



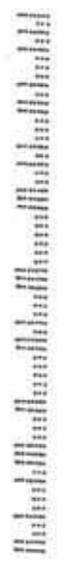
Raymond Paulk
11657 Harvest Moon Ct.
South Jordan, UT 84095-8006

SALT LAKE CITY UT 841
12 SEP 2011 PM 2 V



*Bureau of Land Management
Gateway West Project
P.O. Box 20879
Cheyenne, WY. 82003*

82003-7012



Dear, Bureau Of Land Management,

I Raymond Gene Paulk and my father Doyle Gene Paulk are the proud owners of 20 acres of unspoiled land, a creek on it pine trees, Pinion Pines, within walking distance from South Fork of the fountain Fontelle Creek. My father and I are avid hunters & fishermen.

We purchased this beautiful land 11 yrs ago have for all those years contributed to the State of Wyoming.

We will not just stand by and let you destroy what we have purchased and enjoy.

These lands are a gateway to the Bridger Mountains.

So find another route for your foreign made monsters.

I will see you in the meetings opposing your Northern route in section 14. without modifications.

P.S. owners for 11 yrs.

Signed, Raymond Gene Paulk

11657 south Harvest
Moon Cot.
South Jordan, Ut. 84095

2011 SEP 14 AM 10:00

RECEIVED
001-BLM
CHEYENNE, WYOMING

R Bishop
PO Box 1773
Glenrock, WY
82637



Bureau of Land Management
Gateway West Project
PO Box 20879
Cheyenne, WY
82003



8200337014





2011 OCT 31 AM 10:50
RECEIVED
DOI-BLM
CHEYENNE, WYOMING
10/27/11

To: Bureau of Land Management

Re: Gateway West Transmission Line Project

Wyoming's environment is already impacted by numerous easements for pipelines, utilities, etc. The best way for The Gateway project to have the least environmental impact would be to follow existing utility right of ways without crossing additional municipal or private property. If it is absolutely necessary to cross private property, it should be parallel and adjacent to existing utility easements.

This approach will have the least impact upon private and public lands and will not spoil existing uses or undeveloped terrain.

I am opposed to the project's plan to cross Glenrock's south municipal boundary and the creation of a new utility corridor through the Laramie Range and Shirley Basin.

Sincerely,

Rick Bishop
P. O. Box 1773
833 South Second
Glenrock, WY 82637

From: jmclain@blm.gov
Sent: Wednesday, October 19, 2011 6:53 AM
To: blm@gwcomment.com
Subject: Fw: gateway project

----- Forwarded by Joy McLain/WYSO/WY/BLM/DOI on 10/19/2011 07:52 AM -----

"rockenc"
<rockenc@vistabea
m.com> To
<Gateway West WYMail@blm.gov> cc
10/18/2011 05:18 PM Subject
gateway project

Dear BLM

We the people that have lived here in Wyoming do not need any more junk. "transmissions lines" We have plenty of power lines now and do not need any more lines running in, out and through our Scenic State, that is know for its beauty. This would include personal property, BLM grounds and government lands. These lines will only bring in a abundance of low poverty stricken workers. The workers of the gateway transmissions lines will carry diseases, crime and low intellectual mentality into our Wyoming communities thus, infecting and impregnating our towns, cities and communities with this cancerous mentality. Proof of this can be seen and felt in communities such as Arlington, Casper and Hanna Wyoming. This same synopsis also applies to the oil and gas industries working in and around our Wyoming communities.

Regards
Rick Cronk



"Chris Gertschen"
<cgertschen@cox.net>

10/25/2011 03:39 PM

Please respond to
<cgertschen@cox.net>

To <Gateway_West_WYMail@blm.gov>

cc

bcc

Subject BLM, Gateway West Project

Walt George
Bureau of Land Management

Dear Mr. George:

Please include our comments in the record of the public poll and your National Environmental Policy Act study concerning the Gateway West transmission line project.

While we remain dubious about the need and the cost of the overall project, we strongly support the alternative with the lowest deleterious environmental impacts. We own a home and property in Hagerman, Idaho and are particularly concerned about that portion of the project. Segment Alt. 8A has far more impact than "Segment 8". We strongly support Segment 8 which minimizes deleterious impacts on:

Plant and wildlife habitat
Public access and recreation (Billingsly State Park and Wildlife Management Area)
Visual resources, and
Developed property.

