From:	jvolker
To:	Hingtgen, Robert J
Cc:	"svolker"
Subject:	Scoping Comments of The Protect Our Communities Foundation et al. for the Soitec Solar Project PEIR (Email 3 of 3)
Date:	Thursday, October 10, 2013 4:14:17 PM
Attachments:	Exhibit 6 to Soitec Solar PEIR Scoping Comments of POC et al.pdf Exhibit 7 to Soitec Solar PEIR Scoping Comments of POC et al.pdf Exhibit 8 to Soitec Solar PEIR Scoping Comments of POC et al.pdf Exhibit 9 to Soitec Solar PEIR Scoping Comments of POC et al.pdf Exhibit 10 to Soitec Solar PEIR Scoping Comments of POC et al.pdf Exhibit 11 to Soitec Solar PEIR Scoping Comments of POC et al.pdf

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Dear Mr. Hingtgen,

Attached to this email please find Exhibits 6-11 to the Scoping Comments of The Protect Our Communities Foundation, Backcountry Against Dumps and Donna Tisdale for the Programmatic Environmental Impact Report being prepared for the Soitec Solar Development Project. Due to their size, the Comments and Exhibits 1-5 are transmitted separately.

Our Comments and Exhibits 1-11 have also been mailed to you via Priority mail today.

Please let us know if you have any difficulty opening the attached documents.

Best regards,

Jamey Volker
Attorney for The Protect Our Communities Foundation,
Backcountry Against Dumps, and Donna Tisdale

Jamey M.B. Volker
Law Offices of Stephan C. Volker
436 - 14th Street, Suite 1300
Oakland, CA 94612
Tel: (510) 496-0600
Fax: (510) 496-1366
jvolker@volkerlaw.com

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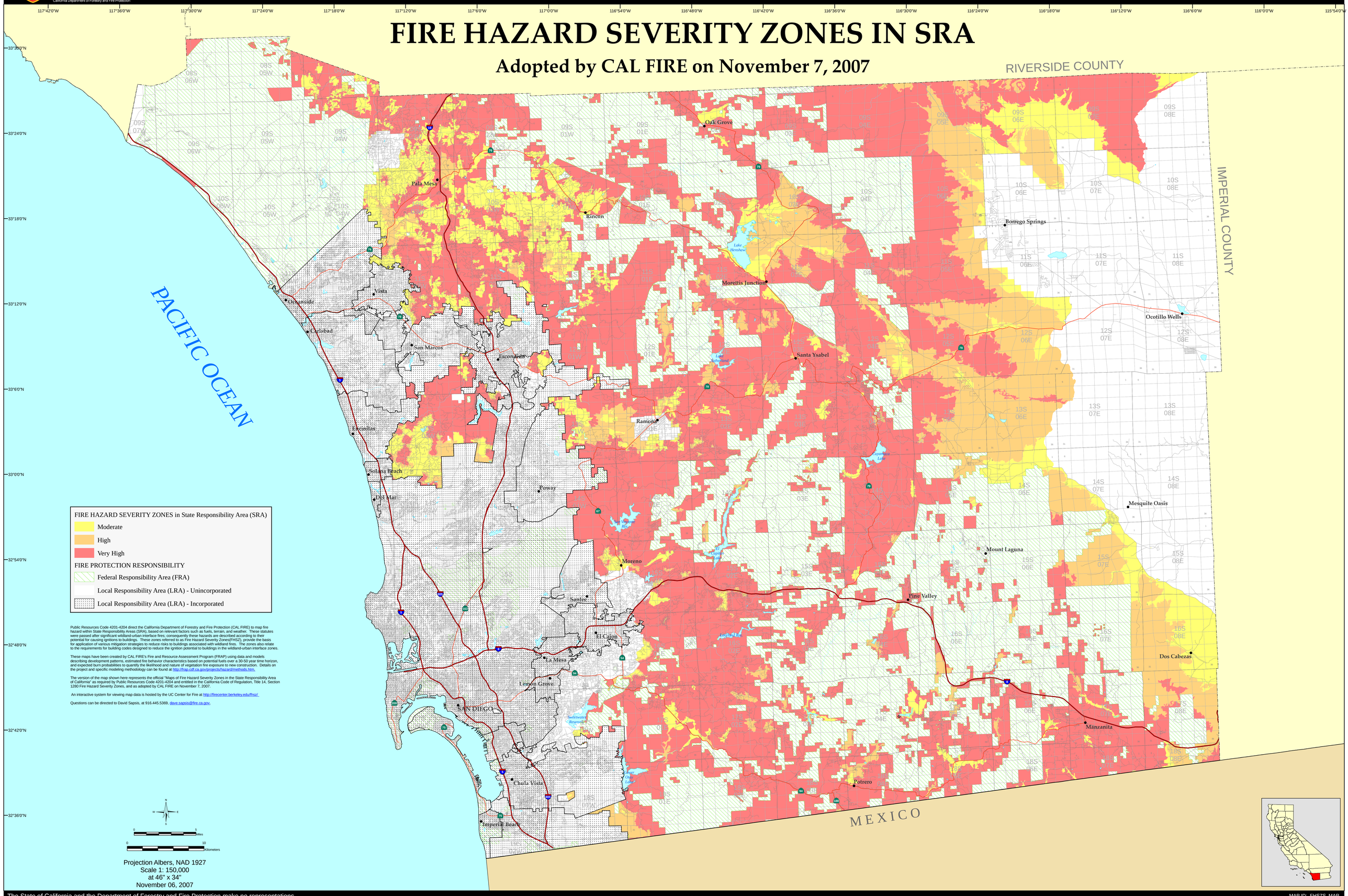
EXHIBIT 6

SAN DIEGO COUNTY

FIRE HAZARD SEVERITY ZONES IN SRA

Adopted by CAL FIRE on November 7, 2007



Public Resources Code 4201-4204 direct the California Department of Forestry and Fire Protection (CAL FIRE) to map fire hazard within State Responsibility Areas (SRA), based on relevant factors such as fuels, terrain, and weather. These statutes were passed after significant wildland-urban interface fires; consequently these hazards are described according to their potential for causing ignitions to buildings. These zones referred to as Fire Hazard Severity Zones(FHSZ), provide the basis for application of various mitigation strategies to reduce risks to buildings associated with wildland fires. The zones also relate to the requirements for building codes designed to reduce the ignition potential to buildings in the wildland-urban interface zones.

These maps have been created by CAL FIRE's Fire and Resource Assessment Program (FRAP) using data and models describing development patterns, estimated fire behavior characteristics based on potential fuels over a 30-50 year time horizon, and expected burn probabilities to quantify the likelihood and nature of vegetation fire exposure to new construction. Details on the project and specific modeling methodology can be found at http://frap.cdf.ca.gov/projects/hazard/methods.htm.

The version of the map shown here represents the official "Maps of Fire Hazard Severity Zones in the State Responsibility Area of California" as required by Public Resources Code 4201-4204 and entitled in the California Code of Regulation, Title 14, Section 1280 Fire Hazard Severity Zones, and as adopted by CAL FIRE on November 7, 2007.

An interactive system for viewing map data is hosted by the UC Center for Fire at http://firecenter.berkeley.edu/fhsz/

Questions can be directed to David Sapsis, at 916.445.5369, dave.sapsis@fire.ca.gov.

Projection Albers, NAD 1927
Scale 1: 150,000
at 46" x 34"
November 06, 2007

The State of California and the Department of Forestry and Fire Protection make no representations or warranties regarding the accuracy of data or maps. Neither the State nor the Department shall be liable under any circumstances for any direct, special, incidental, or consequential damages with respect to any claim by any user or third party on account of, or arising from, the use of data or maps.

Obtain FRAP maps, data, metadata and publications on the Internet at http://frap.cdf.ca.gov
For more information, contact CAL FIRE-FRAP, PO Box 944246, Sacramento, CA 94244-2460, (916) 327-3939.

Arnold Schwarzenegger, Governor,
State of California
Mike Chrisman, Secretary for Resources,
The Resources Agency
Ruben Grijalva, Director,
Department of Forestry and Fire Protection

MAP ID: FHSZS_MAP

DATA SOURCES
CAL FIRE Fire Hazard Severity Zones (FHSZS06_3)
CAL FIRE State Responsibility Areas (SRA05_5)
CAL FIRE Incorporated Cities (Incorp07_3)
PLSS (1:100,000 USGS, Land Grants with CAL FIRE grid)

EXHIBIT 7

STEPHAN C. VOLKER (CSB #63093)
JOSHUA A.H. HARRIS (CSB #226898)
STEPHANIE L. ABRAHAMS (CSB #257961)
JAMEY M.B. VOLKER (CSB # 273544)
LAW OFFICES OF STEPHAN C. VOLKER
436 14th Street, Suite 1300
Oakland, California 94612
Tel: 510/496-0600
Fax: 510/496-1366

Attorneys for Plaintiffs
BACKCOUNTRY AGAINST DUMPS, et al.

IN THE UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF CALIFORNIA

BACKCOUNTRY AGAINST DUMPS, et al., Plaintiffs, v. JIM ABBOTT, in his official capacity as California State Director of the United States Bureau of Land Management, et al., Defendants, and SAN DIEGO GAS & ELECTRIC CO., Intervenor-Defendant.	Civ. No. **3:10-CV-01222-AJB-BGS** **DECLARATION OF MARK OSTRANDER IN SUPPORT OF PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION AND APPLICATION FOR TEMPORARY RESTRAINING ORDER** Hearing: May 6, 2011 Time: 2:00 p.m. Court: 12 Judge: Hon. Anthony J. Battaglia

I, Mark Ostrander, declare as follows:

1. I am a recently retired Battalion Chief with the California Department of Forestry and Fire Protection ("Cal Fire") with 36 years' experience working in the field of wildfire suppression throughout Southern California.[1] I submit this declaration in support of the plaintiffs' motion for a preliminary injunction to prevent construction of the Sunrise Powerlink project. I make this declaration based on my personal knowledge and extensive

[1] I make this declaration in my capacity as an independent wildfire suppression expert. The views expressed herein are not intended to reflect the official views of the California Department of Forestry and Fire Protection.

professional expertise in the field of wildfire suppression.

BACKGROUND AND EXPERIENCE

2. I was employed by Cal Fire for the period 1974 through 2010. During the entire period of my employment by Cal Fire – an all risk fire response agency – I worked in the field of wildfire suppression, performing numerous essential tasks including both "hands-on" fire suppression activities in the field and later, in a supervisorial capacity as a manager of wildfire response campaigns on major conflagrations. I have worked on over 2,000 fires – including 12 of the 20 largest wildfires in California history – during my 36 years of employment with Cal Fire.

3. I started work with Cal Fire as a Firefighter and as a Firefighter I learned the use of the primary tools for construction of fire lines, including the Pulaski, the McCloud, double-bitted axe and, of course, shovels, rakes and grub hoes. I also became proficient in the removal of "ladder fuels" that give rise to "crown fires" in heavy timber, and as a feller, in the removal of trees that either had caught fire or were in imminent danger of doing so, in order to help prevent the rapid spread of wildfires. I was promoted to the position of Heavy Fire Equipment Operator in 1985, where my duties included construction of fire lines utilizing heavy equipment such as D-6C Caterpillar tractors. After 15 years, I became proficient in all these hands-on areas of fire suppression, and was promoted to Fire Station Captain. My responsibilities as Captain included management of a Fire Station and supervision of the personnel assigned to the Station. Our Station responded to all types of emergencies including the highly hazardous first phase, or "initial attack" on wildland fires for which I assumed management responsibility as Incident Commander until a Chief Officer arrived on scene.

4. In addition to my responsibilities as a Fire Station Captain, I also worked as a Crew Captain from 1996 to 2008. In this capacity, I led fire suppression crews numbering from 14 to 17 inmate men under extremely hazardous conditions in the initial attack on wildland fires where we quickly built fire lines to contain and ultimately control wildfires. As a Crew Captain, my tasks included training, supervising, and disciplining of Inmate

Firefighters. In addition to supervising and directing Inmate Fire Crews on wildland fires, I also kept them in a high state of readiness to respond to wildfires, floods, and earthquakes.

5. During my employment as a Fire Captain, I supervised Cal Fire's fire suppression response to approximately 200 plus fires, including the large, recent Harris and Pines fires. I was responsible for the supervision of 600 or more fire suppression personnel. Over time, as I became more proficient as a Fire Captain, my responsibilities grew to include the positions of Respiratory Protection Administrator for San Diego Cal Fire, Facility Safety Program Manager, Cal Fire San Diego Safety Committee Member, and Administrative Captain and Project Manager for the McCain Valley Conservation Camp.

6. After 23 years as a Fire Captain, I was promoted to the position of Battalion Chief for the Campo Battalion in eastern San Diego County. In that capacity I supervised operations for 7 fire stations, 1 Conservation Camp, and the San Diego Unit's Dozer and Heavy Equipment Program. I served as Incident Commander for Multi-Causality Incidents, Structure Fires, Hazardous Materials Incidents and Cal Fire wildfire suppression response to all wildfires on state or private land in eastern San Diego County. I also assisted in the supervision of fire suppression responses by the United States Forest Service and the U.S. Bureau of Land Management on lands managed by those federal agencies. I was Logistic Section Chief for Cal Fire's suppression response to the Cedar fire in 2003, a fire that engulfed 285,000 plus acres of land throughout central San Diego County. This fire ultimately burned 3000 plus structures and resulted in serious injuries or death to 14 people. I also managed the following additional major wildfire response campaigns as part of a Major Incident Command Team, Cal Fire Team 8: Saw Tooth, Eagle, Border 50, Shekel, Martin, Esperanza, among others. Our Team was also Certified in Complex Incident Management.

PROFESSIONAL OPINION

7. As a result of my 36 years' experience as a fire suppression specialist with Cal Fire, I have developed substantial expertise in understanding the causes of wildfire ignition and spread. At the same time, I have developed substantial expertise in the areas of

wildfire prevention, containment and control. Based on my nearly 4 decades of training and hands-on experience in these areas, I have formed the professional opinion that the Sunrise Powerlink project poses an extremely hazardous impediment to effective wildfire suppression in eastern San Diego County. I base this conclusion on my thorough familiarity with the Sunrise Powerlink project, which I have studied and on which I have submitted comments to the responsible agencies on numerous occasions.

8. The Sunrise Powerlink project will, if built, prevent effective fire suppression response for three reasons. First, modern fire suppression response depends heavily on retardant and water drops by large aircraft and helicopters as close as possible to the leading edge of wildfires. To be effective, retardant and water drops must be made as close to the surface as possible, and as quickly following ignition as possible. Delay in the delivery of retardant or water drops, or failure to deliver retardant or water directly to the leading edge of a wildfire, allows the fire to build momentum, size and speed. Once a wildfire has reached a critical size, temperature and speed, it is extremely difficult to contain much less control. Construction of the Sunrise Powerlink project will prevent the delivery of retardant and water drops along the many ridgelines and valley crossings that this powerline will span. Over 110 miles of high tension powerline located upwards of several hundred feet above the terrain will effectively create a large zone in which low flying aircraft cannot operate for the purpose of making retardant and water drops. This zone will increase in width when wildfires create smoky conditions in which low level flying is at best difficult, and in the face of potential ensnarement by high tension powerlines, completely impossible. The resulting inability to deliver retardant and water drops with speed and pinpoint accuracy onto the many ridges and exposed valleys that the Sunrise Powerlink project would span will cause a severe impairment of our fire suppression abilities.

9. Second, competent fire suppression leading to containment and ultimate control of fires requires construction of mineral earth fire lines by skilled fire crews deploying, where possible, heavy equipment. The deployment of fire crews within 100 to

1000 feet (depending on conditions) of high tension power lines is a serious safety concern and requires altering Strategies and Tactics which generally means the fire will get larger as we wait for it to pass thru the power line area to a safer distance to work the fire. These are well-established safety practices and procedures by all of the major fire suppression agencies, including Cal Fire. The reason for this is that wildfires can cause high tension powerlines to snap, either due to heat or wind. A powerline that has snapped not only can cause severe physical injury from the powerlines' whiplash motion to any crew member within several hundred yards of the line but also heavy smoke around the lines can cause phase-to-ground arcing due to the carbon in the smoke which reduces the air resistance allowing a path to ground. This increases the risk for electrocution or serious injury to fire personnel caused by the arcing and grounding of a "hot" electrical line. For this reason, construction of the Sunrise Powerlink project will prevent effective hand crew and heavy equipment fire suppression response, including the essential construction of fire lines needed to contain and control wildfires, within a 2,000-foot wide swath of land underlying the Sunrise Powerlink's designated route.

