation of GTPase activating protein. Nature 342: pp. 711-714, 1989.

376. Kraus, M. H., Issing, W., Miki, T., Popescu, N. C., and Aaronson, S. A.: Isolation and characterization of *erb*B-3, a third member of the erbB/epidermal growth factor receptor family: Evidence for over-expression in a subset of human mammary tumors. Proc. Natl. Acad. Sci. USA 86: pp. 9193-9197, 1989.

377. Fleming, T. P., Pech, M. W., Di Marco, E., Di Fiore, P. P., Falco, J. P., and Aaronson, S. A. Gene encoding growth factors as oncogenes. *In* Cavenee, W., Hastie, N. and Stanbridge, E. (Eds.). Current Communications in Molecular Biology. New York, Cold Spring Harbor Press, 1989, pp. 203-209.

378. Molloy, C. J., Fleming, T. P., Bottaro, D. P., and Aaronson, S. A.: PDGF signaling pathways in malignancy. *In* Papas, T.S. (Ed.): Oncogenesis: Oncogenes in Signal Transduction and Cell Proliferation. Houston Gulf Publishing, 1990, pp. 13-23.

379. Di Fiore, P. P., Segatto, O., Taylor, W. G., Aaronson, S. A., and Pierce, J. H.: EGF-receptor and *erb*B-2 tyrosine kinase domains confer cell specificity for mitogenic signaling. Science 248: pp. 79-83, 1990.

380. Di Fiore, P. P., Segatto, O., Lonardo, F., Fazioli, F., Pierce, J. H., and Aaronson, S. A.: The carboxy-terminal domains of *erb*B-2 and epidermal growth factor receptor exert different

regulatory effects on intrinsic receptor tyrosine kinase function and transforming activity. Mol. Cell. Biol. 10: pp. 2749-2756, 1990.

381. Melchiori, A., Carlone, S., Allavena, G., Aresu, O., Parodi, S., Aaronson, S. A., and Albini, A.: Invasiveness and chemotactic activity of oncogene transformed NIH/3T3 cell. Anticancer Research 10: pp. 37-44, 1990.

382. LaRochelle, W. J., Giese, N., May-Siroff, M., Robbins, K. C., and Aaronson, S. A.: Molecular localization of the transforming and secretory properties of PDGF A and PDGF B. Science 248: pp. 1541-1544, 1990.

383. Di Marco, E., Pierce, J. H., Aaronson, S. A., and Di Fiore, P. P.: Mechanisms by which EGF receptor and TGF@@contribute to malignant transformation. Nat. Immun. Cell Growth Regul. 9: pp. 209-221, 1990.

384. Bottaro, D. P., Rubin, J. S., Ron, D., Finch, P. W., Florio, C., and Aaronson, S. A.: Characterization of the receptor for keratinocyte growth factor: evidence for multiple fibroblast growth factor receptors. J. Biol. Chem. 265: pp. 12767-12770, 1990.

385. Miki, T., Matsui, T., Heidaran, M. A., and Aaronson, S. A.: Automatic directional cloning: an efficient method to construct eukaryotic expression cDNA libraries. *In* Alitalo, K.K., Huhtala, M.-L., Knowles, J. and Vaheri, A. (Eds.). Proceedings of the International Symposium on Recombinant Systems in Protein Expression. Amsterdam, Elsevier Science Publishers BV, 1990, pp. 125-136.

386. Aaronson, S. A., and Pierce, J. H.: Molecular mimicry of growth factors by products of tumor viruses. Cancer Cells 2: pp. 212-214, 1990.

387. Marchese, C., Rubin, J. S., Ron, D., Faggioni, A., Torrisi, M. R., Messina, A., Frati, L. and Aaronson, S. A.: Human keratinocyte growth factor activity on proliferation and differentiation of human keratinocytes: differentiation response distinguishes KGF from EGF family. J. Cell. Physiol. 144: pp. 326-332, 1990.

388. Heidaran, M. A., Fleming, T. P., Bottaro, D. P., Bell, G. I., Di Fiore, P. P., and Aaronson, S. A.: Transformation of NIH/3T3 fibroblasts by an expression vector for the human epidermal growth factor precursor. Oncogene 5: pp. 1265-1270, 1990.

389. Kruh, G. D., Perego, R., Miki, T., and Aaronson, S. A.: The complete coding sequence of *arg* defines the Abelson subfamily of cytoplasmic tyrosine kinases. Proc. Natl. Acad. Sci. USA 87: pp. 5802-5806, 1990.

390. Pierce, J. H., Di Marco, E., Cox, G. W., Lombardi, D., Ruggiero, M., Varesio, L., Wang, L. M., Choudhury, G. G., Sakaguchi, A. Y., Di Fiore, P. P., and Aaronson, S. A.: Macrophage-colony-stimulating factor (CSF-1) induces proliferation, chemotaxis and reversible monocytic differentiation in myeloid progenitor cells transfected with the human c-*fms*/CSF-1 receptor cDNA. Proc. Natl. Acad. Sci. USA 87: pp. 5613-5617, 1990.

391. Giese, N., LaRochelle, W. J., May-Siroff, M., Robbins, K. C., and Aaronson, S. A.: A small v-*sis*/platelet-derived growth factor (PDGF) B- protein domain in which subtle conformational changes abrogate PDGF receptor interaction and transforming activity. Mol. Cell. Biol. 10: pp. 5496-5501, 1990.

392. McKinnon, R. D., Matsui, T., Dubois-Dalcq, M., and Aaronson, S. A.: FGF modulates the PDGF-driven pathway of oligodendrocyte development. Neuron 5: pp. 603-614, 1990.

393. Crescenzi, M., Fleming, T. P., Lassar, A. B., Weintraub, H., and Aaronson, S. A.: MyoD induces growth arrest independent of differentiation in normal and transformed cells. Proc. Natl. Acad. Sci. USA 87: pp. 8442-8446, 1990.

394. Heidaran, M. A., Pierce, J. H., Jensen, R. A., Matsui, T., and Aaronson, S. A.: Chimeric ⊛- and ⊛-platelet-derived growth factor (PDGF) receptors define three immunoglobulin-like domains of the α-PDGF receptor that determine PDGF-AA binding specificity. J. Biol. Chem. 265: pp. 18741-18744, 1990.

395. Aaronson, S. A., Rubin, J. S., Finch, P. W., Wong, J., Marchese, C., Falco, J., Taylor, W. G., and Kraus, M. H.: Growth factor regulated pathways in epithelial cell proliferation. Am. Rev. Respir. Dis. 142: pp. S7-S10, 1990.

396. Lonardo, F., Di Marco, E., King, C. R., Pierce, J. H., Segatto, O., Aaronson, S. A., and Di Fiore, P. P. The normal *erb*B-2 product is an atypical receptor-like tyrosine kinase with constitutive activity in the absence of ligand. New Biol. 2: pp. 992-1003, 1990.

397. LaRochelle, W. J., Giese, N., May-Siroff, M., Robbins, K. C., and Aaronson, S. A.: Chimeric molecules map and dissociate the potent transforming and secretory properties of PDGF A and PDGF B. J. Cell Sci. Suppl. 13: pp. 31-42, 1990.

398. Rubin, J. S., Chan, A. M.-L., Bottaro, D. P., Burgess, W. H., Taylor, W. G., Cech, A. C., Hirschfield, D. W., Wong, J., Miki, T., Finch, P. W., and Aaronson, S. A.: A broad-spectrum human lung fibroblast-derived mitogen is a variant of hepatocyte growth factor. Proc. Natl. Acad. Sci. USA 88: pp. 415-419, 1991.

399. Heidaran, M. A., Pierce, J. H., Lombardi, D., Ruggiero, M., Gutkind, J. S., Matsui, T., and Aaronson, S. A.: Deletion or substitution within the ⊛ platelet-derived growth factor receptor kinase insert domain: effects on functional coupling with intracellular signaling pathways. Mol. Cell. Biol. 11: pp. 134-142, 1991.

400. Miki, T., Fleming, T. P., Bottaro, D. P., Rubin, J. S., Ron, D., and Aaronson, S. A.: Expression cDNA cloning of the KGF receptor by creation of a transforming autocrine loop. Science 251: pp. pp. 72-75, 1991.

401. Bottaro, D. P., Rubin, J., Faletto, D. L., Chan, A. M-L., Kmiecik, T. E., Vande Woude, G. F., and Aaronson, S. A.: Identification of the hepatocyte growth factor receptor as the c-*met* proto-oncogene product. Science 251: pp. 802-804, 1991.

402. Molloy, C. J., Fleming, T. P., Bottaro, D. P., Cuadrado, A., Pangelinan, M. J., and Aaronson, S. A.: Oncogenes and signal transduction in malignancy. *In* Brinkley, W., Lechner, J. and Harris, C. (Eds.): Current Communications in Molecular Biology: Cellular and Molecular Aspects of Fiber Carcinogenesis. New York, Cold Spring Harbor Laboratory Press, 1991, pp. 67-81.

403. LaRochelle, W. J., Giese, N., Robbins, K. C., and Aaronson, S. A.: Variant PDGF ligands and receptors-structure/function relationship. News in Physiol. Sci. 6: pp. 56-60, 1991.

404. Ron, D., Zannini, M., Lewis, M., Wichner, R. B., Hunt, L. T., Graziani, G., Tronick, S. R., Aaronson, S. A., and Eva. A.: A region of proto-*dbl* essential for its transforming activity shows sequence similarity to a yeast cell cycle gene, CDC24 and the human breakpoint cluster gene, *bcr*. New Biol. 3: pp. 372-379, 1991.

405. Aaronson, S. A., and Tronick, S.R.: Growth factor signalling pathways and their alterations in human tumors. FORUM 1.1: pp. 14-35, 1991.

406. Smith, A., Seldin, M.F., Martinez, L., Watson, M.L., Choudhury, G.G., Lalley, P.A., Pierce, J., Aaronson, S.A., Barker, J., Naylor, S.L., and Sakaguchi, A.Y.: Mouse platelet-derived growth factor receptor α gene is deleted in W^{19H} and patch mutations on chromosome 5. Proc. Natl. Acad. Sci. USA 88: 4811-4815, 1991.

407. LaRochelle, W. J., May-Siroff, M., Robbins, K. C., and Aaronson, S. A.: A novel mechanism regulating growth factor association with the cell surface: identification of a PDGF retention domain. Genes Dev. 5: pp. 1191-1199, 1991.

408. Miki, T., Fleming, T. P., Crescenzi, M., Molloy, C. J., Blam, S. B., Reynolds, S. H., and Aaronson, S. A.: Development of a highly efficient expression cDNA cloning system: Application to oncogene isolation. Proc. Natl. Acad. Sci. USA 88: pp. 5167-5171, 1991.

409. Yu, J-C., Heidaran, M. A., Pierce, J. H., Gutkind, J. S., Lombardi, D., Ruggiero, M., and Aaronson, S. A.: Tyrosine mutations within the ⊛ platelet-derived growth factor receptor kinase insert domain abrogate receptor-associated phosphatidylinositol-3 kinase activity without affecting mitogenic or chemotactic signal transduction. Mol. Cell. Biol. 11: pp. 3780-3785, 1991.

410. Takahashi, R., Hashimoto, T., Xu, H-J., Hu, S-X., Matsui, T., Bigo-Marshall, H., Aaronson, S. A., and Benedict, W. F.: The retinoblastoma gene functions as a growth and tumor suppressor in human bladder carcinoma cells. Proc. Natl. Acad. Sci. USA. 88: pp. 5257-5261, 1991.

411. Kraus, M. H. and Aaronson, S. A.: Detection and isolation of novel protein-tyrosine kinase genes employing reduced stringency hybridization. *In* Hunter, T. and Sefton, B. M. (Eds.). Methods in Enzymology "Protein Phosphorylation." San Diego, Academic Press, Inc., 1991, pp. 546-556.

412. Pierce, J. H., Arnstein, P., DiMarco, E., Artrip, J., Kraus, M. H., Lonardo, F., Di Fiore, P. P., and Aaronson, S. A.: Oncogenic potential of *erb*B-2 in human mammary epithelial cells. Oncogene 6: pp.1189-1194, 1991.

413. Heidaran, M.A., Pierce, J.H., Yu, J-C., Lombardi, D., Artrip J.E., Fleming, T.P., Thomason, A., and Aaronson, S.A.: Role of ⊛⊛ receptor heterodimer formation in ⊛⊛platelet-derived growth factor (PDGF) receptor activation by PDGF-AB. J. Biol. Chem. 266: pp. 20232-20237, 1991.

414. Aaronson, S.A.: Growth factors and cancer. Science 254: pp. 1146-1153, 1991.

415. Chan, A., Rubin, J., Bottaro, D., Hirschfield, D., Chedid, M. and Aaronson, S. A.: Identification of a competitive HGF antagonist encoded by an alternative transcript. Science 254: pp. 1382-1385, 1991.

416. Tronick, S. R. and Aaronson, S. A.: Oncogenes. *In* Cossman, J. (Ed.). Mol. Genetics in Cancer Diagnosis. Amsterdam, Elsevier Science Publishers BV, 1991, pp. 29-41.

417. Aaronson, S. A. and Tronick, S. R.: Constitutive activation of growth factor signalling pathways in cancer cells. *In* Broder, S. (Ed.): Molecular Foundations of Oncology. Hanover, Sheridan Press, 1991, pp. 41-53.

418. Hart, M.J., Eva, A., Evans, T., Aaronson, S.A., and Cerione, R.A.: Catalysis of guanine nucleotide exchange on the CDC42HS protein by the *dbl* oncogene product. Nature. 354: pp. 311-314, 1991.

419. Di Fiore, P. P., Segatto, O., and Aaronson, S. A.: Cloning, expression and biological effects of the *erb*B2/*neu* gene in mammalian cells. Methods in Enzymology 198: pp. 272-277, 1991.

420. Aaronson, S. A., Bottaro, D. P., Miki, T., Ron, D., Finch, P. W., Fleming, T. P., Ahn, J., Taylor, W. G., and Rubin, J. S.: Keratinocyte growth factor, a fibroblast growth factor family member with unusual target cell specificity. Ann. NY Acad. Sci. 638: pp. 62-77, 1991.

421. Miki, T., Fleming, T.P., and Aaronson, Stuart A.: Expression cDNA cloning of growth control genes. *In* Verna, R. and Nishizuka, Y. (Eds.); Biotechnology of Cell Regulation. New York, Raven Press, 1992, pp. 193-203.

422. Miki, T., Bottaro, D.P., Fleming, T.P., Smith, C., Burgess, W.H., Chan, A. M.-L., and Aaronson, S.A.: Determination of ligand-binding specificity by alternative splicing:Two

distinct growth factor receptors encoded by a single gene. Proc. Natl. Acad. Sci. USA 89: pp. 246-250, 1992.

423. Heidaran, M.A., Yu, J.-C., Jensen, R.E., Pierce, J.H. and Aaronson, S.A.: A deletion in the extracellular domain of the ⊛⊛platelet-derived growth factor (PDGF) receptor differentially impairs PDGF-AA and PDGF-BB binding affinities. J. Biol. Chem. 267: pp. 2884-2887, 1992.

424. Miki, T., Bottaro, D.P., Fleming, T.P., Smith, C.L., Rubin, J.S., Chan, A. M.-L., and Aaronson, S.A. KGF receptor: transforming potential on fibroblasts and epithelial cell-specific expression by alternative splicing. *In* Papa, S., Azzi, A. and Trager, J.M. (Eds.): Molecular and Cell Biology Updates. Basel, Switzerland, Birkhauser Verlag, 1992, pp. 289-300.

425. Fleming, T. P., Matsui, T., Heidaran, M.A., Molloy, C. J., Artrip, J., and Aaronson, S. A.: Demonstration of an activated platelet-derived growth factor autocrine pathway and its role in human tumor cell proliferation *in vitro*. Oncogene 7: pp. 1355-1359, 1992.

426. Fleming, T. P., Saxena, A., Clark, W. C., Robertson, J. T., Oldfield, E. H., Aaronson, S. A. and Ali, I. U.: Amplification and/or overexpression of platelet-derived growth factor receptors and epidermal growth factor receptor in human glial tumors. Cancer Research 52: pp. 4550-4553, 1992.

427. Molloy, C. J., Fleming, T. P., Bottaro, D. P., Cuadrado, A., and Aaronson, S. A.: Platelet-derived growth factor stimulation of GTPase-activating protein tyrosine phosphorylation in control and c-H^{ras}-expressing NIH/3T3 cells correlates with p21^{ras} activation. Mol. Cell. Biol. 12: pp. 3903-3909, 1992.

