

Case No. 67315-7-I

COURT OF APPEALS, DIVISION I
OF THE STATE OF WASHINGTON

CATHERINE LAKEY, a single woman; GERTHA RICHARDS, a single woman; MICHAEL HESLOP, a single man; TROY FREEMAN and CAROLINA AYALA de FREEMAN, husband and wife; PATRICK McCLUSKY and MICHELLE McCLUSKY, husband and wife; SHAHNAZ BHUIYAN and ANN RAHMAN, husband and wife; STEVEN RYAN and NORA RYAN, husband and wife; KEVIN CORBETT and MARGARET CORBETT, husband and wife; KATHRYN McGIFFORD, a single woman; and JACQUELYN MILLER, a single woman,

Appellants,

v.

PUGET SOUND ENERGY INC., a Washington corporation; and CITY OF KIRKLAND, a Washington municipal corporation,

Respondents.

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APPELLANTS' OPENING BRIEF

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TABLE OF CONTENTS

I. INTRODUCTION AND OVERVIEW.....	1
II. APPEAL AS TO THE CITY OF KIRKLAND	6
A. Standard of Review.....	6
B. Statement of the Case.....	7
C. Applicable Authority and Discussion.	10
III. APPEAL AS TO PUGET SOUND ENERGY INC.....	17
A. Standard of Review.....	17
B. Statement of the Case.....	17
C. Applicable Authority and Discussion.	29
1. The Legal Standard for Nuisance Liability.	29
2. The Trial Court’s Resolution of the <i>Frye</i> Hearing Is Not Consistent With Either the Controlling Law or the Facts.	36
a. A Literature Review is a Generally-Recognized Method of Evaluating Health Risks from Environmental Exposures.	36
b. The Trial Court was Obviously Confused About the Difference Between ER 703 and ER 702.....	39
IV. CONCLUSION.....	46

TABLE OF AUTHORITIES

Cases

<u>Asche v. Bloomquist</u> , 132 Wn. App. 784, 133 P. 3d 475 (2006)	14
<u>Burns v. PACCAR, Inc.</u> , 77 Wn. App. 201, 890 P.2d 469 (1995)	36
<u>Daubert v. Merrill Dow Pharmaceuticals Inc.</u> , 509 U.S. 579, 113 S.Ct. 2768 (1993).....	21
<u>Deluca by Deluca v. Merrill Dow, Inc.</u> , 911 F.2d 941 (C.A. 3 1990)	37
<u>Dowling v. United States</u> , 493 U.S. 342, 110 S.Ct. 668, 673 n. 3, 107 L.Ed.2d 708 (1990).....	38
<u>Eakins v. Huber</u> , 154 Wn. App. 592, 225 P.3d 1041 (2010).....	20, 34
<u>Everett v. Paschall</u> , 61 Wash. 47, 111 P. 879 (1910)	30, 31
<u>Ferry v. City of Seattle</u> , 116 Wash. 648, 203 P. 40 (1922).....	30
<u>Gen. Elec. Co. v. Joiner</u> , 522 U.S. 136, 118 S.Ct. 512, 139 L.Ed.2d 508 (1997).....	43
<u>Granite Beach Holdings, L.L.C. v. Dep’t of Natural Resources</u> , 103 Wn. App. 186, 11 P.3d 847 (2000).....	12
<u>Grundy v. Thurston County</u> , 155 Wn.2d 1, 117 P.3d 1089 (2005).....	14
<u>Hall v. Baxter Healthcare Corp.</u> , 947 F.Supp. 1387 (D.Or.1996).....	43
<u>Harris v. Skirving</u> , 41 Wn.2d 200, 248 P.2d 408 (1952).....	30
<u>Hisle v. Todd Pac. Shipyards Corp.</u> , 151 Wn.2d 853, 93 P.3d 108 (2004).....	6
<u>In re Silicon Breast Implant Litigation</u> , 318 F.Supp. 879 (C.D. Cal 2004).....	19, 34, 43
<u>Indiana Michigan Power Co. v. Runge</u> , 717 NE.2d 216 (Indiana 1999)	31
<u>Intalco Aluminum</u> , 66 Wn. App. 644, 833 P.2d 390.....	20, 34, 37
<u>Lambier v. City of Kennewick</u> , 56 Wn. App. 275, 783 P.2d 596 (1989).....	12
<u>Manufactured Housing Communities of Washington v. State</u> , 142 Wn.2d 347, 13 P.3d 183 (2000).....	15
<u>Mercer Island Citizens for Fair Process v. Tent City 4</u> , 156 Wn. App. 393, 232 P.3d 1163 (2010), review granted 170 Wn.2d 1020, 245 P.3d 774 (2011), review withdrawn ___ Wn.2d ___, 247 P.3d 421 (2011).....	12, 13
<u>Phillips v. King County</u> , 136 Wn.2d 946, 968 P.2d 871 (1998)	11
<u>Richardson v. Richardson-Merrell, Inc.</u> , 857 F.2d 823 (D.C.Cir.1988), cert. denied, 493 U.S. 882, 110 S.Ct. 218, 107 L.Ed.2d 171 (1989).....	19, 43
<u>State v. Cauthron</u> , 120 Wn.2d 879, 846 P.2d 502 (1993).....	17

<u>State v. Evans</u> , 26 Wn. App. 251, 612 P.2d 442 (1980), <i>reversed</i> on other grounds 96 Wn.2d 119	31
<u>State v. Gore</u> , 143 Wn.2d 288, 21 P.3d 262 (2001).....	36
<u>State v. Jones</u> , 130 Wn.2d 302, 922 P.2d 806 (1996).....	17
<u>State v. Kunze</u> , 97 Wn. App. 832, 988 P.2d 977 (1999).....	37
<u>Tingey v. Haisch</u> , 159 Wn.2d 652, 152 P.3d 1020 (2007).....	6, 12
<u>Turpin v. Merrell Dow Pharms., Inc.</u> , 959 F.2d 1349, 1360 (6th Cir.1992)	43
<u>Vallandigham v. Clover Park Sch. Dist. No. 400</u> , 154 Wn.2d 16, 109 P.3d 805 (2005).....	6
Federal Statutes	
42 U.S.C. § 1983.....	13
State Statutes	
Land Use Protection Act, Chap. 36.70C RCW.....	<i>passim</i>
RCW 54.16.020	9
Other Authorities	
17 Wash. Prac., Real Estate § 4.25 (2d ed.).....	10
83 Am. Jur. 2d Zoning and Planning	10
Article I, § 16, of the State Constitution	<i>passim</i>

TABLE OF SCIENTIFIC EVIDENCE

I. EXPERT TESTIMONY

Declaration of Dr. David O. Carpenter, dated January 11, 2011.....	Ex. 2; CP 186-313
Deposition of Dr. David O. Carpenter, taken March 22, 2011.....	CP 1047-1104
Declaration of De-Kun Li, MD, PhD, MPH, dated January 11, 2011.....	CP 418-467

II. EPIDEMIOLOGICAL STUDIES

Electrical Wiring Configurations and Childhood Cancer (Wertheimer and Leeper), dated May 11, 1978	CP 318-329
Case-Control Study of Childhood Cancer and Exposure to 60-Hz Magnetic Fields (Savitz et al), dated November 25, 1987	CP 331-348
Magnetic Fields and Cancer in Children Residing Near Swedish High-Voltage Power Lines (Feychting and Ahlbom), dated June 10, 1993.....	CP 350-364
Electromagnetic Fields and Cancer in Children Residing Near Norwegian High-Voltage Power Lines (Tynes and Haldorsen), dated October 28, 1996	CP 597-604
A Population-Based Prospective Cohort Study of Personal Exposure to Magnetic Fields During Pregnancy and the Risk of Miscarriage (Li et al), dated March 20, 2001	Ex. 21; CP 259-270
Maternal Occupational Exposure to Extremely Low Frequency Magnetic Fields During Pregnancy and Childhood Leukemia (Infante-Rivard and Deadman), dated January 14, 2003	Ex. 8; CP 279-283
Childhood Leukemia and EMF: Review of the Epidemiologic Evidence (Kheifets and Shimkhada), dated April 11, 2005	Ex. 9; CP 1388-1396
Childhood Leukemia and Magnetic Fields in Japan: A Case-Control Study of Childhood Leukemia and Residential Power-Frequency Magnetic Fields in Japan (Kabuto et al), dated May 31, 2005.....	Ex. 22; CP 285-292

Childhood Cancer in Relation to Distance from High Voltage Power Lines in England and Wales: A Case-Control Study (Draper et al), dated July 4, 2005.....	Ex. 10; CP 294-298
The Sensitivity of Children to Electromagnetic Fields (Kheifets et al), dated August 2005.....	CP 606-618
Magnetic Field Exposure and Long-Term Survival Among Children With Leukaemia (Foliart et al), dated November 18, 2005	CP 588-591
Magnetic Fields and Acute Leukemia in Children With Down Syndrome (Mejia-Arangure), dated July 17, 2006	CP 307-310
Briefing Report on Electromagnetic Fields: Health Effects, Public Policy and Site Planning (Sage and Sage), dated August 2006.....	CP 154-158
Exposure to Magnetic Fields and Survival After Diagnosis of Childhood Leukemia: a German Cohort Study. Epidemiol. (Svendsen et al), dated 2007.....	CP 593-595
Residential Exposure to Electric Power Transmission Lines and Risk of Lymphoproliferative and Myeloproliferative Disorders: A Case-Control Study (Lowenthal et al), dated February 1, 2007	CP 300-305
Setting Prudent Public Health Policy for Electromagnetic Field Exposures (Carpenter and Sage), dated 2008	Ex. 3; CP 219-245
Exposure to Magnetic Fields and the Risk of Poor Sperm Quality (Li et al), dated September 4, 2009	CP 454-460
Maternal Exposure to Magnetic Fields During Pregnancy in Relation to the Risk of Asthma in Offspring (Li et al), dated August 2, 2011.....	Appendix 1

III. POOLED ANALYSES

A Pooled Analysis of Magnetic Fields and Childhood Leukaemia (Ahlbom et al), dated May 16, 2000	Ex. 6; CP 1110-1116
A Pooled Analysis of Magnetic Fields, Wire Codes, and Childhood Leukemia (Greenland et al), dated May 30, 2000.....	Ex. 7; CP 247-257
Pooled Analysis of Recent Studies on Magnetic Fields and Childhood Leukaemia (Kheifets et al), dated July 12, 2010.....	Ex. 11; CP 1118-1125

IV. GOVERNMENTAL STUDIES AND FINDINGS

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Southern California Edison Company, dated
September 12, 1990 CP 712-766
- California Public Utilities Commission, Order re
Potential Health Effects of Electric and Magnetic
Fields of Utility Facilities, dated November 2,
1993 CP 674-710
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Services, National Institutes of Health, NIEHS
Report on Health Effects from Exposure to
Power-Line Frequency Electric and Magnetic
Fields, dated May 4, 1999 CP 73-152
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the Evaluation of Carcinogenic Risks to Humans,
Non-Ionizing Radiation, Part 1: Static and
Extremely Low-Frequency (ELF) Electric and
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An Evaluation of the Possible Risks from
Electric and Magnetic Fields (EMFs) from
Power Lines, Internal Wiring, Electrical
Occupations, and Appliance, dated June 2002Ex. 14; CP 272-277
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for Research on Cancer, IARC Monographs on
the Evaluation of Carcinogenic Risks to Humans,
Preamble, dated 2006Ex. 4; CP 1307-1333
- World Health Organization, Environmental Health
Criteria 238, Extremely Low Frequency Fields,
dated 2007Ex. 12; CP 1127-1148
- Centers for Disease Control and Prevention
webpage printout regarding EMF (Electric and
Magnetic Fields) information,
last updated July 26, 2010 Ex. 13
- U.S. Department of Health and Human Services,
National Institute for Occupational Safety and
Health webpage printout regarding EMFs in the
Workplace, printed April 26, 2011 Ex. 15
- U.S. Environmental Protection Agency webpage
printout regarding Electric and Magnetic Fields

(EMF) Radiation from Power Lines, printed
April 26, 2011..... Ex. 16

V. ADDITIONAL MATERIALS REFERENCED BY EXPERTS

Power Line and Cancer: Public Health and Policy
Implications (Carpenter and Ahlbom), dated Winter
1983

Exposure to Residential Electronic and Magnetic Fields
and Risk of Childhood Leukemia (London et al), dated
November 1, 1991

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Intrachromosomal Recombination in Dividing Yeast
Cells (Schiestl), dated December 1993

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Canine Lymphoma (Reif et al), dated 1995

Brodeur Testifies Before Nebraska Legislature: Provides
Evidence that Power Line EMF’s Cancer Link is
Conclusive, dated Spring 1995.....