10. Third, powerlines cause the ignition of wildfires. Several of the worst conflagrations in San Diego County history, including the Pines and Witch fires, were ignited by powerlines. Although it is true that high tension powerlines such as the Sunrise Powerlink are less likely to ignite wildfires than smaller powerlines located closer to the ground, it is nonetheless true that high tension lines pose a very significant threat of wildfire ignition. This threat is exacerbated by the location of the Sunrise Powerlink project in remote areas of eastern San Diego County where inaccessibility by fire suppression response vehicles would allow the wildfires to grow into major conflagrations before effective suppression activities could commence.

11. For the foregoing reasons, I strongly urge this Court to halt construction of the Sunrise Powerlink project on federal land pending consideration of alternatives that would not pose such severe wildfire hazards to public and private land and to the health and safety of San Diego's citizens and wildlife.

I declare, under penalty of perjury, that the foregoing is true and correct based on my personal knowledge, and that this declaration was executed on April 14, 2011 in Jacumba, California.

Mark Ostrander

EXHIBIT 8

Attention Deficit Hyperactivity Disorder and Dirty Electricity

To the Editor:

In February 2010, while studying a cancer cluster in teachers at a California elementary school, a fourth-grade teacher complained that her students were hyperactive and unteachable. The classroom levels of high-frequency voltage transients (dirty electricity) in the radio frequencies (RF) between 4 and 100 kHz measured in the outlets of her classroom with a Graham/Stetzer Microsurge meter were very high. Dirty electricity is a term coined by the electrical utilities to describe electrical pollution contaminating the 60 Hz electricity on the electrical grid. A cell phone tower on campus a few feet from this classroom and unshielded fluorescent lights both contributed to the electrical pollution in this room. Cell tower transmitters, like most modern electrical equipment, operate on direct current. The electrical current brought to the tower is alternating current that needs to be changed to direct current. This is done by a switching power supply. These devices interrupt the alternating current and are the likely major source of the dirty electricity in the classroom.

On a Friday afternoon after school, I filtered the 5 outlets in this room with Graham/Stetzer plug-in capacitive filters, reducing the measured dirty electricity in the room wiring from more than 5000 Graham/Stetzer units to less than 50 units. With no change in either the lighting or the cell tower radiation, the teacher reported an immediate dramatic improvement in the behavior of her students in the following week. They were calmer, paid more attention, and were teachable all week except for Wednesday when they spent part of the day in the library.

In his 1973 book, Health and Light,[1] John N. Ott described a 1973 study of 4 first-grade classrooms in a windowless Sarasota, Florida school. Two of the rooms had standard white fluorescent lighting and the other two had full-spectrum fluorescent lighting with a grounded aluminum wire screen to remove the RF radiation produced by fluorescent bulbs and ballasts. Concealed time-lapse cameras recorded student behavior in classrooms for 4 months.[2] In the unshielded rooms, the first graders developed, "... nervous fatigue, irritability, lapses of attention, and hyperactive behavior." "... students could be observed fidgeting to an extreme degree, leaping from their seats, flailing their arms, and paying little attention to their teachers." In the RF-shielded rooms, "Behavior was entirely different. Youngsters were calmer and far more interested in their work."

The Old Order Amish live without electricity. A pediatric group practice in Jasper, Indiana, which cares for more than 800 Amish families has not diagnosed a single child with attention deficit hyperactivity disorder (ADHD).[3] Dozens of cases of childhood ADHD have been "cured" with no further need for drugs by simply changing their electrical environments (Stetzer D, personal communication [www.Stetzerelectric.com]). AQ:A

Before children are treated with drugs for ADHD, the dirty electricity levels in their homes and school environments should first be examined and reduced if needed.

I present the epidemiologic evidence linking dirty electricity to the other diseases of civilization in a recent book.[4,5] AQ:B

Disclosure: The authors declare no conflict of interest. AQ:C

Samuel Milham, MD, MPH AQ:D
Washington State Health Department
Tumwater, WA

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3. Ruff ME. Attention deficit disorder and stimulant use: an epidemic of modernity. *Clin Pediatr (Phila).* 2005;44:557–563.
4. Milham S. *Dirty Electricity.* Bloomington, IN: iUniverse; 2010. AQ:F
5. www.sammilham.com.

IN PRESS: JOURNAL OF DEVELOPMENTAL AND BEHAVIORAL PEDIATRICS

EXHIBIT 9

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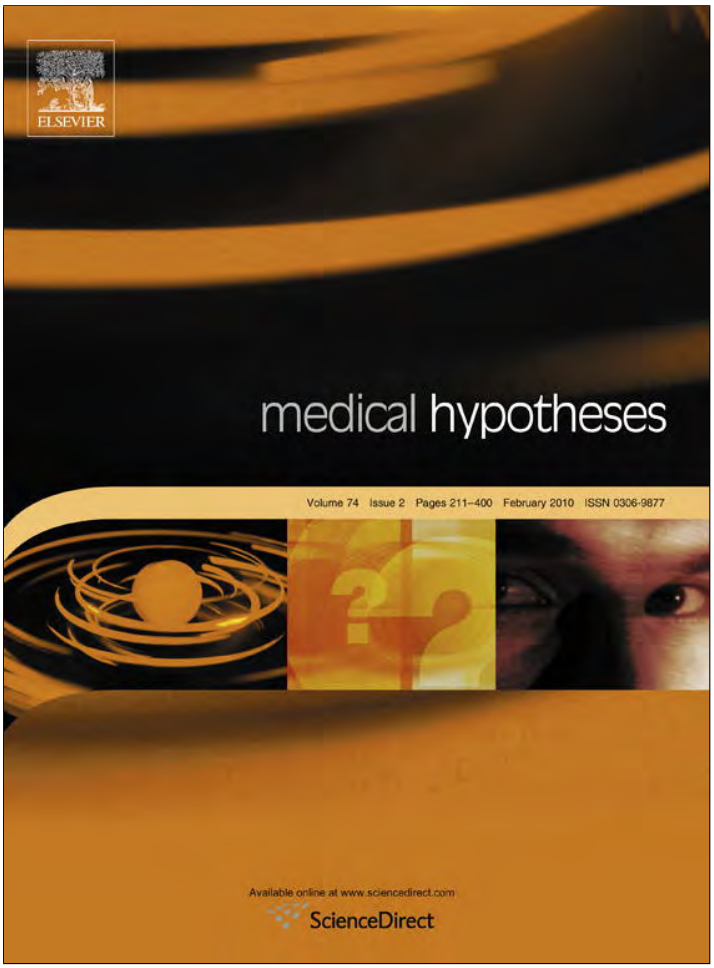

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Historical evidence that electrification caused the 20th century epidemic of "diseases of civilization" ☆

Samuel Milham *

Washington State Department of Health, Olympia, WA, USA

ARTICLE INFO

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SUMMARY

The slow spread of residential electrification in the US in the first half of the 20th century from urban to rural areas resulted by 1940 in two large populations; urban populations, with nearly complete electrification and rural populations exposed to varying levels of electrification depending on the progress of electrification in their state. It took until 1956 for US farms to reach urban and rural non-farm electrification levels. Both populations were covered by the US vital registration system. US vital statistics tabulations and census records for 1920–1960, and historical US vital statistics documents were examined. Residential electrification data was available in the US census of population for 1930, 1940 and 1950. Crude urban and rural death rates were calculated, and death rates by state were correlated with electrification rates by state for urban and rural areas for 1940 white resident deaths. Urban death rates were much higher than rural rates for cardiovascular diseases, malignant diseases, diabetes and suicide in 1940. Rural death rates were significantly correlated with level of residential electric service by state for most causes examined. I hypothesize that the 20th century epidemic of the so called diseases of civilization including cardiovascular disease, cancer and diabetes and suicide was caused by electrification not by lifestyle. A large proportion of these diseases may therefore be preventable.

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Background

In 2001, Ossiander and I [1] presented evidence that the childhood leukemia mortality peak at ages 2–4 which emerged in the US in the 1930s was correlated with the spread of residential electrification in the first half of the 20th century in the US. While doing the childhood leukemia study, I noticed a strong positive correlation between level of residential electrification and the death rate by state due to some adult cancers in 1930 and 1940 vital statistics. At the time, a plausible electrical exposure agent and a method for its delivery within residences was lacking. However, in 2008 I coauthored a study of a cancer cluster in school teachers at a California middle school [2] which indicated that high frequency voltage transients (also known as dirty electricity), were a potent universal carcinogen with cancer risks over 10.0 and significant dose–response for a number of cancers. They have frequencies between 2 and 100 kHz. These findings are supported by a large cancer incidence study in 200,000 California school employees which showed that the same cancers and others were in excess in California teachers statewide [3]. Power frequency magnetic fields (60 Hz) measured at the school were low and not related to cancer incidence, while classroom levels of high frequency voltage transients measured at the electrical outlets in the classrooms accurately predicted a teacher's cancer risk. These fields are potentially present in all wires carrying electricity and are an important component of ground currents returning to substations especially in rural areas. This helped explain the fact that professional and office workers, like the school teachers, have high cancer incidence rates. It also explained why indoor workers had higher malignant melanoma rates, why melanoma occurred on part of the body which never are exposed to sunlight, and why melanoma rates are increasing while the amount of sunshine reaching earth is stable or decreasing due to air pollution. A number of very different types of cancer had elevated risk in the La Quinta school study, in the California school employees study, and in other teacher studies. The only other carcinogenic agent which acts like this is ionizing radiation.

Among the many devices which generate the dirty electricity are compact fluorescent light bulbs, halogen lamps, wireless routers, dimmer switches, and other devices using switching power supplies. Any device which interrupts current flow generates dirty electricity. Arcing, sparking and bad electrical connections can also generate the high frequency voltage transients. Except for the dimmer switches, most of these devices did not exist in the first half of the 20th century. However, early electric generating equipment

☆ Supported by a small grant from Children with Leukemia.

* Address: 2318 Gravelly Beach Loop NW, Olympia, WA 98502-8837, USA. Tel.: +1 360 866 0256.
E-mail address: smilham2@comcast.net

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and electric motors used commutators, carbon brushes, and split rings, which would inject high frequency voltage transients into the 60 Hz electricity being generated and distributed.

With a newly recognized electrical exposure agent and a means for its delivery, I decided to examine whether residential electrification in the US in the first half of the last century was related to any other causes of death. Most cancers showed increasing mortality in this period, and many are still increasing in incidence in the developed world.

Thomas Edison began electrifying New York City in 1880, but by 1920, only 34.7% of all US dwelling units and 1.6% of farms had electric service (Table 1). By 1940, 78% of all dwelling units and 32% of farms had electric service [4]. This means that in 1940 about three quarters of the US population lived in electrified residences and one quarter did not. By 1940, the US vital registration system was essentially complete, in that all the 48 contiguous United States were included. Most large US cities were electrified by the turn of the century, and by 1940, over 90% of all the residences in the northeastern states and California were electrified. In 1940 almost all urban residents in the US were exposed to electromagnetic fields (EMFs) in their residences and at work, while rural residents were exposed to varying levels of EMFs, depending on the progress of rural electrification in their states. In 1940, only 28% of residences in Mississippi were electrified, and five other southern states had less than 50% of residences electrified (Table 2). Eleven states, mostly in the northeast had residential electrification rates above 90%. In the highly electrified northeastern states and in California, urban and rural residents could have similar levels of EMF exposure, while in states with low levels of residential electrification, there were potentially great differences in EMF exposure between urban and rural residents. It took the first half of the 20th century for these differences to disappear. I examined US mortality records by urban and rural residence by percent of residences with electric service by state.

Hypothesis

The diseases of civilization or lifestyle diseases include cardiovascular disease, cancer and diabetes and are thought to be caused by changes in diet, exercise habits, and lifestyle which occur as countries industrialize. I think the critical variable which causes the radical changes in mortality accompanying industrialization is electrification. Beginning in 1979, with the work of Wertheimer and Leeper [5], there has been increasing evidence that some facet of electromagnetic field exposure is associated epidemiologically with an increased incidence of leukemia, certain other cancers and non-cancers like Alzheimer's disease, amyotrophic lateral sclerosis, and suicide. With the exception of a small part of the electromagnetic spectrum from infra red through visible light, ultraviolet light and cosmic rays, the rest of the spectrum is man-made and foreign to human evolutionary experience. I suggest that from the time that Thomas Edison started his direct current electrical distribution system in the 1880s in New York City until now, when most of the world is electrified, the electricity carried high frequency voltage transients which caused and continue to cause what are considered to be the normal diseases of civilization. Even today, many of these diseases are absent or have very low incidence in places without electricity.

Table 1
Growth of residential electric service US 1920–1956 percent of dwelling units with electric service.

Year	All		Urban and rural non-farm
	Dwellings	Farm	
1920	34.7	1.6	47.4
1925	53.2	3.9	69.4
1930	68.2	10.4	84.8
1935	68.0	12.6	83.9
1940	78.7	32.6	90.8
1945	85.0	48.0	93.0
1950	94.0	77.7	96.6
1956	98.8	95.9	99.2

Table 2
Percent of residences with electric lighting 1930 and 1940 by state.

Code	State	1930	1940
AL	Alabama	33.9	43.3
AZ	Arizona	68.8	70.5
AR	Arkansas	25.3	32.8
CA	California	93.9	96
CO	Colorado	69.6	77.6
CT	Connecticut	95.3	96.5
DE	Delaware	78.4	81.8
FL	Florida	60.9	66.5
GA	Georgia	35.5	46.6
ID	Idaho	64.5	79.1
IL	Illinois	86.1	89.9
IN	Indiana	74.8	84
IA	Iowa	65.6	76.7
KS	Kansas	62	71.5
KY	Kentucky	44.2	54.2
LA	Louisiana	42.2	48.9
ME	Maine	76.1	80.4
MD	Maryland	81.8	85.9
MA	Massachusetts	97.1	97.6
MI	Michigan	84.8	92.1
MN	Minnesota	65.9	75.8
MS	Mississippi	19.4	28.3
MO	Missouri	65.5	70.6
MT	Montana	58.2	70.7
NE	Nebraska	61	70.5
NV	Nevada	76.2	80.8
NH	New Hampshire	84.9	87
NJ	New Jersey	95.8	96.6
NM	New Mexico	39.8	49.2
NY	New York	94.5	96.4
NC	North Carolina	40.8	54.4
ND	North Dakota	41.6	53.8
OH	Ohio	85.2	90.6
OK	Oklahoma	45.3	55.1
OR	Oregon	79.5	85.8
PA	Pennsylvania	89.5	92.3
RI	Rhode Island	97.3	97.7
SC	South Carolina	34.3	46.2
SD	South Dakota	44.4	56.6
TN	Tennessee	42	50.9
TX	Texas	*	59
UT	Utah	88.4	93.9
T	Vermont	71.9	80.2
VA	Virginia	50.5	60.6
WA	Washington	86.3	90.9
WV	West Virginia	63.4	69.1
WI	Wisconsin	74.5	83.9
WY	Wyoming	60	70.9

*No data.

Evaluation of the hypothesis

To evaluate the hypothesis, I examined mortality in US populations with and without residential electrification. Vital statistics tabulations of deaths [6], US census records for 1920–1970 [7], and historical US documents [8,9] were examined in hard copy or downloaded from the internet. The same state residential electrification data used in the childhood leukemia study [1] was used in this study. Crude death rates were calculated by dividing number of deaths by population at risk, and death rates by state were then correlated with electrification rates by state using downloaded software [10]. Time trends of death rates for selected causes

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of death by state were examined. Most rates were calculated by state for urban and rural residence for whites only in 1940 deaths, since complete racial data was available by urban/rural residence by state for only 13 of 48 states. Data was available for 48 states in the 1940 mortality tabulations. District of Columbia was excluded because it was primarily an urban population. Excel graphing software [11] and "Create a Graph" [12] software was used.