428. LaRochelle, W. J., Pierce, J. H., May-Siroff, M., Giese, N., and Aaronson, S. A.: Five PDGF B amino acid substitutions convert PDGF A to a PDGF B-like transforming molecule. J. Biol. Chem. 267: pp. 17074-17077, 1992.

429. Kelley, M. J., Pech, M., Seuanez, H. N., Rubin, J. S., O'Brien, S. J., and Aaronson, S. A.: Emergence of the keratinocyte growth factor multigene family during the great ape radiation. Proc. Natl. Acad. Sci. USA. 89: pp. 9287-9291, 1992.

430. Fleming, T. P., Matsui, T., and Aaronson, S. A.: Platelet-derived growth factor (PDGF) receptor activation in cell transformation and human malignancy. Exp. Gerontol. 27: pp. 523-527, 1992.

431. Rong, S., Bodescot, M., Blair, D., Dunn, J., Nakamura, T., Mizuno, K., Park, M., Chan, A., Aaronson, S., and Vande Woude, G. F.: Tumorigenicity of the *met* protooncogene and the gene hepatocyte growth factor. Mol. Cell. Biol. 12: pp. 5152-5158, 1992.

432. Ishibashi, T., Bottaro, D. P., Chan, A., Miki, T., and Aaronson, S.A.: Expression cloning of a novel human dual-specificity phosphatase. Proc. Natl. Acad. Sci. USA 89: pp. 12170-12174, 1992.

433. LaRochelle, W.J., Fleming, T., and Aaronson, S.A.: PDGF in cell transformation. *In* Westermark, B. and Sorg, C. (Eds.): Biology of Platelet-Derived Growth Factor. Basel, Karger Medical and Scientific Publishers, 1993, pp. 129-145.

434. Chan, A., Rubin, J., Bottaro, D., Hirschfield, D., Chedid, M., and Aaronson, S. A.: Isoforms of human HGF and their biological activities. *In* Goldberg, I. D. and Rosen, E. M. (Eds.): Hepatocyte Growth Factor-Scatter Factor and the c-*MET* Receptor. Birkhauser Verlag, 1993, pp. 67-79.

435. Chan, A. M.-L., Fleming, T. P., McGovern E. S., Chedid, M., Miki, T., and Aaronson, S. A.: Expression cDNA cloning of a transforming gene encoding the wild-type G$_{\alpha\alpha}$ gene product. Mol. Cell. Biol. 13: pp. 762-768, 1993.

436. Ron, D., Bottaro, D. P., Finch, P. W., Morris, D., Rubin, J. S., and Aaronson, S.A.: Expression of biologically active recombinant keratinocyte growth factor: structure/function analysis of amino-terminal truncation mutants. J. Biol. Chem. 268: pp. 2984-2988, 1993.

437. Aaronson, S. A. and Tronick, S. R.: Growth factors. *In* Holland, J. F., Frei, III, E., Bast, Jr., R. C., Kufe, D. W., Morton, D. L. and Weichselbaum, R. R. (Eds.): Cancer Medicine, 3rd Ed. Philadelphia, Lea & Febiger, 1993, pp. 33-48.

438. Kraus, M. H., Fedi, P., Starks, V., Muraro, R., and Aaronson, S. A.: Demonstration of ligand-dependent signaling by the *erb*B-3 tyrosine kinase and its constitutive activation in human breast tumor cells. Proc. Natl. Acad. Sci. USA 90: pp. 2900-2904, 1993.

439. Chan, A. M.-L., Chedid, M., McGovern, E. S., Popescu, N. C., Miki, T., and Aaronson, S. A.: Expression cDNA cloning of a serine kinase transforming gene. Oncogene 8: pp. 1329-33, 1993.

440. Wang, L.-M., Keegan, A. D., Li, W., Lienhard, G. E., Pacini, S., Gutkind, J. S, Myers, Jr., M. G., Sun, X-J., White, M. F., Aaronson, S. A., Paul, W. E., and Pierce, J. H.: Common elements in IL-4 and insulin signaling pathways in factor-dependent hematopoietic cells. Proc. Natl. Acad. Sci. USA 90: pp. 4032-4036, 1993.

441. Bottaro, D. P., Fortney, E., Rubin, J. S., and Aaronson, S. A.: A keratinocyte growth factor receptor-derived peptide antagonist identifies part of the ligand binding site. J. Biol. Chem. 268: pp. 9180-9183, 1993.

442. Miki, T. and Aaronson, S. A.: cDNA cloning method using stable expression in mammalian cells. *In* Adolph, K.W. (Ed.): Methods in Molecular Genetics, Vol. 1. Orlando, FL, Academic Press, Inc., 1993, pp. 3-22.

443. Heidaran, M. A., Beeler, J. F., Yu, J.-C., Ishibashi, T., LaRochelle, W. J., Pierce, J. H., and Aaronson, S.A.: Differences in substrate specificities of ⊚ and ⊚ platelet-derived growth factor (PDGF) receptors. J. Biol. Chem. 268: pp. 9287-9295, 1993.

444. LaRochelle, W. J., Jensen, R. A., Heidaran, M. A., May-Siroff, M., Wang, L.-M., Aaronson, S. A., and Pierce, J. H.: Inhibition of platelet-derived growth factor autocrine growth stimulation by a monoclonal antibody to the human ⊚ platelet-derived growth factor receptor. Cell Growth Differ. 4: pp. 547-553, 1993.

445. Cuadrado, A., Bruder, J. T., Heidaran, M. A., App, H., Rapp, U. R., and Aaronson, S. A.: H-*ras* and *raf*-1 cooperate in transformation of NIH/3T3 fibroblasts. Oncogene 8: pp. 2443-2448, 1993.

446. Aaronson, S. A.: Influences of growth factors and their signaling pathways in malignancy. The Harvey Lectures, Series 87. New York, NY, Wiley-Liss, 1993, pp. 17-34.

447. Chan, M.-L. and Aaronson, S. A.: An efficient expression cDNA cloning approach to screen human tumors for oncogenes. *In* Cittadini, A. (Ed.): Molecular Oncology and Clinical Applications. Basel, Switzerland, Birkhauser Verlag, 1993, pp. 111-122.

448. Panos, R. J., Rubin, J. S., Aaronson, S. A., and Mason, R. J.: Keratinocyte growth factor and hepatocyte growth factor/scatter factor are heparin-binding growth factors for alveolar type II cells in fibroblast conditioned medium. J. Clin. Invest. 92: 969-977, 1993

449. Staiano-Coico, L., Krueger, J. G., Rubin, J. S., D'limi, S., Vallat, V. P., Valentino, L., Fahey III, T., Hawes, A., Kingston, G., Madden, M. R., Mathwich, M., Gottlieb, A., and Aaronson, S.A.: Human keratinocyte growth factor effects in a porcine model of epidermal wound healing. J. Exp. Med. 178: pp. 865-878, 1993.

450. Suzuki, M., Itoh, T., Osada, H., Rubin, J. S., Aaronson, S. A., Suzuki, T., Koga, N., Saito, T., and Mitsui, Y.: Spleen-derived growth factor, SDGF-3, is identified as keratinocyte growth factor (KGF). FEBS Lett. 328: pp. 17-20, 1993.

451. May, M., Aaronson, S. A., and LaRochelle, W. J.: Platelet-derived growth factor AB heterodimer interchain interactions influence secretion as well as receptor binding and activation. Biochemistry 32: pp. 11734-11740, 1993.

452. Wang, L-M., Myers Jr., M. G., Sun, X-J., Aaronson, S. A., White, M., and Pierce, J. H.: IRS-1: Essential for insulin- and IL-1-stimulated mitogenesis in hematopoietic cells. Science. 261: pp. 1591-1594, 1993.

453. Miki, T., and Aaronson, S. A.: Use of⊚⊚-plasmid composite vectors for expression cDNA cloning. *In* Hardy, K. G. (Ed.): Plasmids: A Practical Approach. Geneva, Switzerland, Oxford University Press, 1993, pp. 177-196.

454. Aaronson, S. A., Miki, T., Meyers, K., and Chan, A.: Growth factors and malignant transformation. *In* Zappia, V., Salvatore, M. and Della Ragione, F. (Eds.): Advances in Nutrition and Cancer. New York, Plenum Press, 1993, pp. 7-22.

455. Hart, M. J., Eva, A., Zangrilli, D., Aaronson, S. A., Evans, T., Cerione, R. A., and Zheng, Y.: Cellular transformation and guanine nucleotide exchange activity are catalyzed by a common domain on the *dbl* oncogene product. J. Biol. Chem. 269: pp. 62-65, 1994.

456. Beeler, J.F., LaRochelle, W.J., Chedid, M., Tronick, S.R. and Aaronson, S.A.: Prokaryotic expression cloning of a novel human tyrosine kinase. Mol. Cell. Biol. 14: pp. 982-988, 1994.

457. Rubin, J.S., Bottaro, D.P., and Aaronson, S.A.: Hepatocyte growth factor/scatter factor and its receptor, the *c-met* proto-oncogene product. Biochim. Biophys. Acta. 1155: pp. 357-371, 1994.

458. Cheon, H-G., LaRochelle, W. J., Bottaro, D. P., Burgess, W. H., and Aaronson, S.A.: High affinity binding sites for related fibroblast growth factor ligands reside within different receptor immunoglobulin-like domains. Proc. Natl. Acad. Sci. USA. 91: pp. 989-993, 1994.

459. Strain, A. J., McGuiness, G., Rubin, J. S., and Aaronson, S. A.: Keratinocyte growth factor and fibroblast growth factor action on DNA synthesis in rat and human hepatocytes: modulation by heparin. Exp. Cell. Res. 210: pp. 253-259, 1994.

460. Alarid, E. T., Rubin, J. S., Young, P., Chedid, M., Ron, D., Aaronson, S. A., and Cunha, G. R.: Keratinocyte growth factor functions in epithelial induction during seminal vesicle development. Proc. Natl. Acad. Sci. USA. 91: pp. 1074-1078, 1994.

461. Chedid, M., Rubin, J. S., Csaky, K. G., and Aaronson, S. A.: Regulation of keratinocyte growth factor gene expression by interleukin 1. J. Biol. Chem. 269: pp. 10753-10757, 1994.

462. Koji, T., Chedid, M., Rubin, J.S., Slayden, Ov D., Csaky, K.G., Aaronson, S.A., and Brenner, R.M.: Progesterone dependent expression of keratinocyte growth factor (KGF) mRNA in stromal cells of the primate endometrium: KGF as a progestomedin. J. Cell Biol. 125: pp. 393-401, 1994.

463. Chan, A.M.L., Miki, T., Meyers, K.A., and Aaronson, S.A.: A human oncogene of the *ras* superfamily unmasked by expression cDNA Cloning. Proc. Natl. Acad. Sci. USA 91 (16): pp. 7558-7562, 1994.

464. Kruh, G.D., Chan, A.M., Meyers, K.A., Gaughan, K., Miki, T., and Aaronson, S. A.: Expression complementary cDNA Library Transfer Establishes *mrp* as a Multidrug Resistance Gene. Cancer Research 54 (7): pp. 1649-1652, 1994.

465. Saez, R., Chan, A.M.-L., Miki, T., and Aaronson, S.A.: Oncogenic activation of human R-*ras* by point mutations analogous to those of prototype H-*ras* oncogenes. Oncogene 9: pp. 2977-2982, 1994.

466. Cunha, G.R., Foster, B.A., Donjacour, A.A., Rubin, J.S., Sugimura, Y., Finch, P.W., Brody, J.R., and Aaronson, S.A.: Keratinocyte growth factor: a mediator of mesenchymal-epithelial interactions in the development of androgen target organs. *In* Motta, M., and Serio, L. (Eds.): Sex hormones and antihormones in endocrine dependent pathology: Basic and clinical aspects. New York, Elsevier Science B.V., 1994, pp. 45-57.

467. Ishibashi, T., Bottaro, D.P., Michieli, P., Kelley, C.A., and Aaronson, S.A.: A novel dual-specificity phosphatase induced by serum stimulation and heat shock. J. Biol. Chem. 269: 47, pp. 29897-29902, 1994.

468. Goldstein, D.J., Li, W., Wang, L.-M., Heidaran, M.A., Shinn, R., Aaronson, S.A., Schlegel, R., and Pierce, J.H.: The BPV-1 E5 transforming protein specifically binds and activates the β-type receptor for the PDGF but not other related tyrosine kinase-containing receptor to induce cellular transformation. J. Virol. 68 (7): pp. 4432-4441, 1994.

469. Bottaro, D. P., Chan, A. M.-L., Rubin, J. S., Gak, E., Fortney, E., Schindler, J., Chedid, M. and Aaronson, S. A.: The HGF-c-*met* signaling pathway. In Kitagawa, Y., & Tartakoff, A. M. (Ed.): Advance in Biochemistry and Biology of Cells. Vol. 3, pp 1-14, JAI Press, Inc., Greenwich, CT., 1994

470. Tronick, S.R. and Aaronson, S.A.: Growth factors and signal transduction. In Mendelsohn, J., Howley, P., Liotta, L. and Israel, M. (Eds.): The Molecular Basis of Cancer. Philadelphia, W. B. Saunders Co., 1995, pp. 117-140.

471. La Rochelle, W.J., Dirsch, O.R., Cheon H.G., May, M., Marchese, C., Pierce, J.H. and Aaronson, S.A.: Specific receptor detection by a functional KGF-immunoglobulin chimera. Journal of Cell Biology 129 (2): pp. 357-366, 1995.

472. Lorenzi, M.V., Long, J.E., Miki, T. and Aaronson, S.A.: Expression cloning, developmental expression and chromosomal localization of fibroblast growth factor-8. Oncogene 10: pp. 2051-2055, 1995.

473. Aaronson, S.A.: Lung Cancer: Emerging therapeutic approaches based on molecular genetics. The Mount Sinai Journal of Medicine 62 (3): pp. 204-205, 1995.

474. Alimandi, M., Romano, A., Curia, M.C., Muraro, R., Fedi, P., Aaronson, S.A., Di Fiore, P.P. and Kraus, M.H.: Cooperative signaling of *erb*B-3 and *erb*B-2 in neoplastic transformation and human mammary tumors. Oncogene 10: pp. 1813-1821, 1995.

475. Aroca, P., Bottaro, D.P., Ishibashi, T., Aaronson, S.A. and Santos, E.: Human dual specificity phosphatase VHR activates MPF and triggers meiotic maturation in *Xenopus* oocytes. J. Biol. Chem. 270 (23): pp. 14229-14234, 1995.

476. Miki, T. and Aaronson, S.A.: Isolation of oncogenes by expression cDNA cloning. *In* Peter K. Vogt and Inder M. Verma, (Eds.): Methods in Enzymology: Oncogene Techniques. San Diego, California, Academic Press, pp. 196-206, 1995.

477. Huang, Y., Saez, R., Chao, L., Santos, E., Aaronson, S.A. and Chan, A.M.-L.: A novel insertional mutation in the *TC21* gene activates its transforming activity in a human leiomyosarcoma cell line. Oncogene 11: pp. 1255-1260, 1995.

478. Cunha, G.R., Sugimura, Y., Foster, B., Rubin, J.S. and Aaronson, S.A.: The role of growth factors in the development and growth of the prostate and seminal vesicle. Biomed. Pharmacotherapy 48: S9-S19, 1995.

479. Marchese, C., Chedid, M., Dirsch, O.R., Csaky, K.G., Santanelli, F., Latini, C., LaRochelle, W.J., Torrisi, M.R. and Aaronson, S.A.: Modulation of keratinocyte growth factor and its receptor in reepithelializing human skin. J. Exp. Med. 182: pp. 1369-1376, 1995.

480. Breuninger, L.M., Paul, S., Gaughan, K., Miki, T., Chan, A., Aaronson, S.A. and Kruh, G.D.: Expression of MRP in NIH/3T3 cells confers multidrug resistance associated with increased drug efflux and altered intracellular drug distribution. Cancer Research 55 (22): pp. 5342-5347, 1995.

481. Cioce, V., Csaky, K.G., Chan, A., Bottaro, D.P., Taylor, W.G., Jensen, R., Aaronson, S.A. and Rubin, J.S.: Hepatocyte growth factor (HGF)/NK1 is a naturally occurring HGF/scatter factor variant with partial agonist/antagonist activity. The Journal of Biological Chemistry 271 (22): pp. 13110-13115, 1996.