Thank you for providing the opportunity to comment and thank you for your time.

Sincerely,

Robert and Christine Gertschen
2567 Vineyard Alley
Hagerman, Idaho 83332
Mail to: P.O. Box 2166, Sun Valley, Idaho 83353

From: info@gatewayeis.com
Sent: Sunday, October 23, 2011 4:19 PM
To: Gateway BLM
Subject: A comment from gatewayeis.com

Name:
Robert and Joanne Holland

Organization:

Mailing Address:
609 W Lakeshore Dr

Mailing Address 2:

City:
Cocoa

State:
FL

Zip:
32926

Daytime Phone:
321-338-0333

E-mail:
rjholl0323@gmail.com

Confidential:
No

DEIS Location:

Comment:

My wife and I have been coming to Idaho since 1989 in order to paraglide and hang glide. We fly the area south of Declo, from Mt Harrison down through the East Hills. Your lines would impact the airspace such that we would no longer be able to pursue our sport. Our instructor, Frank Gillette, lives at the base of the Albion grade and very frequently has students out training in this area. Your lines need to be routed away from the hills and mountains south of Declo, ID. Our lives would be endangered by possibly getting tangled in your power lines. Pilots from all over the world have come here to fly. Your action would negatively impact the tourist trade in the area relating to free flight. Please consider routing your lines away from the hills and mountains.

Bureau of Land Management
Gateway West Project
PO Box 20879
Cheyenne, WY 82003

BOISE ID 83725
24 OCT 2011 PM 2 J



100496

Bureau of Land Management
Gateway West Project
PO Box 20879
Cheyenne, WY 82003

6200337018



100496

In Reference To:
Gateway West Transmission Line
Segment 9

CHEYENNE, WYOMING

RECEIVED
DOI-BLM

2011 OCT 28 AM 10:00

To Whom It May Concern:

My name is Robert A. Thomas, and I reside at 17947 Shortcut Rd. in Oreana, Idaho. Here, my family ranches in the Brown and Catherine Creek valleys. This area was settled some 140-150 years ago and several early homesteads are still visible today. Typically, these developments are crude living quarters called dugouts. There are at least four of these early dwellings still standing today. In 1905, a family named Adcock bought this ranch and operated it until 1996 when my family purchased it from Shirley (Adcock) Murdoch. It is my family's intentions to own and operate this ranch for several generations to come. I have two sons who both live and work on the ranch. My oldest son has a 5 month old daughter and I am sure my youngest, Logan, anticipates on having long and productive lives in stewardship of the ground.

The little community of Oreana is just one area directly and adversely affected by the proposed route in segment 9. As a member of the Owyhee County Task Force—we were unified in our goal of developing alternatives to the proposed route. The current proposed route dissected valleys like ours and forever disfigures communities such as Bruneau, Little Valley, Grand View as well as Castle Creek and Catherine Creek, which comprise Oreana. Little thought was given to the proposed route as evidenced by the fact that this route, chosen by the BLM—Idaho Power—Rocky Mountain power, transverses privately owned ground—where in fact Owyhee County is nearly 80% publically owned. This astonished me, particularly when the corridors within which this transmission line lies, according to SEC.368 Energy Right- of -Way Corridors, should be placed on Federal grounds whenever possible. It appears to me that the BLM in particular, was not fond at the idea of placing this on said Federal ground. Our committee developed two routes 9D & 9E which nearly eliminated the project crossing private property. Our belief was that since the project was of a benefit to the public, the public i.e. BLM, should bear the burden where possible, of accommodating Gateway West. This seems logical and we wondered why the parties concerned adopted a route so adversarial to the private landowner. It was apparent that little consideration was given to our isolated valleys and communities. As I have alluded to earlier, these valleys are important to us and represent the corridors of our lives. To permanently disfigure these privately owned valleys with steel towers of 200 feet in height and power lines transcending for miles across these ribbons of green would be a travesty. Remember, a development of these monumental proportions is a forever project... it will not go away with time.

1/2

In closing, I am optimistic that the determining bodies will look at two alternate routes available, 9D & 9E. The proposed route, Segment 9, is definitely the least appealing. Thank you for your time and consideration.