I had hoped to further test this hypothesis by studying mortality in individual US farms with and without electrification, when the 1930 US census 70 year quarantine expired in 2000. Unfortunately, the 1930 US farm census schedules had been destroyed.

Findings

Rural residential electrification did not reach urban levels until 1956 (Table 1). Table 2 shows the level of residential electrification for each state for 1930 and 1940. In 1930 and 1940 only 9.5% and 13%, respectively, of all generated electricity was used in residences. Most electricity was used in commercial and industrial applications.

Figs. 1–4 were copied and scanned from "Vital statistics rates in the United States 1940–1960", by Robert Grove Ph.D. and Alice M. Henzel. This volume was published in 1968. Fig. 1 shows a gradual decline in the all causes death rate from 1900 to 1960 except for a spike caused by the 1918 influenza pandemic. Death rates due to tuberculosis, typhoid fever, diphtheria, dysentery, influenza and pneumonia and measles all fell sharply in this period, and account for most of the decline in the all causes death rate. Figs. 2–4 show that in the same time period when the all causes death rate was declining, all malignant neoplasms (Fig. 2), cardiovascular diseases (Fig. 3), and diabetes (Fig. 4) all had gradually increasing death rates. In 1900, heart disease and cancer were 4th and 8th in a list of 10 leading causes of death. By 1940 heart disease had risen to first and cancer to second place, and have maintained that position ever since. Table 3 shows that for all major causes of death examined, except motor vehicle accidents, there was a sizable urban excess in 1940 deaths. The authors of the extensive 69 page introduction to the 1930 mortality statistics volume noted that the cancer rates for cities were 58.2% higher than those for rural areas. They speculated that some of this excess might have been due to rural residents dying in urban hospitals. In 1940, deaths by place of residence and occurrence are presented in separate volumes. In 1940 only 2.1% of all deaths occurred to residents of one state dying in another state. Most non-resident deaths were residents of other areas of the same state. Table 4 presents correlation coefficients for the relationship between death rates by urban rural areas of each state and the percent of residences in each state with

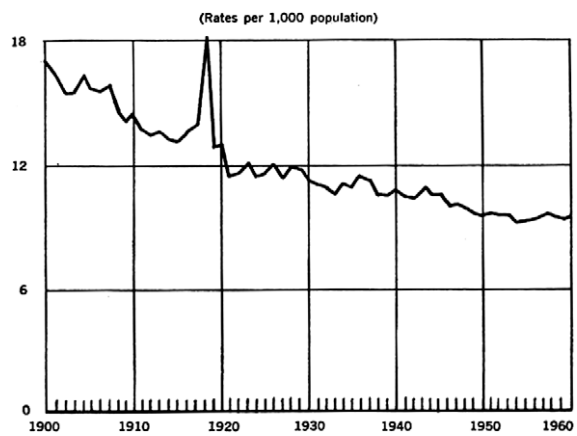

Fig. 1. Death rates: death registration states, 1900–32, and United States, 1933–60.

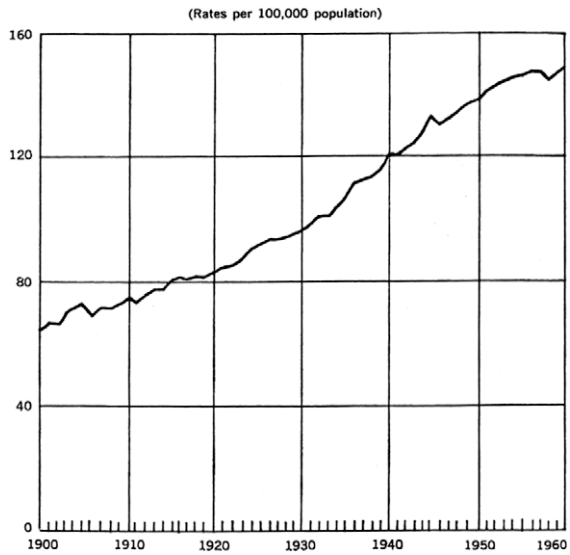

Fig. 2. Death rates for malignant neoplasms: death registration states, 1900–32, and United States, 1933–60.

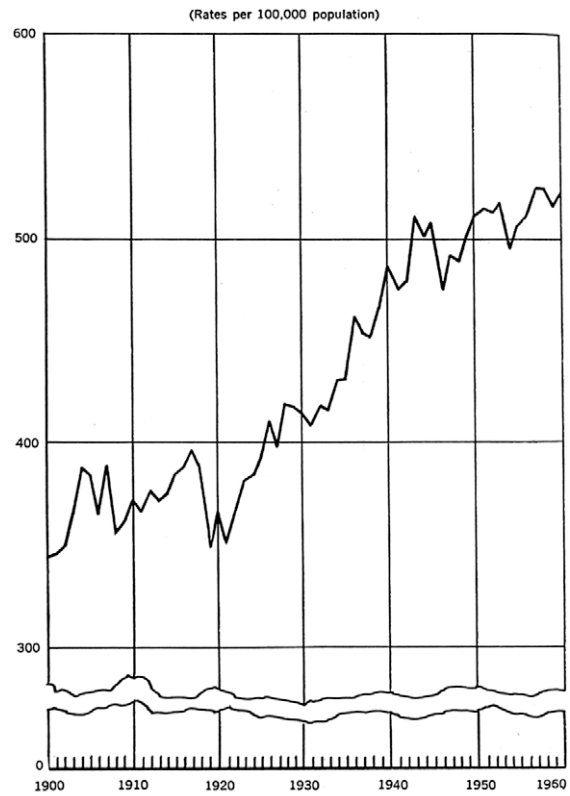

Fig. 3. Death rates for major cardiovascular renal diseases: death registration states, 1900–32, and United States, 1933–60.

electric service. In 1940 urban and rural residence information was not available for individual cancers as it was in 1930, but death rates for each cancer were available by state. They were used to calculate correlations between electric service by state and respiratory cancer, breast cancer and leukemia mortality.

All causes of death

There was no correlation between residential electrification and total death rate for urban areas, but there was a significant

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correlation for rural areas ($r = 0.659$, $p = <0.0001$). Fig. 5 shows the 1940 resident white death rates for urban and rural areas of states having greater than 96% of residences electrified and states having less than 50% of residences electrified. In the highly electrified states, urban and rural death rates were similar, but in low electrification states, the urban death rates were systematically higher than the rural death rates. The urban death rates were similar in both high and low electrification states.

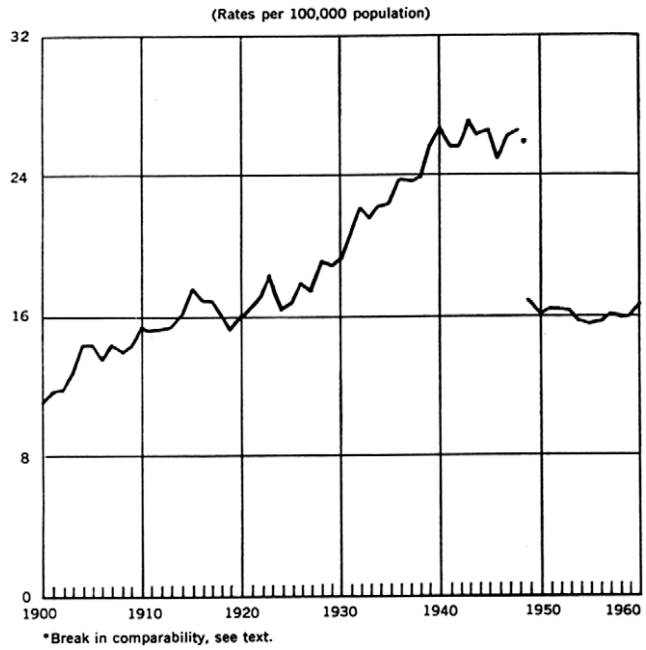

Fig. 4. Death rates for diabetes mellitus: death registration states, 1900–32, and United States, 1933–60.

All malignant neoplasms

In 1940, the urban total cancer rate was 49.2% higher than the rural rate. Both urban and rural cancer deaths rates were significantly correlated with residential electrification. Fig. 6 shows the 1940 resident white total cancer rates for urban and rural areas of states having greater than 96% of residences electrified and states having less than 50% of residences electrified. Four of the five high electrification states had similar urban and rural total cancer rates, while all the low electrification states had urban rates about twice as high as rural rates. Both urban and rural total cancer rates were lower in low electrification states than in high electrification states. Fig. 7 shows the time trend of the total cancer rate between 1920 and 1960 for Massachusetts (1940 electrification rate = 97.6%) and Louisiana (1940 electrification rate = 48.9%). The Massachusetts cancer rate was about twice that of Louisiana between 1920 and 1945. The Massachusetts rate leveled off in 1945, but the Louisiana rate increased steadily between 1920 and 1960. A declining urban–rural gradient for cancer is still evident in 1980–1990 US cancer incidence data [13]. Swedish investigators [14] have reported increasing cancer mortality and incidence time trend breaks in the latter half of the 20th century.

Table 3
1940 US white resident crude death rates per 100,000 by urban/rural residence.

Cause of death	ICD No.[a]	Urban rate	Rural rate	(%) Urban excess
All	1-200	1124.1	929.5	20.9
All cancers	47-55	145.8	97.7	49.2
Coronary disease	94	92.4	69.1	33.7
Other diseases of heart	90b,91,92a,d,e 93a,b,d,e 95a,c	217.0	162.8	33.3
Diabetes	61	33.2	20.0	66.0
Suicide	163-164	17.1	13.2	29.5
Motor vehicle accidents	170	26.6	26.3	1.1

[a] 1938 Revision International classification of disease.

Table 4
Correlation coefficients (r) 1940 crude US death rates by state by electrification for white resident deaths.

Cause	ICD No.[A]	Residence	r	r^2	p One tailed	Slope	Y intercept
All causes	1-200	Urban	0.083	0.007	0.285	0.007	11.114
		Rural	0.659	0.434	<0.0001	0.070	4.185
All cancers	45-55	Urban	0.667	0.445	<0.0001	0.883	75.970
		Rural	0.758	0.575	<0.0001	1.502	−10.040
Respiratory cancer[B]	47	State	0.611	0.374	<0.0001	0.071	1.020
Breast cancer female	50	State	0.794	0.630	<0.0001	0.170	−1.506
Diabetes	61	Urban	0.666	0.444	<0.0001	0.278	8.168
		Rural	0.693	0.480	<0.0001	0.366	−6.184
Leukemia[B]	72a	State	0.375	0.140	0.0042	0.021	1.980
Coronary artery	94	Urban	0.400	0.160	0.0024	0.494	61.570
Disease		Rural	0.781	0.610	<0.0001	1.252	25.319
Other diseases of the heart	90b, 91	Urban	0.449	0.202	0.0006	1.236	100.35
	92a,d,e	Rural	0.799	0.639	0.0001	2.887	−48.989
	93a,b,d,e						
	95a,c						
Suicide	163-4	Urban	0.077	0.006	0.2993	0.028	16.235
		Rural	0.729	0.532	<0.0001	0.181	0.299
Motor vehicle	170	Urban	−0.254	0.064	0.0408	−0.171	44.572
Accidents		Rural	0.451	0.203	0.0006	0.195	12.230

[A] International classification of diseases 1938 revision.
[B] Age adjusted death rate both sexes.

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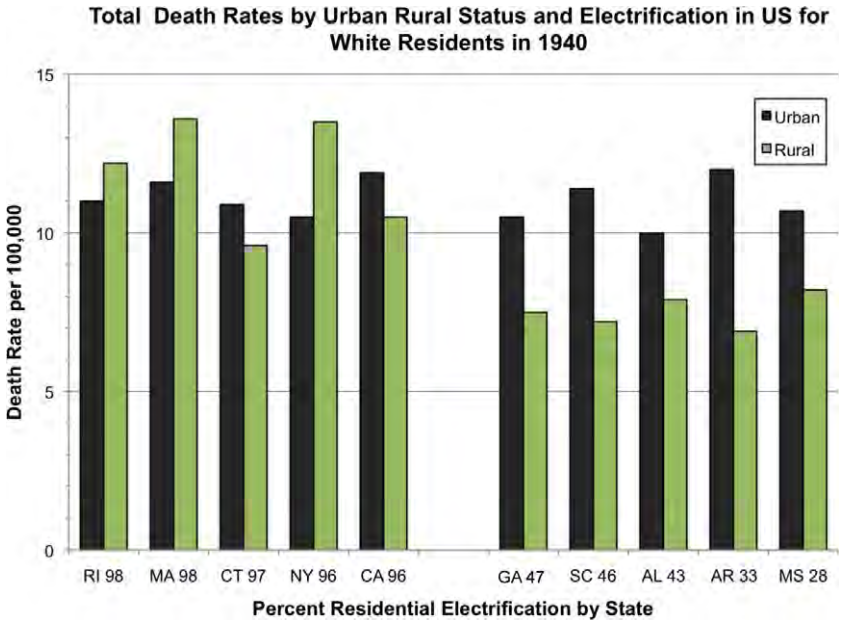

Fig. 5. All causes death rates by urban rural status and electrification in the US for white residents in 1940.

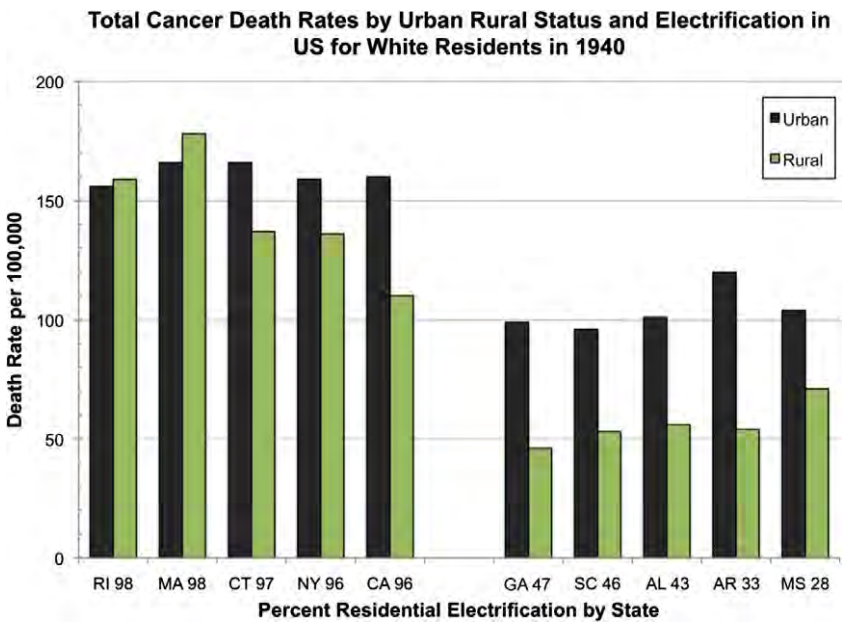

Fig. 6. Total cancer death rates by urban rural status and electrification in the US for white residents in 1940.

Respiratory cancer

No urban rural information was available for respiratory cancer, but the correlation between residential electrification and state death rates was $r = 0.611$; $p = <0.0001$. This cancer is etiologically strongly related to cigarette smoking, so the correlation with electrification is surprising. A large electrical utility worker cohort study found a high respiratory cancer incidence related to high frequency EMF transient exposure independent of cigarette smoking with a significant dose–response relationship [15].

Breast cancer

Although urban/rural information was not available for breast cancer, the 1940 state breast cancer death rates have a correlation of $r = 0.794$; $p = <0.0001$ with residential electrification. Fig. 8 shows the typical time trend of breast cancer death rates for a state with a high level of electrification (96%) and one with a low level of electrification (<50) in 1940. The California breast cancer death rate increased from 1920 to 1940, and then gradually decreased until 1960. The Tennessee breast cancer death rate is less than half of the California rate in 1920 and continues a steady increase until 1960.