482. Tachibana, M., Takeda, K., Nobukuni, Y., Urabe, K., Meyers, K., Aaronson, S. A., and Miki, T.: Ectopic expression of *MITF*, a gene for Waardenberg syndrome type 2, converts fibroblasts to cells with melanocyte characteristics. Nature Genetics 14: pp. 50-54, 1996.

483. Sugimura, Y., Foster, B.A., Hom, Y.K., Lipschutz, J.H., Rubin, J.S., Finch, P.W., Aaronson, S.A., Hayashi, N., Kawamura, J., and Cunha, G.R.: Keratinocyte growth factor (KGF) can replace testosterone in the ductal branching morphogenesis of the rat ventral prostate. Int. J. Dev. Biol. 40: pp. 1-11, 1996.

484. Fedi, P., Tronick, S. and Aaronson, S.A.: Growth factors. *In* Holland, J. F., Frei, Ill, E., Bast, Jr., R. C., Kufe, D. W., Morton, D. L. and Weichselbaum, R. R. (Eds.): Cancer Medicine 4th Ed. Baltimore, Williams & Wilkins, 1996.

485. da Medina, S.G.D., Chopin, D., Marjon, A.E., Delouvee, A., LaRochelle, W.J., Hoznek, A., Abbou, C., and Aaronson, S.A., Thiery, J.P., Radvanyi, F.: Decreased expression of

keratinocyte growth factor receptor in a subset of human transitional cell bladder carcinomas. Oncogene 14: pp. 1-8, 1997.

486. Takayama, H., LaRochelle, W.J., Sharp, R., Otsuka, T, Kriebel, P., Anver, M., Aaronson, S.A., and Merlino, G.: Diverse tumorigenesis associated with aberrant development in mice overexpressing hepatocyte growth factor/scatter factor. Proc. Natl. Acad. Sci. USA 94: pp. 701-706, 1997.

487. Shin, D-Y., Ishibashi, T., Choi, T.S., Chung, E., Chung, I.Y., Aaronson, S.A., and Bottaro, D.P.: A novel human ERK phosphatase regulates H-*ras* and *v-raf* signal transduction. Oncogene 14: pp. 2633-2639, 1997.

488. Kirschenbaum, A., Wang, J-P., Ren, M., Schiff, J.D., Aaronson, S.A., Droller, M.J., Ferrara, N., Holland, J.F., and Levine, A.C.: Inhibition of vascular endothelial cell growth factor suppresses the *in vivo* growth of tumors. Urologic Oncology 3(1): pp. 3-10, 1997.

489. Finch, P.W., He, X., Kelley, M.J., Uren, A., Schaudies, P., Popescu, N.C., Rudikoff, S., Aaronson, S.A., Varmus, H.E., and Rubin, J.S.: Purification and Molecular Cloning of a Secreted, Frizzled-Related Antagonist of Wnt Action. Proc. Natl. Acad. Sci. USA 94: pp. 6770-6775, 1997.

490. Sugrue, M., Shin, D.Y., Lee, S.W., and Aaronson, S.A.: Wild-type p53 triggers a rapid senescence program in human tumor cells lacking functional p53. Proc. Natl. Acad. Sci. USA 94: pp. 9648-9653, 1997.

491. Zimonjic, D.B., Kelley, M.J., Rubin, J.S., Aaronson, S.A., and Popescu, N.C.: FISH anaysis of keratinocyte growth factor (KGF) gene amplification and dispersion in evolution of great apes and humans. Proc. Natl. Acad. Sci. USA 94: pp. 11461-11465, 1997.

492. Ouchi, T., Monteiro, A.N.A., August, A., Aaronson, S.A., Hanafusa, H.: BRCA1 regulates p53-dependent gene expression. Proc. Natl. Acad. Sci. USA 95: pp. 2302-2306, 1998.

493. Montesano, R., Soriano, J.V., Malinda, K.M., Ponce, M.L., Bafico, A., Kleinman, H.K., Bottaro, D.P., Aaronson, S.A.: Differential Effects of Hepatocyte Growth Factor Isoforms on Epithelial and Endothelial Tubulogenesis. Cell Growth and Differentiation 9: pp. 355-365, 1998.

494. Igarashi, M., Finch, P.W., and Aaronson, S.A.: Characterization of recombinant human FGF-10 reveals functional similarities with keratinocyte growth factor (FGF-7). J. Biol. Chem. 273(18): pp. 13230-13235, 1998.

495. Bafico, A., Gazit, A., Wu-Morgan, S.S., Yaniv, A., and Aaronson, S.A.: Characterization of Wnt-1 and Wnt-2 induced growth alterations and signalling pathways in NIH3T3 fibroblasts. Oncogene 15: pp. 2819-2825, 1998.

496. Levin, A.C., Liu, X-H., Ren, M., Wang, J-P., Schiff, J.D., Diamond, E.J., Finch, P.W., Aaronson, S.A., Kirschenbaum, K.: Adrogenic effects on prostate specific antigen secretion by human fetal prostate are mediated by keratinocyte growth factor. Molec. Urol. 2(2): pp. 65-71, 1998.

497. Kurdistani, S.K., Arizti, P., Sugrue, M.M., Aaronson, S.A., and Lee, S.W.: Inhibition of tumor cell growth by *RTP/rit42* and its responsiveness to p53 and DNA damage. Cancer Research 58: pp. 1-6, 1998.

498. Marmorstein, L.Y., Ouchi, T., and Aaronson, S.A.: The BRCA2 gene product functionally interacts with p53 and RAD51. Proc. Natl. Acad. Sci. USA 95: pp. 13869-13874, 1998.

499. Levin, A.C., Liu, X-H., Greenberg, P.D., Eliashili, M., Schiff, J.D., Aaronson, S.A., Holland, J.F., and Kirschenbaum, A.: Androgens induce the expression of vascular endothelial growth factor in human fetal prostatic fibroblasts. Endocrinology 139(11): pp. 4672-4678, 1998.

500. Aaronson, S.A.: Robert Huebner Obituary. Cancer Research 58, 1998.

501. Fang, L., Igarashi, M., Leung, J., Sugrue, M.M., Lee, S.W., and Aaronson, S.A.: $p21^{Waf1/Cip1/Sdi1}$ induces permanent growth arrest with markers of replicative senescence in human tumor cells lacking functional p53. Oncogene 18: pp. 2789-2797, 1999.

502. Bafico, A., Gazit, A., Pramila, T., Finch, P.W., Yaniv, A., and Aaronson, S.A.: Interaction of FRP with Wnt ligands and the frizzled receptor suggests alternative mechanisms for FRP inhibition of Wnt signalling. J. Biol. Chem. 274: pp. 16180-16187, 1999.

503. Fedi, P., and Aaronson, S.A.: Signal transduction through tyrosine kinase growth factor receptors. *In* "Signal Transduction, Cell Cycle and their Inhibitors" Edited by Gutkind et al., Humana Press, 1999, pp. 27-38.

504. Reimer, C.L., Borras, A.M., Kurdistani, S.K., Garreau, J.R., Chung, M., Aaronson, S.A., and Lee, S.W.: Altered regulation of cyclin G in human breast cancer and its specific localization at replication foci in response to DNA damage in p53+/+ cells. J. Biol. Chem. 274 (16): pp. 11022-11029, 1999.

505. Fedi, P., Bafico, A., Soria, A.N., Burgess, W.H., Miki, T., Kraus, M.H., and Aaronson, S.A.: Isolation and biochemical characterization of the human Dkk-1 homologue, a novel inhibitor of mammalian Wnt signaling. J. Biol. Chem. 274: pp. 19465-19471, 1999.

506. Gazit, A., Yaniv, A., Bafico, A., Pramila, T., Igarashi, M., Kitajewsky, J., and Aaronson, S.A.: Human frizzled 1 interacts with transforming Wnts to transduce a TCF dependent transcriptional response. Oncogene 18: pp. 5959-5966, 1999.

507. Fang, L. Lee, S.W., and Aaronson, S.A., Comparative analysis of p73 and p53 regulation and effector functions. J. Cell Biol. 147: pp. 824-830, 1999.

508. Blaho, J.A., Aaronson, S.A.: Convicting of a human tumor virus: Guilt by association? Proc. Natl. Acad. Sci. USA 96: pp. 7619-7621, 1999.

509. Lee, S.W., Reimer, C., Fang, L., Iruela-Arispe, M.L., and Aaronson, S. A.: Overexpression of kinase-associated phosphatase (KAP) in breast and prostate cancer and inhibition of the transformed phenotype by antisense KAP expression. Mol. Cell. Biol. 20: pp. 1723-1732, 2000.

510. Hazan R, Phillips, Qia, Norton, L, Ossowski, L., Aaronson S. A.: Exogenous expression of N-cadherin in Breast Cancer cells induces cell migration, invasion and metatasis. J. Cell. Biol. 148: pp. 779-790, 2000.

511. Lee, S.W., Fang, L., Igarashi, M., Ouchi, T., Lu, K.P., and Aaronson, S.A.: Sustained activation of Ras/RaF/MAP kinase cascade by the tumor suppressor p53. Proc. Natl. Acad. Sci. USA 97 (15): pp. 8302-8305, 2000.

512. Kimmelman, A., Bafico, A., and Aaronson, S.A.: Oncogenes and Signal Transduction: Molecular Basis of Cancer pp. 396-444, 2000.

513. Fedi, P., Kimmelman, A., and Aaronson, S.: Growth Factor Signal Transduction in Cancer. *In* Holland, J. F., Frei, Ill, E., Bast, Jr., R. C., Kufe, D. W., Pollock, R. E. and Weichselbaum, R. R. (Eds.): Cancer Medicine 5th Ed. Hamilton, B.C. Decker pp. 33-55, 2000.

514. Ouchi, T., Lee, S.W., Aaronson, S.A., and Horvath, C.: Collaboration of STAT1 and BRCA1 in differential regulation of IFNγ target genes, Proc. Natl. Acad. Sci. USA 97: 5208-5213, 2000.

515. Ariziti, P., Fang, L., Park, I., Yin, Y., Solomon, E., Ouchi, T., Aaronson, S., Lee, S.W., Tumor suppressor p53 is required to modulate BRCA1 expression, Mol. Cell. Biol. 20: pp. 7540-7459, 2000.

516. Fang, L., Li, G., Liu, G., Lee, S.W., Aaronson, S.A., p53 induction of heparin-binding EGF-like growth factor counteracts p53 growth suppression through activation of MAPK and PI3K/Akt signaling cascades. EMBO J. 20 (8): pp. 1931-1939, 2001.

517. Bafico, A., Liu, G., Yaniv, A., Gazit, A., Aaronson, S. A., Novel mechanism of Wnt signaling inhibition mediated by Dickkopf-1 interaction with LRP6/Arrow, Nature Cell Biology 3 (7): pp. 683-686, 2001.

518. Bafico, A. and Aaronson, S. A.,: "Autocrine/Paracrine", Edited by Brenner, et al., Encyclopedia of Genetics pp. 126-128, 2001.

519. Macip, S., Igarashi, M., Fang, L., Chen, A., Pan, Z-Q., Lee, S.W., Aaronson, S.A., Inhibition of p21-mediated ROS accumulation can rescue p21-induced senescence, EMBO J. 21 (9): pp. 2180-2188, 2002.

520. Spiegelmann, V. S., Tang, W., Chan, A. M., Igarashi, M., Aaronson, S.A., Sassoon, D. A., Kotah, M., Slaga, T. J., Fuchs, S. Y. Induction of homologue of slimb ubiquitin ligase receptor by mitogen signaling, J. Biol. Chem. 277 (39): pp. 36624-36630, 2002.

521. Y. Ramanathan, Haibo Zhang, Virginie Aris, Patricia Soteropoulos, Stuart A. Aaronson, and Peter P. Tolias. Functional cloning, sorting and expression profiling of nucleic-acid binding proteins. Genome Research 12: pp. 1175-1189, 2002.

522. Jeong A. Han, Jong-Il Kim, Pat, P. Ongusaha, Daniel H. Hwang, Leslie R. Ballou, Alka Mahale, Stuart A. Aaronson, and Sam W. Lee. p53-mediated induction of Cox-2 counteracts p53- or genotoxic stress-induced apoptosis. EMBO J. 21 (21): pp. 5635-5644, 2002.

523. Ongusaha, P.P., Kim, J., Fang, L., Wong, T. W., Yancopoulos, G.D., Aaronson, S. A., Lee, S. W. p53 induction and activation of DDR 1 kinase counteract p53-mediated apoptosis and influence p53 regulation through a positive feedback loop. EMBO J. 22 (6): pp.1289-1301, 2003.

524. Yeh, B. K., Igarashi, M., Eliseenkovo, A. V., Plotnikov, A. N., Sher, I., Ron, D., Aaronson, S. A., and Mohammadi, M. Structural basis by which alternative splicing confers specificity in fibroblast growth factor receptors. Proc. Natl. Acad. Sci. USA 100 (5): pp. 2266-2271, 2003.

525. Bafico, A. and Aaronson, S.A.: Growth Factors And Signal Transduction in Cancer. *In* Holland, J. F., Frei, Ill, E., Bast, Jr., R. C., Kufe, D. W., Pollock, R. E. and Weichselbaum, R. R. (Eds.): Cancer Medicine 6th Ed. Hamilton, B.C. Decker, Vol. 1, pp. 53-71, 2003.

526. Liu, G., Bafico, A., Harris, V.K., Aaronson, S.A. A Novel Mechanism for Wnt Activation of Canonical Signaling through the LRP6 Receptor. Mol. Cell. Biol. 23 (16): pp. 5825-5835. 2003.

527. Dong, J., Phelps, R. G., Qiao, R., Shen, Y., Benard, O., Ronai, Z., Aaronson, S.A. BRAF oncogenic mutations correlate with progression rather than initiation of human melanoma. Cancer Research, 63: pp. 3883-3885, 2003.

528. Macip, S., Igarashi, M., Berggren, P., Yu, J., Lee, S. W., Aaronson, S.A. Influence of Induced ROS in Determining p53-Mediated Cell Fate Decisions. Mol. Cell. Biol. 23 (23): pp. 8576-8585, 2003.

529. Ohtsuka, T., Ryu, H., Minamishima, Y. A., Macip, S., Sagara, J., Aaronson, S.A., Lee, S.W. ASC Functions as an Adaptor for Bax and Regulates a p53-Bax Mitochondrial Pathway of Apoptosis. Nature Cell Biology 6 (2) pp. 121-128, 2004.

William H. Bailey, Ph.D.

Principal Scientist and Director, New York Office

Professional Profile

Dr. William H. Bailey is a Principal Scientist in the Health practice and Director of Exponent's New York office. Before joining Exponent, Dr. Bailey was President of Bailey Research Associates, Inc., the oldest research and consulting firm with specialized expertise in electromagnetic fields and health. Dr. Bailey specializes in applying state-of-the-art assessment methods to environmental health and impact issues. His 30 years of training and experience include laboratory and epidemiologic research, health risk assessment, and comprehensive exposure analysis. Dr. Bailey is particularly well known for his research on potential health effects of electromagnetic fields and is active in setting IEEE standards for human exposure to electromagnetic fields. He uses advanced analytical and statistical methods in the design and analysis of both experimental studies and epidemiology and survey research studies. Such methods include Monte Carlo and other probabilistic methods to characterize uncertainties pertaining to exposure impacts and the determination of "safe" environmental exposures.

In addition to his training and experience in health risk assessment, Dr. Bailey's postgraduate training is helpful in assessing the important effects of social, economic, and community factors on health risks and vulnerability to environmental impacts in health and environmental justice research. Currently, he is directing research projects on effects of electrical charge on the deposition of aerosols in the respiratory tract and an epidemiologic evaluation of air pollution impacts on community health from mobile sources. He is a member of a working group that advises a committee of the World Health Organization on risk assessment, perception, and communication. Dr. Bailey is also a visiting scientist at the Cornell University Medical College and has lectured at Rutgers University, the University of Texas (San Antonio), and the Harvard School of Public Health. He was formerly Head of the Laboratory of Neuropharmacology and Environmental Toxicology at the New York State Institute for Basic Research, Staten Island, New York, and an Assistant Professor and NIH postdoctoral fellow in Neurochemistry at The Rockefeller University in New York.

