

SUPPLEMENTAL DIRECT TESTIMONY OF
STUART AARONSON, M. D.

PHYSICIAN
MOUNT SINAI SCHOOL OF MEDICINE

Subject: EMF: Oncology

INTRODUCTION

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25

Q. Please state your name and business address.

A. My name is Stuart Aaronson, M. D.

Q. What is your occupation?

A. I am a physician employed by the Mount Sinai School of Medicine, where I hold the Jane B. and Jack R. Aron Professorship and serve as Chairman, Department of Oncological Sciences.

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 (HECO-ST-1100B). Briefly, I became Chief, Laboratory of Cellular and Molecular Biology in the National Cancer Institute (NCI) 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 lead the Ruttenberg Cancer Center, a research

1 department now formally designated as the Department of Oncological Sciences,
2 at Mount Sinai, where I have been involved in building a nationally recognized
3 cancer program. I am responsible for hiring faculty members, developing disease
4 focused multidisciplinary cancer research efforts, and serving as a senior
5 academic leader within the Mount Sinai School of Medicine. I have my own
6 grant-supported research program as well. This program involves investigation of
7 cancer genes and the signaling pathways in which they act as well as the multistep
8 process of carcinogenesis. In the course of my work, I train graduate and medical
9 school students as well as postdoctoral investigators in the area of cancer biology.
10 I have published over 520 articles primarily related to cancer and have more than
11 50 patents or patent applications arising from my discoveries, one of which has
12 led to an approved drug and with others of which are at different stages of clinical
13 development.

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

15 A. I am presently a member of the American Association for Cancer
16 Research, and serve as a member of its Public Relations and Communications
17 Committee. I also serve as a Member of the National Neurofibromatosis
18 Foundation Research Advisory Board. I am an Associate Editor or Editorial
19 Board member of a large number of cancer focused scientific journals. These
20 include Cancer Research, Oncogene, International Journal of Cancer, and Cancer
21 and Metastasis Reviews. I serve on the Scientific Advisory Boards of the Kimmel
22 Cancer Center, Thomas Jefferson University, and the Georgetown University
23 Breast Cancer Specialized Program of Research Excellence (SPORE). I have
24 previously served as organizer of a number of scientific meetings including the
25 Princess Takamatsu Symposium. I have served as an elected officer of scientific

1 societies including Councilor of the Society for Experimental Biology and
2 Medicine and President of the Harvey Society.

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

5 A. I have followed this field for a number of years, although I have not published in
6 it. Specifically, I have reviewed the literature related to efforts to study whether
7 power frequency EMF may initiate and/or promote cancer by methods that have
8 been utilized to test other environmental agents.

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

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

18 Q. What do you refer to as “power frequency” electric and magnetic fields?

19 A. The “frequency” of electromagnetic energy is expressed in hertz (Hz), which is a
20 measure of the rapidity at which the field varies. Electric and magnetic fields do
21 not vary at 0 Hz. The fields associated with alternating current (AC) electric
22 power transmission are 60-Hz (50-Hz in Europe), which means that they oscillate
23 60 times per second. This frequency is in the Extremely Low Frequency portion
24 of the electromagnetic spectrum. Toward the top of the spectrum, where we find
25 ultraviolet radiation and X-rays, the frequencies are much higher. For instance,

1 the frequency of X-rays is about 1 billion billion Hz.

2 Q. What is cancer?

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

5 Q. How is cancer caused?

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

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

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

1 act as oncogenes.

2 Q. What is a carcinogen?

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

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

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

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

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

20 A. There have been many laboratory studies aimed at assessing whether power
21 frequency EMF could cause or in some manner promote the development of
22 cancer. The laboratory assessment of the carcinogenicity of EMF, as of that for
23 any other suspected carcinogen, has involved both long term studies in which
24 whole animals are chronically exposed to EMF, and studies of cancer-related
25 changes in genes or other cellular processes observed in isolated cells. The

1 traditional term for the whole animal studies is *in vivo*, Latin for “in life, or alive.”
2 Since isolated cells are traditionally studied in a culture contained in a glass
3 vessel, and the type of test is called *in vitro* (“in glass”).

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

6 A. There have been several large, well conducted long term studies (called bioassays)
7 in which laboratory mice and rats have been chronically exposed to very high
8 doses of 60-Hz EMF for long periods, in some cases for almost their entire
9 lifetimes. These types of studies have a proven record for predicting the
10 carcinogenicity of chemicals, physical agents, and other suspected cancer-causing
11 agents. Typically, one group of animals is exposed to a controlled, high 60-Hz
12 magnetic field and another group of the same size is not so exposed. Any animals
13 that die during the experiment are autopsied, and at the conclusion of a
14 predetermined time, all remaining animals are sacrificed and autopsied. Tumors
15 are carefully noted by type and number, and the tumor incidence between the two
16 groups is compared.

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

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

25 A. The whole animal experiments are overwhelmingly negative. As a whole, they

1 provide no consistent or convincing evidence of any relationship between EMF
2 and cancer, including brain cancer, breast cancer and leukemia.

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

5 A. A great many studies of different types have been performed. Some of these
6 studies have looked for evidence that power frequency EMF is “genotoxic,” that
7 is, that it damages DNA directly; others have looked for evidence that EMF
8 promotes the development of cancer.

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

11 A. Because of the very substantial funding through the federal RAPID research
12 program, this literature is massive. These studies involve controlled exposures of
13 normal cells to EMF, in various controlled environments, and then examining
14 them for evidence of damage, such as cell transformation, chromosome
15 aberrations, the breaking and rejoining of chromosomes, detached pieces of
16 chromosomes, and DNA strand breaks. The field intensities used in these
17 experiments ranged from less than 1 microtesla (or 10 milligauss) to more than
18 1,000 microtesla (or 10,000 milligauss).

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

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

25 Q. Please describe the *in vitro* studies that have investigated whether power

1 frequency magnetic fields are cancer promoters?

2 A. There have been a great many laboratory experiments aimed at assessing possible
3 biologic effects of power frequency fields that might conceivably cause them to
4 act as cancer promoters or to enhance the effectiveness of genotoxic agents. Thus,
5 experiments have been performed to test whether such fields inhibit programmed
6 cell death or DNA repair, affect cell proliferation or differentiation, or affect gene
7 expression or enzyme activity. Like the whole animal studies, the cell studies
8 have produced no consistent or convincing evidence that power frequency electric
9 or magnetic fields promote the development of cancer.

10 Q. Please describe laboratory studies that have specifically examined leukemia?

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

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

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

22 Q. Does this conclude your testimony?

23 A. Yes.

24

25