Diabetes

This cause has a 66% urban excess. In spite of this, the correlation coefficients for urban and rural areas are similar at $r = 0.66$; $p = <0.0001$. There is some animal and human evidence that EMFs can effect insulin production and blood glucose levels [16]. Fig. 9

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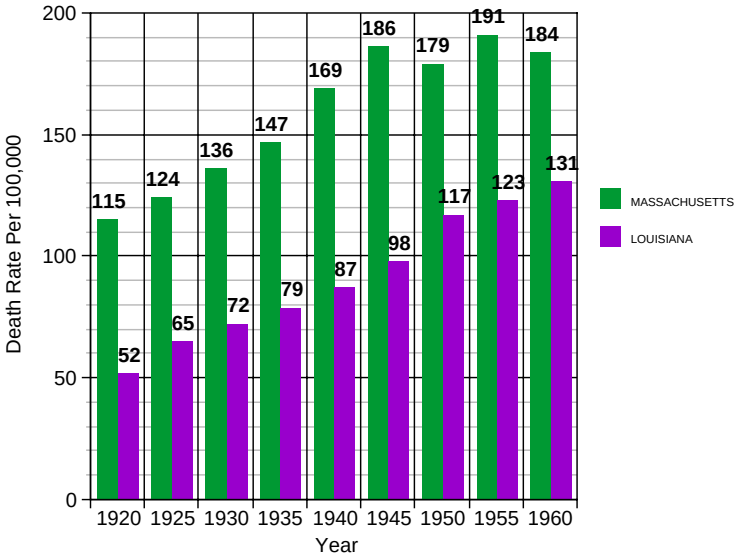

Fig. 7. US white resident total cancer death rates for Massachusetts (97.6% elect.) and Louisiana (48.9% elect.) by year.

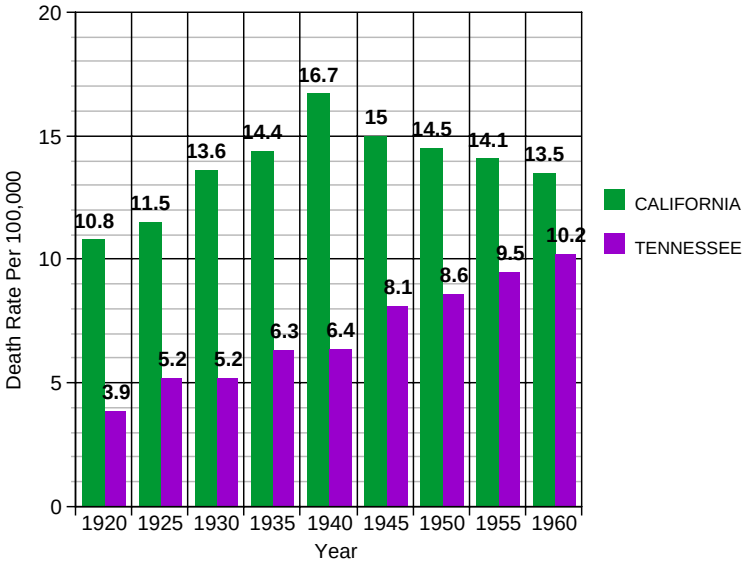

Fig. 8. US white resident breast cancer death rates for California (96% elect.) and Tennessee (50% elect.) by year.

shows that in states with low levels of electrification in 1940, the urban diabetes death rates are consistently higher than the rural rates, but are always lower than the urban and rural rates in the high electrification states.

Leukemia

Since the childhood leukemia age peak is strongly associated with residential electrification, it was interesting that the all leukemia death rate correlation was $r = 0.375$; $p = 0.0042$. Most of these deaths are in adults and are of different types of leukemia. A study of amateur radio operators showed a selective excess only of acute myelogenous leukemia [17].

Coronary artery disease and other heart disease

These two cause groups had the same percentage urban excess (33%), and very similar patterns of urban and rural correlation coefficients with residential electrification. The urban correlations were about $r = 0.4$ and rural deaths had correlations of 0.78 and 0.79, respectively. Fig. 10 shows the 1940 resident white coronary artery disease death rates for urban and rural areas of states having greater than 96% of residences electrified and states having less than 50% of residences electrified. Four of the five high electrification states had similar urban and rural total cancer rates, while all the low electrification states had urban rates about twice as high as rural rates. Urban and rural coronary artery death rates were lower in low electrification states than in high electrification states.

Suicide

The urban suicide death rate is about 30% higher than the rural rate. The urban suicide rate is not correlated with residential electrification ($r = 0.077$; $p = 0.299$), but the rural death rate is correlated with 1940 state residential electrification levels ($r = 0.729$; $p = <0.0001$). Fig. 11 shows the 1940 resident white suicide for

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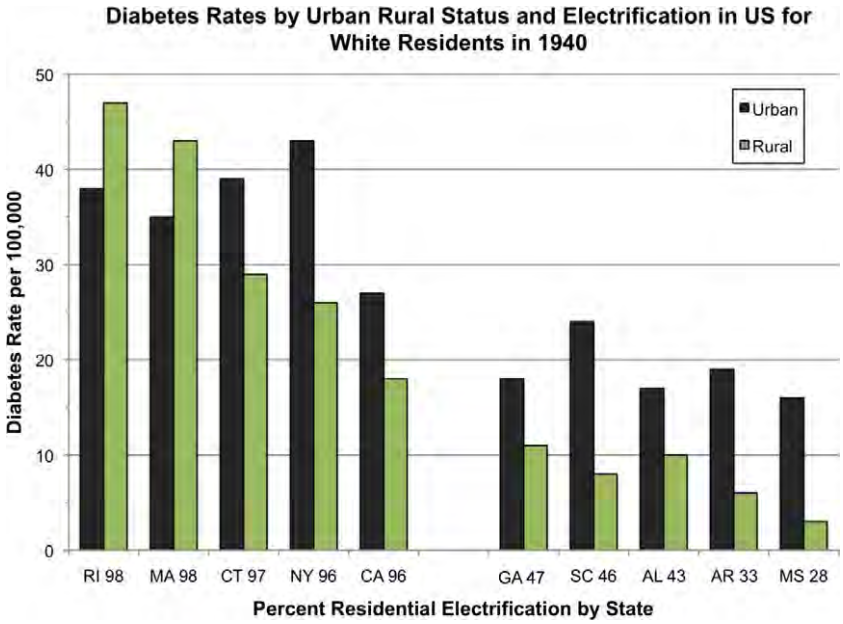

Fig. 9. Total diabetes rates by urban rural status and electrification in the US for white residents in 1940.

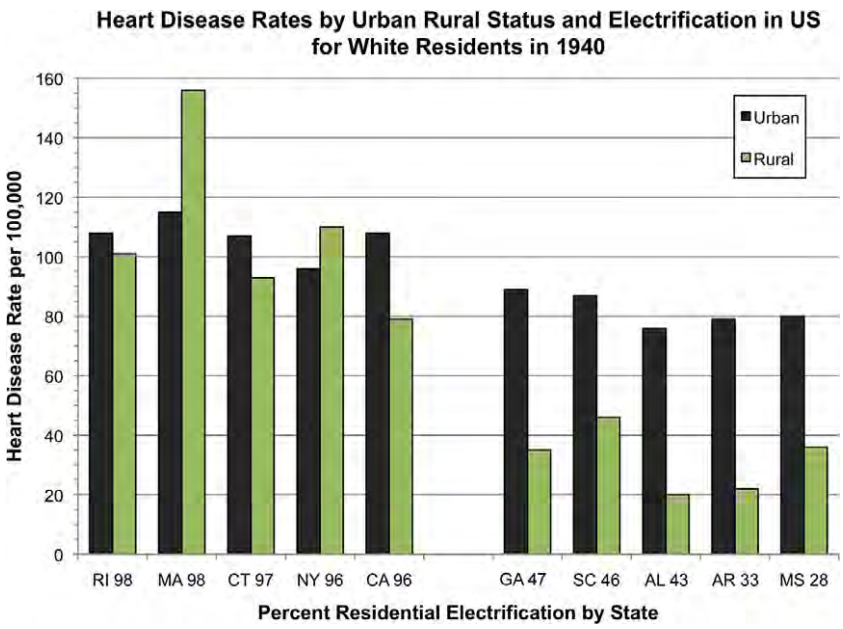

Fig. 10. Total heart disease rates by urban rural status and electrification in the US for white residents in 1940.

urban and rural areas of states having greater than 96% of residences electrified and states having less than 50% of residences electrified. In four of five high electrification states, rural suicide rates are higher than the urban rates. In all of the low electrification states, the urban rate is higher. The rural rates in the high electrification states are higher than the rural rates in the low electrification states. Fig. 12 shows X Y scatter plots for urban and rural suicide by electrification for 48 states. Suicide has been associated with both residential [18] and occupational [19] EMF exposure. Suicide is probably the visible peak of the clinical depression iceberg.

Motor vehicle accidents

Although the mortality rates are similar in urban and rural areas, the correlations with residential electrification levels are different. There is a slight negative correlation ($r = -0.254$) in urban areas and a positive correlation ($r = 0.451$) in rural areas. Since motor vehicle fatality is related to access to a vehicle and to speed. It may be that in the larger cities it was difficult to go fast enough for a fatal accident, and in rural areas especially on farms, a farmer who could afford electrification could also afford a car.

Discussion

When Edison and Tesla opened the Pandora's box of electrification in the 1880s, the US vital registration system was primitive at best, and infectious disease death rates were falling rapidly. City residents had higher mortality rates and shorter life expectancy than rural residents [8]. Rural white males in 1900 had an expectation of life at birth of over 10 years longer than urban residents.

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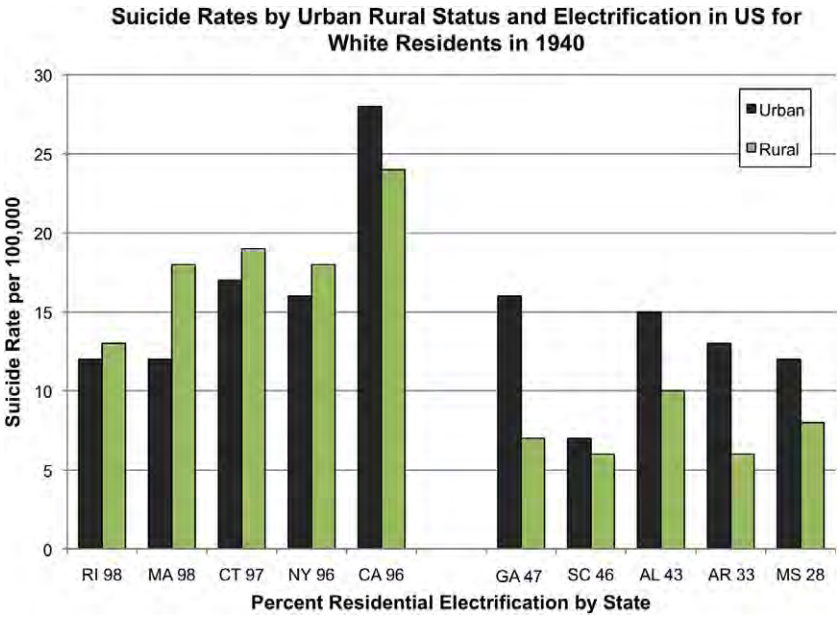

Fig. 11. Total suicide death rates by urban rural status and electrification in the US for white residents in 1940.

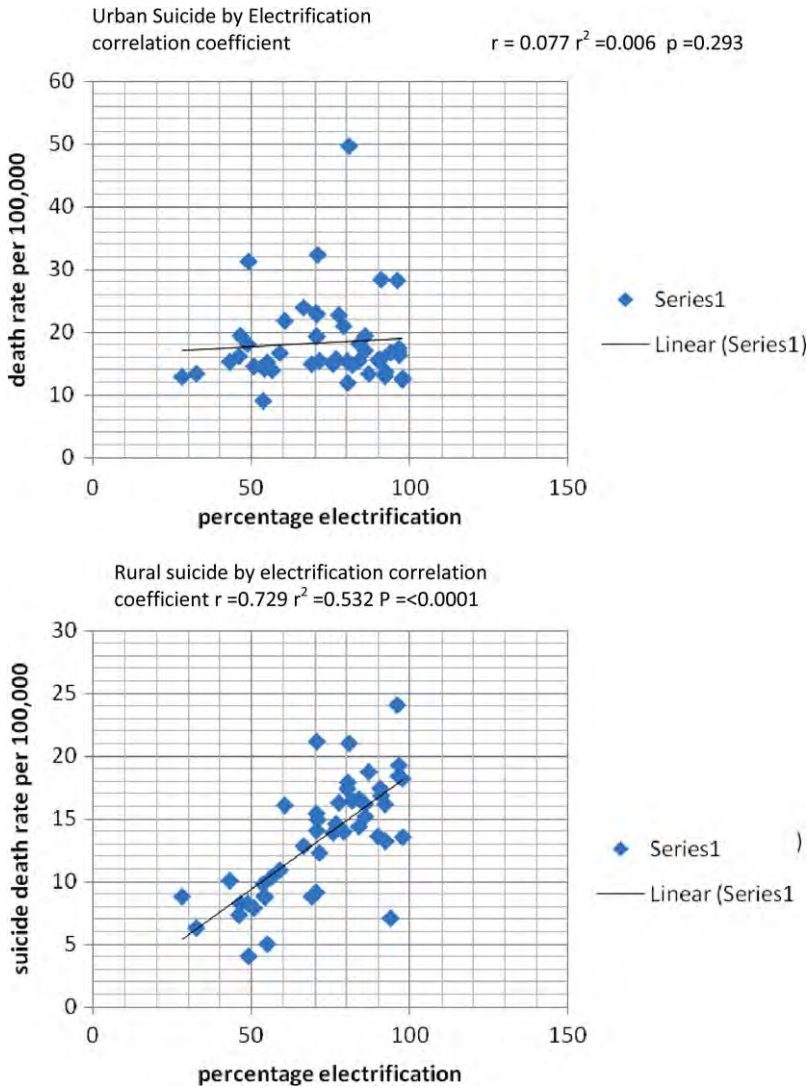

Fig. 12. 1940 US white resident urban rural suicide death rates by state and electrification.

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Although the authors of the 1930 US vital statistics report noted a 58.2% cancer mortality excess in urban areas, it raised no red flags. The census bureau residential electrification data was obviously not linked to the mortality data. Epidemiologists in that era were still concerned with the communicable diseases.

Court Brown and Doll reported [20] the appearance of the childhood leukemia age peak in 1961, forty years after the US vital statistics mortality data on which it was based was available. I reported a cluster of childhood leukemia [21] a decade after it occurred, only because I looked for it. Real time or periodic analysis of national or regional vital statistics data is still only rarely done in the US.

The real surprise in this data set is that cardiovascular disease, diabetes and suicide, as well as cancer seem to be strongly related to level of residential electrification. A community-based epidemiologic study of urban rural differences in coronary heart disease and its risk factors was carried out in the mid 1980s in New Delhi, India and in a rural area 50 km away [22]. The prevalence of coronary heart disease was three times higher in the urban residents, despite the fact that the rural residents smoked more and had higher total caloric and saturated fat intakes. Most cardiovascular disease risk factors were two to three times more common in the urban residents. Rural electrification projects are still being carried out in parts of the rural area which was studied.

It seems unbelievable that mortality differences of this magnitude could go unexplained for over 70 years after they were first reported and 40 years after they were noticed. I think that in the early part of the 20th century nobody was looking for answers. By the time EMF epidemiology got started in 1979 the entire population was exposed to EMFs. Cohort studies were therefore using EMF-exposed population statistics to compute expected values, and case-control studies were comparing more exposed cases to less exposed controls. The mortality from lung cancer in two pack a day smokers is over 20 times that of non-smokers but only three times that of one pack a day smokers. After 1956, the EMF equivalent of a non-smoker ceased to exist in the US. An exception to this is the Amish who live without electricity. Like rural US residents in the 1940s, Amish males in the 1970s had very low cancer and cardiovascular disease mortality rates [23].

If this hypothesis and findings outlined here are even partially true, the explosive recent increase in radiofrequency radiation, and high frequency voltage transients sources, especially in urban areas from cell phones and towers, terrestrial antennas, wi-fi and wi-max systems, broadband internet over power lines, and personal electronic equipment, suggests that like the 20th century EMF epidemic, we may already have a 21st century epidemic of morbidity and mortality underway caused by electromagnetic fields. The good news is that many of these diseases may be preventable by environmental manipulation, if society chooses to.

Conflicts of interest statement

None declared.