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and Leukemia and Central Nervous System Tumors
(Feychting et al.), dated February 3, 1997

The EMF Biochip™ Technology – Neutralizing the
Effects of EMF Field, dated October 22, 2003

Electromagnetic Fields Stress Living Cells (Blank and
Goodman), dated 2009

Electromagnetic Fields and Cancer: The Cost of Doing
Nothing (Carpenter), dated January 27, 2009

A Precautionary Public Health Protection Strategy for the
Possible Risk of Childhood Leukaemia from Exposure
to Power Frequency Magnetic Fields (Maslanyj et al),
dated 2010

I. INTRODUCTION AND OVERVIEW

On February 9, 2009, the City of Kirkland (“City”) granted variances to Puget Sound Energy Inc. (“PSE”) to construct, in a quiet, middle-class, residential neighborhood, the largest electrical substation ever constructed by PSE in a residential neighborhood (the “Substation”). The City and PSE are the Respondents here. Appellants are nine of the ten families whose homes are contiguous with the Substation (the “Homeowners”).

As related by homeowner Kevin Corbett:

PSE’s Roque Bamba told us: “the largest substation in a residential area in Puget Sound Energy’s history” – 300 feet long, 65 feet wide, and 35 feet high – bigger than a football field. The fact that the [City] granted special variances to squeeze it into a community and in between ten houses, means that it[’s] very obvious and obnoxious.

(*CP 1549 at ¶ 4*). Photographs of the completed Substation are CP 1484-1490. “Before and after” aerial photographs are CP 1474-1483.

The more important impact to the Homeowners, however, is that the construction of the Substation resulted in an immediate and significant devaluation of the Homeowners’ properties. In the case of homeowner Michael Heslop, the assessed value of his home went from \$337,000 (*CP 1509*) to \$172,000 (*CP 1511*) – a drop of 51%. While there were multiple factors identified by King County in the decision to lower the assessed value, most of them were related to the Substation. One of the factors was described by King County as “nuisances/stigmas” arising from “electromagnetic fields.” (*CP 1511*). These were, of course, electromagnetic fields (“EMF”) emanating from the Substation.

The Homeowners filed a Complaint naming PSE as a Defendant. The Complaint alleged that a reasonable apprehension of injury from exposure to EMF held by the public caused the loss in value of their properties. The Homeowners asserted that EMF intruding onto their properties was both a nuisance and a trespass. PSE moved for dismissal under CR 12(b)(6).

As discussed below, there is a large body of epidemiological evidence drawing a link between exposure to EMF and a number of human diseases/adverse health conditions. The 1999 National Institute of Environmental Health Services/National Institute of Health (“NIEHS”) report, prepared at the direction of Congress, concludes: “The NIEHS concludes that *ELF-EMF exposure cannot be recognized at this time as entirely safe* because of weak scientific evidence that exposure may pose a leukemia hazard.” (CP 76; *emphasis added*). The International Agency for Research on Cancer, a sub entity of the World Health Organization (“WHO/IARC”), characterized EMF as a possible carcinogen in 2002. (CP 887). A 2002 report on EMF exposure from the California Department of Health Services (“CDHS”) concludes:

To one degree or another, all three of the [C]DHS scientists are inclined to believe that EMFs can cause some degree of increased risk of childhood leukemia, adult brain cancer, Lou Gehrig’s Disease, and miscarriage.

(CP 274). A publication currently available from the Centers for Disease Control (“CDC”) states:

Many studies report small increases in the rate of leukemia or brain cancer in groups of people living or working in high magnetic fields. Other studies have found no such increases. The most important data come from six recent

studies of workers wearing EMF monitors to measure magnetic fields. All but one study found significantly higher cancer rates for men with average workday exposures above 4 milligauss. However, the results of these studies disagree in important ways such as the type of cancer associated with EMF exposures. So scientists cannot be sure whether the increased risks are caused by EMFs or by other factors. A few preliminary studies have also associated workplace EMFs with breast cancer, and one study has reported a possible link between occupational EMF exposure and Alzheimer[']s disease.

(Hearing Ex. 15 at p. 3).

The most recent study published in 2010 of the relation between EMF exposure and childhood leukemia, conducted by Dr. Leeka Kheifets,¹ an epidemiologist *cited by PSE expert Dr. Nancy Lee as authoritative*, concluded: “[T]hat recent studies on magnetic fields and childhood leukaemia do not alter the previous assessment that magnetic fields are possibly carcinogenic [to humans].” (CP 1118).

While it is clear that there is a dispute in the scientific community as to the magnitude of the risk, virtually every governmental authority which has examined the EMF issue has recommended that people conduct themselves in a manner so as to limit exposure to EMF. The CDHS report, for example, concludes:

[T]o put things in perspective, individual decisions about things *like buying a house* or choosing a jogging route should involve the consideration of certain risks, such as those from traffic, fire, flood, and crime, as well as the uncertain comparable risks from EMFs.

¹ Dr. Kheifets is a Professor of Epidemiology in the UCLA School of Public Health. Previously, she was Head of the Radiation Studies Program at the WHO at the time the WHO/IARC concluded EMF was a possible carcinogen.

(CP 275; *emphasis added*). The Environmental Protection Agency (“EPA”) makes available a publication which states that a “*definitive* cause-effect relationship” between EMF and human disease cannot be confirmed *or refuted*. (*Hearing Ex. 16 at p. 1; emphasis in original*). The publication goes on to recommend that:

People concerned about possible health risks from power lines can reduce their exposure by:

Increasing the distance between you and the source – The greater the distance between you and the power lines the more you reduce your exposure.

Limiting the time spent around the source – Limit the time you spend near power lines to reduce your exposure.

(*Hearing Ex. 16 at p. 2; emphasis in original*). Since the Homeowners already owned their homes at the time the Substation was constructed, the alternatives offered by the CDHS or the EPA would be of little help.

In response to PSE’s Motion to Dismiss, the Trial Court entered an Order requiring the Homeowners to submit scientific evidence supporting the claim that the Homeowners were entitled to “stigma damages” based on the allegation that the diminution in value was attributable to a reasonable apprehension of adverse health effects from exposure to EMF generated by the Substation. (CP 185). The Homeowners submitted multiple epidemiological studies and reports showing a correlation between EMF exposure and human disease, together with two expert opinions from Dr. David O. Carpenter² and Dr. De-Kun Li.³

² Dr. Carpenter is the Director of the Institute for Health and the Environment at the University of New York at Albany (*see* CP 193-217 for Dr. Carpenter’s CV).

³ Dr. Li a research epidemiologist with Kaiser Permanente Foundation (*see* CP 425-439 for Dr. Li’s CV).

At PSE's request, the Trial Court ordered a *Frye* hearing. The Homeowners elected to use a single expert, Dr. Carpenter, for reasons of expense. Dr. Carpenter testified that, based on a review of epidemiologic and other scientific literature, there was a statistically-significant relationship between EMF and human disease. During the *Frye* hearing, 16 exhibits consisting of various government reports and epidemiological studies concluding that a correlation existed between EMF and human disease were admitted into evidence without objection by PSE, including the NIEHS, WHO/IARC, CDC, CDHS and EPA reports referenced above.

Following the *Frye* hearing, the Homeowners' claims against PSE were dismissed based on a finding that the opinions of the Homeowners' expert, Dr. Carpenter, did not meet the *Frye* standard. (*CP 1418-1423*).

The Homeowners' appeal that dismissal on the bases that:

1. The Trial Court applied an "actual injury" standard for liability not applicable in this case;
2. The Trial Court ignored the existence of multiple issues of material fact; and
3. The Trial Court's decision on the admissibility of Dr. Carpenter's testimony cannot be reconciled with the *Frye* standard.

The City was added as a Defendant by an amendment to the Complaint. With respect to the City, the Homeowners did not elect to pursue a challenge to the granting of the variances under the Land Use Protection Act, Chap. 36.70C RCW ("LUPA"). Rather, the Homeowners amended the Complaint to assert an inverse condemnation claim against the City.

The Trial Court determined on summary judgment that: “[T]he City is entitled to dismissal of Plaintiffs’ claims as a matter of law based on the conclusion that the claim should have been brought under LUPA.” (CP 1668).

Although the claims against the City and PSE are linked by same event, the approval of the variances necessary to the construction of the Substation, the facts and law applicable to the claims have no further link. In order to avoid confusion, the discussion of the issues are separated.

II. APPEAL AS TO THE CITY OF KIRKLAND

A. Standard of Review.

This appeal is taken from the entry of an Order on the City’s Summary Judgment Motion. On review of an Order for Summary Judgment, the Appellate Court performs the same inquiry as the Trial Court. *Hisle v. Todd Pac. Shipyards Corp.*, 151 Wn.2d 853, 860, 93 P.3d 108 (2004). The standard of review is *de novo* and, under that standard, the reviewing Court must consider all facts in the light most favorable to the non-moving party. *Vallandigham v. Clover Park Sch. Dist. No. 400*, 154 Wn.2d 16, 26, 109 P.3d 805 (2005). The Appellate Court may affirm the summary judgment if the pleadings, affidavits, depositions and admissions on file demonstrate that there are no genuine issues of material fact and that the moving party is entitled to judgment as a matter of law. CR 56(c).

This appeal also presents an issue of the interpretation of LUPA. Issues of statutory interpretation are also reviewed *de novo*. *Tingey v. Haisch*, 159 Wn.2d 652, 152 P.3d 1020 (2007).

B. Statement of the Case.

The action requested in PSE's application to the City to allow construction of the Substation is described in the City's Advisory Report as follows:

As part of the zoning and variance permit application, the applicant [PSE] is proposing to reduce the east and west side yard setbacks from 20 feet to 13 feet, reduce the required east and west landscape buffers from 15 feet to 13 feet, and exceed the maximum allowable height of 30 feet by 5 feet to accommodate termination structures.

(CP 1532).

As shown in the various photographs (CP 1484-1490; CP 1474-1483), the result is a massive structure sitting right on the boundaries of the Homeowners' properties. That structure was adversely impacting the Homeowners' property values before construction started. CP 407 and CP 409-411 are letters from King County progressively lowering the value of Mr. Heslop's property. The Order issued April 9, 2010 lowers the 2008 assessed value by \$38,000 for the following reasons:

Based on the *pending* Puget Sound Energy substation construction, *the associated stigma issues*, and the 2009 construction start, the Board finds that to a degree, the market value of the subject property was negatively impacted.

(CP 407; *emphasis added*). The largest decrease in value did not come until the Order of December 28, 2010 when the 2009 assessed value was lowered an additional \$107,000 for a grand total of \$165,000 in lost value.

As stated by King County:

After weighing the features of the substation, its proximity to the subject properties, *electromagnetic issues*, construction inconveniences, *associated nuisances/*

stigmas, real estate professionals' opinions, comparable sales, and market timing, the Board concludes that the evidence presented demonstrates that the values listed on the attached page represent the subject's true and fair market value for the January 1, 2009, valuation date in question.

(CP 410; *emphasis added*). Both reassessments occurred *before* the Substation had actually gone into operation.

The potential impact on the value of the Homeowners' properties was known to the City *before* it took the action to grant the variances. Exhibit F to the Hearing Examiner's Decision, and also part of the City's record on appeal, is a letter to the City from William Rynd, an experienced real estate agent in the applicable market area, that states construction and operation of the Substation would have a substantial adverse impact on the value of the Homeowners' properties. (CP 1457).

The actual impact on the Homeowners' lives is described in the Declarations of Michael Heslop (CP 365-367; CP 1466-1470), Kevin Corbett (CP 1548-1551) and Steven Ryan (CP 1561-1562) filed in support of the Homeowners' Cross-Summary Judgment Motion.

The City Staff's Conclusions and Recommendations to the Hearing Examiner on PSE's application state that granting the variances:

[I]s consistent with the public health, safety and welfare because it will allow a **Public Utility** Use to replace an existing substation with a new substation that will increase electrical service capacity and improve reliability, benefiting property owners and electrical customers.

(CP 1537; *emphasis added*). The problem is that **PSE is not a "Public Utility"** – it is a privately held, "for profit" corporation as described on PSE's website:

On Feb. 6, 2009, Puget Holdings LLC, a group of long-term infrastructure investors, closed on its acquisition of Puget Energy, transforming the company from being publicly-held and traded on the New York Stock Exchange, to being privately-held with continued financial disclosure and regulatory oversight of it and utility subsidiary, Puget Sound Energy.

(CP 1459).

Public utility districts have the power of eminent domain (*see* RCW 54.16.020) – PSE does not. As a private utility, PSE **could not** have obtained the necessary rights in the Homeowners’ properties without the cooperation of the City. In essence, the City was used as a vehicle to circumvent the necessity of compensating the Homeowners.

However, the City was not exercising a general regulatory authority for the benefit of the public – it was allowing a private company to increase its business at the Homeowners’ expense. In this regard, it was undisputed that, in order to construct the Substation, PSE had to obtain the variances.

Because of the configuration of the Subject Property, PSE applied to the City for a variance to construct the new substation. Hearing Examiner’s Decision, pp. 2, 3 & 9, Findings of Fact Nos. 1 & 10; Conclusion No. 8. PSE sought a variance from the City with respect to setbacks, landscape buffering and maximum height. *Id.*, p.1.