Credentials and Professional Honors

Ph.D., Neuropsychology, City University of New York, 1975

M.B.A., University of Chicago, 1969

B.A., Dartmouth College, 1966

Sigma Xi; The Institute of Electrical and Electronics Engineers/International Committee on Electromagnetic Safety (Subcommitee 3, Safety Levels with Respect to Human Exposure to

Fields (0 to –3 kHz) and Subcommitte 4, Safety Levels with Respect to Human Exposure to Radiofrequency Fields (3 kHz to3 GHz); Elected member of the Committee on Man and Radiation (COMAR) of the IEEE Engineering in Medicine and Biology Society (1998–present); Invited Speaker, First Institute of Neurological Sciences Symposium in Neurobiology, University of Pennsylvania (1980); Invited Speaker, National Heart and Lung Institute (1977).

Publications

Bailey WH. Dealing with uncertainty in formulating occupational and public exposure limits. Health Phys 2002; 83: 402–408.

Bailey WH. Health effects relevant to the setting of EMF exposure limits. Health Phys 2002; 83:376–386.

Kavet R, Stuchly MA, Bailey WH, Bracken TD. Evaluation of biological effects, dosimetric models, and exposure assessment related to ELF electric- and magnetic-field guidelines. Applied Occupational and Environmental Hygiene; 2001; 16:1118–1138.

Bailey WH. ICNIRP recommendation for limiting public exposure to 4 Hz–1 kHz electric and magnetic fields. Health Phys 1999; 77:97–98.

Bailey WH. Principles of risk assessment with application to current EMF risk communication issues. In: EMF Risk Perception and Communication, Repacholi MH, Muc, AM (eds.), World Health Organization, Geneva, 1999.

De Santo RS, Bailey, WH. Environmental justice tools and assessment practices. Proceedings, 1999 American Public Transit Association, 1999.

Bailey WH, Su SH, Bracken TD. Probabilistic approach to ranking sources of uncertainty in ELF magnetic field exposure limits. Health Phys 1999; 77:282–290.

Bailey WH. Field parameters. Proc. EMF Engineering Review Symposium, Status and Summary of EMF Engineering Research, Bracken TD, Montgomery JH (eds.), Oak Ridge National Laboratory, Oak Ridge, TN, April 28–29, 1998.

Bailey WH. Policy implications. Proceedings, EMF Engineering Review Symposium, Status and Summary of EMF Engineering Research, Bracken TD, Montgomery JH (eds.), Oak Ridge National Laboratory, Oak Ridge, TN, April 28–29, 1998.

Bailey WH. Probabilistic approaches to deriving risk-based exposure guidelines: application to extremely low frequency magnetic fields. In: Non-Ionising Radiation, Dennis JA and Stather JW (eds.), Special Issue of Radiation Protection Dosimetry 1997; 72:327–336.

Bailey WH, Su SH, Bracken TD, Kavet R. Summary and evaluation of guidelines for occupational exposure to power frequency electric and magnetic fields. Health Phys 1997; 73:433–453.

Bracken TD, Senior RS, Rankin RF, Bailey WH, Kavet R. Magnetic field exposures in the electric utility industry relevant to occupational guideline levels. Appl Occupat Environ Hyg 1997; 12:756–768.

Blondin J-P, Nguyen D-H, Sbeghen J, Goulet D, Cardinal C, Maruvada P-S, Plante M, and Bailey WH. Human perception of electric fields and ion currents associated with high voltage DC transmission lines. Bioelectromagnetics 1996; 17:230–241.

Bailey WH, Charry JM. Acute exposure of rats to air ions: effects on the regional concentration and utilization of serotonin in brain. Bioelectromagnetics 1987; 8:173–181.

Bailey WH, Charry JM. Measurement of neurotransmitter release and utilization in selected brain regions of rats exposed to dc electric fields and atmospheric space charge. Proceedings, Twenty-Third Hanford Life Sciences Symposium, Interaction of Biological Systems with Static and ELF Electric and Magnetic Fields, 1987.

Pavildes C, Aoki C, Chen J-S, Bailey WH, Winson J. Differential glucose utilization in the parafascicular region during slow-wave sleep, the still-alert state and locomotion. Brain Res 1987; 423:399–402.

Bailey WH, Charry JM. Behavioral monitoring of rats during exposure to air ions and DC electric fields. Bioelectromagnetics 1986; 7:329–339.

Charry JM, Shapiro MH, Bailey WH, Weiss JM. Ion-exposure chambers for small animals. Bioelectromagnetics 1986; 7:1–11.

Charry JM, Bailey WH. Regional turnover of norepinephrine and dopamine in rat brain following acute exposure to air ions. Bioelectromagnetics 1985; 6:415–425.

Bracken TD, Bailey WH, Charry JM. Evaluation of the DC electrical environment in proximity to VDTs. J Environ Sci Health Part A 1985; 20:745–780.

Gross SS, Levi R, Bailey WH, Chenouda AA. Histamine modulation of cardiac sympathetic responses: a physiological role. Fed Proc 1984; 43:458.

Gross SS, Guo ZG, Levi R, Bailey WH, Chenouda AA. 1984. Release of histamine by sympathetic nerve stimulation in the guinea pig heart and modulation of adrenergic responses. Circulation Res 1984; 54:516–526.

Dahl D, Bailey WH, Winson J. Effect of norepinephrine depletion of hippocampus on neuronal transmission from perforant pathway through dentate gyrus. J Neurophysiol 1983; 49:123–135.

Guo ZG, Gross SS, Levi R, Bailey WH. Histamine: modulation of norepinephrine release from sympathetic nerves in guinea pig heart. Fed Proc 1983; 42:907.

Bailey WH. Biological effects of air ions on serotonin metabolism: fact and fancy. In: Conference on Environmental Ions and Related Biological Effects, pp. 90–120, Charry JM (ed.), American Institute of Medical Climatology, Philadelphia, PA, 1982.

Weiss JM, Goodman PA, Losito BG, Corrigan S, Charry JM, Bailey WH. Behavioral depression produced by an uncontrollable stressor: relationship to norepinephrine, dopamine, and serotonin levels in various regions of rat brain. Brain Res Rev 1981; 3:167–205.

Bailey WH. Ion-exchange chromatography of creatine kinase isoenzymes: a method with improved specificity and sensitivity. Biochem Med 1980; 24:300–313.

Bailey WH, Weiss JM. Evaluation of a 'memory deficit' in vasopressin-deficient rats. Brain Res 1979; 162:174–178.

Bailey WH, Weiss JM. Effect of ACTH 4-10 on passive avoidance of rats lacking vasopressin (Brattleboro strain). Hormones and Behavior 1978; 10:22–29.

Pohorecky LA, Newman B, Sun J, Bailey WH. Acute and chronic ethanol injection and serotonin metabolism in rat brain. J Pharmacol Exper Therap 1978; 204:424–432.

Koh SD, Vernon M, Bailey WH. Free-recall learning of word lists by prelingual deaf subjects. J Verbal Learning and Verbal Behavior 1971; 10:542–574.

Book Chapters

Bailey WH. Principles of risk assessment and their limitations. In: Risk Perception, Risk Communication and Its Application to EMF Exposure, Matthes R, Bernhardt JH, Repacholi MH (eds.), International Commission on Non-Ionizing Radiation Protection, Oberschleißheim, Germany, 1998.

Bailey, WH. Biological responses to air ions: is there a role for serotonin? In: Air Ions: Physical and Biological Aspects, pp. 151–160, Charry JM, Kavet R (eds.), CRC Press, Boca Raton, FL, 1987.

Weiss JM, Bailey WH, Goodman PA, Hoffman LJ, Ambrose MJ, Salman S, Charry JM. A model for neurochemical study of depression. In: Behavioral Models and the Analysis of Drug Action, pp. 195–223, Spiegelstein MY, Levy A (eds.), Elsevier Scientific, Amsterdam, 1982.

Bailey WH. Mnemonic significance of neurohypophyseal peptides. In: Changing Concepts of the Nervous System, pp. 787–804, Morrison AR, Strick PL (eds.), Academic Press, New York, NY, 1981.

Bailey WH, Weiss, JM. Avoidance conditioning and endocrine function in Brattleboro rats. In: Endogenous Peptides and Learning and Memory Process, pp. 371–395, Martinez JL, Jensen RA, Messing RB, Rigter H, McGaugh JL (eds.); Academic Press, New York, NY, 1981.

Weiss JM, Glazer H, Pohorecky LA, Bailey WH, Schneider L. Coping behavior and stress-induced behavioral depression: studies of the role of brain catecholamines. In: The Psychobiology of the Depressive Disorders: Implications for the Effects of Stress, pp. 125–160, Depue R (ed.), Academic Press, New York, NY, 1979.

Reports

Bailey WH. Probabilistic approach to ranking sources of uncertainty in ELF magnetic-field exposure limits. In: Evaluation of Occupational Magnetic Exposure Guidelines, Interim Report, EPRI Report TR-111501, 1998.

Bailey WH, Weil DE, Stewart JR. HVDC Power Transmission Environmental Issues Review. Oak Ridge National Laboratory, Oak Ridge, TN, 1997.

Bailey, WH. Melatonin responses to EMF. In: Proc. Health Implications of EMF Neural Effects Workshop, Report TR-104327s, Electric Power Research Institute, 1994.

Bailey, WH. Recent neurobiological and behavioral research: overview of the New York State powerlines project. In: Power-Frequency Electric and Magnetic Field Research, Electric Power Research Institute, 1989.

Bailey WH, Bissell M, Dorn CR, Hoppel WA, Sheppard AR, Stebbings, JH. Comments of the MEQB Science Advisors on Electrical Environment Outside the Right of Way of CU-TR-1, Report 5. Science Advisor Reports to the Minnesota Environmental Quality Board, 1986.

Bailey WH, Bissell M, Brambl RM, Dorn CR, Hoppel WA, Sheppard AR, Stebbings JH. A Health and Safety Evaluation of the +/- 400 KV Powerline. Science Advisor's Report to the Minnesota Environmental Quality Board, 1982.

Charry JM, Bailey WH, Weiss JM. Critical Annotated Bibliographical Review of Air Ion Effects on Biology and Behavior. Rockefeller University, New York, NY, 1982.

Bailey WH. Avoidance Behavior in Rats with Hereditary Hypothalamic Diabetes Insipidus. Dissertation, City University of New York, 1975.

Presentations

Bailey, WH, Erdreich, L, Waller, L, Mariano, K. Childhood leukemia in relation to 25-Hz and 60-Hz magnetic fields along the Washington DC—Boston rail line. Society for Epidemiologic Research, 35th Annual Meeting, Palm Desert CA, June 2002. American Journal of Epidemiology. 155:S38, 2002.

De Santo, RS, Coe, M, Bailey, WH. Environmental justice assessment and the use of GIS tools and methods. National Association of Environmental Professionals, 27th Annual Conference, Dearborn, MI, June 2002.

Bailey WH. Applications to enhance safety: research to understand and control potential risks. Human Factors and Safety Research, Volpe National Transportation Systems Center/Dutch Ministry of Transport, Cambridge, MA, November 2000.

Bailey WH. EMF health effects review. EMF Exposure Guideline Workshop, Brussels Belgium, June 2000.

Bailey WH. Dealing with uncertainty when formulating guidelines. EMF Exposure Guideline Workshop, Brussels Belgium, June 2000.

Bailey WH. Field parameters: policy implications. EMF Engineering Review Symposium, Status and Summary of EMF Engineering Research, Charleston, SC, April 1998.

Bailey WH. Principles of risk assessment: application to current issues. Symposium on EMF Risk Perception and Communication, World Health Organization, Ottawa, Canada, August 1998.

Erdreich L, Klauenberg BJ, Bailey WH, Murphy MR. Comparing radiofrequency standards around the world. Health Physics Society 43rd Annual Meeting, Minneapolis, MN, July 1998.

Bailey WH. Current guidelines for occupational exposure to power frequency magnetic fields. EPRI EMF Seminar, New Research Horizons, March 1997.

Bailey WH. Methods to assess potential health risks of cell telephone electromagnetic fields. IBC Conference—Cell Telephones: Is there a Health Risk? Washington, DC, June 1997.

Bailey WH. Principles of risk assessment and their limitations. Symposium on Risk Perception, Risk Communication and its Application to EMF Exposure, International Commission on Non-Ionizing Radiation Protection, Vienna, Austria, October 1997.

Bailey WH. Probabilistic approach for setting guidelines to limit induction effects. IEEE Standards Coordinating Committee 28: Non-Ionizing Radiation, Subcommittee 3 (0–3 kHz), June 1997.

Bracken TD, Senior RS, Rankin RF, Bailey WH, Kavet R. Relevance of occupational guidelines to utility worker magnetic-field exposures. Second World Congress for Electricity and Magnetism in Biology and Medicine, Bologna, Italy, June 1997.

Bailey WH. Epidemiology and experimental studies. American Industrial Hygiene Conference, Washington, DC, May 1996.

Bailey WH. Power frequency field exposure guidelines. IEEE Standards Coordinating Committee 28: Non-Ionizing Radiation, Subcommittee 3 (0–3 kHz), June 1996.

Weil DE, Erdreich LS, Bailey WH. Are 60-Hz magnetic fields cancer causing agents? Mechanisms and Prevention of Environmentally Caused Cancers, The Lovelace Institutes 1995 Annual Symposium, La Fonda, Santa Fe, NM, October 1995.

Bailey WH. Neurobiological research on extremely-low-frequency electric and magnetic fields: a review to guide future research. Sixteenth Annual Meeting of the Bioelectromagnetics Society, Copenhagen, Denmark, June 1994.

Blondin J-P, Nguyen D-H, Sbeghen J, Maruvada PS, Plante M, Bailey WH, Goulet D. The perception of DC electric fields and ion currents in human observers. Annual Meeting of the Canadian Psychological Association, Penticton, British Columbia, Canada, June 1994.

Erdreich LS, Bailey WH, Weil DE. Science, standards and public policy challenges for ELF fields. American Public Health Association 122nd Annual Meeting, Washington, DC, October 1994.

Bailey WH. Review of 60 Hz epidemiology studies. EMF Workshop, Canadian Radiation Protection Association, Ontario, Canada, June 1993.

Bailey WH. Biological and health research on electric and magnetic fields. American Industrial Hygiene Association, Fredrickton, New Brunswick, Canada, October 1992.

Bailey WH. Electromagnetic fields and health. Institute of Electrical and Electronics Engineers, Bethlehem, PA, January 1992.

Bailey WH, Charry JM. Particle deposition on simulated VDT operators: influence of DC electric fields. Tenth Annual Meeting of the Bioelectromagnetics Society, June 1988.

Charry JM, Bailey WH. Contribution of charge on VDTs and simulated VDT operators to DC electric fields at facial surfaces. Tenth Annual Meeting of the Bioelectromagnetics Society, June 1988.

Bailey WH, Charry, JM. Dosimetric response of rats to small air ions: importance of relative humidity. EPRI/DOE Contractors Review, November 1986.

Charry JM, Bailey WH, Bracken TD. DC electric fields, air ions and respirable particulate levels in proximity to VDTs. International Conference on VDTs and Health, Stockholm, Sweden, June 12–15 1986.

Charry JM, Bailey WH. Air ion and DC field strengths at 10^4 ions/cm^3 in the Rockefeller University Small Animal Exposure Chambers. EPRI/DOE Contractors Review, November 1985.

Charry JM, Bailey WH. DC Electrical environment in proximity to VDTs. Seventh Annual Meeting of the Bioelectromagnetics Society, June 1985.

Bailey WH, Collins RL, Lahita RG. Cerebral lateralization: association with serum antibodies to DNA in selected bred mouse lines. Society for Neuroscience, 1985.

Kavet R, Bailey WH, Charry JM. Respiratory neuroendocrine cells: a plausible site for air ion effects. Seventh Annual Meeting of The Bioelectromagnetics Society, June 1985.

Bailey WH, Charry JM. Measurement of neurotransmitter release and utilization in selected brain regions of rats exposed to DC electric fields and atmospheric space charge. Twenty-third Hanford Life Sciences Symposium, Richland, WA, October 1984.

Bailey WH, Charry JM, Weiss JM, Cardle K, Shapiro M. Regional analysis of biogenic amine turnover in rat brain after exposure to electrically charged air molecules (air ions). Society for Neuroscience, 1983.

Bailey WH. Biological effects of air ions: fact and fancy. American Institute of Medical Climatology Conference on Environmental Ions and Related Biological Effects, October 1982.

Goodman PA, Weiss JM, Hoffman LJ, Ambrose MJ, Bailey WH, Charry, JM. Reversal of behavioral depression by infusion of an A2 adrenergic agonist into the locus coeruleus. Society for Neuroscience, November 1982.