Sincerely yours,

Robert A. Thomas





TINA HARPER
535 W. Yellowstone
Casper, WY 82601
307-266-1600



*Bureau of Land Management
Hatchery Wild Project
P.O. Box 20875
Cheyenne, WY 82003*

#200337014





2011 OCT 27 AM 10:00
RECEIVED
DOI-BLM
CHEYENNE, WYOMING

11

October 25, 2011

Bureau of Land Management
Gateway West Project
P.O. Box 20879
Cheyenne, WY 82003

RE: Transmission Line Project Comment

We would like to encourage the management team for the Gateway West Project to use an alternate route for the line near Glenrock, Wyoming. Moving the line further south, Route 1E-A, along existing high power routes, would affect fewer people for this portion of the project.

The proposed route between the town of Glenrock and I-25 will definitely be visible from homes in Glenrock. This is also the main road for the residents and visitors coming into and leaving Glenrock which commands a picture of pride. The town of Glenrock has planned development of that area for future growth and we're pretty sure a high powered transmission line isn't in their beautification concept. The visual impact alone will be enormous for the residents of Glenrock and visitors alike with the current route.

We appreciate all the work you've done to provide power to the masses but we would not appreciate having a high power transmission line as a part of our view of the mountains on a daily basis.

Thank you for the opportunity to comment,

Tina & Bob Harper
835 Grove
P.O. Box 844
Glenrock, WY 82637

Bureau of Land Management
Gateway West Project
PO Box 20879
Cheyenne, WY 82003



PO BOX 20879
CHEYENNE WY 82003

Bureau of Land Management
Gateway West Project
PO Box 20879
Cheyenne, WY 82003

100321

8200347016



Draft EIS Comment Form

Gateway West Transmission Line Project

Draft EIS comment period: July 29, 2011 - October 28, 2011

100321

2011 NOV - 1 AM 10:00
RECEIVED
DOI-BLM
CHEYENNE, WYOMING

BLM

Date: 10/28/2011

First Name: Robert LaDean

Last Name: Barnard

Organization or Office Name: _____

Mailing Address: P.O. Box 305

City: Rockland State: ID Zip: 83271

Daytime Phone: 208-548-2643

Email: barnard-RL1@msn.com

Please check here if you wish for your personal information to remain confidential*

*If you wish for your contact information to remain confidential, BLM will protect the personal information that you submit to the extent allowed by law. However, the information may be subject to the Freedom of Information Act (U.S.C. etc.). See privacy note on reverse.

Please submit your comments by October 28, 2011. Information submitted on this form is being voluntarily provided solely for the purpose of commenting on the Gateway West Transmission Line Project.

Comment:

Rockland, Idaho is a small, rural farming community where we all stick together to do the right thing for the betterment of the whole Rockland Valley.

When this project began and found out about possible towers (electric transmission) running across our farm ground, I was very opposed to this. Besides it is de-valuing our farm ground, but it also will cause access road entries as well so these towers will have no benefit to our town, region, or even state.

Our farm is in Segment 7 on your maps



To mail this comment form please send to:

Bureau of Land Management | Gateway West Project | P.O. Box 20879 | Cheyenne, WY 82003

Comments may also be submitted via email to: Gateway_West_WYMail@blm.gov or online at www.wy.blm.gov/nepa/cfodocs/gateway_west

1/2

continued on back

Name:

Robert L. Barnard

between East Fork and Sand Hollow Roads. We will be in line to get multiple towers on our farm land on the "MILE RADIUS" projection.

We bluntly do not want towers on our land nor do we want anything to do with this project. Our land is posted with NO TRESPASSING and we will prosecute if needed. We hope that this demonstrates our position on this matter.

Privacy Note: Comments, including names and addresses of respondents, will be made available for public review after the close of the official comment period. Before including your address, phone number, email address or other personal identifying information with your comments, please be advised that your entire comment, including your personal identifying information, may be made publicly available at any time. Although you may ask the BLM in your comment to withhold your personal identifying information from public review, we cannot guarantee that we will be able to do so. All submissions from organizations and businesses, and from individuals identifying themselves as representatives or officials of organizations or businesses, will be available for public inspection in their entirety.