(CP 1568:1-8).

But a variance is not an exercise of general authority to zone under the police power – a variance is an exception to zoning regulations granted to benefit a single landowner. As stated in the Washington Practice Manual on Real Estate:

Whereas a conditional use is a permitted use, one listed in the zoning ordinance as permitted upon a special permit, a variance permit allows the applicant to do something the zoning ordinance would otherwise forbid.

17 Wash. Prac., Real Estate § 4.25 (2d ed.). *See, also*, 83 Am. Jur. 2d Zoning and Planning.

Following the Hearing Examiner's Decision, an appeal was taken to the City Council pursuant to City ordinances and state law. On February 17, 2009, the City Council denied the appeal and adopted the Hearing Examiner's Findings and Conclusions.

The Homeowners did not elect to pursue a LUPA action because they were told by their attorney that the action would be too costly. (*CP 366 at ¶ 4*).

C. Applicable Authority and Discussion.

This appeal is taken from the Trial Court's Order granting the City's Summary Judgment Motion on the basis that the Homeowners' claim was barred as a result of a failure to bring a LUPA appeal of the grant of the variances. Specifically, the Trial Court concluded that: "[T]he City is entitled to dismissal of Plaintiffs' claims as a matter of law based on the conclusion that the claim should have been brought under LUPA." (*CP 1668*).

The Trial Court's Decision was in error for the following reasons:

1. Under RCW 36.70C.030(1)(c), LUPA does not apply to "claims provided by any law for monetary damages or **compensation**." (*Emphasis added*). Since an inverse condemnation claim is a claim for **compensation** under Article I, § 16, of the State Constitution, an inverse condemnation claim is exempt from LUPA.

2. The invalidity or illegality of governmental act which is the basis for an inverse condemnation claim has never been recognized as a required element of an inverse condemnation claim. The Trial Court's Decision imposes a new element previously unrecognized and inconsistent with the principles underlying an inverse condemnation claim.

The question here is whether the Homeowners were required to initiate review under LUPA as a pre-condition to asserting an inverse condemnation claim. This is a pure issue of law involving the interpretation of the relevant provision of LUPA.

Article I, § 16, of the State Constitution provides:

§ 16 EMINENT DOMAIN. Private property shall not be taken for private use, except for private ways of necessity, and for drains, flumes, or ditches on or across the lands of others for agricultural, domestic, or sanitary purposes. No private property shall be taken or damaged for public or private use without **just compensation** having been first made, or paid into court for the owner, and no right-of-way shall be appropriated to the use of any corporation other than municipal until **full compensation** therefore be first made in money, or ascertained and paid into court for the owner, irrespective of any benefit from any improvement proposed by such corporation, which **compensation** shall be ascertained by a jury, unless a jury be waived, as in other civil cases in courts of record, in the manner prescribed by law.

(Emphasis added). The term “inverse condemnation” is used to describe an action alleging a governmental “taking” or “damaging” that is brought to recover the value of property which has been appropriated in fact, but with no formal exercise of the power of eminent domain. *Phillips v. King County*, 136 Wn.2d 946, 957, 968 P.2d 871, 876 (1998) (*quoting Lambier v. City of Kennewick*, 56 Wn. App. 275, 279, 783 P.2d 596

(1989)); Granite Beach Holdings, L.L.C. v. Dep't of Natural Resources, 103 Wn. App. 186, 205, 11 P.3d 847 (2000).

The relevant provision in the statute is RCW 36.70C.030(1)(c):

(1) This chapter replaces the writ of certiorari for appeal of land use decisions and shall be the exclusive means of judicial review of land use decisions, except that this chapter does not apply to:

(c) Claims provided by any law for monetary damages or ***compensation***.

(*Emphasis added*). On its face, the statute is explicit that it does not extend to “claims provided by any law for...compensation.” There is no issue that Article I, § 16, of the State Constitution would fall within the scope of the phrase “any law.” Thus, the dispositive issue is whether the Legislature intended a different meaning for the term “compensation” in LUPA than the meaning of the same term in the State Constitution.

Given the context of LUPA - land use decisions – the specific area of governmental action typically spawning inverse condemnation claims for compensation – it would be impossible to conclude that the Legislature did not intend the term “compensation” in LUPA to mean the same thing as “compensation” in the State Constitution. Such an interpretation would violate a basic principle of interpretation that statutes should not be construed so as to produce unlikely, unreasonable, absurd or strained results. Tingey v. Haisch, 159 Wn.2d 652, 152 P.3d 1020 (2007).

Relying on Mercer Island Citizens for Fair Process v. Tent City 4, 156 Wn. App. 393, 232 P.3d 1163 (2010), the City asserted that a plaintiff cannot assert an inverse condemnation claim unless it first prevails on a

LUPA appeal challenging the validity of the land use decision.⁴ What the City leaves out of the discussion is that the Supreme Court granted review, 170 Wn.2d 1020, 245 P.3d 774 (2011), which Petition was later withdrawn by the appellant; ___ Wn.2d ___, 247 P.3d 421 (2011). So, there is reason to believe that the Supreme Court was inclined to see the issues differently than the Court of Appeals.

Nevertheless, *Mercer Island* is clearly distinguishable because *Mercer Island* did not involve an inverse condemnation claim – the claims were predicated on a violation of constitutional due process rights for which an award of damages may be made under 42 U.S.C. § 1983. As the *Mercer Island* Court noted, unless the land use decision at issue violated the plaintiffs’ due process rights (“each of these claims was based on the alleged illegality of the TUA and challenged its approval process;” *id.* at 401) – in other words was constitutionally infirm, the plaintiffs had no claim under 42 U.S.C. § 1983 and no right to an award of damages.

The substantive and procedural due process rights in the State and Federal Constitution at issue in *Mercer Island* are absolute prohibitions on certain kinds of conduct by a government. Unconstitutional or illegal conduct by a government or governmental agent is an element of the cause of action for damages under 42 U.S.C. § 1983. This special circumstance took the claim in *Mercer Island* outside the exception in LUPA.

By way of contrast, Article I, § 16, of the State Constitution does not stop a government from taking private property – rather it authorizes

⁴ The City’s position is literally an oxymoron. If a landowner prevails in opposition to a proposed land use action, the land use action does not occur precluding a taking.

the taking of property. What Article I, § 16, of the State Constitution requires is that, unless the action can be justified as an exercise of police power, the landowner must be fairly compensated. The elements required to establish inverse condemnation are: (1) a taking or damaging (2) of private property (3) for public use (4) without just compensation being paid (5) by a governmental entity that has not instituted formal proceedings. *Fitzpatrick v. Okanogan County*, 169 Wn.2d 598, 605, 238 P.3d 1129 (2010) (quoting *Dickieser v. State*, 153 Wn.2d 530, 534-35, 105 P.3d 26 (2005)). Invalidity of the government action is *not* an element of an inverse condemnation claim. There is no authority in Washington – and neither the City nor the Trial Court identified any – holding that the conduct of the government must be illegal or unconstitutional as a pre-condition for compensation under Article I, § 16, of the State Constitution.

In *Asche v. Bloomquist*, 132 Wn. App. 784, 800, 133 P. 3d 475 (2006), the Court specifically held:

Claims that do not depend on the validity of a land use decision are not barred. That LUPA allows such nuisance claims is confirmed by the Washington Supreme Court's recent decision in *Grundy*. There, the Court determined that whether a land owner had a valid permit was irrelevant to the landowner's private nuisance action. *Grundy v. Thurston County*, 155 Wn.2d 1, 8, 10, 117 P.3d 1089 (2005).

As the right to seek compensation under Article I, § 16, of the State Constitution is likewise not dependent on the validity of the land use decision involved, the same rule should be applicable here.

Here is where the twist comes in – Article I, § 16, of the State Constitution provides:

Private property shall not be taken for private use, except for private ways of necessity, and for drains, flumes, or ditches on or across the lands of others for agricultural, domestic, or sanitary purposes.

Other than the exception emphasized above, the prohibition against appropriating private property is absolute:

The eminent domain provision of the Washington State Constitution provides a complete restriction against taking private property for private use: “Private property shall not be taken for private use....” Const. art. I, § 16 (amend.9). This absolute language is further strengthened by the enumeration of specific, but here inapplicable, exceptions “for private ways of necessity, and for drains, flumes, or ditches on or across the lands of others for agricultural, domestic, or sanitary purposes.” Const. art. I, § 16 (amend.9). These specific exceptions are incorporated into an otherwise absolute prohibition precluding taking private property for private use. This prohibition is not conditioned on payment of compensation.

Manufactured Housing Communities of Washington v. State, 142 Wn.2d 347 at 362, 13 P.3d 183 (2000).

Relying on this case, the City asserted:

Plaintiffs’ theory is that the City’s land use decision is a taking for a private use. Assuming (for the sake of argument only) that this is true, just compensation is not an available remedy under the *Manufactured Housing* and the *In re Seattle* cases.

(*CP 1575:28-33; emphasis in original*). In other words, the City appears to be asserting that, because the City did something it was prohibited from doing irrespective of compensation – an action the City admits is an illegal act – the City should be insulated from liability by its own illegal conduct

because the Homeowners sought compensation rather than to invalidate a facially-illegal variance.

Neither *In re Seattle* nor *Manufactured Housing* actually held that an inverse condemnation remedy was unavailable in part because neither case involved an inverse condemnation claim. *In re Seattle*, 92 Wn.2d 616, was an eminent domain case. As the Court in *Martin v. Port of Seattle*, 64 Wn.2d 309 at 318-19, stated:

[Inverse condemnation] differs from eminent domain only in that the landowner institutes the action, rather than the entity possessing the condemnation power. Also, the plaintiff landowner in the inverse condemnation setting is usually claiming a partial taking or damaging rather than a total loss of the land itself.

Eminent domain is a power granted, in the case of cities, under Chap. 8.12 RCW. As an element of its condemnation power, the City of Seattle was required to demonstrate that the appropriation was for a public use and failed to do so. There is nothing in the eminent domain statute or related case law that says the defendant land owner is required to contest whether the appropriation was for a public use, or that a defendant land owner could not elect to litigate only the issue of compensation. But, compensation was not the remedy this particular defendant was seeking.⁵

Manufactured Housing only held that the government could not take the right involved – it says nothing about whether the landowner could elect to be compensated in lieu of challenging the taking of the right. The case most certainly did not say a landowner was precluded from

⁵ The situation in *Manufactured Housing* was the same. The property owners sought to invalidate the regulation rather than compensation.

accepting just compensation if that landowner elected to do so. In this same regard, there is no law that says the Homeowners here cannot waive the prohibition against appropriation for private use and request compensation.

Proving the invalidity or illegality of the granting of the variances was not a condition to the right to be compensated under Article I, § 16, of the State Constitution. Thus, there are no special circumstances here that would take the Homeowners' inverse condemnation claim outside the express exception for such claims in RCW 36.70C.030. The dismissal of the Homeowners' claims on the basis that the Homeowners had failed to pursue a LUPA appeal to conclusion was simply error.

III. APPEAL AS TO PUGET SOUND ENERGY INC.

A. Standard of Review.

The standard of review applicable to all of the issues here, including the *Frye* issue, is *de novo*. With respect to the *Frye* issue, see *State v. Jones*, 130 Wn.2d 302, 307, 922 P.2d 806 (1996); *State v. Cauthron*, 120 Wn.2d 879, 887, 846 P.2d 502 (1993) (appellate court reviews *de novo* without deference to the Trial Court's determination that scientific process is or is not generally accepted in the scientific community; the Appellate Court may use scientific literature and other appropriate information, whether or not in the Trial Court record).

B. Statement of the Case.

As previously noted, PSE originally brought a Motion to Dismiss the Homeowners' nuisance claim based on exposure to EMF emanating from the Substation under CR 12(b)(6). PSE asserted that there was no

scientific basis supporting the existence of a health risk, as evidenced by the NIEHS Report. However, the NIEHS Report itself concludes: “that *ELF-EMF exposure cannot be recognized at this time as entirely safe...*” (CP 76; *emphasis added*). In fact, throughout the course of these proceedings, *no scientific publication* expressing any opinion on EMF was offered by PSE, much less a publication opining that exposure to EMF is safe.

In addition to the NIEHS Report, the Homeowners submitted “Briefing Report on Electromagnetic Fields: Health Effects, Public Policy and Site Planning,” published by the Journal of the Australasian College of Nutritional & Environmental Medicine in the August 2006 issue. This publication summarized the available epidemiological research on health risks from EMF exposure citing 15 epidemiological studies concluding that childhood leukemia rates increased in children exposed to EMF and 20 epidemiological studies drawing a link between adult exposure and various health impacts including cancer and miscarriage. (CP 154-158).⁶

Epidemiological studies examine the rates of incidence of human disease in human populations based on the amount of exposure to a potentially disease causing agent. Epidemiological studies are distinguishable from toxicological studies in that toxicological studies (including animal and cellular studies) seek to induce effects by exposing the test subject to potentially-harmful substances, whereas epidemiological studies focus on the effects of exposure on existing human populations. In

⁶ The reports referenced in this briefing include many of the epidemiological studies relied on by Dr. Carpenter in framing his opinions.

a toxicological study, the exposure can be controlled. In an epidemiological study, exposure must be measured or estimated. (*CP 1086-1087 at 159:20-163:10*).