Charry JM, Bailey WH. Biochemical and behavioral effects of small air ions. Electric Power Research Institute Workshop, April 1981.

Bailey WH, Alsonso DR, Weiss JM, Chin S. Predictability: a psychologic/ behavioral variable affecting stress-induced myocardial pathology in the rat. Society for Neuroscience, November 1980.

Salman SL, Weiss JM, Bailey WH, Joh TH. Relationship between endogenous brain tyrosine hydroxylase and social behavior of rats. Society of Neuroscience, November 1980.

Bailey WH, Maclusky S. Appearance of creatine kinase isoenzymes in rat plasma following myocardial injury produced by isoproterenol. Fed Assoc Soc Exp Biol, April 1978.

Bailey WH, Maclusky S. Appearance of creatine kinase isoenzymes in rat plasma following myocardial injury by isoproterenol. Fed Proc 1978; 37:889.

Bailey WH, Weiss JM. Psychological factors in experimental heart pathology. Visiting Scholar Presentation, National Heart Lung and Blood Institute, March 1977.

Bailey WH, Weiss JM. Effect of ACTH 4-10 on passive avoidance of rats lacking vasopressin (Brattleboro strain). Eastern Psychological Association, April 1976.

Research Appointments

- Visiting Fellow, Department of Pharmacology, Cornell University Medical College, New York, NY (1986–present)
- Visiting Scientist, The Jackson Laboratory, Bar Harbor, ME (1984–1985)
- Head, Laboratory of Neuropharmacology and Environmental Toxicology, NYS Institute for Basic Research in Developmental Disabilities, Staten Island, NY (1983–1987)
- Assistant Professor, The Rockefeller University, New York, NY (1976–1983)
- Postdoctoral Fellow, Neurochemistry, The Rockefeller University, New York, NY (1974–1976)
- Dissertation Research, The Rockefeller University, New York, NY (1972–1974)
- CUNY Research Fellow, Dept. of Psychology, Queens College, City University of New York, Flushing, NY (1969–1971)
- Clinical Research Assistant, Department of Psychiatry, University of Chicago; Psychiatric Psychosomatic Inst., Michael Reese Hospital, and Illinois State Psychiatric Inst, Chicago, IL (1968–1969)

Teaching Appointments

- Lecturer, University of Texas Health Science Center, Center for Environmental Radiation Toxicology, San Antonio, TX (1998)
- Lecturer, Harvard School of Public Health, Office of Continuing Education, Boston, MA (1995, 1997)
- Lecturer, Rutgers University, Office of Continuing Education, New Brunswick, NJ (1991–1995)
- Adjunct Assistant Professor, Queens College, CUNY, Flushing, NY (1978)
- Lecturer, Queens College, CUNY, Flushing, NY (1969–1974)

Advisory Positions

- National Institute of Environmental Health Sciences, Review Committee Role of Air Pollutants in Cardiovascular Disease (2004)
- Working Group on Non-Ionizing Radiation, Static and Extremely Low-Frequency Electromagnetic Fields, International Agency for Research on Cancer (2000–2002)

- Working Group, EMF Risk Perception and Communication, World Health Organization (1998–present)
- Associate Editor, Non-Ionizing Radiation, *Health Physics* (1996–present)
- Member, International Committee on Electromagnetic Safety, Subcommittee 3 - Safety Levels with Respect to Human Exposure to Fields (0 to 3 kHz) and Subcommitee 4 - Safety Levels with Respect to Human Exposure (3kHz to 3GHz) Institute of Electrical and Electronics Engineers (IEEE) (1996–present)
- Invited participant, National Institute of Environmental Health Sciences EMF Science Review Symposium: Clinical and *In Vivo* Laboratory Findings (1998)
- Working Group, EMF Risk Perception and Communication, International Commission on Non-Ionizing Radiation Protection (1997)
- U.S. Department of Energy, RAPID EMF Engineering Review (1997)
- Oak Ridge National Laboratory (1996)
- American Arbitration Association International Center for Dispute Resolution (1995–1996)
- U.S. Department of Energy (1995)
- National Institute for Occupational Safety and Health (1994–1995)
- Federal Rail Administration (1993–1996)
- U.S. Forest Service (1993)
- New York State Department of Environmental Conservation (1993)
- National Science Foundation
- National Institutes of Health, Special Study Section—Electromagnetics (1991–1993)
- Maryland Public Service Commission and Maryland Department of Natural Resources, Scientific Advisor on health issues pertaining to HVAC Transmission Lines (1988–1989)
- Scientific advisor on biological aspects of electromagnetic fields, Electric Power Research Institute, Palo Alto, CA (1985–1989)
- U.S. Public Health Service, NIMH: Psychopharmacology and Neuropsychology Review Committee (1984)

- Consultant on biochemical analysis, Colgan Institute of Nutritional Science, Carlsbad, CA (1982–1983)
- Behavioral Medicine Abstracts, Editor, animal behavior and physiology (1981–1983)
- Consultant on biological and behavioral effects of high-voltage DC transmission lines, Vermont Department of Public Service, Montpelier, VT (1981–1982)
- Scientific advisory committee on health and safety effects of a high-voltage DC transmission line, Minnesota Environmental Quality Board, St. Paul, MN (1981–1982)
- Consultant on biochemical diagnostics, Biokinetix Corp., Stamford, CT (1978–1980)

Professional Affiliations

- The Health Physics Society (Affiliate of the International Radiation Protection Society)
- Society for Risk Analysis
- New York Academy of Sciences
- American Association for the Advancement of Science
- Air and Waste Management Association
- Society for Neuroscience/International Brain Research Organization
- Bioelectromagnetics Society
- The Institute of Electrical and Electronics Engineers/Engineering in Medicine and Biology Society

Robert E. Carberry
Project Director, Transmission Business
Northeast Utilities Service Company
Hartford, Connecticut

March, 2004

Education:

Bachelor of Science in Electric Power Engineering, June, 1972, Rensselaer Polytechnic Institute, Troy, NY

Master of Engineering in Electric Power Engineering, June 1973, Rensselaer Polytechnic Institute, NY

Management Development Program, Hartford Graduate Center, 1989

Experience:

June 1973 to March 1974 - Bechtel Associates Professional Corp., electrical design of Midland nuclear plant including load flow and voltage studies.

March 1974 to March 1975 - NUSCO, Protection Engineering Section. Performed relay settings and assisted Transmission Line Engineering.

March 1975 to March 1984 - NUSCO, Transmission Line Engineering. Standards, investigations and studies for permanent and temporary grounding, radio and audible noise, electrical/biological effects of AC fields, special insulation, thermal rating studies and research projects, high phase order, HVDC, compact line design, insulated shield wires, and lightning performance.

March 1984 to April 1985 - NUSCO, Substation Project Engineering. Project conceptual development and management plus associated studies and standards activities.

April 1985 to March 1988 - NUSCO, Substation Project Engineering Manager.

March 1988 to November 1992 - NUSCO, Manager of Substation Engineering and Design. (Principal witness at public hearings related to siting of new substations.)

December 1992 to June 1997 - NUSCO, Manager of Transmission Line and Civil Engineering.

June 1997 to October 2000 - NUSCO, Manager of T&D Asset Strategy.

October 2000 to September 2001 - NUSCo, Manager of Transmission Engineering

September 2001 to March 2003 - NUSCO, Project Manager – Bethel to Norwalk Transmission Project

March 2003 to Present - NUSCO, Project Director – Bethel to Norwalk Transmission Project

NU's EMF expert 1975- 2004 and chairman of the NU EMF Task Force established in 1990.

Other Experiences:

Adjunct Faculty Member, University of Hartford, College of Engineering, January to May, 1987. Conducted portions of course in Power Systems Analysis.

T&D Emergency plan assignment as First Deputy to the Director, Electric, a liaison position with the CT Office of Emergency Management, 1985 to 2002.

Member of Advisory Committee serving the Connecticut Interagency EMF Task Force, 1991 to present.

Professional Engineering Registration: Connecticut and Massachusetts

Industry and Professional Society Activities/Senior Member, IEEE (1983)

IEEE Power Engineering Society, Transmission and Distribution Committee memberships.

1) Corona and Field Effects (C&FE) Subcommittee, Member 1976 to present, Vice Chairman 1983 to 1985.

2) C&FE Working Groups on AC Fields and Audible Noise, 1976 to present.

3) Chairman of C&FE Working Group on Design and Environmental Considerations, 1977 to 1985.

4) Secretary and Vice Chairman of Administrative Subcommittee's Coordinating Group on Environment, Safety and Public Affairs, 1981 to 1984.

IEEE Power Engineering Society, Substations Committee memberships

1) Substation Committee, member 1987 to 1995

2) Environmental Subcommittee and Associated Working Groups, member 1985 to 1995.

3) Various Working Groups of the Distribution Substations Subcommittee and the Gas Insulated Substations Subcommittee, member 1985 to 1995.

Edison Electric Institute - Chairman of the Electric Light and Power group delegation to the American National Standards Committee C63 on Electromagnetic Compatibility. 1980 to 1985.

Electric Power Research Institute - Industry advisor on project RP1591, Assessment of AC Transmission Line Field Effects, 1982 to 1984. NU representative on Transmission Line Business Unit Council, 10/95 to 12/96.

International Electrotechnical Commission, CISPR C - Member of an advisory group assisting the Technical Advisor to the U.S. National Committee of the IEC on matters pertaining to interferences from overhead power lines, 1980 to 1988.

Edison Electric Institute - EMF Task Force, 1990 to 2003: EMF Steering Committee 1995 to 2003.

Professional Recognitions:

IEEE PES Working Group Recognition and/or Prize Paper Awards

- AC Fields Working Group (1992)
- Working Group on Design and Location of Substation for Community Acceptance (1992)
- Substation Security Working Group
- "A Survey of Methods for Calculating Transmission Line Conductor Surface Voltage Gradients," 1980
- "Corona and Field Effects of AC Overhead Transmission Lines: Information for Decision Makers," 1986

CURRICULUM VITAE

Name: Philip Cole
509 Carnoustie Drive, Box 65
Shoal Creek, Alabama 35242
Tel: 205-408-9355
Fax: 205-408-9850
E-mail: pcole@uab.edu

Academic Appointment: Professor, *Emeritus*
Department of Epidemiology
School of Public Health
Univ. of Alabama at Birmingham

Education:

Michigan State University	B.A.	1960
University of Vermont	M.D.	1965
Harvard University	M.P.H.	1967
Harvard University	Dr.P.H.	1970

Previous positions:

Professor (Chairman, 1981-94) Department of Epidemiology School of Public Health University of Alabama at Birmingham	1979-99
Senior Scientist	1979-99
Associate Director for Epidemiology Comprehensive Cancer Center University of Alabama at Birmingham	1979-93
Department of Epidemiology Harvard School of Public Health	
Assistant and Associate Professor	1969-78
Professor	1978-79
Consultant in Epidemiology and Biostatistics International Agency for Research on Cancer	1977-78
Surgical Intern, Royal Victoria Hospital Montreal	1965-66

Prepared: January 2, 2004

Certification and Professional Societies:

	Licensed, Alabama Medical Licensure Commission	1981
Licensed,	Board of Registration in Medicine, Commonwealth of Massachusetts	1966-80
	Diplomate, National Board of Medical Examiners	1966
	Certified, American Board of Preventive Medicine	1971
	Member, American Epidemiologic Society	1973-79
	Honorary Fellow, American College of Epidemiology	1997

Honors:	American Cancer Society, Faculty Research Award	1973-78
	Visiting Lecturer on Epidemiology Harvard School of Public Health	1979-99
	Gordon Richards Memorial Lecturer Ontario Cancer Treatment and Research Foundation	1979
	John Whittick Memorial Lecturer Canadian Cancer Society	1980
	Kammer Merit in Authorship Award American Occupational Medical Association	1982
	John Rankin Visiting Professor of Occupational and Preventive Medicine University of Wisconsin, Madison	1983
	Eleanor Leader Memorial Lecturer University of Toronto, Toronto	1985
	Grand Prix Lacassagne du La Ligue Nationale Francaise contre le Cancer, (with B. MacMahon, J. Brown and D. Trichopoulos)	1986
	First Annual President's Award Outstanding Teacher School of Public Health, UAB	1991
	Cutter Lecturer Harvard School of Public Health	1996

Myrick Lecturer
Injury Control Research Center, UAB 1996

Lecturer
Delta Omega Society, UAB 1997

Honors (continued):

First Recipient
Distinguished Faculty Investigator Award
School of Public Health, UAB 1998

Distinguished Academic Achievement Award
College of Medicine, University of Vermont 2000

Major Committees:

Epidemiology and Disease Control Study Section
National Institutes of Health 1973-77

Scientific Advisory Committee
Division of Cancer Cause and Prevention
National Cancer Institute 1978-80

General Motors-United Auto Workers
Occupational Health Advisory Board 1982-87

Prevention, Cancer Control (Chairman)
Steering Committee, National Planning Effort
National Cancer Institute 1984-85

Mott Prize Selection Committee
General Motors Cancer Research Foundation 1985

Board of Scientific Counselors
Division of Cancer Prevention and Control
National Cancer Institute 1986-90

Advisory Council on Epidemiology
Electric Power Research Institute 1986-90

Program Project Review Committee
National Cancer Institute of Canada 1993

Research Professor Selection Committee
American Cancer Society 1994

EPA-Dow Elanco Review Committee
Health Effects of Chlorpyrifos 1997

American Council on Science and Health
Committee on Phthalates 1999

Teaching: Harvard School of Public Health

The epidemiology of chronic diseases	1969-72
The epidemiology of neoplastic diseases	1973-77
Epidemiologic methods	1976
Principles of epidemiology	1978-79

Teaching (continued):

University of Minnesota-Graduate Summer Session

The epidemiology of cancer	1971-80
Principles of epidemiologic research	1985
Fundamentals of epidemiology	1986-91

International Agency for Research on Cancer

Cancer epidemiology	1974-80

University of Massachusetts-Graduate Summer Session

Principles of epidemiology	1981-85
Cancer epidemiology	1982

Tufts University-Graduate Summer Session

Epidemiologic bases of public health policy and law	1986,87
Principles of epidemiology	1994-96

University of Alabama at Birmingham

Epidemiology of cancer	1980
Principles of epidemiologic research	1980-95
Advanced epidemiologic methods	1981
Doctoral seminar	1981-91,99,02
Introduction to epidemiology	1996,97,00

Mediterranean School of Epidemiology and Biostatistics

National Research Council of Italy, Siracusa, Sicily
Introduction to Epidemiology 2003

Research Interests:

The moral bases for preventive interventions
Causality in epidemiology, health policy and law
Innovative approaches to smoking cessation
Occupational and chemical carcinogenesis
Health effects of electromagnetic fields

Editorships:

Associate Editor, *Cancer Research*	1982-85
Associate Editor, *American Journal of Epidemiology*	1982-88
Editorial Board, *International Journal of Breast and Mammary Pathology*	1984-90
Editorial Board, *Fundamental and Applied Toxicology*	1984-90
Editorial Board, *Breast Diseases*	1987-93
Editorial Board, *Southern Medical Journal*	1990-99
Editorial Board, *Cancer Epidemiology, Biomarkers and Prevention*	1991-98
Editorial Board, *Regulation*	1999-01

Other: Chairman of the Faculty, School of Public Health, UAB 1991-95

Publications

1. Cole P, MacMahon B, Aisenberg A: Mortality from Hodgkin's disease in the United States: Evidence for the multiple-aetiology hypothesis. *Lancet* 2:1371-1376, 1968.

2. Cole P, Gutelius J: Paraplegia resulting from the use of the subclavian artery as a shunt source during resection of the descending thoracic aorta. *Ann Surg* 169:293-294, 1969.

3. Rapoport A, Cole P, Mason J: Correlates of survival after initiation of chemotherapy in 142 cases of Hodgkin's disease. *Cancer* 24:377-381, 1969.

4. Cole P, MacMahon B: OEstrogen fractions during early reproductive life in the aetiology of breast cancer. *Lancet* 1:604-606, 1969.

5. MacMahon B, Cole P: Endocrinology and epidemiology of breast cancer. *Cancer* 24:1146-1150, 1969.

6. Cole P, Gutelius J: Neurologic complications of surgery on the descending thoracic aorta. *Can J Surg* 12:435-443, 1969.

7. Kaplan S, Cole P: Factors affecting response to postal questionnaires. *Br J Prev Soc Med* 24:245-247, 1970.

8. MacMahon B, Cole P, Lin TM, et al: Age at first birth and breast cancer risk. *Bull WHO* 43:209-221, 1970.

9. Cole P, Monson RR, Haning H, Friedell GH: Smoking and cancer of the lower urinary tract. *N Engl J Med* 284:129-134, 1971.