From: info@gatewayeis.com
Sent: Friday, October 21, 2011 1:33 PM
To: Gateway BLM
Subject: A comment from gatewayeis.com

Name:
Robert Love

Organization:

Mailing Address:
303 Morning Glory Lane

Mailing Address 2:

City:
Sandpoint

State:
ID

Zip:
83864

Daytime Phone:
208 255 4048

E-mail:
rblove2519@yahoo.com

Confidential:
No

DEIS Location:
chapter 2 section 4 page 2-43

Comment:

I am writing to express concern that the route proposed in the DEIS for Section 4 undermines the state of Wyoming's efforts at sage grouse conservation. Recognizing the problems with sage grouse habitat faced by Gateway West in Section 4, the governor specifically modified the state's sage grouse conservation plan to establish a corridor for power lines through sage grouse core areas. The DEIS rejects these efforts in favor of the proposed route which destroys an entirely undamaged swath of sage grouse habitat and crosses two core areas. This is the kind of reckless exploitation of the state which has brought us to the point where something like the listing of sage grouse as an endangered species will seriously limit future development. Please make the responsible decision and use the 4A Alternate route instead.

Robert Love
Homeowner - parcel number 2415-063-00-006

From: info@gatewayeis.com
Sent: Wednesday, October 26, 2011 1:09 PM
To: Gateway BLM
Subject: A comment from gatewayeis.com

Name:
Robert Love

Organization:

Mailing Address:
303 Morning Glory Lane

Mailing Address 2:

City:
Sandpoint

State:
ID

Zip:
83864

Daytime Phone:
208 255 4048

E-mail:
rblove2519@yahoo.com

Confidential:
No

DEIS Location:
chapter 2 section 4 page 62

Comment:

I am writing in regard to the assertion that using the existing power line corridor (the 4A Alternative) in Section 4 will ruin the Oregon Trail. For starters whatever is there to be ruined was already ruined by the existing power lines. Additional lines would do minimal additional damage and would spare areas on the proposed route which have not been damaged at all. Secondly what could be a more fitting tribute to the brave pioneers who triumphed over adversity, or died trying, than a massive army of towers following the path they made and carrying electrical power west to make life easier for the descendants of the survivors. The vast majority of these pioneers saw the Oregon Trail and particularly Wyoming as an obstacle to be overcome not something to be cherished. If they could have, they would have built something like an interstate highway. As it is, the scars they left with total disregard to any environmental concerns are still clearly visible after more than 150 years. Furthermore it's not as though the proposed route does no damage to the Oregon Trail and other archeologically and historically significant locations. The damage will just be in other locations many of which are not as well identified as those in the existing corridor.

It seems the BLM employs certain archeologists who find glory and reward in making a fuss about anything to do with the Oregon Trail. It's the job they were hired for. They can summon emotions which, if I were to use, would cause you to think or even say "get a grip" or "be civil." Don't let these people manipulate you into a bad choice locating this power line.

Robert Love

Homeowner - parcel number 2415-063-00-006



BLM
Gateway West Project
Box 20879
Cheyenne, WY 82003

82003#7018



Draft EIS Comment Form

Gateway West Transmission Line Project

Draft EIS comment period: July 29, 2011 - October 28, 2011



BLM

Date: 10/26/11

First Name: Robin ~~Brown~~

Last Name: Brown

Organization or Office Name: _____

Mailing Address: 120 W Maple St.

City: Bowling State: WY Zip: 82301

Daytime Phone: 321-2378

Email: nibor-22@yahoo.com

Please check here if you wish for your personal information to remain confidential*

*If you wish for your contact information to remain confidential, BLM will protect the personal information that you submit to the extent allowed by law. However, the information may be subject to the Freedom of Information Act (U.S.C. etc.). See privacy note on reverse.

Please submit your comments by October 28, 2011. Information submitted on this form is being voluntarily provided solely for the purpose of commenting on the Gateway West Transmission Line Project.

Comment:

The BLM mission Statement
states that:

" To sustain health, diversity
and productivity of public lands
for the use and enjoyment of
present and future generations."

With that said, I am
in favor of the "red route"
on the Project Overview Figure
A-1 map away from Fort
S Steele and the human and



To mail this comment form please send to:

Bureau of Land Management | Gateway West Project | P.O. Box 20879 | Cheyenne, WY 82003

Comments may also be submitted via email to: Gateway_West_WYMail@blm.gov or
online at www.wy.blm.gov/nepa/cfdocs/gateway_west

continued on back

Name:

Robin Brown



2011 OCT 31 AM 10:00

RECEIVED
DOI-BLM
CHEYENNE, WYOMING

would like currently living there.