Basically, an exposed population is compared to an unexposed population. If there is a larger occurrence in the rate of the disease under consideration in the exposed population, that difference is evidence that the exposure is a cause of the disease under consideration. The very point of an epidemiological study is to obtain evidence on the causation of human disease by environmental factors, something PSE cannot conceivably deny as it relied on the testimony of an epidemiologist on the issue of causation, Dr. Lee.

Epidemiological studies have been recognized as valid evidence of causation in toxic tort cases:

“When [epidemiological] studies are available and relevant, and particularly when they are numerous and span a significant period of time, ***they assume a very important role in determinations of questions of causation.***” *Richardson v. Richardson-Merrell, Inc., supra.*⁷ See also *Ref. Manual* at 335 n. 2 (“Epidemiologic studies have been well received by courts trying mass tort suits. Well-conducted studies are uniformly admitted.”)

In re Silicon Breast Implant Litigation, 318 F.Supp. 879 at 892-893 (C.D. Cal 2004) (*emphasis added*). Various Washington Courts have recognized that epidemiological studies can be, but are not required as, evidence of a causal link between environmental contaminants and human health risks. See *Eakins v. Huber*, 154 Wn. App. 592, 225 P.3d 1041

⁷ *Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 830 (D.C.Cir.1988), *cert. denied*, 493 U.S. 882, 110 S.Ct. 218, 107 L.Ed.2d 171 (1989).

(2010); *Intalco Aluminum*, 66 Wn. App. 644 at 661-662, 833 P.2d 390 (a cause-effect relationship need not be proven by epidemiological studies before a doctor can testify to his opinion that such a relationship exists).

Following the hearing on the Motion to Dismiss, the Trial Court entered an Order requiring that the Homeowners: “[S]ubmit evidence establishing some scientific basis to support their claims.” (CP 185).

The Homeowners submitted multiple epidemiological studies and reports showing a correlation between EMF exposure and human disease together with two expert opinions from Drs. Carpenter and Li. These materials are CP 186-313, CP 314-364 and CP 418-467.

The specific expert opinion expressed by Dr. Carpenter at issue here is:

Contrary to the representations made by [PSE] that there is no scientific evidence supporting a link between EMF exposure and human disease, there is actually strong evidence.

(CP 187:11-13). Dr. Carpenter further opined:

In my opinion, based on the available-scientific evidence, an apprehension on the part of the public that exposure to EMF could cause adverse health impacts is clearly warranted.

(CP 189:6-8).

The fundamental issue in a *Frye* hearing is whether the **methodology** used by Dr. Carpenter to generate his opinions was generally accepted in the scientific community. As Dr. Carpenter testified both in his Declaration (CP 186-313) and in his deposition (CP 1047-1104 at 48:7-58:15, 63:12-68:20, 70:4-72:23, 171:20-173:16), the basis for Dr. Carpenter’s opinions is a substantial body of peer-reviewed scientific

literature reporting the results of epidemiological studies, which studies have consistently shown a significant-statistical correlation between EMF exposure and human disease, particularly childhood leukemia:

Q And was there independent research conducted as part of that or was that basically the kind of literature review that is the subject of the preamble?

A It was primarily a literature review.

Q And, again, your opinions are based upon a similar literature review?

A That's correct.

Q Would that kind of literature review be generally accepted in the scientific community as a mechanism for assessing the health risk of an environmental agent?

A Well, yes. This is basically what all of our government agencies do when they issue documents that determine whether or not something is a carcinogen, whether it poses a risk to society.

(4/25/11 T.P. at 32:4-18).

In *Daubert v. Merrill Dow Pharmaceuticals Inc.*, 509 U.S. 579 at 593-94, 113 S.Ct. 2768 (1993), the Court commented on the significance of peer review as follows:

But submission to the scrutiny of the scientific community is a component of “good science,” in part because it increases the likelihood that substantive flaws in methodology will be detected. *See* J. Ziman, *Reliable Knowledge: An Exploration of the Grounds for Belief in Science* 130-133 (1978); Relman & Angell, *How Good Is Peer Review?*, 321 *New Eng.J.Med.* 827 (1989). The fact of publication (or lack thereof) in a peer reviewed journal thus will be a relevant, though not dispositive, consideration in assessing the scientific validity of a

particular technique or methodology on which an opinion is premised.

Thus, the fact that these studies were published establishes that the data collection and analysis methodologies were accepted by the scientific community. So, Dr. Carpenter's opinions were the interpretation of data collected by methodologies the scientific validity of which had already been addressed in the peer review process.

Dr. Li, an epidemiologist and the principal author of a study concluding that there is relationship between EMF and increased rates of miscarriage cited in the briefing and the CDHS Report, based his opinion that EMF posed a potential health risk on the same epidemiological data. His conclusion was:

It should be noted that regarding EMF health effects, we are dealing with an issue of uncertainty. However, uncertainty does not mean that EMF is safe. Uncertainty about EMF adverse health effect[s] means that so far, there are inconsistent reports in scientific findings regarding whether EMFs have adverse health effects. Some studies reported an increased risk of adverse EMF health effects, but other studies failed to find the same results. This inconsistency in scientific findings is nothing unique for adverse EMF health effect[s].

(*CP 419:8-14*). Dr. Li's bottom line: "[G]iven the available scientific evidence, I would not choose to live in a house close to a utility substation." (*CP 420:6-7*).

The principal studies of powerline EMF pre-dating the NIEHS Report are Wertheimer-Leeper (*CP 318-329*), Savitz *et al.* (*CP 331-348*), and Feychting-Ahlbom (*CP 350-364*). All three studies show a

statistically-significant relationship between EMF exposure and childhood leukemia.

The Wertheimer-Leeper study states:

Power carried at higher voltage is stepped down to produce increased current at two points in our electrical distribution system: at the distribution substation [like the Substation at issue here], and again at the neighborhood transformers. ***As indicated, cancer cases were found in excess close to the “first span” wires issuing from the transformers. An even stronger trend was found for substations.***

(CP 327; *emphasis added*). This study specifically links proximity to substation EMF to “excess cancer cases.”

Savitz reports: “Measured magnetic fields under low power use conditions had a modest association with cancer incidence.” (CP 331).

Fechting-Ahlbom conclude: “For leukemia in children and exposure defined from calculated historical fields, this study shows elevated estimated relative risks, which increase with level of exposure.” (CP 361).

Post-NIEHS studies in Japan (Kabuto *et al.*; CP 285-292) and Great Britain (Draper *et al.*; CP 294-298) produced the results establishing a definitive link between childhood leukemia and powerline exposure. As Dr. Carpenter notes, the Draper study found a “significant ($P < .01$) trend was found in relation to closeness to the power line,” in children living within 200 meters (approximately 600 feet) had elevated incidences of disease. (CP 223). The children in the Homeowners’ homes are about 30 feet from the source of exposure.

A study conducted by Infante-Rivard *et al.* (CP 279-283) found a correlation between EMF exposure during pregnancy and childhood

leukemia. A study conducted by Lowenthal *et al.* (CP 300-305) found the same correlation for adult cancers. As Dr. Carpenter wrote:

The observations of Lowenthal *et al.* and Infante-Rivard and Deadman are very important in that they demonstrate clearly that the fetus and young children are at greater risk than are adults, and that early life exposure may result in cancer many years later.

(CP 223; footnotes omitted). In other words, the cancer caused by childhood exposure may not manifest itself until adulthood. Dr. Li found exactly this kind of pattern in a study published after the *Frye* hearing between maternal exposure to EMF during pregnancy and asthma in the offspring. (*App. I*).

However, childhood leukemia is not the only health impact associated with EMF. The Executive Summary of the CDHS Report on the health risks arising from EMF exposure focuses on powerline EMF and states:

To one degree or another, all three of the DHS scientists are inclined to believe that EMFs can cause some degree of increased risk of childhood leukemia, adult brain cancer, Lou Gehrig's Disease, and miscarriage.

(CP 274). On the issue of miscarriage, the CDHS Report states:

[T]wo new epidemiology studies in humans suggest that EMFs might cause a substantial portion of miscarriages. Miscarriages are common in any case (about 10 per 100 clinically diagnosed pregnancies) and the theoretical added risk for an EMF-exposed pregnant woman might be an additional 10 per 100 pregnancies according to these two studies.^[8] If truly causal this could clearly be of concern to

⁸ Or, a doubling of the risk. This is in addition to the increased from EMF exposure during pregnancy for childhood leukemia found in the study by Infante-Rivard *et al.* (CP 279-283).

individuals and regulators. However, the type of EMF exposures implicated by these two new epidemiological studies (short, very high exposures) probably come from being within a few inches of appliances and unusual configurations of wiring in walls and grounded plumbing, and only *rarely from power lines*.

(CP 275; *emphasis added*). Except, of course, if you are living 30 feet from a massive substation like the Homeowners here.

Dr. Li's study, one of the "epidemiological studies" referenced above, found a positive correlation between EMF exposure during pregnancy and increased incidence of miscarriage. (CP 441-452).

Perhaps the most effective way to point out that these studies are recognized as valid is to point out that other scientists have consistently relied on these same studies to reach the same conclusion as Dr. Carpenter.⁹ As Dr. Carpenter testified in his deposition (CP 1059-1061 at 52:4-58:22), the most significant of these documents were two pooled analyses performed by, respectively:

1. Ahlbom *et al.*, A Pooled Analysis of Magnetic Fields and Childhood Leukaemia, *British Journal of Cancer* 83(5) (2000) (CP 1110-1116); and
2. Greenland *et al.*, A Pooled Analysis of Magnetic Fields, Wire Codes, and Childhood Leukemia, *Epidemiology* 11(6) (2000) (CP 247-257).

These pooled analyses took the data from a significant group of prior epidemiological studies and combined it, hence the term "pooled," to assess the correlation between EMF exposure and childhood leukemia

⁹ Wertheimer, Leeper, Savitz, Fechting and Ahlbom are all cited references in the NIEHS Report. (CP 870-876). Ahlbom, Fechting, Li, Savitz, Wertheimer and Leeper are all cited references in the CDHS Report. (CP 860-869).

over a larger population. This includes many of the studies relied on by Dr. Carpenter, as well as additional studies not referenced by Dr. Carpenter.

Dr. Kheifets characterizes the results of these pooled analyses as:

Two pooled analyses by Ahlbom *et al*, (2000) and Greenland *et al*, (2000), based on 9 and 12 studies, respectively, published up to 1999, have provided a basis for concluding that a consistent epidemiological association exists between residential exposure to magnetic fields and the risk of childhood leukaemia.

(CP 1118). In short, Dr. Kheifets agrees with Dr. Carpenter that the epidemiological data as of 2000 showed that “a consistent epidemiological association exists between residential exposure to magnetic fields and the risk of childhood leukaemia.” (CP 1118).

Dr. Kheifets goes on to note that the prior epidemiological studies, including these pooled analyses, were the principal basis for the characterization of EMF exposure as *possibly carcinogenic* by the WHO/IARC:

Thus, largely on the basis of epidemiological association of residential magnetic field exposure and childhood leukaemia, the International Agency for Research on Cancer [an arm of the WHO] has classified extremely low-frequency magnetic field exposure as being possibly carcinogenic to humans.

(CP 1118). Thus, the WHO/IARC drew the same conclusion *from the same data* as Dr. Carpenter, that EMF exposure is a potential human health risk.

The same is true of the CDHS Report. (CP 272-277). All three of the authors of the CDHS Report, all of whom were epidemiologists, were:

“[I]nclined to believe that EMFs can cause some degree of increased risk of childhood leukemia, adult brain cancer, Lou Gehrig’s Disease, and miscarriage.” (CP 274). The set of epidemiological studies in existence as of 2002 cited by Dr. Carpenter was relied on by the three CDHS epidemiologists in reaching the same conclusion from the same data as Dr. Carpenter – that EMF exposure is a potential human health risk. (CP 860-869).

In 2010, Drs. Kheifets and Ahlbom, among others, published the results of another pooled analysis based on peer-reviewed epidemiological research published after 2000, essentially an update of the two prior pooled analyses based on more recent data; published in the *British Journal of Cancer* 103 (2010). (CP 1118-1125). This pooled analysis includes epidemiological research by Draper in Great Britain (CP 294-298), Kabuto in Japan (CP 285-292) and Lowenthal in Tasmania (CP 300-305). The conclusion in this pooled analysis is the same as in the prior two: “[T]hat recent studies on magnetic fields and childhood leukaemia do not alter the previous assessment that magnetic fields are possibly carcinogenic [to humans].” (CP 1118). Once again, these scientists all drew the same conclusion from the same data as Dr. Carpenter – that EMF exposure creates a potential human health risk.