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EXHIBIT 10

A New Electromagnetic Exposure Metric: High Frequency Voltage Transients Associated With Increased Cancer Incidence in Teachers in a California School

Samuel Milham, MD, MPH[*,†] **and L. Lloyd Morgan, BS**[‡]

Background *In 2003 the teachers at La Quinta, California middle school complained that they had more cancers than would be expected. A consultant for the school district denied that there was a problem.*
Objectives *To investigate the cancer incidence in the teachers, and its cause.*
Method *We conducted a retrospective study of cancer incidence in the teachers' cohort in relationship to the school's electrical environment.*
Results *Sixteen school teachers in a cohort of 137 teachers hired in 1988 through 2005 were diagnosed with 18 cancers. The observed to expected (O/E) risk ratio for all cancers was 2.78 ($P = 0.000098$), while the O/E risk ratio for malignant melanoma was 9.8 ($P = 0.0008$). Thyroid cancer had a risk ratio of 13.3 ($P = 0.0098$), and uterine cancer had a risk ratio of 9.2 ($P = 0.019$). Sixty Hertz magnetic fields showed no association with cancer incidence. A new exposure metric, high frequency voltage transients, did show a positive correlation to cancer incidence. A cohort cancer incidence analysis of the teacher population showed a positive trend ($P = 7.1 \times 10^{-10}$) of increasing cancer risk with increasing cumulative exposure to high frequency voltage transients on the classroom's electrical wiring measured with a Graham/Stetzer (G/S) meter. The attributable risk of cancer associated with this exposure was 64%. A single year of employment at this school increased a teacher's cancer risk by 21%.*
Conclusion *The cancer incidence in the teachers at this school is unusually high and is strongly associated with high frequency voltage transients, which may be a universal carcinogen, similar to ionizing radiation.* Am. J. Ind. Med. 2008. *© 2008 Wiley-Liss, Inc.*

KEY WORDS: high frequency voltage transients; electricity; dirty power; cancer; school teachers; carcinogen

BACKGROUND

Since the 1979 Wertheimer–Leeper study [Wertheimer and Leeper, 1979] there has been concern that exposure to power frequency (50/60 Hz) EMFs, especially magnetic fields, may contribute to adverse health effects including cancer. Until now, the most commonly used exposure metric has been the time-weighted average of the power-frequency magnetic field. However, the low risk ratios in most studies suggest that magnetic fields might be a surrogate for a more important metric. In this paper we present evidence that a

Abbreviations: EMF, electromagnetic fields; O, observed cases; E, expected cases; O/E, risk ratio; p, probability; Hz, Hertz or cycles per second; OSHA, Occupational Safety and Health Administration; OCMAP, occupational mortality analysis program; AM, amplitude modulation; GS units, Graham/Stetzer units; G/S meter, Graham/Stetzer meter; MS II, Microsurge II meter; mG, milligauss; EKG, electrocardiogram; LQMS, La Quinta Middle School.

Washington State Department of Health, Tumwater, Washington
[†]Retired.
[‡]Retired Electronic Engineer.
*Correspondence to: Samuel Milham, 2318 Gravelly Beach Loop NW, Olympia, WA 98502. E-mail: smilham2@comcast.net

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new exposure metric, high frequency voltage transients existing on electrical power wiring, is an important predictor of cancer incidence in an exposed population.

The new metric, GS units, used in this investigation is measured with a Graham/Stetzer meter (G/S meter) also known as a Microsurge II meter (MS II meter), which is plugged into electric outlets [Graham, 2005]. This meter displays the average rate of change of these high frequency voltage transients that exist everywhere on electric power wiring. High frequency voltage transients found on electrical wiring both inside and outside of buildings are caused by an interruption of electrical current flow. The electrical utility industry has referred to these transients as "dirty power."

There are many sources of "dirty power" in today's electrical equipment. Examples of electrical equipment designed to operate with interrupted current flow are light dimmer switches that interrupt the current twice per cycle (120 times/s), power saving compact fluorescent lights that interrupt the current at least 20,000 times/s, halogen lamps, electronic transformers and most electronic equipment manufactured since the mid-1980s that use switching power supplies. Dirty power generated by electrical equipment in a building is distributed throughout the building on the electric wiring. Dirty power generated outside the building enters the building on electric wiring and through ground rods and conductive plumbing, while within buildings, it is usually the result of interrupted current generated by electrical appliances and equipment.

Each interruption of current flow results in a voltage spike described by the equation $V = L \times di/dt$, where V is the voltage, L is the inductance of the electrical wiring circuit and di/dt is the rate of change of the interrupted current. The voltage spike decays in an oscillatory manner. The oscillation frequency is the resonant frequency of the electrical circuit. The G/S meter measures the average magnitude of the rate of change of voltage as a function of time (dV/dT). This preferentially measures the higher frequency transients. The measurements of dV/dT read by the meter are defined as GS (Graham/Stetzer) units.

The bandwidth of the G/S meter is in the frequency range of these decaying oscillations. Figure 1 shows a two-channel oscilloscope display. One channel displays the 60 Hz voltage on an electrical outlet while the other channel with a 10 kHz hi-pass filter between the oscilloscope and the electrical outlet, displays the high frequency voltage transients on the same electrical outlet [Havas and Stetzer, 2004, reproduced with permission].

Although no other published studies have measured high frequency voltage transients and risk of cancer, one study of electric utility workers exposed to transients from pulsed

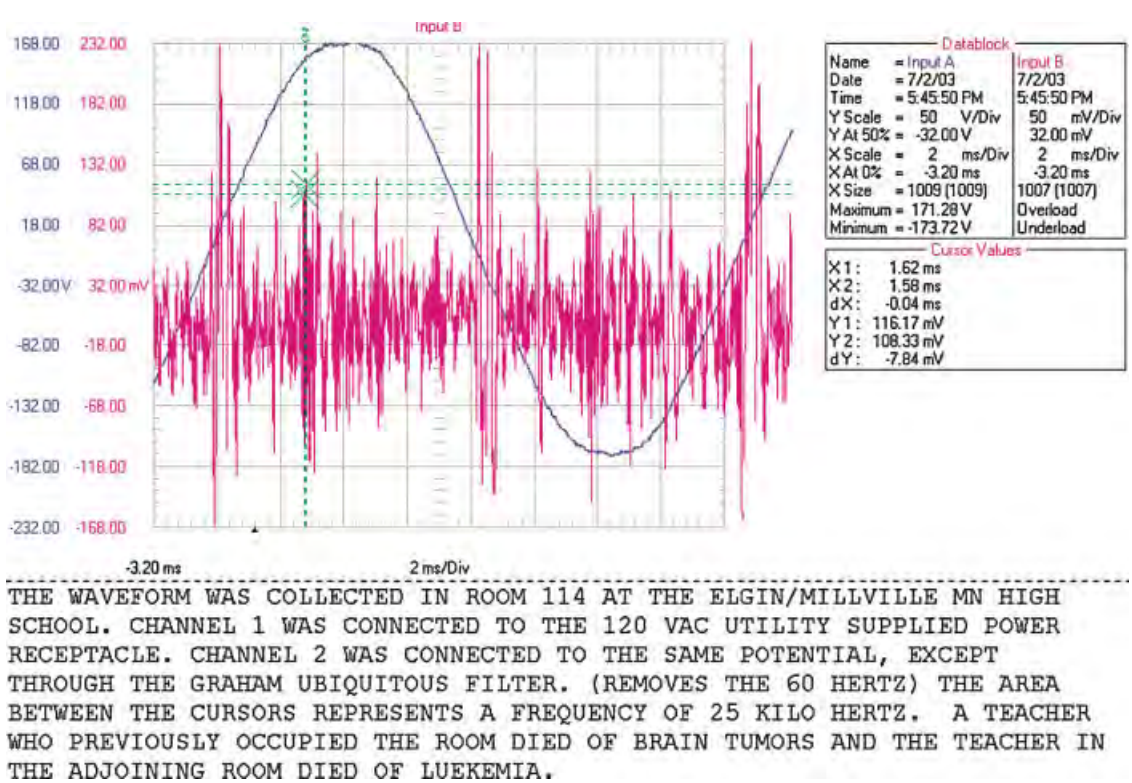

FIGURE 1. Oscilloscope display of dirty power: 60 Hz electrical power (channel 1) with concurrent high frequency voltage transients (channel 2). A 10 kHz hi-pass filter was used on channel 2 in order to filter out the 60 Hz voltage and its harmonics. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

electromagnetic fields found an increased incidence of lung cancer among exposed workers [Armstrong et al., 1994].

INTRODUCTION

In February 2004, a Palm Springs, California newspaper, The Desert Sun, printed an article titled, ''Specialist discounts cancer cluster at school,'' in which a local tumor registry epidemiologist claimed that there was no cancer cluster or increased cancer incidence at the school [Perrault, 2004]. An Internet search revealed that the teacher population at La Quinta Middle School (LQMS) was too small to generate the 11 teachers with cancer who were reported in the article. The school was opened in 1988 with 20 teachers hired that year. For the first 2 years, the school operated in three temporary buildings, one of which remains. In 1990, a newly constructed school opened. In 2003, the teachers complained to school district management that they believed that they had too many cancers. Repeated requests to the school administration for physical access to the school and for teachers' information were denied. We contacted the teachers, and with their help, the cancers in the group were characterized. One teacher suggested using yearbooks to develop population-at-risk counts for calculating expected cancers. We were anxious to assess the electrical environment at the school, since elevated power frequency magnetic field exposure with a positive correlation between duration of exposure and cancer incidence had been reported in first floor office workers who worked in strong magnetic fields above three basement-mounted 12,000 V transformers [Milham, 1996]. We also wanted to use a new electrical measurement tool, the Graham/Stetzer meter, which measures high frequency voltage transients.

The Graham/Stetzer Microsurge II meter measures the average rate of change of the transients in Graham/Stetzer units (GS units). Anecdotal reports had linked dirty power exposure with a number of illnesses [Havas and Stetzer, 2004]. We decided to investigate whether power frequency magnetic field exposure or dirty power exposure could explain the cancer increase in the school teachers.

METHODS

After the school administration (Desert Sands Unified School District) had refused a number of requests to assist in helping us evaluate the cancers reported by the teachers, we were invited by a teacher to visit the school after hours to make magnetic field and dirty power measurements. During that visit, we noted that, with the exception of one classroom near the electrical service room, the classroom magnetic field levels were uniformly low, but the dirty power levels were very high, giving many overload readings. When we reported this to Dr. Doris Wilson, then the superintendent of schools (retired December, 2007), one of us (SM) was threatened with prosecution for ''unlawful.. trespass,'' and the teacher who had invited us into the school received a letter of reprimand. The teachers then filed a California OSHA complaint which ultimately lead to a thorough measurement of magnetic fields and dirty power levels at the school by the California Department of Health Services which provided the exposure data for this study. They also provided comparison dirty power data from residences and an office building, and expedited tumor registry confirmation of cancer cases.

Classrooms were measured at different times using 3 meters: an FW Bell model 4080 tri-axial Gaussmeter, a Dexsil 310 Gaussmeter, and a Graham-Stetzer (G/S) meter. The Bell meter measures magnetic fields between 25 and 1,000 Hz. The Dexsil meter measures magnetic fields between 30 and 300 Hz. The G/S meter measures the average rate of change of the high frequency voltage transients between 4 and 150 KHz.

All measurements of high frequency voltage transients were made with the G/S meter. This meter was plugged into outlets, and a liquid crystal display was read. All measurements reported were in GS units. The average value was reported where more than one measurement was made in a classroom.

We measured seven classrooms in February 2005 using the Bell meter and the G/S meter. Later in 2005, the teachers measured 37 rooms using the same meters. On June 8, 2006, electrical consultants for the school district and the California Department of Health Services (Dr. Raymond Neutra) repeated the survey using the G/S meter and a Dexsil 320 Gaussmeter, measuring 51 rooms. We used results of this June 8, 2006 sampling in our exposure calculations, since all classrooms were sampled, multiple outlets per room were sampled, and an experienced team did the sampling. Additionally, GS readings were taken at Griffin Elementary school near Olympia, Washington, and Dr. Raymond Neutra provided GS readings for his Richmond California office building and 125 private California residences measured in another Northern California study.

All the cancer case information was developed by personal, telephone, and E-mail contact with the teachers or their families without any assistance from the school district. The local tumor registry verified all the cancer cases with the exception of one case diagnosed out of state and the two cases reported in 2007. The out-of state case was verified by pathologic information provided by the treating hospital. The teachers gathered population-at-risk information (age at hire, year of hire, vital status, date of diagnosis, date of death, and termination year) from yearbooks and from personal contact. The teachers also provided a history of classroom assignments for all teachers from annual classroom assignment rosters (academic years 1990–1991 to 2006–2007) generated by the school administration. The school administration provided a listing of school employees, including

the teachers, to the regional tumor registry after the teachers involved the state health agency by submitting an OSHA complaint. The information we obtained anecdotally from the teachers, yearbooks, and classroom assignment rosters was nearly identical to that given to the tumor registry. None of the cancer cases were ascertained initially through the cancer registry search.

Published cancer incidence rates by age, sex, and race for all cancers, as well as for malignant melanoma, thyroid, uterine, breast, colon, ovarian cancers, and non-Hodgkin's lymphoma (NHL) were obtained from a California Cancer Registry publication [Kwong et al., 2001]. We estimated the expected cancer rate for each teacher by applying year, age, sex, and race-specific cancer incidence rates from hire date until June 2007, or until death. We then summed each teacher's expected cancer rate for the total cohort.

Using the California cancer incidence data, the school teacher data, and the GS exposure data, we calculated cancer incidence and risks. A replicate data set was sent to Dr. Gary Marsh and to Mike Cunningham at the University of Pittsburgh School of Public Health for independent analysis using OCMAP software. We calculated cancer risk ratios by duration of employment and by cumulative GS unit-years of exposure. We calculated an attributable risk percent using the frequencies of total observed and expected cancers, and performed trend tests [Breslow and Day, 1987] for cancer risk versus duration of employment and cumulative GS unit-years of exposure. Poisson P values were calculated using the Stat Trek website (Stat Trek, 2007). We also performed a linear regression of cancer risk by duration of employment in years and by time-weighted exposure in GS unit-years.

Since neither author had a current institutional affiliation, institutional review board approval was not possible. The teachers requested the study, and their participation in the study was both voluntary and complete. All the active teachers at the school signed the Cal OSHA request. The authors fully explained the nature of the study to study participants and offered no remuneration to the teachers for participation in the study. The authors maintained strict confidentiality of all medical and personal information provided to us by the teachers, and removed personal identifiers from the data set which was analyzed by the University of Pittsburgh. Possession of personal medical information was limited to the two authors. No patient-specific information was obtained from the tumor registry. With the individual's permission we provided the registry with case information for a teacher with malignant melanoma diagnosed out of state. The exposure information was provided by the California Department of Health Services. The basic findings of the study were presented to the Desert Sands Unified School District School Board and at a public meeting arranged by the teachers.

RESULTS

Electrical Measurements

In our seven-room survey of the school in 2005, magnetic field readings were as high as 177 mG in a classroom adjacent to the electrical service room. A number of outlets had overload readings with the G/S meter. Magnetic fields were not elevated (>3.0 mG) in the interior space of any of the classrooms except in the classroom adjacent to the electrical service room, and near classroom electrical appliances such as overhead transparency projectors. There was no association between the risk of cancer and 60 Hz magnetic field exposures in this cohort, since the classroom magnetic field exposures were the same for teachers with and without cancer (results not shown).

This school had very high GS readings and an association between high frequency voltage transient exposure in the teachers and risk of cancer. The G/S meter gives readings in the range from 0 to 1,999 GS units. The case school had 13 of 51 measured rooms with at least one electrical outlet measuring "overload" (≥2,000 GS units). These readings were high compared to another school near Olympia Washington, a Richmond California office building, and private residences in Northern California (Table I). Altogether, 631 rooms were surveyed for this study. Only 17 (2.69%) of the 631 rooms had an "overload" (maximum, ≥2,000 GS units) reading. Applying this percentage to the 51 rooms surveyed at the case school, we would expect 1.4 rooms at the school to have overload GS readings ($0.0269 \times 51 = 1.37$). However, thirteen rooms (25%) measured at the case school had "overload" measurements above the highest value (1,999 GS units) that the G/S meter can

TABLE I. Graham/Stetzer Meter Readings: Median Values in Schools, Homes and an Office Building

Place	Homes	Office bldg	Olympia WA School	LQMS	Total
No. of rooms surveyed	500	39	41	51	531
Median GS units	159	210	160	750	<270[a]
Rooms with overload GS units (≥2,000)	4	0	0	13*	17

[a]Excludes homes as specific room data was not available.
*$P = 3.14 \times 10^{-9}$.