10. Mirra A, Cole P, MacMahon B: Breast cancer in an area of high parity: Sao Paulo, Brazil. *Cancer Res* 31:77-83, 1971. Reprinted in Portuguese in *Rev Assoc Med Bras* 18:357-364, 1972.

11. Cole P: Coffee-drinking and cancer of the lower urinary tract. *Lancet* 1:1335-1337, 1971.

12. MacMahon B, Cole P, Brown JB, et al: OEstrogen profiles of Asian and North American women. *Lancet* 2:900-902, 1971.

13. Hoover R, Cole P: Population trends in cigarette smoking and bladder cancer. *Am J Epidemiol* 94:409-418, 1971.

14. Cole P, MacMahon B: Attributable risk percent in case-control studies. *Br J Prev Soc Med* 25:242-244, 1971.

15. Allen DW, Cole P: Viruses and human cancer. *N Engl J Med* 286:70-82, 1972. Reprinted in *Ca - Cancer Journal for Clinicians* 23:127-136, 1973 and in *Diagnostic* 4:189-194, 1973.

16. Trichopoulos D, MacMahon B, Cole P: Menopause and breast cancer risk. *J Natl Cancer Inst* 48:605-613, 1972.

17. Cole P, Hoover R, Friedell GH: Occupation and cancer of the lower urinary tract. *Cancer* 29:1250-1260, 1972.

18. MacMahon B, Cole P: The ovarian etiology of human breast cancer. *Current Problems in the Epidemiology of Cancer, Lymphomas and Leukemias. Recent Results Cancer Res* 39:185-192, 1972.

19. Cole P: Epidemiology of Hodgkin's disease. *JAMA* 222:1636-1639, 1972.

20. MacMahon B, Cole P, Brown J: Etiology of human breast cancer: A review. *J Natl Cancer Inst* 50:21-42, 1973.

21. Hoover R, Cole P: Temporal aspects of occupational bladder carcinogenesis. *N Engl J Med* 288:1040-1043, 1973.

22. Cole P: Epidemiologic studies and surveillance of human cancers among personnel of virus laboratories. In Hellman A, Oxman MN, Pollack R (eds), *Biohazards in Biological Research*. Cold Spring Harbor Laboratory, New York 1973.

23. Cole P: Hypotheses regarding the etiology of breast cancer. *SeventhNational Cancer Conference Proceedings*. JB Lippincott Company, Philadelphia, 1973.

24. Cole P: A population-based study of bladder cancer. In Doll R, Vodopija I (eds), *Host Environment Interactions in the Etiology of Cancer in Man*. International Agency for Research on Cancer, Lyon, France, pp 83-87, 1973.

25. Schmauz R, Cole P: Epidemiology of cancer of the renal pelvis and ureter. *J Natl Cancer Inst* 52:1431-1434, 1974.

26. Cole P: Epidemiology of human breast cancer. *J Invest Dermatol* 63:133-137, 1974.

27. MacMahon B, Cole P, Brown JB, et al: Urine oestrogen profiles of Asian and North American women. *Int J Cancer* 14:161-167, 1974.

28. Cole P: Epidemiologic aspects of mammary tumours. Proceedings of the Fifth International Symposium on the Biological Characterization of Human Tumours. *Excerpta Medica*, Amsterdam, 1974.

29. Dickinson L, MacMahon B, Cole P, Brown JB: Estrogen profiles of Oriental and Caucasian women in Hawaii. *N Engl J Med* 291:1211-1213, 1974.

30. Cole P: Morbidity in the United States. Chapter four in Erhardt CL, Berlin JE (eds), *A Review of Mortality and Morbidity in the United States*. American Public Health Association Monograph, Harvard University Press, 1974.

31. Cole P: Primary prevention of cancer. *Bull NY Acad Med* 51:75-79, 1975.

32. Cole P: Cancer of the lower urinary tract. In Schottenfeld D (ed), *Cancer Epidemiology and Prevention: Current Concepts*. Charles C. Thomas, Springfield, Illinois, 1975.

33. Gutensohn N, Li F, Johnson R, Cole P: Hodgkin's disease, tonsillectomy and family size. *N Engl J Med* 292:22-25, 1975.

34. Simon D, Yen S, Cole P: Coffee drinking and cancer of the lower urinary tract. *J Natl_Cancer Inst* 54:587-591, 1975.

35. Cole P: Environmental factors in breast cancer: The epidemiologic evidence. Proceedings of the XIth International Cancer Congress. *Excerpta Medica*, 1975.

36. Cole P, Goldman M: Occupation. Chapter 11 in Fraumeni JF (ed), *Persons at High Risk of Cancer: An Approach to Cancer Etiology and Control*. Academic Press, New York, 1975.

37. Ory H, Cole P, MacMahon B, Hoover R: Oral contraceptives and reduced risk of benign breast diseases. *N Engl J Med* 294:419-422, 1976.

38. Cole P: What should the physician ask? *Cancer* 37:434-436, 1976.

39. Morrison A, Cole P: Epidemiology of bladder cancer. In Prout GR (ed), *Urologic Clinics of North America*, Vol 3, No 1. WB Saunders, Philadelphia, February 1976.

40. Bunker JP, Donahue VC, Cole P, Notman M: Elective hysterectomy: Pro and con. *N Engl J Med* 295:264-268, 1976.

41. Hoover R, Gray LA Sr, Cole P, MacMahon B: Menopausal estrogens and breast cancer. *N Engl J Med* 295:401-405, 1976.

42. Cole P, MacMahon B, Brown J: OEstrogen profiles of parous and nulliparous women. *Lancet* 2:596-599, 1976.

43. Grufferman S, Duong T, Cole P: Occupation and Hodgkin's disease. *J Natl Cancer Inst* 57:1193-1195, 1976.

44. Herbst A, Cole P, Colton T, et al: Age-incidence and risk of DES-related clear cell adenocarcinoma of the vagina and cervix. *Am J Obstet Gynecol* 128:43-50, 1977.

45. Grufferman S, Cole P, Smith P, Lukes RJ: Hodgkin's disease in siblings. *N Engl J Med* 296:248-250, 1977.

46. Cole P: Cancer and occupation: Status and needs of epidemiologic research. *Cancer* 39:1788-1791, 1977.

47. Cole P: Oral contraceptives and breast neoplasia. *Cancer* 39:1906-1908, 1977.

48. Gutensohn N, Cole P: Epidemiology of Hodgkin's disease in the young. *Int J Cancer* 19:595-604, 1977.

49. Sloan GM, Cole P, Wilson RE: Risk indicators of de novo malignancy in renal transplant recipients. *Transplant Proc* 9:1129-1132, 1977.

50. Isselbacher KJ, Cole P: Saccharin - the bitter sweet (editorial), *N Engl J Med* 296:1348-1350, 1977.

51. Cole P, Cramer D: Diet and cancer of endocrine target organs. *Cancer* 40:434-437, 1977.

52. Cole P, Berlin J: Elective hysterectomy. *Am J Obstet Gynecol* 129:117-123, 1977.

53. Elwood JM, Cole P, Rothman K, Kaplan S: Epidemiology of endometrial cancer. *J Natl Cancer Inst* 59:1055-1060, 1977.

54. Peyster RG, Kalisher L, Cole P: Mammographic parenchymal patterns and prevalence of breast cancer. *Radiology* 125:387-391, 1977.

55. Herbst AL, Scully RE, Robboy SJ, et al: Abnormal development of the human genital tract following prenatal exposure to diethylstilbestrol. In Hiatt H, Watson J, Winsten I (eds), *Origins of Human Cancer*. Cold Spring Harbor Laboratory, pp 399-412, 1977.

56. Herbst AL, Cole P: Epidemiologic and clinical aspects of clear cell adenocarcinoma in young women. In Herbst AL (ed), *Intrauterine Exposure to Diethylstilbestrol in the Human. Proceedings of "Symposium on DES," 1977*. The American College of Obstetricians and Gynecologists, February 1978.

57. Cole P, Elwood JM, Kaplan S: Incidence rates and risk factors of benign breast neoplasms. *Am J Epidemiol* 108:112-120, 1978.

58. Hoover R, Bain C, Cole P, MacMahon B: Oral contraceptive use: Association with frequency of hospitalization and chronic disease risk indicators. *Am J Public Health* 68:335-341, 1978.

59. Cole P, Cramer D, Yen S, et al: Estrogen profiles of premenopausal women with breast cancer. *Cancer Res* 38:745-748, 1978.

60. Cole, P: Epidemiology. In Gusberg SB, Frick HC (eds), *Gynecologic Oncology*. The Williams and Wilkins Company, Baltimore, 1978.

61. Cole P, Morrison A: Basic issues in cancer screening. In Miller AB (ed), *Screening in Cancer*. UICC Technical Report Series, Vol 40, Union Internationale Contre le Cancer, Geneva, 1978.

62. Cooper J, Saracci R, Cole P: Describing the validity of carcinogen screening tests. *Brit J Cancer* 39:87-89, 1979.

63. Cole P: Screening - An epidemiologic approach. In Fair R (ed.), *Selected Proceedings from the Symposium on Ocular and Systemic Disorders*. American Optometric Association, St. Louis, Missouri, 1978.

64. Cole P: The evolving case-control study. *J Chronic Dis* 32:15-27, 1979.

65. Grufferman S, Cole P, Levitan TR: Evidence against transmission of Hodgkin's disease in high schools. *N Engl J Med* 300:1006-1011, 1979.

66. Johnson L, Driscoll S, Hertig A, et al: Vaginal adenosis in stillborns and neonates exposed to diethylstilbestrol and steroidal estrogens and progestins. *Obstet and Gynecol* 53:671-679, 1979.

67. Morrison A, Cole P: Epidemiology of urologic cancers. In Javadpour N (ed), *Principles and Management of Urologic Cancer*. The Williams and Wilkins Company, Baltimore, 1979.

68. Austin H, Wynder E, Cole P: Breast cancer among Black American women. *Int J Cancer* 24:541-544, 1979.

69. Herbst A, Cole P, Norusis M, et al: An analysis of 384 registry cases of clear cell adenocarcinoma. *Am J Obstet Gynecol* 135:876-883, 1979.

70. Cole P: Some epidemiological aspects of cancer prevention (1979 Gordon Richards Memorial Lecture). *Cancer in Ontario*, 1979. Report of the Ontario Cancer Treatment and Research Foundation.

71. Herbst A, Scully R, Robboy S, et al: Diethylstilbestrol in pregnancy. *Proceedings of the Eppley Institute for Cancer Research* 1979:5-14. Omaha, Nebraska.

72. Cole P: Oral contraceptives and endometrial cancer (editorial). *N Engl J Med* 302:575-576, 1980.

73. Cole P, Morrison A: Basic issues in population screening for cancer. *J Natl Cancer Inst* 64:1263-1272, 1980.

74. Cole P, Merletti F: Chemical agents and occupational cancer. *J of Environ Path and Toxicol* 3:399-417, 1980.

75. Gutensohn N, Cole P: The epidemiology of Hodgkin's disease. *Sem Oncol* 7:92-102, 1980.

76. Trichopoulos D, Cole P, Brown JB, et al: OEstrogen profiles of primiparous and nulliparous women in Athens. *J Natl Cancer Inst* 65:43-46, 1980.

77. Cole P: Major aspects of the epidemiology of breast cancer. *Cancer* 46:865-867, 1980.

78. Cole P: Introduction. Chapter one in Breslow N, Day N (eds), *Statistical Methods in Cancer Research, Volume 1 - The Analysis of Case-Control Studies*. IARC Scientific Publications No. 32, Lyon, France, 1980.

79. Hoar S, Morrison A, Cole P, et al: An occupation and exposure linkage system for the study of occupational carcinogenesis. *J Occ Med* 22:722-726, 1980.

80. MacMahon B, Andersen A, Brown J, et al: Urine estrogen profiles in European countries with high or low breast cancer rates. *European Journal of Cancer* 16:1627-1632, 1980.

81. Cole P: Analytic epidemiology. In *Proceedings of the 1980 International Symposium on Cancer*, pp. 25-34, Grune and Stratton, New York, New York, 1980.

82. Gutensohn N, Cole P: Epidemiology of Hodgkin's Disease. In Coltman CA, Golomb HM (eds) *Hodgkin's and Non-Hodgkin's Lymphomas*, Grune and Stratton, New York, New York, 1980.

83. Gutensohn N, Cole P: Childhood social environment and Hodgkin's disease. *N Engl J Med* 304:135-140, 1981.

84. Cole P: Estrogens and progesterone in human breast cancer. *Banbury Report 8: Hormones and Breast Cancer*, pp. 109-113, Cold Spring Harbor, New York, 1981.

85. Cole P, Austin H: Breast self-examination: An adjuvant to early cancer detection (editorial). *Am J Pub Health* 71:572-574, 1981.

86. Brisson J, Merletti F, Sadowsky N, et al: Mammographic features of the breast and breast cancer risk. *Am J Epidemiol* 115:428-437, 1982.

87. Brisson J, Sadowsky N, Twaddle J, et al: The relation of mammographic features of the breast to breast cancer risk factors. *Am J Epidemiol* 115:438-443, 1982.

88. Morrison A, Cole P: Urinary tract. Chapter 54 in Schottenfeld D, Fraumeni JF (eds), *Cancer Epidemiology and Prevention*, W. B. Saunders Company, Philadelphia, PA, 1982.

89. MacMahon B, Trichopoulos D, Brown J, et al: Age at menarche, probability of ovulation and breast cancer risk. *Int J Cancer* 30:427-431, 1982.

90. MacMahon B, Trichopoulos D, Cole P, et al: Cigarette smoking and urinary estrogens. *N Engl J Med* 307: 1062-1065, 1982.

91. Cole P: Epidemiologic clues to host factors in human carcinogenesis. In Armstrong B, Bartsch H (eds), *Host Factors in Human Carcinogenesis*. IARC Scientific Publication No. 39, Lyon, France, 1982.

92. MacMahon B, Cole P, Brown J, et al: Urine estrogens, frequency of ovulation and breast cancer risks: A case-control study in premenopausal women. *J Natl Cancer Inst* 70:247-250, 1983.

93. Greenberg R, Grufferman S, Cole P: An evaluation of space-time clustering in Hodgkin's disease. *J Chron Dis* 36:257-262, 1983.

94. Simard, A, Vauclair R, Cole P, et al: Pulmonary cytology in foundry workers. *J Chron Dis* 36:617-623, 1983.

95. Cole P, Austin H: The Role of the Epidemiologist. In Newell G (ed), *The Practice of Cancer Prevention in Clinical Medicine*, pp. 5-17, The Raven Press, New York, New York, 1983.

96. Morrison A, Cole P, Maclure K: Epidemiology of urologic cancers. Chapter two in Javadpour N (ed), *Principles and Management of Urologic Cancer*, Second Edition, Williams and Wilkins, Baltimore, Maryland, 1983.

97. Cole P, Merletti, F: Occupational Cancer. Chapter 15 in Bourke GJ (ed), *The Epidemiology of Cancer*. Croome Helm, Ltd., Kent, England, 1983.

98. Trichopoulos D, Yen S, Brown J. et al: The effect of westernization on urine estrogens, frequency of ovulation and breast cancer risks: A study of ethnic Chinese women in the Orient and the U.S.A. *Cancer* 53:187-192, 1984.

99. Brisson J, Morrison A, Kopans D, et al: Height and weight, mammographic features of breast tissue, and breast cancer risk. Am J Epidemiol 119:371-381, 1984.

100. Scherr P, Gutensohn N, Cole P: School contact among persons with Hodgkin's disease. *Am J Epidemiol* 120:29-38, 1984.

101. Hochberg F, Toniolo P, Cole P: Head trauma and seizures as risk factors of glioblastoma. *Neurology* 34:1511-1514, 1984.

102. McCraw DS, Joyner RE, Cole P: Leukemia excess in a refinery population. *J Occ Med* 27:220-222, 1985.

103. Sohier R, Cole P, Montesano R: *Report on the IARC Research Training Fellowships Programme (1966-1984)*. IARC Internal Technical Report No. 86/002, WHO. Lyon, April 1986.

104. Austin H, Delzell E, Grufferman S, et al: A case-control study of hepatocellular carcinoma and the hepatitis B virus, cigarette smoking, and alcohol consumption. *Cancer Research* 46:962-966, 1986.