PSE’s Response to the Homeowners’ submissions was to refer to the materials as “the *pseudoscience* Plaintiffs present to support their claims that the Juanita substation constitutes a nuisance because of EMF levels.” (CP 541:13-15; *emphasis added*). Eneritech Consultants Inc., the company owned by PSE’s expert on measuring EMF, J. Michael Silva,

actually conducted the exposure assessment in Dr. Li's study of the relationship between EMF exposure and miscarriage. (CP 261).

We did the exposure assessment for the paper that you just asked me about, De-Kun Li. If you'll look more carefully you will see one of my employee's named as a coauthor. In fact, my wife was pregnant and picked for the study that we were doing.

(4/27/11 T.P. at 60:3-8). Enertech Consultants Inc. also conducted the exposure assessment in a recent published report by Dr. Li linking increased asthma in children whose mothers were exposed to EMF during pregnancy. (App. I).¹⁰ Pseudoscience indeed!

The Trial Court then ordered a Frye hearing (CP 645-647) with respect to Dr. Carpenter's opinions expressed in his Declaration and attached materials. (CP 186-313). In addition to the testimony of four expert witnesses – Dr. Carpenter on behalf of the Homeowners, and Dr. Mark Israel, Dr. Lee and Mr. Silva on behalf of PSE – 22 exhibits were admitted into evidence including many of the materials previously submitted by the Homeowners. The testimony added virtually nothing new.

PSE's contentions as to the admissibility of the testimony of Dr. Carpenter were as follows:

- Dr. Carpenter's "opinions are based on the novel scientific theory that power frequency electric and/or magnetic fields ("EMF") at the very low levels from the substation in this case can cause disease and illness, and therefore should be avoided." (CP 1154:9-13).

¹⁰ This study was released *after* the Order on Frye hearing. However, in reviewing an Order based on Frye, an Appellate Court may use scientific literature and other appropriate information, whether or not in the Trial Court record. State v. Cauthron, 120 Wn.2d 879, 887, 846 P.2d 502 (1993).

- Dr. Carpenter’s “non-epidemiologist approach to evaluating this research does not follow methods generally accepted in the scientific community.” (CP 1155:9-13).
- “Dr. Carpenter failed to address the large body of animal and cellular research relevant to cancer and EMF, which for the ‘mainstream cancer research community’ is ‘fundamental to our understanding of how cancers develop and what exposures may cause or contribute to cancer.’” (CP 1155:21-27).
- Lastly, PSE asserted that “Dr. Carpenter’s testimony is inadmissible on evidentiary grounds *apart from Frye*” because Dr. Carpenter could not “state to a reasonable degree of medical certainty that EMF at any level can cause” human disease. (CP 1155:33-34, 42-44; *emphasis added*).

The Trial Court essentially adopted PSE’s contentions in whole in its Summary Decision dismissing the Homeowners’ claims against PSE. (CP 1418-1422).

C. Applicable Authority and Discussion.

1. The Legal Standard for Nuisance Liability.

The Trial Court never adopted, much less articulated, what standard of liability was applicable to Plaintiffs’ claims until it entered the Order dismissing those claims. The basic problem with the Trial Court’s dismissal of the Homeowners’ nuisance claims against PSE is that the dismissal was based on an inappropriate standard for liability. The Trial Court stated in its Order on the *Frye* hearing that:

[T]he question raised on [PSE’s] Motion for a *Frye* hearing was whether Plaintiff’s [*sic*] claims (that the presence of EMFs on their property *is injurious* to the health of Plaintiffs and their families) are based on reliable medical or scientific opinion.

(CP 1418; *emphasis added*). The Trial Court went on to conclude that Dr. Carpenter’s testimony was deficient because Dr. Carpenter could not

testify “to a reasonable degree of medical certainty that EMF at any level *causes*” any human disease. (*CP 1420; emphasis in original*).¹¹

As discussed below, the issue under *Frye* was whether the *methodology* used by Dr. Carpenter to collect the data on which his opinions were based was recognized by the scientific community. Instead, the Trial Court usurped the prerogative of the finder of fact and weighed the various opinions, exactly what a Trial Court is not supposed to do – to reach a conclusion on medical causation without even articulating a standard for liability.

In point of fact, however, the Homeowners did not have to establish that EMF causes human disease – *only that it causes a reasonable apprehension of injury*. The issue in a nuisance case is not whether the plaintiff is being physically injured – the issue is whether the plaintiff’s right to possession and quiet enjoyment of real property is interfered with by the defendant’s conduct. An allegation of actual, existing physical injury to persons is not necessary to show a nuisance – all that is necessary is a showing of a reasonable apprehension of interference with the enjoyment of property. *Ferry v. City of Seattle*, 116 Wash. 648, 662-63, 203 P. 40 (1922); *Everett v. Paschall*, 61 Wash. 47, 51-53, 111 P. 879 (1910); *Harris v. Skirving*, 41 Wn.2d 200, 202, 248 P.2d 408 (1952). Conduct causing a fear of injury to health “not

¹¹ As discussed below, even PSE’s expert Dr. Lee conceded that a statistical association can be evidence of causation, as is recognized by Washington Courts. In fact, a significant-statistical association involves a higher degree of correlation than reasonable-medical certainty.

entirely unreasonable” was first held to be a private nuisance over 100 years ago in *Everett v. Paschall*, 61 Wash. 47, 111 P. 879 (1910):

We conceive the case of *Stotler v. Rochelle* (Kan.) 109 Pac. 788, to be directly in point. There we find the same contentions made as here. The question was whether the ***fear of cancer*** was sustained in the light of medical authority. The court said: ‘In the present state of accurate knowledge on the subject, it is quite within bounds to say that, whether or not there is actual danger of the transmission of the disease under the conditions stated, the fear of it is not entirely unreasonable.’

(Emphasis added).

In *Indiana Michigan Power Co. v. Runge*, 717 NE.2d 216 (Indiana 1999), the Court upheld the denial of summary judgment on both nuisance and trespass claims based on exposure to EMF on the basis that: “[A]ctual physical damage to person or property need not be alleged” to establish a nuisance. At 228-229. The fear of injury from electrical transmission lines has been recognized as a basis for “stigma” damages:

The psychological effect of an adverse condition, real or imagined, on a potential buyer may have a material influence on the market value of property. These effects and their impact on the market value have been recognized in cases involving ***the inherent fear of electricity and gas transmission lines***... It is not the landowner’s fault the adverse conditions exist; he has been damaged in the value of his property by the mere existence of a mental attitude which had a material influence on the market value of his feedlot.

State v. Evans, 26 Wn. App. 251, 612 P.2d 442 (1980), *reversed on other grounds* 96 Wn.2d 119 (*Citations omitted; emphasis added*).

From there, the issue is whether, on the basis of all the information available publicly, a reasonable person would be apprehensive of EMF

exposure. PSE has argued that the lack of regulation of EMF exposure is evidence of the lack of health risk from EMF. The argument is that, given the lack of regulation, an apprehension of risk is not reasonable. The public policy issues relating to regulation of EMF involve an entirely different set of considerations from the set of considerations which might dictate private decision making about EMF exposure. (*CP 1091-1093 at 180:4-185:10*). Typically, at the public policy level, cost-benefit is a significant issue. (*Id.*). In the CDHS Report, the discussion of regulation focused on a lack of certainty as to what potentially-costly regulation would be effective. (*CP 277*).

However, even in the arena of public policy, lack of regulation does not mean lack of recognition of risk. In recognition of risk from EMF exposure, the WHO/IARC, for example, advocates precautionary measures (*Hearing Ex. 12*), as have scientists active in the arena of EMF exposure. The EPA's publication states that a "*definitive* cause-effect relationship" between EMF and human disease cannot be confirmed *or refuted*. (*Hearing Ex. 16 at p. 1; emphasis in original*). The publication goes on to recommend that:

People concerned about possible health risks from power lines can reduce their exposure by:

Increasing the distance between you and the source – The greater the distance between you and the power lines the more you reduce your exposure.

Limiting the time spent around the source – Limit the time you spend near power lines to reduce your exposure.

(*Hearing Ex. 16 at p. 2; emphasis in original*). Unfortunately, the Homeowners don't have these options. Any potential buyer for a

residence owned by the Homeowners can exercise the options by looking elsewhere, or the Homeowners can sell their home for less.

But, the issue here is not what constitutes prudent public policy, but whether it is reasonable for private individuals to avoid exposure. Drs. Carpenter and Li were certainly of that opinion. As is, once again, Dr. Kheifets:

[W]ith regard to childhood exposure to EMFs (and exposure during pregnancy), several factors argue for the adoption of precautionary measures, including the possibility that EMFs may affect children; the dread with which some of the diseases raised in this context, such as leukemia and brain cancer, are perceived; the involuntary nature of some of the exposure; its extensiveness; and its likely rapid growth in the future.

(CP 614-615).

Precautionary measures may also be adopted at an individual level, depending on the degree of concern felt by the exposed person. In giving advice to their patients, physicians should weigh the strength of the scientific evidence for the risk, if any, of an adverse outcome, the benefits of the technology, and the feasibility of reducing exposure, as well as the overall health of the patient, which includes freedom from worry and anxiety...

Some simple options include reducing exposure by minimizing the use of certain electrical appliances or changing work practices to increase distance from the source of exposure. People living near overhead power lines should be advised that such proximity is only an indicator of exposure...

(CP 615). So were the scientists at the CDHS:

[T]o put things in perspective, individual decisions about things like *buying a house* or choosing a jogging route should involve the consideration of certain risks, such as

those from traffic, fire, flood, and crime, as well as the uncertain comparable risks from EMFs.

(*CP 275; emphasis added*).

The 18 different epidemiological studies in the record reporting a correlation between EMF exposure and human disease are in and of themselves evidence of causation:

“When [epidemiological] studies are available and relevant, and particularly when they are numerous and span a significant period of time, they assume a very important role in determinations of questions of causation.” *Richardson v. Richardson-Merrell, Inc., supra.*¹² See also *Ref. Manual* at 335 n. 2 (“Epidemiologic studies have been well received by courts trying mass tort suits. Well-conducted studies are uniformly admitted.”)

In re Silicon Breast Implant Litigation, 318 F.Supp. 879 at 892-893 (C.D. Cal 2004); *Eakins v. Huber*, 154 Wn. App. 592, 225 P.3d 1041 (2010); *Intalco Aluminum*, 66 Wn. App. 644 at 661-662, 833 P.2d 390 (a cause-effect relationship need not be proven by epidemiological studies before a doctor can testify to his opinion that such a relationship exists). And, even PSE’s expert on the causes of cancer ultimately had to admit that the epidemiological evidence should not be ignored. (4/26/11 a.m. *T.P. at 45:1-9*). Irrespective of whether Dr. Carpenter’s testimony was admissible under *Frye*, there was a substantial body of scientific evidence admitted into the record from which a finder of fact could conclude that an apprehension of injury from exposure to EMF is reasonable.

¹² *Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 830 (D.C.Cir.1988), cert. denied, 493 U.S. 882, 110 S.Ct. 218, 107 L.Ed.2d 171 (1989).

Reasonableness is, by definition, an issue of fact. The Trial Court granted the dismissal in the face of a substantial issue of fact that should have been sent to the trier of fact.

In dismissing the Homeowners' claims against PSE on the basis that Dr. Carpenter could not testify that EMF exposure caused human disease to a medical certainty, the Trial Court simply got it wrong. The issue is whether the unresolved issue of the capacity of EMF to cause human disease warrants a reasonable apprehension of exposure. Even recognizing that there is a substantial disagreement as to the level of risk, if any, from exposure to EMF, any fair-minded person reviewing the available evidence would conclude that it is, at a minimum, prudent to avoid exposure. The simple fact of the matter here is that five separate governmental agencies charged with providing information regarding public health issues – the CDC, EPA, NIEHS, WHO/IARC and CDHS – have gone on record as saying that EMF exposure may be a health risk particularly as to childhood leukemia. Would any reasonable parent, for example, take the chance that their child would contract leukemia by living in one of the Homeowners' houses? This is exactly why the Homeowners' houses are now worth substantially less than before construction of the Substation.

2. The Trial Court’s Resolution of the *Frye* Hearing Is Not Consistent With Either the Controlling Law or the Facts.

a. A Literature Review is a Generally-Recognized Method of Evaluating Health Risks from Environmental Exposures.

A *Frye* hearing is intended to determine whether scientific evidence complies with the requirements of ER 703:

The facts or data in the particular case upon which an expert bases an opinion or inference may be those perceived by or made known to the expert at or before the hearing. If of a type reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject, the facts or data need not be admissible in evidence.

The purpose of a *Frye* hearing is not to resolve differences of opinion between opposing experts but, rather, is focused on whether the data collection and analytical *methodologies* used by the expert are accepted by the scientific community as valid:

ER 703 requires the facts and data relied on by expert witnesses to be of a type reasonably relied upon by experts in the field. A *Frye inquiry addresses novel scientific methodology*; it does not deal with medical opinion based on established scientific technique.