TABLE II. Risk of Cancer by Type Among Teachers at La Quinta Middle School

Cancer	Observed	Expected	Risk ratio (O/E)	*P*-value
All cancers	18	6.51	2.78*	0.000098
Malignant melanoma	4	0.41	9.76*	0.0008
Thyroid cancer	2	0.15	13.3*	0.011
Uterus cancer	2	0.22	9.19*	0.019
Female breast cancer	2	1.5	1.34	0.24
All cancers less melanoma	14	6.10	2.30*	0.0025

*$P \leq 0.05$.

measure. This is a highly statistically significant excess over expectation (Poisson $P = 3.14 \times 10^{-9}$).

We noticed AM radio interference in the vicinity of the school. A teacher also reported similar radio interference in his classroom and in the field near his ground floor classroom. In May 2007, he reported that 11 of 15 outlets in his classroom overloaded the G/S meter. An AM radio tuned off station is a sensitive detector of dirty power, giving a loud buzzing noise in the presence of dirty power sources even though the AM band is beyond the bandwidth of the G/S meter.

Cancer Incidence

Three more teachers were diagnosed with cancer in 2005 after the first 11 cancer diagnoses were reported, and another former teacher (diagnosed out-of-state in 2000) was reported by a family member employed in the school system. One cancer was diagnosed in 2006 and two more in 2007. In the years 1988–2005, 137 teachers were employed at the school. The 18 cancers in the 16 teachers were: 4 malignant melanomas, 2 female breast cancers, 2 cancers of the thyroid, 2 uterine cancers and one each of Burkitt's lymphoma (a type of non-Hodgkins lymphoma), polycythemia vera, multiple myeloma, leiomyosarcoma and cancer of the colon, pancreas, ovary and larynx. Two teachers had two primary cancers each: malignant melanoma and multiple myeloma, and colon and pancreatic cancer. Four teachers had died of cancer through August 2007. There have been no non-cancer deaths to date.

The teachers' cohort accumulated 1,576 teacher-years of risk between September 1988 and June 2007 based on a 12-month academic year. Average age at hire was 36 years. In 2007, the average age of the cohort was 47.5 years.

When we applied total cancer and specific cancer incidence rates by year, age, sex, race, and adjusted for cohort ageing, we found an estimate of 6.5 expected cancers, 0.41 melanomas, 0.15 thyroid cancers, 0.22 uterine cancers, and 1.5 female breast cancers (Table II). For all cancers, the risk ratio (Observed/Expected = 18/6.5) was 2.78 ($P = 0.000098$, Poisson test); for melanoma, (O/E = 4/0.41) was 9.8 ($P = 0.0008$, Poisson test); for thyroid cancer (O/E = 2/0.15) was 13.3 ($P = 0.0011$, Poisson test); for uterine cancer (O/E = 2/0.22), was 9.19 ($P = 0.019$, Poisson test).

Table III shows the cancer risk among the teachers by duration of employment. Half the teachers worked at the school for less than 3 years (average 1.52 years). The cancer risk increases with duration of employment, as is expected when there is exposure to an occupational carcinogen. The cancer risk ratio rose from 1.7 for less than 3 years, to 2.9 for 3–14 years, to 4.2 for 15+ years of employment. There was a positive trend of increasing cancer incidence with increasing duration of employment ($P = 4.6 \times 10^{-10}$). A single year of employment at this school increases a teacher's risk of cancer by 21%.

Using the June 8, 2006 survey data (Table IV), the cancer risk of a teacher having ever worked in a room with at least one outlet with an overload GS reading (≥2000 GS units) and employed for 10 years or more, was 7.1 ($P = 0.00007$, Poisson test). In this group, there were six teachers diagnosed

TABLE III. Cancer Risk by Duration of Employment

Time at school	Average time	Teachers	% of teachers	Cancer observed	Cancer expected	Risk ratio (O/E)	Poisson p
<3 years	1.52 years	68	49.6	4	2.34	1.72	0.12
3–14 years	7.48 years	56	40.9	9	3.14	2.87*	0.0037
15+ years	16.77 years	12	8.8	5	1.02	4.89*	0.0034
Total		137	100	18	6.51	2.78*	0.000098

Positive trend test (Chi square with one degree of freedom = 38.8, $P = 4.61 \times 10^{-10}$).
*$P \leq 0.05$.

TABLE IV. Cancer in Teachers Who Ever Taught in Classrooms With at Least One Overload GS Reading (≥2000 GS Units) by Duration of Employment

Ever in a room >2,000 GS units	Employed 10 + years	Total teachers	Cancers observed	Cancers expected	Risk ratio (O/E)	Poisson p
Yes	Yes	10	7[a]	0.988	7.1*	0.00007
Yes	No	30	3[a]	0.939	3.2	0.054
Total		40	10	1.93	5.1*	0.00003
No	Yes	19	2	1.28	1.6	0.23
No	No	78	6	3.25	1.8	0.063
Total		97	8	4.56	1.8*	0.047
Grand total		137	18	6.49	2.8*	0.000098

[a]One teacher had two primary cancers.
*$P < 0.05$.

with a total of seven cancers, and four teachers without a cancer diagnosis, who were employed for 10 or more years and who ever worked in one of these rooms. Five teachers had one primary cancer and one teacher had two primary cancers. These teachers made up 7.3% of the teachers' population (10/137) but had 7 cancers or 39% (7/18) of the total cancers. The 10 teachers who worked in an overload classroom for 10 years or more had 7 cancers when 0.99 would have been expected ($P = 6.8 \times 10^{-5}$ Poisson test). The risk ratio for the 8 teachers with cancer and 32 teachers without cancer, who ever worked in a room with an overload GS reading, regardless of the time at the school, was 5.1 ($P = 0.00003$, Poisson test). The risk ratio for 8 teachers with cancer and 89 teachers without cancer who never worked in a room with an overload G-S reading was 1.8 ($P = 0.047$, Poisson test). Teachers who never worked in an overload classroom also had a statistically significantly increased risk of cancer.

A positive dose-response was seen between the risk of cancer and the cumulative GS exposure (Table V). Three categories of cumulative GS unit-years of exposure were selected: <5,000, 5,000 to 10,000, and more than 10,000 cumulative GS unit-years. We found elevated risk ratios of 2.0, 5.0, and 4.2, respectively, all statistically significant, for each category. There was a positive trend of increasing cancer incidence with increasing cumulative GS unit-years of exposure ($P = 7.1 \times 10^{-10}$). An exposure of 1,000 GS unit-years increased a teacher's cancer risk by 13%. Working in a room with a GS overload (≥2,000 GS units) for 1 year increased cancer risk by 26%.

An attributable risk percentage was calculated: (observed cancers-expected cancers)/observed cancers = (18−6.51)/18 = 63.8%.

The fact that these cancer incidence findings were generated by a single day of G/S meter readings made on June 8, 2006 suggests that the readings were fairly constant over time since the school was built in 1990. For example, if the 13 classrooms which overloaded the meter on June 8, 2006 were not the same since the start of the study and constant throughout, the cancer risk of teachers who ever worked in the overload rooms would have been the same as the teachers who never worked in an overload room.

Although teachers with melanoma and cancers of the thyroid, and uterus, had very high, statistically significant risk ratios, there was nothing exceptional about their age at hire, duration of employment, or cumulative GS exposure. However, thyroid cancer and melanoma had relatively short latency times compared to the average latency time for all 18 cancers. The average latency time between start of

TABLE V. Observed and Expected Cancers by Cumulative GS Exposure (GS Unit-Years)

Exposure group	<5,000 GS unit-years	5,000 to 10,000	>10,000 GS unit-years	Total
Average GS unit-years	914	7,007	15,483	
Cancers obs.	9	4	5	18
Cancers exp.	4.507	0.799	1.20	6.49
Risk ratio (O/E)	2.01*	5.00*	4.17*	2.78*
Poisson p	0.0229	0.0076	0.0062	0.000098

Positive trend test (Chi square with one degree of freedom = 38.0, $P = 7.1 \times 10^{-10}$).
*$P < 0.05$.

employment at the school and diagnosis for all cancers was 9.7 years. The average latency time for thyroid cancer was 3.0 years and for melanoma it was 7.3 years (with three of the four cases diagnosed at 2, 5, and 5 years).

An independent analysis of this data set by the University of Pittsburgh School of Public Health using OCMAP software supported our findings.

DISCUSSION

Because of access denial, we have no information about the source, or characterization of the high frequency voltage transients. We can assume, because the school uses metal conduit to contain the electrical wiring, that any resultant radiated electric fields from these high frequency voltage transients would radiate mainly from the power cords and from electrical equipment using the power cords within a classroom.

The school's GS readings of high frequency voltage transients are much higher than in other tested places (Table I). Also, teachers in the case school who were employed for over 10 years and who had ever worked in a room with an overload GS reading had a much higher rate of cancer. They made up 7.3% of the cohort but experienced 39% of all cancers.

The relatively short latency time of melanoma and thyroid cancers suggests that these cancers may be more sensitive to the effects of high frequency voltage transients than the other cancers seen in this population.

In occupational cohort studies, it is very unusual to have a number of different cancers with an increased risk. An exception to this is that cohorts exposed to ionizing radiation show an increased incidence of a number of different cancers. The three cancers in this cohort with significantly elevated incidence, malignant melanoma, thyroid cancer and uterine cancer, also have significantly elevated incidence in the large California school employees cohort [Reynolds et al., 1999].

These cancer risk estimates are probably low because 23 of the 137 members of the cohort remain untraced. Since exposure was calculated based on 7 days a week for a year, this will overstate the actual teachers' exposure of 5 days a week for 9 months a year.

We could not study field exposures in the classrooms since we were denied access to the school. We postulate that the dirty power in the classroom wiring exerted its effect by capacitive coupling which induced electrical currents in the

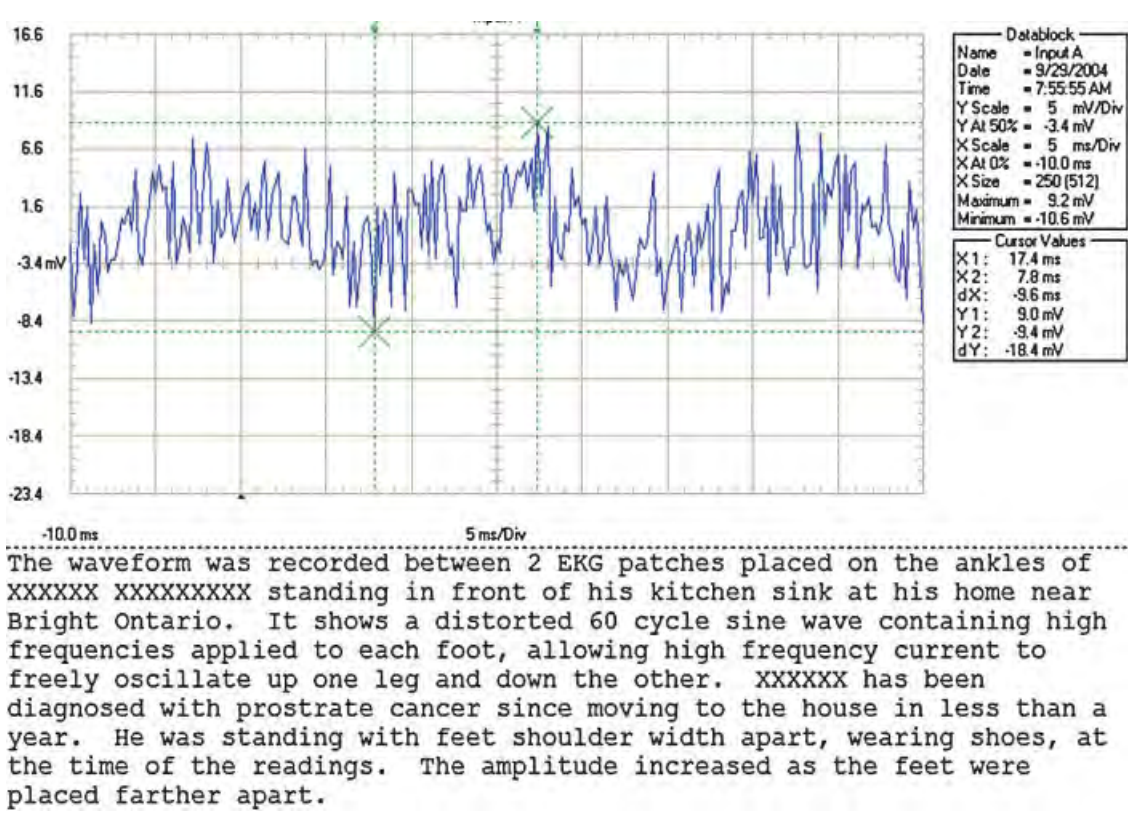

FIGURE 2. Oscilliscope display of 60 Hz current distorted with high frequencies taken between EKG patches applied to the ankles of a man standing with shoes on at a kitchen sink. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

teachers' bodies. The energy that is capacitively coupled to the teachers' bodies is proportional to the frequency. It is this characteristic that highlights the usefulness of the G/S meter. High frequency dirty power travels along the electrical distribution system in and between buildings and through the ground. Humans and conducting objects in contact with the ground become part of the circuit. Figure 2 [Havas and Stetzer, 2004, reproduced with permission] shows an oscilloscope tracing taken between EKG patches on the ankles of a man wearing shoes, standing at a kitchen sink. The 60 Hz sine wave is distorted by high frequencies, which allows high frequency currents to oscillate up one leg and down the other between the EKG patches.

Although not demonstrated in this data set, dirty power levels are usually higher in environments with high levels of 60 Hz magnetic fields. Many of the electronic devices which generate magnetic fields also inject dirty power into the utility wiring. Magnetic fields may, therefore, be a surrogate for dirty power exposures. In future studies of the EMF-cancer association, dirty power levels should be studied along with magnetic fields.

The question of cancer incidence in students who attended La Quinta Middle School for 3 years has not been addressed.

CONCLUSION

The cancer incidence in the teachers at this school is unusually high and is strongly associated with exposure to high frequency voltage transients. In the 28 years since electromagnetic fields (EMFs) were first associated with cancer, a number of exposure metrics have been suggested. If our findings are substantiated, high frequency voltage transients are a new and important exposure metric and a possible universal human carcinogen similar to ionizing radiation.

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EXHIBIT 11

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Perception-based protection from low-frequency sounds may not be enough

Alec N. Salt[a)]
Jeffery T. Lichtenhan[b)]
Department of Otolaryngology, Washington University School of Medicine, St. Louis, MO, 63110, USA.

Hearing and perception in the mammalian ear are mediated by the inner hair cells (IHC). IHCs are fluid-coupled to mechanical vibrations and have been characterized as velocity-sensitive, making them quite insensitive to low-frequency sounds. But the ear also contains more numerous outer hair cells (OHC), which are not fluid coupled and are characterized as displacement sensitive. The OHCs are more sensitive than IHCs to low frequencies and respond to very low-frequency sounds at levels below those that are perceived. OHC are connected to the brain by type II afferent fibers to networks that may further attenuate perception of low frequencies. These same pathways are also involved in alerting and phantom sounds (tinnitus). Because of these anatomic configurations, low-frequency sounds that are not perceived may cause influence in ways that have not yet been adequately studied. We present data showing that the ear's response to low-frequency sounds is influenced by the presence of higher-frequency sounds such as those in the speech frequency range, with substantially larger responses generated when higher-frequency components are absent. We conclude that the physiological effects of low-frequency sounds are more complex than is widely appreciated. Based on this knowledge, we have to be concerned that sounds that are not perceived are clearly transduced by the ear and may still affect people in ways that have yet to be fully understood.