105. Austin H, Cole P: Cigarette smoking and leukemia. *J Chron Dis* 39:417-421, 1986.

106. Austin H, Cole P, McCraw D: A case-control study of leukemia in an oil refinery. *J Occ Med* 28:1169-1173, 1986.

107. Mueller N, Swanson GM, Hsieh C, et al: Tonsillectomy and Hodgkin's disease: Results from companion population-based studies. *J Natl Cancer Inst* 78:1-5, 1987.

108. Melnick S, Cole P, Anderson D, Herbst A: Rates and risks of diethylstilbestrol-related clear-cell adenocarcinoma of the vagina and cervix. An update. *N Engl J Med* 316:514-516, 1987.

109. Carpenter A, Flanders W, Frome E, et al: Brain cancer and nonoccupational risk factors: A case-control study among workers at two nuclear facilities. *Am J Public Health* 77:1180-1182, 1987.

110. Austin H, Delzell E, Grufferman S, et al: Case-control study of hepatocellular carcinoma, occupation, and chemical exposures. J Occup Med 29:665-669, 1987.

111. Austin H, Delzell E, Cole P: Benzene and leukemia: A review of the literature and a risk assessment. *Am J Epidemiol* 127:419-439, 1988.

112. Cole P: Saccharin and Bladder Cancer. Chapter nineteen in Gordis L (ed), *Epidemiology and Health Risk Assessment*. Oxford University Press, New York, New York, 1988.

113. Butterworth CE, Hatch K, Cole P, et al: Zinc concentration in plasma and erythrocytes of subjects receiving folic acid supplementation. *Am J Clin Nutr* 47:484-486, 1988.

114. Waterbor J. Cole P, Delzell E, et al: The mortality experience of major-league baseball players. *N Engl J Med* 318:1278-1280, 1988.

115. Cole P: Cancer: Risk factors and mortality trends. In Maulitz RC and Lear AC (eds), *Unnatural causes*. Philadelphia College of Physicians. Philadelphia, Pennsylvania, 1988.

116. Delzell E, Austin H, Cole P: Epidemiologic studies of the petroleum industry. In Weaver NK (ed), *Occupational Medicine: State of the Art Reviews*, Volume 3. Hanley and Belfus, Inc., Philadelphia, Pennsylvania, 1988.

117. Austin H, Keil J, Cole P: A prospective study of cancer mortality in relation to serum DDT levels. *Am J Public Health* 79:43-46, 1989.

118. Delzell E, Macaluso M, Cole P: A follow-up study of mortality among chemical workers. *J Occ Med* 31:273-278, 1989.

119. Wongsrichanalai C, Delzell E, Cole P: Mortality from leukemia and other diseases among workers at a petroleum refinery. *J Occ Med* 31:106-111, 1989.

120. Tamura T, Soong S, Sauberlich H, et al: Evaluation of the deoxyuridine suppression test using whole blood samples from folic acid-supplemented subjects. *Am J Clin Nutr* 51:80-86, 1990.

121. Hochberg F, Toniolo P, Cole P: Nonoccupational risk indicators of glioblastoma in adults. *Journal of Neuro-Oncology* 8:55-60, 1990.

122. Sharp G, Cole P: Vaginal bleeding and diethylstilbestrol exposure during pregnancy: Relationship to genital tract clear-cell adenocarcinoma and vaginal adenosis in daughters. *Am J Obstet Gynecol* 162:994-1001, 1990.

123. Sharp G, Cole P, Anderson D, et al: Clear cell adenocarcinoma of the lower genital tract. Correlation of mother's recall of diethylstilbestrol history with obstetrical records. *Cancer* 66:2215-2220, 1990.

124. Ibrahim MA, Bond GG, Burke TA, et al: The weight of the evidence on the human carcinogenicity of 2,4-D. Harvard School of Public Health, Program on Risk Analysis and Environmental Health. Final report, January 1990.

125. Cole P: The epidemiologist as an expert witness. *J Clin Epid* 44, Suppl. I, 35S-39S, 1991.

126. Macaluso M, Delzell E, Cole P, Cowles S: An evaluation of a corporate health surveillance system. *J Occ Med* 33:1180-1186, 1991.

127. Cole, P: The value of an indirect index to select groups for a health education message (editorial). *Epidemiology* 2:401-402, 1991.

128. Cole, P, Hovinga ME: Hormone residues in meat products. *Cancer Prevention* May 1991, pp. 1-7.

129. Sharp G, Cole P: Identification of risk factors for diethylstilbestrol-associated clear cell adenocarcinoma of the vagina: Similarities to endometrial cancer. *Am J Epidemiol* 134:1316-1324, 1991.

130. Butterworth CE, Hatch KD, Macaluso M, Cole P, et al: Folate deficiency and cervical dysplasia. *JAMA* 267:528-533, 1992.

131. Butterworth CE, Hatch KD, Soong S, Cole P, et al: Oral folic acid supplementation for cervical dysplasia: A clinical intervention trial. *Am J Obstet Gyn* 166:803-809, 1992.

132. Sathiakumar N, Delzell E, Cole P, Austin H: A follow-up study of agricultural chemical production workers. *Am J Ind Med* 21:321-330, 1992.

133. Chen F, Cole P, Mi Z, Xing L: Dietary trace elements and esophageal cancer mortality in Shanxi, China. *Epidemiology* 3:402-406, 1992.

134. Funkhouser E, Cole P: Declining mortality rates for cancer of the rectum in the United States: 1940-1985. *Cancer* 70:2597-2601, 1992.

135. Liu T, Wilson NP, Craig CB, et al: Evaluation of three nutritional assessment methods in a group of women. *Epidemiology* 3:496-502, 1992.

136. Barbone F, Delzell E, Austin H, Cole P: A case-control study of lung cancer at a dye and resin manufacturing plant. *Am J Ind Med* 22:835-849, 1992.

137. Carlo GL, Cole P, Miller AB, et al: Review of a study reporting an association between 2,4-dichlorophenoxyacetic acid and canine malignant lymphoma: Report of an expert panel. *Regul Toxicol Pharmacol* 16:245-252, 1992.

138. Cole P, Delzell E, Acquavella J: Exposure to butadiene and lymphatic and hematopoietic cancer. *Epidemiology* 4:96-103, 1993.

139. Funkhouser E, Waterbor J, Cole P, Rubin E: Mammographic patterns and breast cancer risk factors among women having elective screening. *South Med J* 86:177-180, 1993.

140. Hayes R, Friedell G, Zahm S, Cole P: Are the known bladder cancer risk-factors associated with more advanced bladder cancer? *Cancer Causes & Control* 4:157-162, 1993.

141. Chen F, Cole P, Mi Z, Xing L: Corn and wheat flour consumption and mortality from esophageal cancer in Shanxi, China. *Int J Cancer* 53:902-906, 1993.

142. Cole P: The hypothesis generating machine. *Epidemiology* 4:271-273, 1993.

143. Jones TL, Shih CH, Thurston DH, et al: Residential wire code associations and implications for case-control study bias. In Blank M (ed), *Electricity and Magnetism in Biology and Medicine*. San Francisco Press, Inc., San Francisco CA, 1993.

144. Jones TL, Shih CH, Thurston DH, et al: Selection bias from differential residential mobility as an explanation for associations of wire codes with childhood cancer. *J Clin Epidemiol* 46:545-548, 1993.

145. Liu T, Soong S-J, Wilson NP, et al: A case control study of nutritional factors and cervical dysplasia. *Cancer Epidemiol Biomarkers Prev* 2:525-530, 1993.

146. Cole P, Amoateng-Adjepong Y: Cancer prevention: Accomplishments and prospects. *Am J Public Health* 84:8-10, 1994.

147. Chen F, Cole P, Wen L, et al: Eight trace elements in Chinese farmers' household food supply, Shanxi Province, China. *J Nutr* 124:196-201, 1994.

148. Rodu B, Cole P: Tobacco-related mortality. *Nature* 370: 184, 1994.

149. Funkhouser E, Waterbor J, Cole P: Mammographic patterns and breast cancer risk. A meta-analysis. *Breast Dis* 6:277-284, 1994.

150. Barbone F, Delzell E, Austin H, Cole P: Exposure to epichlorohydrin and central nervous system neoplasms at a resin and dye manufacturing plant. *Arch Env Hlth* 49:355-358, 1994.

151. Cole P: The moral bases for public health interventions. *Epidemiology* 6:78-83, 1995.

152. Cole P, Sateren W: The evolving picture of cancer in America. *J Natl Cancer Inst* 87:159-160, 1995.

153. Honda Y, Delzell E, Cole P: An updated study of mortality among workers at a petroleum manufacturing plant. *JOEM* 37:194-200, 1995.

154. Amoateng-Adjepong Y, Sathiakumar N, Delzell E, Cole P: Mortality among workers at a pesticide manufacturing plant. *JOEM* 37:471-478, 1995.

155. Cole P, Sateren W, Delzell E: Epidemiologic perspectives on myelodysplastic syndromes and leukemia. *Leuk Res* 19:361-365, 1995.

156. Vigneswaran N, Dent M, Tilashalski K, et al.: Tobacco use and cancer - A reappraisal. *Oral Surg Oral Med Oral Pathol* 80:178-182, 1995.

157. Sathiakumar N, Delzell E, Cole P, et al.: A case-control study of leukemia among petroleum workers. *JOEM* 37:1269-1277, 1995.

158. Beall C, Delzell E, Cole P, Brill I: Brain tumors among electronics industry workers. *Epidemiology* 7:125-130, 1996.

159. Sathiakumar N, Delzell E, Cole P: Mortality among workers at two triazine herbicide manufacturing plants. *Am J Ind Med* 29:143-151, 1996.

160. Cole P, Rodu B: Declining cancer mortality in the United States. *Cancer* 78:2045-2048, 1996.

161. Cole P: Cancer screening strategies for the twenty-first century. In Brown H, Seffrin J (eds), *Horizons 2013* American Cancer Society, 1996.

162. Pinkston J, Cole P: Cigarette smoking and Warthin's tumor. *Amer J Epidemiol* 144:183-187, 1996.

163. Delzell E, Sathiakumar N, Hovinga M, et al: A follow-up study of synthetic rubber workers. *Toxicology* 113:182-189, 1996.

164. Macaluso M, Larson R, Delzell E, et al: Leukemia and cumulative exposure to butadiene, styrene and benzene among workers in the synthetic rubber industry. *Toxicology* 113:190-202, 1996.

165. Pinkston J, Cole P: ABO blood groups and salivary gland tumors. *Cancer Causes & Control* 7:572-574, 1996.

166. Cole P: Causality in epidemiology, health policy and law. *Environmental Law Reporter* 26(6):10279-10285, 1997.

167. Sathiakumar N, Delzell E, Amoateng-Adjepong Y, et al: Epidemiologic evidence on the relationship between mists containing sulfuric acid and respiratory tract cancer. *Crit Rev Toxicol* 27(3):233-251, 1997.

168. Acquavella J, Olsen G, Cole P, et al: Cancer among farmers: A meta-analysis. *Ann Epidemiol* 8:64-74, 1998.

169. Cole P: The only paper I ever wrote. *Cancer Causes & Control* 9:229-231, 1998.

170. Tilashalski K, Rodu B, Cole P: A trial of smokeless tobacco acceptance. *Amer J Med* 104:456-458, 1998.

171. Coggon D, Cole P: Acrylonitrile and human cancer - an overview. *Scan J Work Environ Health* 24 suppl 2:81-82, 1998.

172. Sathiakumar N, Delzell E, Hovinga M, et al: Mortality from cancer and other causes of death among synthetic rubber workers. *Occ & Env Med* 5:230-235, 1998.

173. Rodu B, Cole P: Nicotine maintenance for inveterate smokers. *Technology* 6:17-21, 1999.

174. Pinkston J, Cole P: Incidence rates of salivary gland tumors. Results from a population-based study. *Otolaryngol - Head and Neck Surg* 120:834-840, 1999.

175. Delzell E, Beall C, Rodu B, et al: Case-series investigation of intracranial neoplasms at a petrochemical research facility. *Am J Ind Med* 36:450-458, 1999.

176. Cole P, Green L, Lash T: Lifestyle determinants of cancer among Danish mastic asphalt workers. *Reg Toxicol & Pharm* 30:1-8, 1999.

177. Albers J, Cole P, Greenberg R, et al: Analysis of chlorpyrifos exposure and human health: Expert panel report. *J Toxicol Environ Hlth* 2:301-324, 1999.

178. Sathiakumar N, Delzell E, Cole P, Larson R: Mortality among workers at a plastics manufacturing plant. *J Environ Med*, 1:241-248, 1999.

179. Cole P, Rodu B, Delzell E: Moneychangers in the temple. *Epidemiology*, 11:84-90, 2000.

180. Rodu B, Cole P: The fifty-year decline of cancer in America. *J Clin Oncol*, 19:239-241, 2001.

181. Cole P, Rodu B: Descriptive Epidemiology: Cancer Statistics. Chapter 14, Section 2. In: DeVita V, Hellman S, Rosenberg S (eds.), *Cancer: Principles & Practice of Oncology*. 6th edition. New York: Lippincott-Raven, 2001.

182. Cole P, Rodu B: Analytic Epidemiology: Cancer Causes. Chapter 14, Section 3. In: DeVita V, Hellman S, Rosenberg S (eds.), *Cancer: Principles & Practice of Oncology*. 6th edition. New York: Lippincott-Raven, 2001.

183. Rodu B, Cole P: The impact of the anti-smoking campaign on lung cancer mortality. *International J Cancer*, 97:804-806, 2002.

184. Rodu B, Cole P: Smokeless tobacco use and cancer of the upper respiratory tract. *Oral Surg, Oral Med, Oral Path and Endo*. 93:511-515, 2002.

185. Lane C, Cole P: Dangers of unevaluated reversing alarms on construction equipment. *Construction Site News*, November 2002, pp. 10-11.

186. Rodu B, Stegmayr B, Nasic S, Cole P, Asplund K: Evolving patterns of tobacco use in northern Sweden. *J Int Med*. 253:660-665, 2003.

187. Cole P, Rodu B, Mathisen A: Alcohol-containing mouthwash and oropharyngeal cancer: A review of the epidemiology. *J Amer Dental Assoc*. 134:1079-1088, 2003.

188. Cole P, Trichopoulos D, Pastides H, Starr T, Mandel JS: Dioxin and cancer: A critical review. Regul Toxicol Pharmacol. 38:378-88, 2003.

189. Rodu B, Stegmayr B, Nasic S, Cole P, Asplund K: The influence of smoking and smokeless tobacco use on weight amongst men. *J of Int Med*. 255:102-107, 2004.

190. Tilashalski K, Rodu B, Cole P: Long-term follow-up of smokeless tobacco as a smoking cessation method. *J Psych Drugs*. In press.

191. Rodu B, Cole P: The burden of mortality from smoking in the European Union. In press.

Letters and Miscellany

1. Cole P: Cohorts and conclusions. *N Engl J Med* 278:1126-1127, 1968.

2. Cole P, MacMahon B: Urinary oestrogen profiles and aetiology of breast cancer. *Lancet* 2:153, 1970.

3. MacMahon B, Cole P, Newell GR: Hodgkin's disease: One entity or two? *Lancet* 1:240-241, 1971.

4. Cole P, Hoover R: Letters to the editor. *J Natl Cancer Inst* 46:1111- 1113, 1971.

5. Cole P, et al: Tonsillectomy and Hodgkin's disease. *N Engl J Med* 288:634, 1973.

6. MacMahon B, Cole P, Brown JB: Estrogen profiles and breast cancer. *N Engl J Med* 292:974-975, 1975.

7. Gutensohn N, Cole P, Li F: Hodgkin's disease and sibship size. *N Engl J Med* 292:1025-1038, 1975.

8. Cole P: Costs and benefits of hysterectomy. *N Engl J Med* 295:1086, 1976.

9. Grufferman S, Cole P, Smith P, Lukes RJ: Reasons for familial aggregation in Hodgkin's disease. *N Engl J Med* 296:940-941, 1977.

10. Gutensohn N, Cole P: Response to Vianna et al. *Int J Cancer* 20:633-634, 1977.

11. Gutensohn N, Cole P: Immunity in Hodgkin's disease. *Ann Intern Med* 91:316-317, 1979.

12. Gutensohn N, Cole P: Letter to the editor. *N Engl J Med* 304:1171, 1981.

13. Merletti F and Cole P: Detection bias and endometrial cancer. *Lancet* ii:579-580, 1981.

14. Cole P, Austin H: Reply to letter on breast self-examination. *Am J Pub Health* 71:1277, 1981.

15. Cole P, Austin H: Reply to letter on breast self-examination. *Am J Pub Health* 72:499-500, 1982.

16. MacMahon B, Trichopoulos D, Cole P and Brown J: Smoking and urinary estrogens. Letter to the Editor. *N Engl J Med* 308:591, 1983.