Burns v. PACCAR, Inc., 77 Wn. App. 201 at 215-216, 890 P.2d 469 (1995) (*Emphasis added*); *State v. Gore*, 143 Wn.2d 288, 302, 21 P.3d 262 (2001) (The *Frye* test is to determine “whether the evidence offered is based on *established scientific methodology*.” *Emphasis added*). Novel conclusions are admissible when the *methodology* used to reach them is generally accepted within the relevant scientific community. *Intalco*

Aluminum, 66 Wn. App. 644 at 660, 833 P.2d 390. As PSE noted, for the purposes of a Frye hearing:

General acceptance may be found from testimony that asserts it, from articles and publications, from widespread use in the community, or from the holdings of other courts. State v. Kunze, 97 Wn. App. 832, 853, 988 P.2d 977 (1999).

(CP 1151:27-33).

It is very important to focus on the actual **methodology** by which Dr. Carpenter reached his conclusions/opinions – in other words, what were his investigational methods and are those methods recognized? In this regard, Dr. Carpenter undertook no original research – rather, Dr. Carpenter conducted a literature review of the epidemiological literature bearing on the risk of exposure to EMF. (CP 186-313; CP 1047-1104 at 48:7-58:15, 63:12-68:20, 70:4-72:23, 171:20-173:16). The fundamental issue is whether it is a generally-accepted methodology to base expert testimony on a review of scientific literature. If not, then the WHO/IARC, NIEHS and CDHS Reports would all fail to pass muster as each was based on a review of scientific literature. PSE is hardly in a position to complain that this is not an accepted methodology because it is precisely the methodology undertaken by Dr. Lee in her Declaration challenging Dr. Carpenter’s opinions. (CP 572-580).

It is also a methodology recognized by the Courts. Perhaps the most analogous case is Deluca by Deluca v. Merrill Dow, Inc., 911 F.2d 941 (C.A. 3 1990). The ruling at issue was described as follows:

The district court held Dr. Done’s testimony to be inadmissible, citing the requirement of Federal Rule of Evidence 703, that expert opinion be based on data

reasonably relied upon by experts in the relevant field. The district court reached this conclusion despite the fact that most of the data relied upon by Dr. Done was data from peer reviewed articles in medical journals that was relied upon by the authors of these articles, as well as by Merrell Dow's own expert.

At 943-944. As here, the testifying expert was not an epidemiologist. The Court stated:

Rule 703 is satisfied once there is a showing that an expert's testimony is based on the type of data a reasonable expert in the field would use in rendering an opinion on the subject at issue; it does not address the reliability or general acceptance of an expert's methodology. When a statistician refers to a study as "not statistically significant," he is not making a statement about the reliability of the data used, rather he is making a statement about the propriety of drawing a particular inference from that data. [The inference of a causal relationship].

At oral argument, counsel for Merrell Dow conceded that Merrell Dow had not specifically challenged the data Dr. Done relied upon. Indeed, with respect to most of Dr. Done's data, Merrell Dow is hardly in a position to claim that it is not of a type reasonably relied upon by experts in the field since Merrell Dow's expert relied upon the same epidemiological data from the published literature in formulating her opinion. To the extent Merrell Dow wishes to challenge particular sets of data Dr. Done has used, it is free to do so on remand. However, it has not attempted to show that Dr. Done's reliance upon particular epidemiological data is unreasonable, and the DeLucas had no burden to address arguments not made. *Cf. Dowling v. United States*, 493 U.S. 342, 110 S.Ct. 668, 673 n. 3, 107 L.Ed.2d 708 (1990) ("That the burden is on the introducing party to establish relevancy, does not also require the introducing party to anticipate and rebut possible objections to the offered evidence.").

At 953. The Court went on to state that the fact that the testifying expert was not an epidemiologist was not a valid basis for exclusion under

ER 703 as it went to the expert's qualifications under ER 702 and not the basis for his opinions.

b. The Trial Court was Obviously Confused About the Difference Between ER 703 and ER 702.

ER 702 provides:

If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise.

ER 702 goes to the *weight* to be attached to opinion testimony. The Trial Court's analysis does not speak to whether or not Dr. Carpenter's literature review was consistent with generally-accepted practice – rather, it focuses on issues which go to the weight which might be attached to Dr. Carpenter's testimony for the purposes of ER 702. This is exactly what the Trial Court is not supposed to do under *Frye*.

In rejecting Dr. Carpenter's opinions under *Frye*, the Trial Court stated:

Dr. Carpenter, who is not an epidemiologist, disregards and dismisses the majority of studies that find no evidence or insufficient evidence to conclude that EMFs, at the levels found on Plaintiffs' property [*sic*], cause such diseases as leukemia.

(*CP 1420*). The basis for the conclusion that Dr. Carpenter failed to consider the "majority" of studies finding no evidence of a causal effect is unclear as there were no such studies offered into the record. Rather, PSE only offered a conclusory opinion by Dr. Lee of what was in the studies. In other words, PSE is complaining about Dr. Carpenter's opinion of the

epidemiological data on the basis of another opinion *on the same epidemiological data*. The difference, however, is that, in the case of Dr. Carpenter, the Court can reach its own independent conclusion as to the validity of the opinions as the basis, the original epidemiological literature, is in the record.

If you compare, for example, the studies cited by Dr. Lee (*CP 572-580*) to the bibliography of 121 studies in Dr. Carpenter's paper summarizing his opinions (*CP 219-245*), both experts refer to the same studies. For example, at *CP 578*, Dr. Lee cites to studies by Davis, Schoenfeld and London for the proposition that there is no link between EMF and breast cancer. These are references 47, 48 and 49 in Dr. Carpenter's bibliography at *CP 242*. Moreover, many of the studies which Dr. Carpenter found to be significant are the same studies cited in the NIEHS, CDHS, WHO/IARC and CDC reports.

The Trial Court stated:

In addition, to his methodology of approach, Dr. Carpenter is not able to state to a reasonable degree of medical certainty that EMF at any level causes leukemia, Alzheimer's disease, or ALS. At most, he was able to state that he believed there was a statistically significant association or correlation between EMF and the diseases mentioned despite there being no animal studies to support the conclusion or no single mechanism that explains how EMF causes such diseases.

(*CP 1420*).

The discussion of statistical significance demonstrates how fundamentally confused the Trial Court was about the evidence before it.

As PSE's own epidemiologist expert Dr. Lee testified, a "statistically significant relationship" is evidence of a causal relationship:

Q So a statistically significant association between an environmental agent and the human health syndrome is evidence of a potential cause and effect relationship, correct?

A It's possible.

(4/26/11 T.P. at 44:4-8). Epidemiological studies are well recognized by Courts as evidence of causation: *In re Silicon Breast Implant Litigation*, 318 F.Supp. 879 at 892-893 (C.D. Cal 2004) ("they assume a very important role in determinations of questions of causation;" *emphasis added*).

A statistically-significant correlation is actually a higher standard than "to a reasonable medical certainty."

Q Mr. Renner used a term and I want to know if there is an accepted definition of the term. He asked you in a number of circumstances whether or not something could be characterized as creating a risk to a reasonable degree of medical certainty. What did that term mean in your mind?

A Well, to my mind, that means -- again, it's detailed in my report where we discuss levels of evidence. I think the legal term "reasonable degree of medical certainty" is equivalent to "more likely than not". It's a much weaker level of certainty than 95 or 99 percent confidence, obviously. It means greater than 50 percent chance that this is the case.

Q With respect to the significant statistical correlation between EMF and childhood leukemia, where on the scale of more likely than not would you place that correlation?

A Well, the pooled analyses show statistically significant elevations in risk, which means greater than 95 percent, so much, much stronger than more likely than not.

Q Okay.

A Now, for some of the other diseases, brain cancer, for example, it's sort of right at the more likely than not. Most studies -- most meta-analyses show a slightly increased risk but much less strong than childhood leukemia.

Q Okay. How about ALS?

A ALS and Alzheimer's both have consistently statistically significant elevations in risk, which means 95 percent confidence levels, much more stronger than more likely than not.

(*CP 1090-1091 at 176:20-178:2*).¹³

With respect to animal and mechanistic studies, this comment was based on Dr. Israel's Declaration (*CP 517-523*) and hearing testimony that there was no support from animal studies and no identified mechanism whereby EMF induces disease. Dr. Israel's testimony runs afoul of well-established legal standards. The significance of live-animal data has been questioned regularly by Courts, for very good reasons:

There are two significant disadvantages in relying on animal studies. First, when extrapolating from animals to humans, differences in absorption, metabolism, and other factors may confound results. Second, toxicological expert opinions are "almost always" based on animal studies that involve doses of a suspected carcinogen that are significantly higher than animal doses comparable to

¹³ There is an extensive discussion of statistical significance and confidence testing in *Deluca* where the court was faced with epidemiological data showing a correlation which was not statistically significant. *Id.* at 947-949.

expected human exposure. This is often necessary to obtain statistically significant predictions of the effects of realistic doses. *Ref. Manual* at 409. Extrapolation from high-dose animal studies, however, assumes a predictable relationship between dose and the probability that an exposed animal will be diagnosed with cancer.

In re Silicon Breast Implant Litigation, 318 F. Supp. 879 at 891(C.D. Cal 2004). Expert opinions based on animal data have been excluded where the expert did not establish that the test animal represented a valid basis for extrapolating animal results to human. *E.g.*, *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 144, 118 S.Ct. 512, 139 L.Ed.2d 508 (1997); *Turpin v. Merrell Dow Pharms., Inc.*, 959 F.2d 1349, 1360 (6th Cir.1992); *Hall v. Baxter Healthcare Corp.*, 947 F.Supp. 1387, 1410 (D.Or.1996) (“Extrapolations of animal studies to human beings are generally not considered reliable in the absence of a scientific explanation of why such extrapolation is warranted.”).

Animal studies are not generally admissible where contrary epidemiological evidence in humans exists. *See* *Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 830 (D.C.Cir.1988), *cert. denied*, 493 U.S. 882, 110 S.Ct. 218, 107 L.Ed.2d 171 (1989) (finding that animal studies could not establish general causation of birth defects in humans where there was an “overwhelming” amount of contrary epidemiological evidence). The simple fact is that while Dr. Israel testified that animal studies have not shown a link between EMF and cancer, particularly childhood leukemia, the epidemiological studies have.

But, perhaps the most significant testimony by Dr. Israel was the following:

Q. As you sit here today, Dr. Israel, can you tell the Court definitively that the medical community knows what causes childhood leukemia?

A. I can't speak for the whole medical community, but I would even say that I don't know what causes every case of childhood leukemia.

(4/26/11 a.m. T.P. at 43:17-22).

What it all really boils down to is a difference of opinion in the scientific community as to the significance of the various types of data available. Dr. Carpenter is of the opinion that the epidemiological data is more significant. Even PSE's expert on the causation of cancer, Dr. Israel, testified that epidemiological associations should not be ignored just because a mechanism for the induction of disease for which a correlation exists has not been found. *(4/26/11 a.m. T.P. at 45:1-9).*

The difference between Dr. Carpenter on the one hand and Drs. Lee and Israel on the other is in the interpretation and, here, a significant component of the scientific community shares Dr. Carpenter's opinion that the epidemiological data is determinative:

The three [C]DHS scientists thought there were reasons why animal and test tube experiments might have failed to pick up a mechanism or a health problem; hence, the absence of much support from such animal and test tube studies did not reduce their confidence much or lead them to strongly distrust epidemiological evidence from statistical studies in human populations. They therefore had more faith in the quality of the epidemiological studies in human populations and hence gave more credence to them.

(CP 274-275). And by Dr. Kheifets: “[E]ven consistent negative toxicological [animal] data cannot completely overcome consistent epidemiological studies.” (CP 1393).

Dr. Carpenter’s opinions are not novel and were shared by numerous members of the epidemiological community, including every one of the epidemiologists in the 18 studies in the record concluding that EMF increased the risk of human disease including the CDHS scientists:

To one degree or another, all three of the [C]DHS scientists are inclined to believe that EMFs can cause some degree of increased risk of childhood leukemia, adult brain cancer, Lou Gehrig’s Disease, and miscarriage.

(CP 274). Dr. Kheifets noted that the prior epidemiological studies, including these pooled analyses, were the principal basis for the characterization of EMF exposure as *possibly carcinogenic* by the WHO/IARC:

Thus, largely on the basis of epidemiological association of residential magnetic field exposure and childhood leukaemia, the International Agency for Research on Cancer [an arm of the WHO] has classified extremely low-frequency magnetic field exposure as being possibly carcinogenic to humans.

(CP 1118). Thus, the WHO/IARC drew the same conclusion *from the same data* as Dr. Carpenter, that EMF exposure is a potential human health risk. The weight to be attached to these opinions is an issue for a jury.