Sincerely
Robin Brown

Privacy Note: Comments, including names and addresses of respondents, will be made available for public review after the close of the official comment period. Before including your address, phone number, email address or other personal identifying information with your comments, please be advised that your entire comment, including your personal identifying information, may be made publicly available at any time. Although you may ask the BLM in your comment to withhold your personal identifying information from public review, we cannot guarantee that we will be able to do so. All submissions from organizations and businesses, and from individuals identifying themselves as representatives or officials of organizations or businesses, will be available for public inspection in their entirety.

100526

DEIS Comment by R. Thompson:

CC: Governor Butch Otter, Idaho
C/o John Chatburn
Administrator
Idaho Office of Energy Resources
304 N. 8th St. Ste. 250
Boise, Idaho 83720-0199

Rep. Paul Labrador
C/o Kristy Starnes Staff Assistant
33 Broadway Ave. Suite 251
Meridian, Id. 83642

Sen. Jim Risch
350 N. 9th St. Suite 302
Boise, Id 83702

Sen. Mike Crapo
C/o Bryan Pickett Regional Director
251 East Front Street
Suite 205
Boise ID 83702

Draft EIS Comment Form

100526

Gateway West Transmission Line Project

Draft EIS comment period: July 29, 2011 - October 28, 2011

Date: October 22, 2011

First Name: Robyn

Last Name: Thompson

Organization or Office Name: Owyhee County Task Force

Mailing Address: 16990 Short Cut Rd City: Oreana State: ID Zip: 83650

Daytime Phone: 208-834-2337

Email: _____

Please check here if you wish for your personal information to remain confidential*

*If you wish for your contact information to remain confidential, BLM will protect the personal information that you submit to the extent allowed by law. However, the information may be subject to the Freedom of Information Act (U.S.C. etc.). See privacy note on reverse.

Please submit your comments by October 28, 2011. Information submitted on this form is being voluntarily provided solely for the purpose of commenting on the Gateway West Transmission Line Project.

Comment:

All comments will be concerning
Segment 9

Comment: Refers to 1.5.2 West Wide
Energy Corridors Ch.1 pg 1-23.

The initial scoping process for
the Gateway West Transmission Line
Project in Owyhee County was flawed.

I am enclosing the article which
appeared May 28, 2008 in The Owyhee
Avalanche, announcing a public scoping
meeting to authorize ROW for
Gateway West on BLM land. The

To mail this comment form please send to:

Bureau of Land Management | Gateway West Project | P.O. Box 20879 | Cheyenne, WY 82003

Comments may also be submitted via email to: Gateway_West_WYMail@blm.gov or
online at www.wy.blm.gov/nepa/cfdocs/gateway_west

2/90

continued on back

BLM



public was not notified regarding the impact of this public utility project on private land. As a result only 13 people attended the June 3, 2008 public scoping meeting held in Murphy, ID. The citizenry of Owyhee County was not adequately represented in this initial scoping process resulting in considerable time, labor and expense once the consequence of proposed seq 9 came into our awareness spring 2009.

President George Bush signed the Energy Act in 2005 stipulating in sec. 268 the establishment of energy right-of-way corridors on Federal land. These corridors are to be on Federal land following existing ROW's (i.e. roads, existing transmission lines per the Federal Land Policy and Management Act of 1976) and were to be incorporated into agency land use and resource management plans.

This is not what occurred in Owyhee County. To put things in perspective:

Owyhee County is comprised of 4,894,810 acres of which 3,733,499 are administered by the BLM; 352,000 are owned by the state, leaving only 1,126,111 acres to private land. The West Wide Energy corridor within Owyhee County affects 18.4 miles of private land, encompasses 1.1 miles of state land and only 37.6 miles of Federal land. This corridor was supposed to be established on Federal land, which there is plenty of in southwest Idaho, not on our prime farmland and homes; the heart of Owyhee County!

It must be noted that while the WWE corridor was established affecting our most valuable agricultural and residential property the Snake River Birds of Prey National Conservation Area adopted its Resource Management Plan prohibiting

new transmission lines in the
Birds of Prey Area (Sept 2008).
BLM agency land use and
resource management plans
were to incorporate the
designated corridors as per
sec. 368 of the 2005 energy
act.

Therefore - please amend
the SRBOP RMP to
accommodate segment 9D
as outlined in appendix F-1.

100526

Real estate slowdown shutter title office

Business from Alliance's Homedale branch moves to Caldwell



Homedale's branch of the Alliance Title and Escrow Co., apparently has fallen victim to the slumping real estate market.