1 INTRODUCTION

The manner in which the inner ear responds to very low-frequency sounds is still not well characterized. The pertinent anatomy and physiology is diagrammed in Figure 1. When sounds enter the cochlea they stimulate different regions, depending on the frequency, or tone, of the sound. The basilar membrane has a low-pass filter characteristic, such that the basal turn can respond to all frequencies while higher frequency components are progressively filtered out towards the apex. The apical regions are where mechanical-to-electrical transduction occurs for

[a)]email: salta@ent.wustl.edu
[b)] email: lichtenhanj@ent.wustl.edu

only the low-frequency components of acoustic sound. Superimposed on the passive mechanical filtering are cochlear hair cells that amplify low-level sounds for detection. This mechanical amplification is performed by the outer hair cells (OHC) so that the signal can be detected by the inner hair cells (IHC) that are the true sensory cells of the ear. The IHCs are densely innervated by type I primary afferent fibers, which is why it is generally accepted that hearing and perception are mediated by IHC activity. An important feature of the IHCs is that their sensory stereocilia ("hairs") do not contact the overlying tectorial membrane. They are therefore fluid coupled to the mechanical vibrations and have been characterized as velocity-sensitive, or ac-coupled[1]. That is to say, high-frequency vibrations stimulate IHCs but low-frequency vibration is attenuated, making IHCs insensitive to very low-frequency sounds. This contributes to the insensitivity of mammalian hearing to very low-frequency sounds and infrasound, requiring high levels to be heard. However, this is not to say that the ear itself is insensitive to very low-frequency sounds and infrasound. The stereocilia of the OHC are directly coupled to the tectorial membrane so they receive mechanical input in a displacement-sensitive manner. In early studies, Békésy[2] showed that displacements of the basilar membrane by trapezoidal stimuli generated trapezoidal response potentials that were sustained for the duration of the stimulus. The OHCs are thus dc-coupled to input and therefore highly sensitive to low-frequency stimulation. The OHC are, in part, connected to the brain by the type II afferents which make up approximately 5% of the afferent fibers in the auditory nerve. Each type II fiber contacts multiple OHC. Although no one has ever reported recordings from type II fibers with infrasound stimulation, Schermuly and Klinke[3] have shown that similar fibers in the bird, that innervate multiple hair cells, are highly sensitive to infrasound input.

Studies have suggested that the perception of low-frequency sounds by humans is influenced by the presence of higher-frequency sounds. Krahé[4,5] found that the perception of low-frequency noise alone was rated to be more annoying than low-frequency noise presented with higher-frequency sounds. These studies suggest that the perceptual consequences of low-frequency sounds should not be studied without considering the combined effects of higher-frequency sounds such as those in the range of speech.

Here we report objective measures from the low-frequency regions of the cochlea. They offer support for hypotheses that the influence of high-frequency sounds on the perception of low-frequency sounds is rooted in a cochlear mechanism in which the OHCs near the apex are stimulated by low frequency sounds more intensely than previously understood.

2 METHODS

Stimulus generation and response acquisition were performed using Tucker-Davis System 3 hardware controlled by custom-written software on a personal computer. Sound stimuli were delivered in a closed system using a hollow ear bar between the transducers and the external ear canal of anesthetized guinea pigs. Full details of stimulus delivery and presentation are given elsewhere[6]. Cochlear responses were measured from 500 mM KCl-filled glass pipettes inserted into endolymph of the cochlear third turn and connected through a high-input impedance electrometer. All procedures were approved by the Animal Studies Committee of Washington University under protocols 20070147 and 20100135.

3 RESULTS

3.1 Suppression of Infrasonic-Tone-Response by a Higher-Frequency Tone

The response of the apical, low-frequency regions of the cochlea to low-frequency sounds is complex. Responses are large when the sound is dominated by low frequencies and become smaller when higher-frequency sounds are present. In Figure 2, the response to an infrasonic (5Hz, 90 dB SPL) tone was recorded from endolymph of the third cochlear turn while a higher frequency (500 Hz) tone was superimposed after 1 second. As the level of the 500 Hz tone was increased from 50 to 80 dB SPL, the response to the 5 Hz stimulus was dramatically suppressed. Suppression of the infrasound response occurred at stimulus levels well below those that saturate the mechanical-to-electrical hair cell transducers of the inner ear (Figure 2, lower left panel), meaning that the suppression is not a result of transducer saturation.

3.2 Low-Pass Noise: A Variant of Infrasonic-Tone-Response Suppression

Responses to low-pass filtered noise were measured with electrodes located in the basal turn (sensitive to high frequency sounds) and in the third cochlear turn (sensitive to low-frequency sounds). All recordings were made with electrodes located in the endolymphatic compartment of the guinea pig inner ear. The sound stimuli used are shown in Figure 3. White noise stimuli were generated and digitally low-pass filtered with a cutoff slope of approximately 55 dB/octave. The noise was digitally generated "frozen noise" so that it had the characteristics of white noise but was exactly repeatable for each of the low-pass filtered conditions, allowing multiple responses to be time-domain averaged. The spectra here were obtained from 20 responses averaged with the noise at a level of 90 dB SPL for the 4 kHz filtered condition. The low-pass cutoff frequency of the filter was varied in half-octave steps from 125 Hz to 4 kHz. Filtered electrical signals sent to the headphone for sound stimulation are shown in the upper panel of Figure 3. These stimuli were delivered by a Sennheiser HD 580 driver and the spectra measured in the canal are shown in the middle panel of Figure 3. For each cutoff frequency the noise levels were measured either with or without filtering the microphone response with a 22 Hz high-pass filter that reduced ambient room noise. The signals were also measured with A-weighting as shown in the lower panel of Figure 3. The noise level for the 125 Hz low-pass filtered condition had an A-weighted level of 56 dB A.

Spectra shown on an expanded frequency scale (0 to 300 Hz) for three simultaneously recorded conditions are shown in Figure 4. Responses measured from the animal are expressed as dB re. 1V where -72 dB represents ~ 250 μV response amplitude. The microphone ear canal measurements of Figure 4, which are the same data as in Figure 3, show that the low-frequency components are indeed frozen, as all measures overlie each other in the 20 – 100 Hz range. The right column of Figure 4 shows the average spectral level over the 12-125 Hz range for each low-pass filter condition. When measured in the basal region of the cochlea, noise with lower frequency cutoff produced larger responses in the low-frequency range. The spectral average over the 12-125 Hz range was approximately 3 dB greater for the 125 Hz cutoff noise than it was with the cutoff set to 2000 Hz, a characteristic that was not present in the simultaneously measured sound levels in the ear canal demonstrating that our inner-ear measures are not an analysis artifact. This same tendency was more pronounced when responses were measured from the third cochlear turn in that the noise with the lowest cutoff frequency generated a substantially larger response. When the response amplitude from the third turn was averaged across the 12-125 Hz range, an approximately 6 dB decline was seen between the 125 Hz and 2000 Hz low-pass filter settings. The results of similar measurements made in 5 animals are summarized in Figure 5. The responses in the low-frequency spectral region (12-125 Hz) were 8.8 dB greater

from the 125 Hz low-pass cutoff noise as compared to that from the 2 kHz or higher cutoff frequencies. In other words, *responses from low-frequency stimulus components were 2-3x greater in amplitude when high-frequency sounds were not present.*

3.3 The Effect of A-Weighting

Although the low-pass filtered noise with a cutoff frequency of 2 kHz or greater was set to 90 dB SPL, the measured sound levels decreased, as expected, for stimuli with lower cutoff frequencies as higher frequency components were filtered out. The decline with cutoff frequency, measured in dB SPL, is shown in the lower panel of Figure 3. The changes as cutoff frequency was varied become even more pronounced when the sound was A-weighted. The A-weighted level of the noise with the cutoff filter set at 125 Hz was 56 dB A.

The low frequency responses of the ear, measured as the average spectral components from 12 – 125 Hz, as a function of noise levels is summarized in the left panel of Figure 6. As level was increased, the response from the 125 Hz low-pass filtered noise was always larger than the 4 kHz low-pass filtered noise. In this plot, the sound level represents how the data were collected, based on the noise level for the 4 kHz low-pass (i.e., wide-band) condition. In the right panel, we provide a comparison of the two noise–band responses corrected by A-weighted levels. There are two major observations that result from the comparison in Figure 6 (right panel):

1) **Low-pass filtered noise with a cutoff frequency of 125 Hz presented at a level of ~43 dB A stimulated the apical regions of the cochlea to the same degree as noise with a low-pass cutoff frequency of 4 kHz (i.e. wide band) at a level of 90 dB A.**
2) **At stimulus levels above 45 dB A, 125 Hz low-pass filtered noise generated larger responses at the apical regions of the cochlea than were generated by ANY level of 4 kHz low-frequency cutoff (i.e. wide band) noise.**

4 DISCUSSION AND CONCLUSIONS

The sensitivity of the apical regions of the cochlea to low-frequency sounds, and the suppressive influence of higher frequency sounds on this response, is confirmed by this study. We have demonstrated that A-weighted noise levels of as low as 45 dB A can stimulate apical regions to the same degree as wide band noise of much higher levels, as high as 90 dB A. This study shows that it cannot be assumed that noise levels as low as 40 dB A are benign and do not cause strong stimulation of the ear. Low-frequency noise around 40 dB A undoubtedly affects the ear. If the noise consists of predominantly low frequencies, then it will induce greater stimulation of the ear than has hitherto been appreciated. The observation that responses to primarily low-frequency noise stimulation are larger and do not saturate to the degree seen when higher-frequency components are present (Figure 6) is in complete agreement to the behavior previously seen with tonal stimuli[7]. The input/output functions of cochlear responses saturated at progressively higher levels for 500 Hz, 50 Hz and 5 Hz tonal stimuli presented in quiet. This means that the largest electrical responses in the apical regions of the cochlea will occur specifically when very low-frequency sound dominates the stimulus and mid-frequency components (200 – 2000 Hz) are absent.

The responses from inside the inner ear reported here may provide a physiologic basis for Krahé's psychoacoustical studies that showed how low-frequency noises with sharp cutoff slopes

are judged to be more annoying than when presented with higher-frequency sounds, as is the case when lower-cutoff slopes are present[4,5]. Although our studies offer support for Krahé's findings, we do not necessarily agree with his interpretation that the annoyance is mediated by primary type I afferent auditory nerve fibers. We have shown empirically that infrasound rarely leads to direct excitation of single-auditory-nerve afferent fibers [7] and there are many other mechanisms that should not be ruled out, including:

1) Stimulation mediated by type II afferent fibers. Type II fibers innervate multiple OHC and connect to multiple cell types of the cochlear nucleus[8] in the brainstem. These pathways may be inhibitory to conscious hearing[9], and may also be linked to alerting and attention pathways.

2) Cochlear Fluids Disturbances. Low-frequency stimulation at non-damaging levels has been shown to result in a localized endolymphatic hydrops – a swelling of the endolymphatic inner-ear compartment – with associated basally-directed endolymph flow[10]. Wit et al. showed that during endolymph volume increases, pressure changes were consistent with a flow through a narrow duct (the ductus reuniens) into a more compliant chamber (the sacculus)[11]. Histologic studies showed that the sacculus is highly compliant and is one of the first structures affected by endolymphatic hydrops[12]. Low-frequency sound exposure could therefore produce saccular disturbance, with symptoms including unsteadiness and disequilibrium. Furthermore, endolymphatic hydrops has been shown to contribute to an occlusion of the cochlear helicotrema which then makes the ear approximately 20 dB more sensitive to very low-frequency sounds[13]. This leads to the possibility of a positive feedback process, with low-frequency stimulation generating hydrops that in turn makes the ear more sensitive to the low-frequency stimulation. In addition to the mechanical disturbance of the saccule caused by endolymphatic hydrops, saccular enlargement also brings the saccular membranes closer and possibly in contact with the stapes footplate which would result in more efficient, direct stimulation of the saccule.

3) Amplitude modulation of sounds in the acoustic range. We have shown that low-frequency sounds that do not directly stimulate the IHCs and primary afferents, can influence the responses of the auditory nerve (i.e., sounds that will be heard) by inducing a biological form of amplitude modulation[7]. This type of modulation occurs within the cochlea and cannot be measured with a sound level meter. Rather, because the low-frequency displacements of the basilar membrane affect the amplification properties of the OHCs, responses to high-frequency sounds are perceived as being modulated in amplitude.

It is well documented that people find noise with prominent low frequency content annoying[4,5,14]. In the context of wind turbine noise it is known that the larger wind turbines can generate high levels of low frequency noise and infrasound[15,16,17,18,19]. The concern arising from the work we report here is that the cochlear apex of people exposed to such low-frequency sounds will be stimulated to a far greater degree than is suggested by their measured A-weighted sound level. The demonstration that sounds in the range of 40 – 45 dB A may be causing intense stimulation of the cochlear apex has not previously been appreciated. This may account for why the influence of low frequency noise on humans is greater than that estimated from spectral measurements and why consideration of noise crest factors is appropriate[20]. The fact that apical stimulation is maximal when mid- and high-frequency components are absent from the sound may also be important to wind turbine noise effects. It is known that people's houses attenuate sound frequencies in the audible range but have little influence, or may even increase infrasound and low-frequency sound levels[21]. Thus, prolonged periods of exposure to wind turbine noise in an otherwise quiet environment (such as a quiet bedroom) seems to represent a condition in which apical stimulation would be maximized. Intense stimulation of the cochlear apex will certainly have some influence on human physiology. On this basis we think that the concept of

"what you can't hear can't hurt you" is false. Similarly, there are potential mechanisms by which low-frequency sounds could influence vestibular physiology which are being ignored by some[22]. Our measurements showing that the ear generates large electrical responses to low-frequency stimulation suggest that the effects of low-frequency sound on people living near wind turbines should not be dismissed by those with little understanding of how low frequency sounds indeed affect the ear[19,21,22]. More research on this topic is necessary to enlighten the scientific, medical, and legal communities, and the public, some of whom are being chronically exposed to these sounds.

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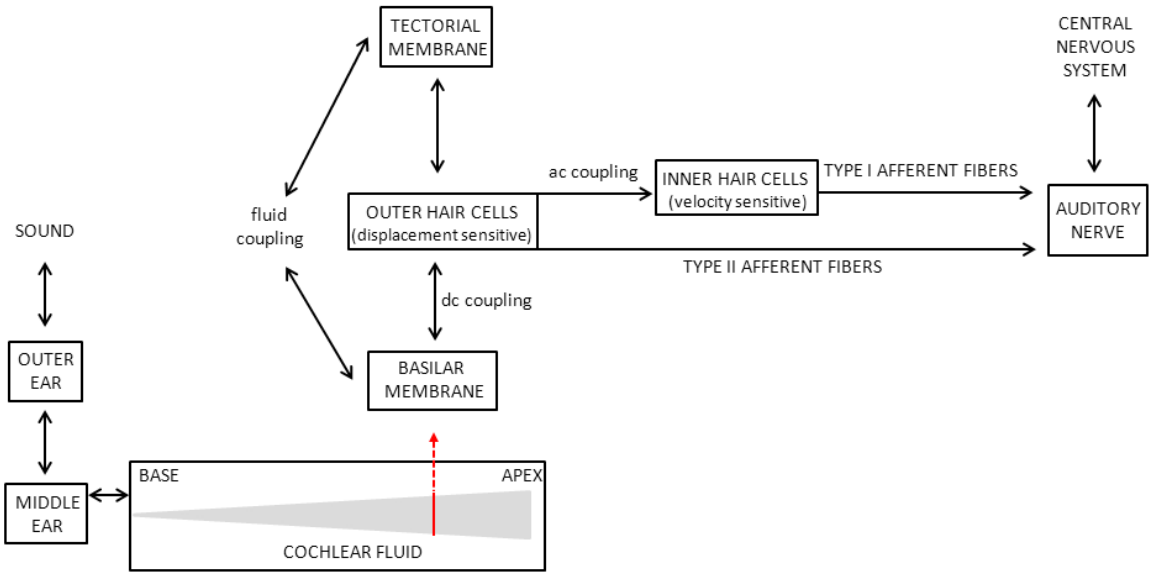

Fig. 1 – Schematic of the auditory periphery. The guinea pig cochlea has 1 row of inner hair cells (IHC) and three rows of outer hair cells (OHC) along its length. The red line and subsequent compartments describe some anatomical and physiological properties of a given segment. After sound propagates through the outer and middle ear, the basilar membrane is set into motion. OHCs in the cochlear apex region respond to low frequencies and are sensitive to the displacement of the basilar membrane motion and are dc-coupled to the sound stimulus. In contrast, the IHC are free within the cochlear fluid causing them to be excited by the velocity of basilar membrane motion, are ac-coupled to the sound stimulus, and are insensitive to low frequency stimulation.