IV. CONCLUSION

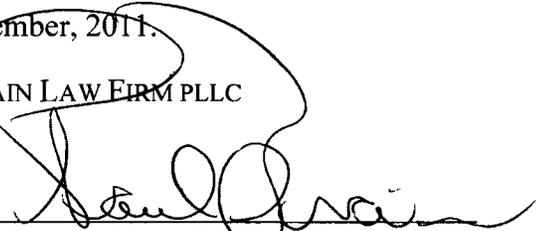
The Trial Court got absolutely nothing right. It adopted an unreasonable and highly-strained interpretation of LUPA based on clearly distinguishable case law.

The Trial Court failed to articulate, much less adopt, a standard for liability governing the issue of liability arising from EMF exposure, applied the wrong evidence rule in evaluating the Frye issues and dismissed the Homeowners' claims in the face of manifest issues of fact.

These rulings by the Trial Court were clearly in error under the applicable standard of review and, should be overruled.

DATED this 28th day of December, 2011.

BRAIN LAW FIRM PLLC

By: 

Paul E. Brain, WSBA #13438

Counsel for Appellants

CERTIFICATE OF SERVICE

I hereby certify that I have this 28th day of December, 2011, served a true and correct copy of the foregoing document upon counsel of record, via the methods noted below, properly addressed as follows:

Counsel for Respondent Puget Sound Energy Inc.:

Jeffrey M. Thomas	<input type="checkbox"/>	Hand Delivery
Jeffrey I. Tilden	<input type="checkbox"/>	U.S. Mail (first-class, postage prepaid)
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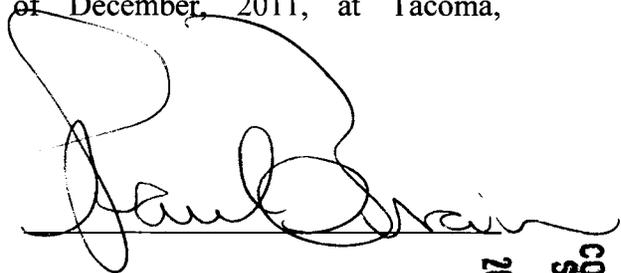
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I declare under penalty of perjury under the laws of the State of Washington that the foregoing is true and correct.

DATED this 28th day of December, 2011, at Tacoma, Washington.



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Appendix 1

ONLINE FIRST

Maternal Exposure to Magnetic Fields During Pregnancy in Relation to the Risk of Asthma in Offspring

De-Kun Li, MD, PhD; Hong Chen, MPH; Roxana Odouli, MSPH

Objective: To determine whether maternal exposure to high levels of magnetic fields (MFs) during pregnancy is associated with the risk of asthma in offspring.

Design: A prospective cohort study.

Setting: Kaiser Permanente Northern California.

Participants: Pregnant Kaiser Permanente Northern California members in the San Francisco area.

Main Outcome Measures: Asthma was clinically diagnosed among 626 children who were followed up for as long as 13 years. All participants carried a meter to measure their MF levels during pregnancy.

Results: After adjustment for potential confounders, a statistically significant linear dose-response relationship was observed between increasing maternal median daily MF exposure level in pregnancy and an increased risk of asthma in offspring: every 1-mG increase of maternal MF level during pregnancy was associated with a

15% increased rate of asthma in offspring (adjusted hazard ratio [aHR], 1.15; 95% confidence interval [CI], 1.04-1.27). Using the categorical MF level, the results showed a similar dose-response relationship: compared with the children whose mothers had a low MF level (median 24-hour MF level, ≤ 0.3 mG) during pregnancy, children whose mothers had a high MF level (> 2.0 mG) had more than a 3.5-fold increased rate of asthma (aHR, 3.52; 95% CI, 1.68-7.35), while children whose mothers had a medium MF level (> 0.3 - 2.0 mG) had a 74% increased rate of asthma (aHR, 1.74; 95% CI, 0.93-3.25). A statistically significant synergistic interaction was observed between the MF effect and a maternal history of asthma and birth order (firstborn).

Conclusion: Our findings provide new epidemiological evidence that high maternal MF levels in pregnancy may increase the risk of asthma in offspring.

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ASTHMA IS THE MOST COMMON chronic condition among children. Approximately 13% of children younger than 18 years (9.4 million children in the United States) have asthma.¹ Based on reports from the Centers for Disease Control and Prevention, asthma is a leading cause of hospitalization and emergency department visits for children younger than 18 years in the United States, with staggering annual costs of more than \$30 billion (<http://www.cdc.gov/HealthyYouth/asthma>).¹ The prevalence of asthma has been steadily rising during the last several decades, with an increase of about 74% from 1980 to 1996. While not ruling out genetic susceptibility, such a secular increase indicates the presence of important environmental risk factors that remain elusive.

Environmental exposures during pregnancy could affect fetal development of the

immune system and lungs and thus have an impact on the risk of asthma in offspring.²⁻⁵ Among the limited research, chemical exposures have represented much of the focus, while the potential of environmental physical exposures has rarely been examined. One such physical exposure is increasing man-made electromagnetic fields (EMFs). In addition to traditional low-frequency EMFs from power lines and appliances, the buildup of increasingly stronger wireless networks both inside and outside living and work spaces and the proliferation of cell phones and other wireless devices have led to human populations being surrounded by EMFs of increasing intensity. This parallel increase in both EMF exposure and asthma prevalence in the past several decades warrants examination.

Studies have shown that EMFs could adversely affect reproductive outcomes and the immune system.⁶⁻¹⁵ A recent study also

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showed an EMF effect on brain cell activities.^{16,17} Therefore, it is conceivable that exposure to high EMFs, especially during pregnancy (the period of fetal development), may have an impact on the risk of asthma in offspring. To examine this hypothesis, we conducted a prospective study based on a cohort of pregnant women whose daily exposure to magnetic fields (MFs) was captured objectively by a meter during their pregnancy and whose offspring from the index pregnancy were followed up for as long as 13 years for their asthma diagnosis.

METHODS

A prospective cohort study was conducted to examine the effect of EMF exposure on the risk of miscarriage among pregnant members of Kaiser Permanente Northern California (KPNC) in the San Francisco area who were recruited from 1996 to 1998.⁶ The members of KPNC are representative of the racially/ethnically diverse underlying population. All pregnant women who submitted a pregnancy test in the KPNC facilities of the San Francisco area were informed of the study, and those with a positive pregnancy test result were recruited for their possible participation. The study was approved by the KPNC institutional review board, and all participants signed an informed consent form.

RECRUITMENT

Women who spoke English and intended to carry the pregnancy to term at the time of recruitment were eligible for participation in the study. We recruited pregnant women early in gestation (5-13 weeks) because miscarriage usually occurs during the first trimester.⁸ All participants were interviewed in person during pregnancy to ascertain risk factors for adverse pregnancy outcomes and potential confounders. Of the original 1063 recruited women, 829 delivered a live birth. Of these offspring, 28 did not have medical records in our KPNC system, which means that they likely received their pediatric care outside the KPNC system and therefore were not included in the study.

EXPOSURE MEASUREMENT: MFs

Electromagnetic field refers to both electric fields and MFs. In this study, because the instrument we used (EMDEX-II meter; EnerTech Consultants, Campbell, California) measures only MFs, hereafter we will refer to our exposure as MFs. All participants were asked to wear an EMDEX-II meter for 24 hours during the first or second trimester so that their actual MF exposure level throughout the day from all sources could be measured objectively. The EMDEX-II meter collected MF measurements in the frequency range of 40 to 800 Hz every 10 seconds. The MF level was measured in milligauss. The meter was programmed to show only the time of day, without displaying any MF exposure level, so that participants were not aware of their MF exposure during the measurement period. This design was implemented to avoid changes of any routine daily activities due to the MF level displayed. At the end of the measurement period, the women were asked to rate their activity patterns during the measurement period as either similar to or quite different from those during a typical day of their pregnancy. Of 801 participants whose children had pediatric care at KPNC, 67 did not have complete 24-hour MF measurements. These mother-child pairs were excluded from the study.

OUTCOME MEASUREMENT: ASTHMA IN OFFSPRING

The children of the remaining 734 pairs with complete maternal 24-hour MF measurements during pregnancy were followed up until (1) they received a diagnosis of asthma, (2) they left the KPNC system (no longer a KPNC member), or (3) the end of the study period (August 31, 2010). To be considered as having a case of asthma, a child had to have received a clinical diagnosis of asthma (*International Classification of Diseases, Ninth Revision*, codes 493.00-493.99) on at least 2 occasions within a 1-year period during follow-up. We excluded those who had either only 1 diagnosis (n=67) or 2 diagnoses that were more than 1 year apart (n=17) or those who used antiasthmatic medications without a clinical diagnosis of asthma (n=24). These children were considered to have suspected asthma and formed a separate outcome group. They were not included in the main analyses but were analyzed separately for comparison. The final analyses included 626 mother-child pairs with both maternal MF measurements and a known asthma status.

POTENTIAL CONFOUNDERS

Although the number of known potential confounders are likely limited because of (1) a lack of association between MF exposure and many commonly known social, demographic, and behavioral factors and (2) the small number of known risk factors for asthma,²⁴ we evaluated many common sociodemographic characteristics and known prenatal and postnatal risk factors for asthma to ensure that they truly did not confound the association between maternal MF exposure during pregnancy and the risk of asthma in offspring. Because most variables evaluated were not confounders, we included the common sociodemographic variables such as maternal age, education, and race/ethnicity as well as the main risk factors for asthma such as a maternal history of asthma and smoking during pregnancy in the final model.

DATA ANALYSIS

We used the Cox proportional hazard regression model to examine the relationship between in utero MF exposure and the risk of asthma in offspring after controlling for potential confounders. Survival analysis has the advantage of taking into account different follow-up times for the offspring with regard to asthma diagnosis. All children were followed up starting from birth until (1) they received diagnoses of asthma (failed), (2) they left the KPNC system (censored), or (3) the end of the study (censored).

To quantify a woman's overall daily MF exposure burden, we used median 24-hour MF exposure to reflect her overall MF exposure during pregnancy to reduce the impact of outliers. Because everyone is exposed to MF at some level, we examined whether an increasing MF exposure during pregnancy is associated with an increased risk of asthma in offspring, a dose-response relationship rather than a dichotomized variable of yes/no. We first examined the dose-response relationship using the median MF level as a continuous variable. To present the association as categorical MF exposure for an easier interpretation, we divided the median MF level into 3 categories: low (≤ 10 th percentile [≤ 0.3 mG]), medium (> 10 th-90th percentile [> 0.3 -2.0 mG]), and high (> 90 th percentile [> 2.0 mG]).

RESULTS

Table 1 presents the characteristics of the study population according to their MF exposure level during pregnancy. We examined maternal, prenatal, genetic, and

Table 1. Characteristics of the Study Population

Characteristic	Median Magnetic Field (MF) Level, %			χ^2 Test (P Value)
	Low, ^a (n=81) ^d	Medium, ^b (n=482) ^d	High, ^c (n=63) ^d	
Sociodemographic factors				
Maternal age, y				.91
≤25	19.7	18.3	19.1	
26-30	32.1	31.5	31.7	
31-35	30.9	32.8	38.1	
>35	17.3	17.4	11.1	
Maternal education				.93
<College	51.8	55.8	57.1	
College	32.1	27.8	28.6	
Postgraduate	16.1	16.4	14.3	
Maternal race/ethnicity				.66
White	40.7	38.4	47.5	
Black	4.9	8.3	4.8	
Hispanic	21.0	19.5	17.5	
Asian/Pacific Islander	24.7	29.1	25.4	
Other	8.6	4.7	4.8	
Maternal prepregnancy BMI				.97
≤25	71.6	71.6	73.0	
>25	28.4	28.4	27.0	
Family income, \$.004
<30 000	24.4	18.4	13.3	
≥30 000	26.9	44.7	60.0	
≥60 000	48.7	36.8	26.7	
Prenatal factors				
Smoke during pregnancy				.90
Yes	8.6	9.5	7.9	
No	91.4	90.5	92.1	
Infection in pregnancy				.66
Yes	34.6	32.6	38.1	
No	65.4	67.4	61.9	
Antibiotic use in pregnancy				.48
Yes	34.6	41.3	42.9	
No	65.4	58.7	57.1	
Mode of delivery				.66
Vaginal birth	77.3	79.7	83.6	
Cesarean section	22.7	20.3	16.4	
Genetic factor				
Maternal history of asthma				.85
Yes	8.6	7.1	6.3	
No	91.4	92.9	93.7	
Infant factors				
Breastfed				.89
Yes	88.9	91.7	90.5	
No	11.1	8.3	9.5	
Sex				.66
Female	44.4	49.4	46.1	
Male	55.6	50.6	53.9	
Parity				.48
First child	51.9	45.6	50.8	
Not first child	48.1	54.4	49.2	
Low birthweight, <2500 g				.07
Yes	9.9	4.1	3.2	
No	90.1	95.9	96.8	
Preterm, <37 wk				.95
Yes	7.4	7.5	6.3	
No	92.6	92.5	93.7	
KPNC member at the end of follow-up				.92
Yes	58.0	60.4	60.3	
No	42.0	39.6	39.7	
NICU admission				.34
Yes	11.8	7.9	5.1	
No	88.2	92.1	94.9	
Use of antibiotics before the first diagnosis of asthma				.10
Yes	84.8	87.3	77.4	
No	15.2	12.7	22.6	
Other factors				
MF level measured on a typical day				.99
Yes	64.2	63.9	63.5	
No	35.8	36.1	36.5	

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); NICU, neonatal intensive care unit; KPNC, Kaiser Permanente Northern California.