Chamber of Commerce secretary Robin Aberasturi, who was senior escrow officer at the title company's Owyhee County office formerly located 7 W. Colorado Ave., explained the closure of the outpost last week.

"(Alliance officials) determined that the growth potential for Owyhee County looked bleak for at least the next couple of years and choose to re-allocate their resources to Canyon County, where there is some growth, and potential for growth, going on," Aberasturi said.

The nearest Alliance Title office now is in Caldwell. The Homedale office, which closed May 2, was open for about two years. Aberasturi said the office still is used on a by-appointment basis for contract signings for Owyhee County customers. All phone calls to the old Homedale number are forwarded to Caldwell, too.

"Essentially the market in Owyhee County is about 30 percent of what it was two years ago when we opened," Aberasturi said.

Messages left at Alliance's corporate office seeking official comment have gone unanswered for two weeks.

Aberasturi chose not to accept a transfer to Canyon County, but she said her assistant in Homedale, Vicky Ramirez, has made the move back to the Caldwell office.

The dive in the market was evident as the number of insurable transactions (property sales)

Title and escrow closes
Above: Alliance Title and Escrow Co. has closed its Owyhee County branch at 7 W. Colorado Ave., in Homedale. Right: A sign on the door refers clients to Caldwell and says the Homedale office is open only by appointment.

plummeted from 80 per month throughout Owyhee County to about 30, she said.

Aberasturi said the market is likely to remain sluggish.

"Unfortunately, with the mortgage-lending crisis reducing the number of potential buyers and the artificially inflated sales prices due to out-of-state investors in the past couple of years, this is probably not going to turn around for a couple years," she said. "There is an adjustment that needs to take place in the market — not just in Owyhee County, but Treasure Valley — and that adjustment is going to take a bit of time."

Aberasturi said that other factors also played a role in the disappearance of the Homedale branch.

She said that developers have had difficulty obtaining Owyhee County Planning and Zoning approval to build subdivisions unless

the new homes were to be next to existing city impact areas.

"As long as the county P&Z is holding tight on their permits to build, making it almost impossible to get a subdivision approval, there is not going to be any growth in the county," Aberasturi said. "Title companies can't survive in that environment."

There may be light at the end of the tunnel in the Alliance situation, though. The company still has the lease on the West Colorado Avenue building and Aberasturi said the company hopes to re-open the office in a couple years when the market turns the corner.

In the meantime, she said, there are plans to either sub-lease or possibly sell the Homedale building.

"I don't want to have another empty building sitting in town," Aberasturi said.



Aberasturi continues to serve as Chamber secretary and has been instrumental in the development of the upcoming Block Party and carnival. She also serves as Homedale's business representative in the Western Alliance for Economic Development board of directors.

"It is my hope that Homedale can find ways to continue to grow in this market and keep the forward momentum that has already been started in the local business environment," Aberasturi said.

— JPB

Boy hurt when car slams tree

A 15-year-old boy was sent to the hospital early May 20 after an automobile accident that the Homedale Police Department still is investigating.

Cpl. Perry Grant responded to the report of a car slamming head-on into a tree at 620 W. Idaho Ave., at 12:38 a.m. on May 20.

Grant said later in the week that he is "positive" that the injured juvenile was driving the car. He said investigators still are trying to determine if a second person was in the vehicle.

Grant also said that he measured 192 feet of tire marks through the front lawn of the residence, which is just east of the Homedale LDS church.

Homedale Ambulance responded to the scene, but Grant said that an adult who arrived on the scene refused to let the boy be taken to West Valley Medical Center in Caldwell.

Instead, the boy was driven by personal car to St. Luke's Medical Center in Meridian where he was treated and released, Grant said.

— JPB

El-Ada plans HIV testing in Homedale

The El-Ada Community Action Partnership has set up another day of free HIV testing at its Owyhee County location in Homedale.

The screening will be held from 10 a.m. to 1 p.m. on Friday, June 6 at the El-Ada office, 15 W. Colorado Ave.

Staffers at the screening will use the Oraquick Rapid Test, and clients will be able to receive results in 20 minutes. There is no blood product involved because the test uses an oral sample to screen for HIV antibodies.

For more information, contact El-Ada HIV prevention specialist Katy Kujawski at (208) 345-2820 or katykujawski@msn.com.