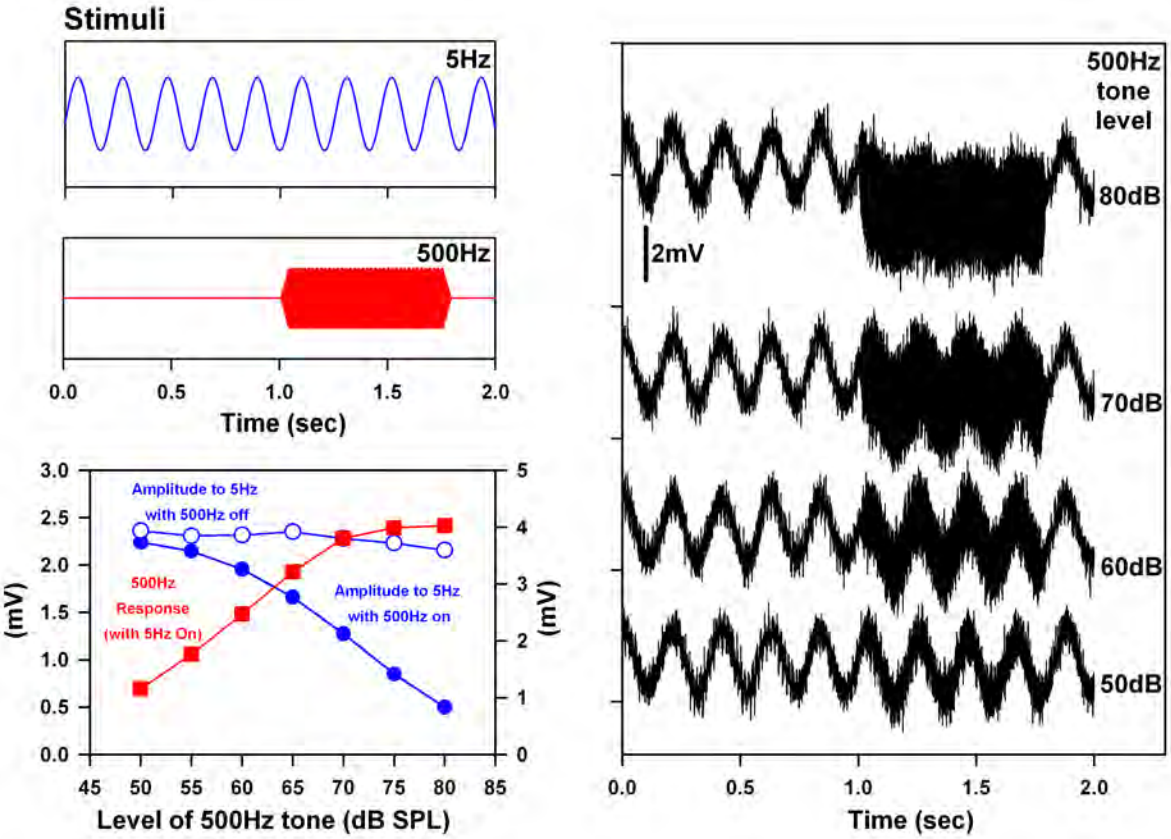

Fig. 2 – Higher-frequency stimuli (500 Hz) suppress the response to a very low-frequency (5 Hz) stimulus. A 500 Hz tone of varied level was superimposed on a 5 Hz, 90 dB stimulus. As the level of the 5 Hz tone was increased, the amplitude of the 5 Hz response declined (right panel).Amplitude measurements (lower left panel) show that 5 Hz stimulation well below saturation (red curve) caused a reduction in response amplitude to the 5 Hz.

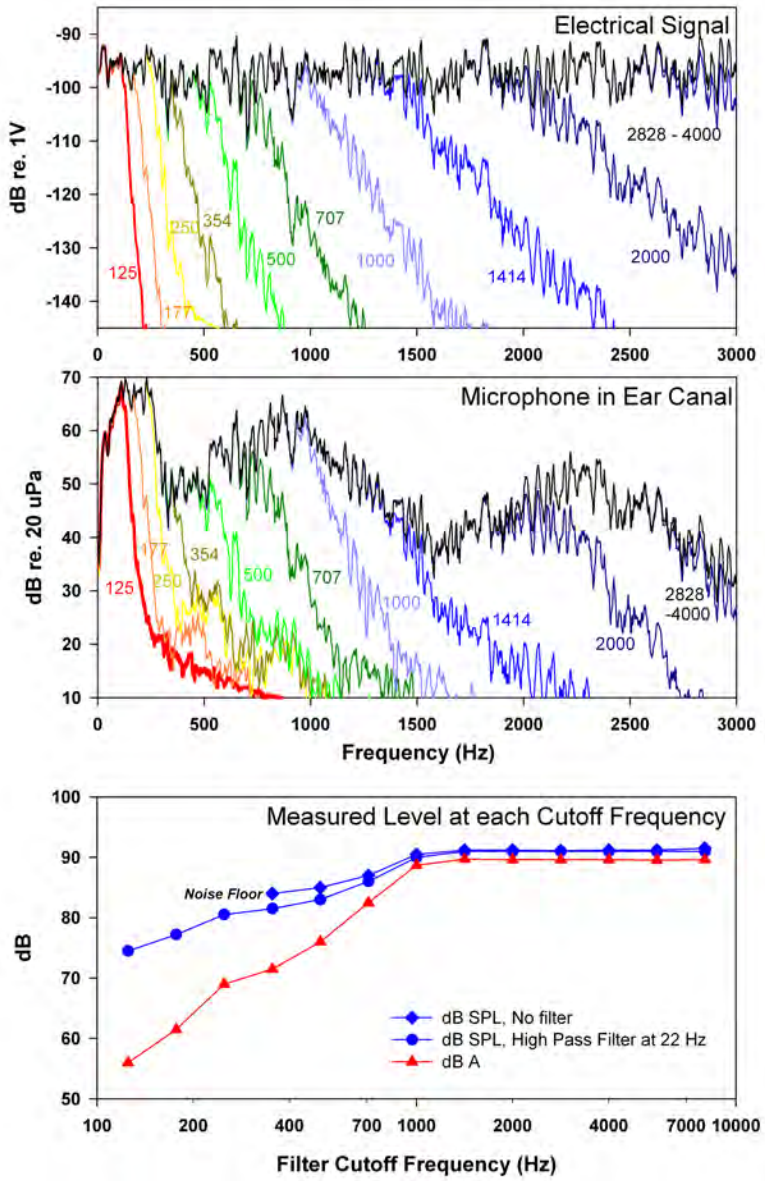

Fig. 3 – Low-pass noise used in this study. The upper panel shows the spectra of low-pass noises with cutoff frequency varied from 125 Hz to 4 kHz. Note that the filter altered the high-frequency component of the noise but had no influence on the low-frequency content (below 100 Hz). The cutoff slope was approximately 55 dB/octave. The middle panel shows the stimuli measured in vivo in the external canal after being delivered by the Sennheiser headphone. The lower panel shows the stimuli with different cutoff frequencies measured in dB SPL and in dB A.

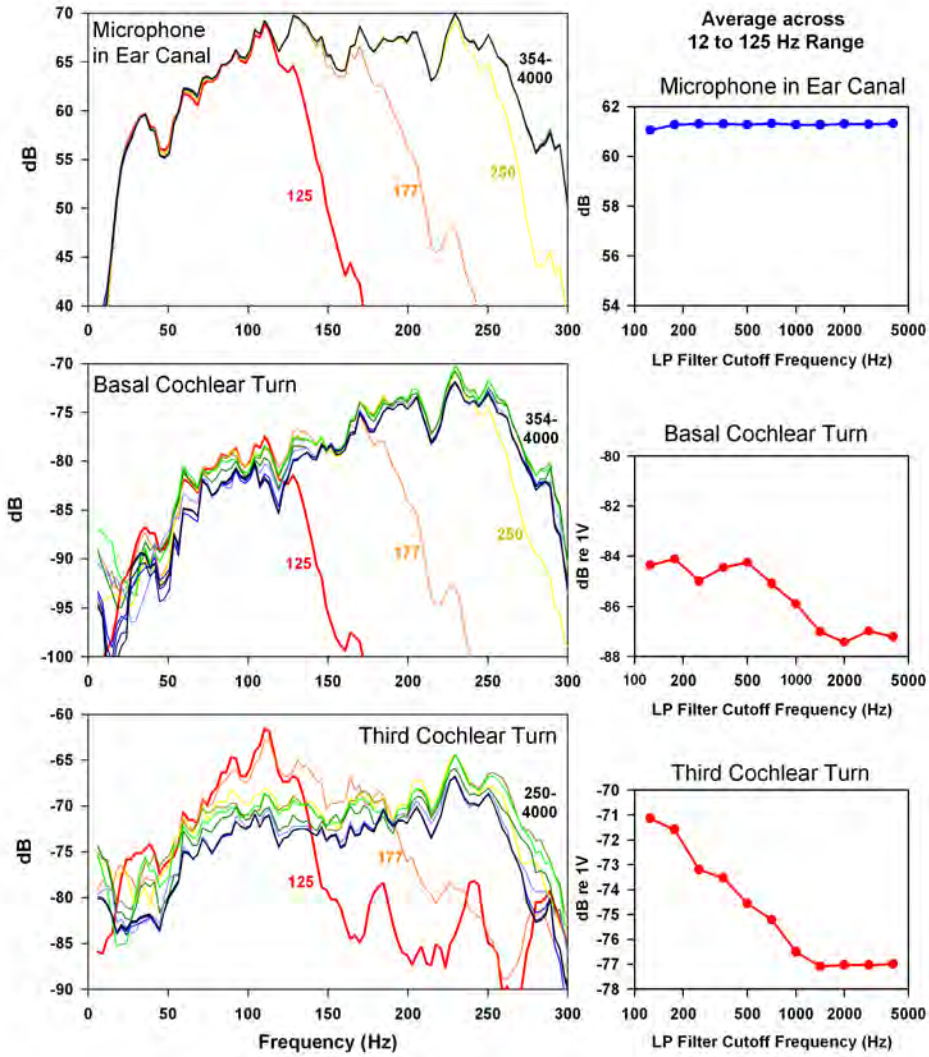

Fig. 4 – Spectra expanded to show the lowest 300 Hz of the frequency range. At each noise filter cutoff frequency, all responses (all 3 panels) were recorded simultaneously to the same stimuli. Top row: The low-frequency region of the sound field showed little variation as cutoff frequency was changed. Middle Row: Responses from the basal cochlear turn were larger when high frequency components were absent. Bottom Row: Apical (Third turn) responses were substantially greater when high frequency components were absent. In this case, the lowest band of noise (125 Hz cutoff) generated ~ 6 dB larger responses than the widest band of noise (4 kHz cutoff). For each condition, the average spectral level in to 12 Hz to 125 Hz range was graphed relative to the noise filter cutoff frequency in the right column.

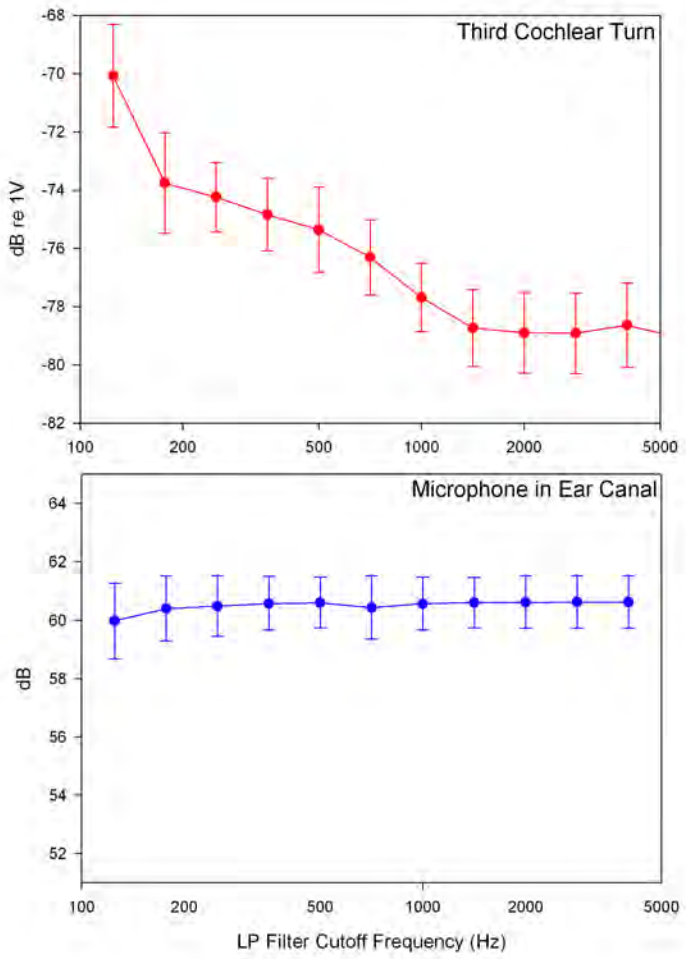

Fig. 5 – Average response amplitudes (+/- SD) in 5 experiments. At each noise cutoff frequency, response amplitude was measured as the average spectral level in the 12 – 125 Hz range. Responses from the apical region of the cochlea showed a systematic decline as noise cutoff frequency is varied, while responses from the microphone, analyzed in an identical manner, did not.

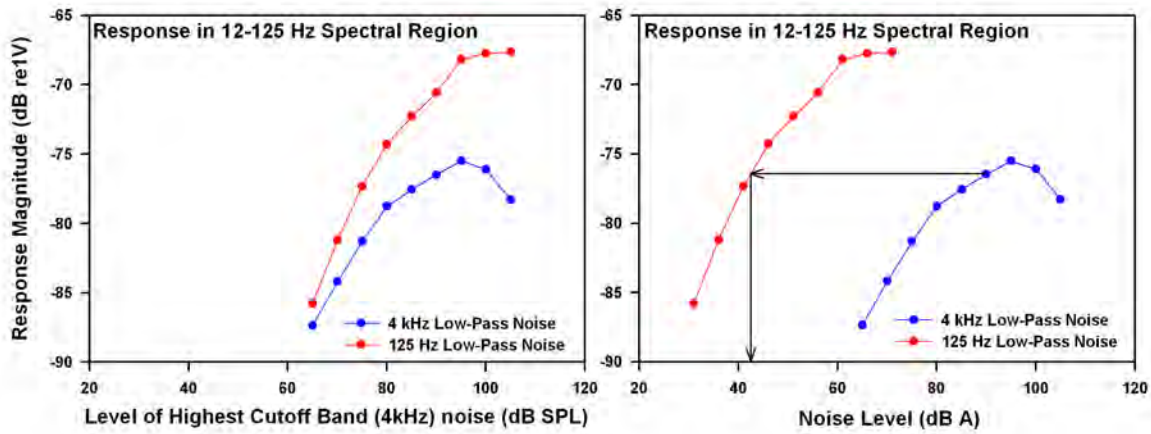

Fig. 6 – ***Left panel:*** *Response amplitudes as the noise level was varied in 5 dB steps. Shown here is only the response amplitudes to the lowest (125 Hz) and highest (4 kHz) noise filter cutoff frequencies used. Amplitudes were the average from the spectrum across the 12 – 125 Hz range (as in Figure 4). Noise with higher frequencies present always generated lower response amplitudes than when higher frequencies were absent. For the 4 kHz band, the responses saturate and decline as level was increased, while the responses to the low-band (125 Hz cutoff) noise keep increasing.* ***Right panel:*** *The same data plotted based on the A-weighted level of the stimuli measured at each cutoff frequency. This shows that low-frequency noise (125 Hz cutoff) of ~43 dB A generated as large of a response at low frequencies as did a 90 dB A wide band (4 kHz cutoff) noise. Indeed, for 125Hz low-pass noise of 45 dB A or greater, an ear's response will be larger than for wide band noise presented at ANY level.*