^a Less than or equal to the 10th percentile (≤0.3 mG).

^b Greater than the 10th percentile to the 90th percentile (>0.3-2.0 mG).

^c Greater than the 90th percentile (>2.0 mG).

^d The following 3 variables had missing data: family income (n=32), maternal mode of delivery (n=22), and NICU admission (n=24).

Table 2. Maternal Exposure to Magnetic Fields (MFs) During Pregnancy and the Risk of Asthma in Offspring

Maternal Daily Median MF Level	Asthma in Children		cHR (95% CI)	aHR* (95% CI)
	Yes	No		
Continuous MF level, mean ^b (SD), mG	1.22 (1.22)	0.98 (1.09)	1.12 (1.02-1.23)	1.15 (1.04-1.27)
MF level in category, No. (%)				
Low, ≤10th percentile	11 (13.6)	70 (86.4)	1 [Reference]	1 [Reference]
Medium, >10th-90th percentile	98 (20.3)	384 (79.7)	1.65 (0.88-3.08)	1.74 (0.93-3.25)
High, >90th percentile	21 (33.3)	42 (66.7)	3.16 (1.52-6.57)	3.52 (1.68-7.35)

Abbreviations: aHR, adjusted hazard ratio (adjusted for maternal age, race, education, smoking during pregnancy, and a history of asthma; further adjustment for the remaining variables in Table 1 did not materially change the results); cHR, crude hazard ratio; CI, confidence interval.

^aTrend test, $P < .001$.

^bMean of median.

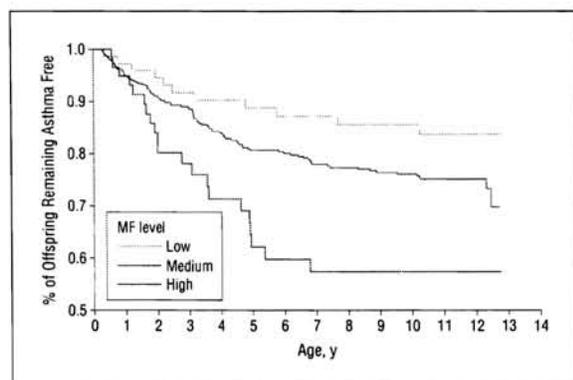


Figure 1. Kaplan-Meier estimates of asthma risk by maternal magnetic field (MF) exposure level during pregnancy.

infant factors that may be related to MF exposure, the risk of asthma, or both (ie, potential confounders). Of the 19 factors examined, none was related to MF exposure level except family income, which did not show a clear pattern of a relationship (Table 1). The percentages of children who were unavailable for follow-up at the end of the study because of their exiting KPNC membership and those whose MF exposure was measured on a typical day during pregnancy were quite similar among all MF exposure levels (Table 1).

Overall, 130 children (20.8%) of the study participants developed asthma during 13 years of follow-up, with most cases (>80%) diagnosed by 5 years of age. **Table 2** presents the results examining the dose-response relationship between increasing maternal MF exposure level in pregnancy and the risk of asthma in offspring using MF exposure level as both a continuous and a categorical variable. After adjustment for maternal age, race, education, smoking during pregnancy, and a history of asthma, a statistically significant linear dose-response relationship was observed between increasing maternal median daily MF exposure level in pregnancy and an increased risk of asthma in offspring (adjusted hazard ratio [aHR], 1.15; 95% confidence interval [CI], 1.04-1.27). In other words, 1 unit (1 mG) of increase in the maternal median MF exposure level during pregnancy was associated with a 15% increased rate of asthma in offspring (Table 2). Using the categorical MF level (low, medium, and high) as dummy variables, the results confirmed the linear dose-response relationship: compared

with children whose mothers had a low MF level (<0.3 mG) during pregnancy, children whose mothers had a medium MF level (>0.3-2.0 mG) had a 74% increased rate of developing asthma (aHR, 1.74; 95% CI, 0.93-3.25). Furthermore, children whose mothers had a high MF level (>2.0 mG) during pregnancy had more than a 3.5-fold increased rate of developing asthma (aHR, 3.52; 95% CI, 1.68-7.35). Further adjustment for the remaining 14 factors, including family income, listed in Table 1 did not materially change the results. Finally, a similar association was also observed using suspected asthma cases, although the association was weaker, perhaps because of the misclassification of asthma cases. The aHRs were 1.24 and 1.41 for medium and high maternal MF exposure levels, respectively.

The **Figure** shows the Kaplan-Meier survival curves for the percentages of offspring who remained free of asthma during the 13-year follow-up period for 3 different maternal MF exposure levels in pregnancy. The cumulative asthma risks (1 - cumulative survival rate) in offspring were 0.16, 0.30, and 0.43 for low, medium, and high maternal MF exposure levels, respectively.

To determine whether other factors would modify the observed association, we examined the association stratified by 2 known risk factors for asthma: maternal history of asthma (a possible genetic risk factor) and firstborn child (a possible environmental risk factor, the hygiene hypothesis).²⁻⁵ **Table 3** shows that the observed association was noticeably stronger among the children whose mothers had a history of asthma (aHR, 6.06; a more than 6-fold increased rate of asthma for 1 unit [1 mG] of increase in MF level in the maternal median MF exposure level during pregnancy) than among those whose mothers did not have a history of asthma (aHR, 1.12). Similarly, the association between increasing maternal MF exposure levels in pregnancy and the risk of asthma in offspring was stronger among firstborn children (aHR, 1.40; a 40% increased rate of asthma for every 1 unit [1 mG] of increase in MF level) than among later-born children (aHR, 1.07) (Table 3). The presence of these 2 risk factors (ie, history of maternal asthma [$P < .005$] and being a firstborn child [$P < .05$]) significantly exacerbated the adverse effect of maternal MF exposure in pregnancy on the risk of asthma in offspring.

Table 3. Maternal Exposure to Magnetic Fields During Pregnancy and the Risk of Asthma in Offspring in Relation to Other Risk Factors for Asthma

Other Risk Factor for Asthma	Total No.	Asthma in Children, Mean (SD)		aHR (95% CI)	P Value
		Yes	No		
Maternal history of asthma					<i>P</i> < .005
Yes	45	1.17 (0.87)	0.65 (0.49)	6.06 (2.20-16.72)	
No	581	1.22 (1.25)	1.01 (1.11)	1.12 (1.01-1.25)	
Birth order					<i>P</i> < .05
First child	294	1.33 (1.31)	0.96 (0.88)	1.40 (1.16-1.70)	
Not first child	332	1.13 (1.14)	1.01 (1.25)	1.07 (0.92-1.25)	

Abbreviations: CI, confidence interval; aHR, adjusted hazard ratio (adjusted for maternal age, race, education, smoking during pregnancy, and a history of asthma; further adjustment for the remaining variables in Table 1 did not materially change the results).

Table 4. The Strengths of the Association in Relation to the Measurement Accuracy of Magnetic Fields (MFs)

Maternal Daily Median MF Level	Asthma in Children, No. (%)		aHR (95% CI)
	Yes	No	
Measured on a typical day			
Low, ≤10th percentile	5 (9.6)	47 (90.4)	1 [Reference]
Medium/high, >10th percentile	73 (21.0)	275 (79.0)	2.52 (1.01-6.30)
Measured on a nontypical day			
Low, ≤10th percentile	6 (20.7)	23 (79.3)	1 [Reference]
Medium/high, >10th percentile	46 (23.3)	151 (76.7)	1.31 (0.55-3.13)

Abbreviations: CI, confidence interval; aHR, hazard ratio (adjusted for maternal age, race, education, smoking during pregnancy, and a history of asthma).

COMMENT

In this prospective cohort study, we found that a high maternal MF exposure level in pregnancy is associated with a significantly increased risk of asthma in offspring. The observed association showed a dose-response relationship. Given the lack of understanding of the causes of asthma, our findings could open up a new research area to elucidate risk factors of asthma that are unknown and have not been examined before. Also, our study provides new findings for the potential adverse health effect of MF exposure on an end point (asthma) that, to our knowledge, has not been previously studied. While the public has been increasingly aware of EMF exposure owing to the increasing presence of infrastructure of wireless networks and the pervasive use of wireless devices, studies on EMF health effects remain limited. Because EMF exposure is ubiquitous and exposure to it is involuntary, these new findings have important public health implications. Nevertheless, they need to be replicated by other studies.

While prenatal risk factors for asthma are not well understood, pregnancy is one of the most influential periods when allergic sensitization (atopy) is developed in the fetus.^{2,18,19} The underlying pathogenesis of asthma is likely structural and due to functional defects in epithelium and an impaired innate immune system.³ Prenatal exposure to high MF levels could interfere with the development of both epithelial cells and normal immune systems. Research by multidisciplinary collaborative studies is needed to understand these mechanisms.

The current study has several methodological strengths that enhanced the validity of the new findings. First, it was

a prospective cohort study in which MF exposure was measured in pregnancy, long before the diagnosis of asthma in offspring. This study design substantially reduces the likelihood of potential biases associated with participation influenced by the presence of outcomes. Second, both the exposure (MF levels) and the outcome (diagnosis of asthma) in this study were measured objectively without the knowledge of each other, thus reducing the concern of recall bias associated with the ascertainment of exposure and outcome variables that has existed in many epidemiological studies. Unlike many case-control studies of the MF health effect, in which MF exposure in the etiologically relevant period of the past was either reconstructed or surrogated by the current exposure measurement (eg, studies of childhood leukemia), MF exposure levels in this study were prospectively measured during the etiologically relevant period (eg, pregnancy). Also, while EMF exposure measurement in past studies was frequently based only on recalls, surrogate measures, and home spot measurements, the current study asked participants to carry an EMDEX-II meter that objectively captured their MF exposure from all sources during pregnancy. Furthermore, all diagnoses of asthma were based on clinical records, not on self-report by the participants, thereby reducing measurement errors of the outcome of interest. Finally, MF exposure is not related to most sociodemographic, behavioral, and commonly known risk factors (Table 1).^{6,9} Given that confounders have to be associated with the exposure of interest, a lack of association between MF exposure and those factors limits the number of potential confounders, making the observed association robust against potential biases.

While, compared with previous studies, we improved the accuracy of measuring MF exposure by asking participants to wear an EMDEX II meter for 24 hours, it was not feasible to measure MF exposure throughout pregnancy. Therefore, the accuracy of the MF measurement in reflecting the MF exposure in pregnancy may still be questioned, although one study has reported that MF exposure levels were relatively stable within 12 to 36 months.²⁰ Assuming that there was some misclassification of MF exposure because of measurement errors, given that this was a cohort study and MF was measured long before the diagnosis of asthma, such misclassification would be non-differential (ie, the same degree of misclassification to both mothers of children with and without asthma). Nondifferential misclassification generally leads to attenuation of observed associations. Without such misclassification, the observed association could have been stronger. In fact, our reanalysis of the association, stratified by whether the MF measurement was conducted on a typical day of pregnancy (more representative of MF exposure in pregnancy) or a nontypical day (less representative of MF exposure in pregnancy, thus more measurement errors) provided evidence supporting this argument. As shown in **Table 4**, we indeed observed that less measurement error (ie, measured on a typical day) led to a stronger observed association (>2.5 times risk of asthma associated with a higher maternal MF exposure level during pregnancy) compared with more measurement error (ie, measured on a nontypical day), a nonstatistically significant 31% increased risk of asthma. Therefore, had we been able to measure participants throughout pregnancy, the observed association between maternal MF exposure in pregnancy and the risk of asthma might have been stronger than that presented in Table 2.

In addition to observing an association between high maternal MF exposure during pregnancy and the risk of asthma in offspring with a dose-response relationship, we also observed a statistically significant interaction between the MF effect on asthma and the other 2 risk factors for asthma: maternal history of asthma and birth order (firstborn). A maternal history of asthma is a well-established risk factor for genetic susceptibility that has been supported by the results of both genome-wide association studies and candidate gene studies.^{2,5} Such an interaction with known risk factors for asthma not only revealed possible synergistic adverse effects between prenatal MF exposure and these 2 risk factors on the risk of asthma but also provided further support for the underlying association between maternal MF exposure in pregnancy and the risk of asthma in offspring. Synergistic factors themselves are often independent risk factors.

In conclusion, the findings of the present study open up a new area in understanding the risk factors for asthma and the health effects of ubiquitous MF exposure, especially during pregnancy. As with any epidemiological study, these findings need to be replicated. If confirmed, they have the potential to inform new intervention strategies to reduce asthma, the most prevalent chronic disease among children.

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