17. Austin H, Cole P: *Early Detection of Testicular Cancer.* (Book Review) *Chemother* 28:311, 1982.

18. Merletti F, Cole P: UICC Technical Report Series, Vol. 41, *Cancer Risks by Site*. (Book Review). *Chemother* 28(4):312, 1982.

19. Grufferman S, Cole P: Interaction between sex and HLA type and Hodgkin's Disease. *Arch Neurol* 41:816, 1984.

20. Austin H, Delzell E, Cole P, Grufferman S, Levene R, Morrison AS and Stolley PD: Reply to a letter regarding a case-control study of hepatocellular carcinoma. *Cancer Res* 48:654-655, 1987.

21. Austin H, Delzell E, Cole P: Benzene and leukemia. Letter to the editor. *N Engl J Med* 317:1027-28, 1987.

22. Cole P: Carpenter D and Ahlbom A: Comments on electromagnetic fields and cancer in human beings, especially children. Letter to the editor (by invitation). *Forum for Applied Research and Public Policy* 1989.

23. Austin H, Cole P: Reply to letter regarding cigarette smoking and leukemia. *J Clin Epidemiol* 42:1027-1028, 1989.

24. Cole P: Comment on a book review. *The Epid Monitor*, 14(4):9, 1993.

25. MacMahon B, Cole P, Rothman K: Tribute to Alan S. Morrison. *Epidemiol Rev* 15:3-6, 1993.

26. Cole P, Delzell E, Acquavella J: Butadiene and cancer. Response to letter by Park. *Epidemiology* 4:560-561, 1993.

27. Acquavella J, Delzell E, Cole P: Butadiene and cancer. Response to letter by Matanoski and Santos-Burgoa. *Epidemiology* 4(2):96-103, 1993.

28. Jones, T, Shih, C, Thurston, D et al: Response to a letter on Bias in studies on electromagnetic fields by Wertheimer, N and Leeper, E. *J Clin Epidemiol* 47:1083, 1994.

29. Rodu, B, Cole P: Absence of effect of smokeless tobacco use on mortality. *Am J Pub Health* 85:118, 1995.

30. Cole, P: The real conflict of interest. *Am J Ind Med* 28:615-616, 1995.

31. Rodu B, Cole P: Would a switch from cigarettes to smokeless tobacco benefit public health? Yes. Point/Counterpoint. *Priorities* 7:24-30, 1995.

32. Rodu B, Cole P: The rewards of smoking cessation. *Epidemiology* 7:111-112, 1996.

33. Cole P: What the doctor orders. Commentary. *Priorities* 8:27-28, 1996.

34. Cole P, Rodu B: Winning the war on cancer. *N Engl J Med* 337:936, 1997.

35. Acquavella J, Olsen G, Cole P, et al: Farming and Cancer: The Authors Respond. *Ann Epidemiol* 9(1):72, 1999.

36. Rodu B, Cole P: Advice and consent. *The Atlantic Monthly* 284:14, 1999.

37. Cole P, Delzell E, Rodu B: Moneychangers in the temple, The author's reply. *Epidemiology* 11:477-478, 2000.

38. Rodu B, Cole P: Response to Letters to the Editor. *Oral Surgery Oral Medicine Oral Pathology* 95:638-641.

- END -

Gary B. Johnson, Ph.D.

Managing Engineer

Professional Profile

Dr. Gary Johnson is a Managing Engineer in Exponent's Electrical practice, and is based in Boston, Massachusetts. He specializes in issues pertaining to electrical engineering particularly as they relate to the electrical environment of power systems. Dr. Johnson has extensive experience with the electric and magnetic fields of transmission and distribution systems as well as the audible noise, radio noise, and ozone that may be produced by high voltage power systems. His work has involved the measurement, modeling, and mitigation of the electrical environment of transmission lines, transformer vaults, and underground cables. His experience also involves issues dealing with lightning, electrical transients, and ground currents.

Dr. Johnson has appeared as an expert witness on the corona and field effects of DC and AC transmission lines and been a lecturer at the EPRI Transmission Line Design Seminars. He has made numerous presentations and led several workshops on power line design and the electrical environment. As part of his investigations of the electrical environment, Dr. Johnson developed an instrumentation system to measure the charge-size distribution of aerosols.

Dr. Johnson has also performed engineering studies related to power system fields, corona, and ground currents for several clients. He helped develop digital magnetic field recorders (STAR, WaveCam, and Pulsar) and techniques for automatic magnetic field mapping. He was a principal investigator in the EPRI research on magnetic field sources and methods of shielding.

His recent efforts have focused on power quality issues. He has investigated the production and magnitude of harmonics and transients on power systems. Prior to joining Exponent, Dr. Johnson was the President of Power Research Engineering, where he worked on engineering issues related to power lines, specializing in the electrical environment and power quality.

Credentials and Professional Honors

Ph.D., Electrical Engineering, University of Illinois, 1979
M.S., Physics, University of Illinois, 1976
B.S., Engineering Physics (Highest Honors), University of Illinois, 1974

Tau Beta Pi; Phi Kappa Phi
Institute of Electrical and Electronic Engineers, Corona and Field Effects Subcommittee (Chairman of AC & DC Fields Working Group); American Association for the Advancement of Science; American Physical Society; BioElectroMagnetics Society

Publications

"Predictive Models of Power-Frequency Residential Magnetic Fields Independent of Outdoor Power Lines," In Press, (with R. Kavet, R.M. Uhlrich, W.T. Kaune, and T. Powers).
"Measurement Instrumentation for Transient Magnetic Fields and Currents," Proceedings, 2001 IEEE Electromagnetic Compatibility Symposium, Montreal, Canada, August 2001, (with J.L. Guttman, J. Niple, and R. Kavet).
"Transient Magnetic Fields and Currents in Residences," Proceedings, 2001 IEEE Electromagnetic Compatibility Symposium, Montreal, Canada, August 2001, (with J.L. Guttman and R. Kavet).
"Instrumentation and Measurement Technology," Proceedings of EMF Engineering Review Symposium- EMF-RAPID Program, Charleston, SC, April 1998.
"Field-Management Technologies," Proceedings of EMF Engineering Review Symposium- EMF-RAPID Program, Charleston, SC, April 1998.
"Residential Magnetic Field Sources" Proceedings, 1995 EPRI EMF Seminar, Santa Clara, CA, March 1995.
"Residential Ground Current Reduction" Proceedings, 1995 EPRI EMF Seminar, Santa Clara, CA, March 1995.
"Low Field Transmission Lines: Design Concepts," Proceedings, 1995 CIGRE Study Committee 36 Colloquium, Foz do Aquacu, Brazil, May 1995 (with B.A. Clairmont).
"Study on the Human Perception of Hybrid Fields," Proceedings, 1995 CIGRE Study Committee 36 Colloquium, Foz do Aquacu, Brazil, May 1995 (with B.A. Clairmont and S. Zelingher).
"HVDC Transmission Line Corona Performance and Conductor Contamination by Insects," Proceedings, 1995 CIGRE Study Committee 36 Colloquium, Foz do Aquacu, Brazil, May 1995.
"Residential Field Sources: EPRI EMF Survey," Proceedings, Pennsylvania Electric Association Transmission and Distribution Meeting, Metamoras, Pennsylvania, May 12, 1994.
"EPRI Magnetic Field Technical Information Center," Proceedings, American Power Conference, Chicago, Illinois, April 26, 1994 (with R.J. Lordan).
"WAVECAM: A Pocket Size Magnetic Field Waveform Capture Device," Proceedings, American Power Conference, Chicago, Illinois, April 26, 1994 (with D.J. Childs and T.P. Sullivan).
"Magnetic Field Sources in Residences: Measurement, Detection, and Options," EMF Management Techniques Training Session, 1994 IEEE/PES Transmission and Distribution Conference and Exposition, Chicago, Illinois, April 14, 1994.
"Magnetic Field Management for Transmission Lines," Proceedings, 1994 Missouri Valley Electric Association Engineering Conference, Kansas City, MO, March 23, 1994 (with R. Lordan, B. Clairmont, K. King, and V. Rashkes).
"Residential Field Sources at Power Frequencies," Proceedings, 1993 IEEE International Symposium on Electromagnetic Compatibility, Dallas, TX, August 1993, pp. 132–137.

"Survey of Residential Magnetic Field Sources: Interim Report," Proceedings, 1993 American Power Conference, Chicago, IL, April 1993, pp. 1669–1673 (with J.H. Dunlap and L.E. Zaffanella).
"Measurements of Magnetic Field Sources in Schools," Proceedings, 1992 American Power Conference, Chicago, IL, April 1992.
"Transmission Line Magnetic Fields: Measurements and Calculations," Proceedings, 1992 American Power Conference, Chicago, IL, April 1992 (with B. Clairmont and J. Dunlap).
"Magnetic Field Sources in Nonresidential Settings" Proceedings, 1991 EPRI Science and Communication Seminar, San Jose, CA, October 1991.
"Magnetic Field Management: Residential Low-Voltage Grounding," Proceedings, 1991 EPRI Science and Communication Seminar, San Jose, CA, October 1991.
"A Comparison of International Grounding Practices and Associated Magnetic Fields," Proceedings, 1991 IEEE T&D Conference, Dallas, TX, September 1991 (with G.B. Rauch, P. Johnson, A. Stamm, S. Tomita, and J. Swanson).
"Research Facility for the Study of Power System Magnetic Fields," Proceedings, 1991 IEEE T&D Conference, Dallas, TX, September 1991 (with L.E. Zaffanella and G.B. Rauch.
"Residential Magnetic Field Sources," Panel Session Paper at the IEEE Power Engineering Society Summer Meeting, Minneapolis, MN, July 1990, and IEEE Power Engineering Society Transmission and Distribution Conference, Dallas, TX, September 1991.
"Studies of Power System Magnetic Fields: Characterization of Sources in Residential Environments, Measurements of Exposure, Influence on Computer Screens," Proceedings, CIGRE 1990 General Conference, Paris, France, August 1990 (with R.S. Baishiki, T.D. Bracken, G.B. Rauch, J.M. Silva, S.S. Sussman, and L.E. Zaffanella,).
"Degree of Corona Saturation for HVDC Transmission Lines," IEEE Transactions on Power Delivery, Vol. PWRD-5, April 1990, pp. 695–707.
"The Effect of HVAC – HVDC Line Separation in a Hybrid Corridor," IEEE Transactions on Power Delivery, PWRD-4, No. 2, pp. 1338–1350, April 1989 (with B.A. Clairmont, L.E. Zaffanella, and S. Zelingher).
"Measurements of AC and DC Field and Corona Effects in a Hybrid Corridor," Proceedings, 1989 American Power Conference, Chicago, IL, April 24–26, 1989 (with B.A. Clairmont).
"Measurement of Space Charge Density Using a Faraday Cage," Proceedings, 6th International Symposium on High Voltage Engineering, Paper 42.32, New Orleans, LA, August 1989.
"Space Charge Measurements Downwind from a Monopolar 500 KV HVDC Test Line," IEEE Transactions on Power Delivery, PWRD-3, No. 4, pp. 2056–2063, October 1988 (with P.J. Carter).
"Electric Field and Ion Density in Proximity of HVDC Transmission Lines: Measurements and Calculations," CIGRE Study Committee Montreal Colloquium, Montreal, Canada, June 1987.
"Small Air Ion Environments," Air Ions: Physical and Biological Aspects, CRC Press, 1987 (with T.D. Bracken).

"Electric Fields and Ion Currents of a +/- 400 kV HVDC Test Line," IEEE Trans. on Power Apparatus and Systems, PAS-102, 1983.
"Techniques for Measurements of the Electrical Environment Created by HVDC Transmission Lines," Proceedings, 4th International Symposium on High Voltage Engineering, Paper 13.05, Athens, Greece, September 1983 (with L.E. Zaffanella).
"HVDC Field and Ion Effects at Project UHV: Results of Electric Field and Ion Current Measurements," IEEE Trans. on Power Apparatus and Systems, PAS-101, 1982 (with M.G. Comber).
"The Electrical Environment and HVDC Transmission Lines," Proceedings, American Institute of Medical Climatology Conference on Environmental Ions and Related Biological Effects, Philadelphia, PA, October 1982.
"Extraction of an Intense Neutralized Ion Beam from a Plasma," Proceedings, 2nd International Conference on Electron Beam Research and Technology, Ithaca, NY, October 1977 (with J.T. Verdeyen and R.J. Kaye).
"Ion Beam Pellet Fusion," Proceedings, 4th Inter-University Conference on Energy, Urbana, IL, April 1977 (with W.L. Johnson, R.J. Kaye, and J.T. Verdeyen).
"Ion Bunching in Electronic Space Charge Regions," J. Appl. Phys., Vol. 47, p. 4442, 1976 (with W.L. Johnson and J.T. Verdeyen).

Workshops/Seminars

"Proposed IEEE Standard – 1556: Public Impacts," Panel Session: Electric and Magnetic Field Exposure Standards for the Public and Workers: 0 – 3 kHz, IEEE Power Engineering Society Summer Meeting, Vancouver, Canada, 2001.
"Power System Magnetic Fields," GPU Workshop, EPRI Power Delivery Center-Lenox, MA 1997.
"Measurement of Residential Magnetic Fields," Yankee Conference, Massachusetts Environmental Health Association, Westborough, MA, 1995.
"Residential Sources and Exposure," EMF Health Research: State of the Science, Harvard School of Public Health, Boston, MA, 1995
"Power System Magnetic Field Management Seminar," HVTRC, Lenox, MA, 1994.
"EMF in Substations," IEEE Workshop, Los Angeles, CA, May 1994.
"Proceedings: Substation Magnetic Field Workshop," EPRI Workshop, Palo Alto, CA, EPRI Report on RP 2942-41, TR 101852, April 1993.
"Distribution Magnetic Field Management Workshop," HVTRC, Lenox, MA, 1992; Washington, DC, 1993.
"End Use Magnetic Field R&D Workshop," EPRI Workshop, Raleigh, NC, 1992.
"EPRI Electrical Potpourri Seminar," Palo Alto, CA, 1990, Haslet, TX, 1991.
"Magnetic Field Considerations: Low Voltage Grounding," EPRI Workshop, Colorado Springs, CO, 1991.
"Power System Magnetic Field Measurement Workshop," HVTRC, Lenox, MA, 1988 to 1995.
"EPRI High Voltage Transmission Line Design Seminar," HVTRC, Lenox, MA, 1982 to 1992.

Kathleen M. Shanley
Process Leader, Environmental, Safety & Real Estate
The United Illuminating Company
Shelton, Connecticut

March, 2004

Education:

Bachelor of Science in Biology, June, 1978, Eastern Connecticut State University, Willimantic, CT

Master of Business Administration, May 2003, University of New Haven, CT

Experience:

September 1978 to July 1980 – American Chemical & Refining, Precious Metal Recovery Laboratory Supervisor

July 1980 to March 1985 - United Illuminating, Assistant Supervisor of Environmental Laboratory

April 1985 to August 1988 – United Illuminating, Supervisor of Environmental Reporting & Support Services.

August 1988 to August 1990 – United Illuminating, Manager of Environmental Licensing & Regulatory Affairs. Siting, environmental permits & licenses, environmental remediation, biological effects of AC fields, legislative and regulatory review.

August 1990 to March 1994 – United Illuminating, Manager of Environmental Audit & Special Projects

March 1994 to April 1999 – United Illuminating, Manager of Environmental Issues & Audit

April 1999 to February 2001 – United Illuminating, Director of Environmental & Safety

February 2001 to present - United Illuminating, Process Leader of Environmental, Safety & Real Estate

Other Experiences:

Member of Advisory Committee serving the Connecticut Interagency EMF Task Force, 1991 to present.

Industry and Professional Affiliations

Edison Electric Institute - EMF Task Force, 1990 to present. EMF Steering Committee, 1995 to present. EMF Task Force/EMF Steering Committee Chair, 1997-1999.

Electric Power Research Institute (EPRI) EMF Research Committee, 1990 to 2004

Member - University of Connecticut Institute of Water Resources Advisory Board

Director – National Audubon, Connecticut Board

Director – Gateway Community College Foundation