Meeting set for power line on BLM land

A public scoping meeting is scheduled for Tuesday in Murphy about the Bureau of Land Management's West Transmission Line Project.

The meeting will run from 3 p.m. to 7 p.m. and give folks a chance to review the project, ask questions and submit comments.

The scoping meeting will take place at the Owyhee County Historical Museum, 17085 Basey St., in Murphy.

For more information visit http://www.wy.blm.gov/nepa/cfdocs/gateway_west or call (307) 775-6116.

A BLM notice of intent document states, the project proposed

by Idaho Power Co. and Rocky Mountain Power of Wyoming entails 1,250 miles of 230 and 500 kilovolt (kV) electric transmission lines stretching from the proposed Windstar Substation near Glenrock, Wyo., to the planned Hemingway substation near Melba.

The notice of intent, which also gives notice that the two utility companies have asked for right-of-way on public lands, states that authorization of the project could trigger amendment of BLM resource management plans.

According to the document, approximately 500 miles of the line would cross public land with a right-of-way in width of between

150 and 250 feet.

Seven BLM offices in Idaho — including Bruneau, Jarbidge and Owyhee — would be affected as 300 miles of the line would cross BLM land in the state.

The document said that through the public scoping process, the BLM will identify issues, potential impacts, mitigation measures and alternatives to the proposal.

Some of the issues already being examined include the effects of wildlife habitat, plants and threatened, endangered and sensitive species; impact on existing view sheds; effects on Native American cultural and sacred resources; the impact of construc-

tion on soil and water; and the potential for the introduction or proliferation of noxious weeds and the ability to rehabilitate land affected by transmission line construction and location.

Keep up with county news in the Avalanche

Farnam or Absorbine
Insect Repellants
10% OFF
Cattle & Horse
Wormers 10% OFF

PSC
Producers Supply

3441 Hwy 95 • Homedale
208-337-5706

6/90

SEC. 368. ENERGY RIGHT-OF-WAY CORRIDORS ON FEDERAL LAND.

(a) WESTERN STATES.—Not later than 2 years after the date of enactment of this Act, the Secretary of Agriculture, the Secretary of Commerce, the Secretary of Defense, the Secretary of Energy, and the Secretary of the Interior (in this section referred to collectively as “the Secretaries”), in consultation with the Federal Energy Regulatory Commission, States, tribal or local units of governments as appropriate, affected utility industries, and other interested persons, shall consult with each other and shall—

- (1) designate, under their respective authorities, corridors for oil, gas, and hydrogen pipelines and electricity transmission and distribution facilities on Federal land in the eleven contiguous Western States (as defined in section 103(o) of the Federal Land Policy and Management Act of 1976 (43 U.S.C. 1702(o));
- (2) perform any environmental reviews that may be required to complete the designation of such corridors; and
- (3) incorporate the designated corridors into the relevant agency land use and resource management plans or equivalent plans.

(b) OTHER STATES.—Not later than 4 years after the date of enactment of this Act, the Secretaries, in consultation with the Federal Energy Regulatory Commission, affected utility industries, and other interested persons, shall jointly—

- (1) identify corridors for oil, gas, and hydrogen pipelines and electricity transmission and distribution facilities on Federal land in States other than those described in subsection

(a); and

- (2) schedule prompt action to identify, designate, and incorporate the corridors into the applicable land use plans.

(c) ONGOING RESPONSIBILITIES.—The Secretaries, in consultation with the Federal Energy Regulatory Commission, affected utility industries, and other interested parties, shall establish procedures

under their respective authorities that—

- (1) ensure that additional corridors for oil, gas, and hydrogen pipelines and electricity transmission and distribution facilities on Federal land are promptly identified and designated as necessary; and

Procedures.

Deadline.

Deadline.

42 USC 15926.

119 STAT. 728 PUBLIC LAW 109-58—AUG. 8, 2005

(2) expedite applications to construct or modify oil, gas, and hydrogen pipelines and electricity transmission and distribution facilities within such corridors, taking into account prior analyses and environmental reviews undertaken during the designation of such corridors.

(d) CONSIDERATIONS.—In carrying out this section, the Secretaries shall take into account the need for upgraded and new electricity transmission and distribution facilities to—

- (1) improve reliability;
- (2) relieve congestion; and
- (3) enhance the capability of the national grid to deliver electricity.

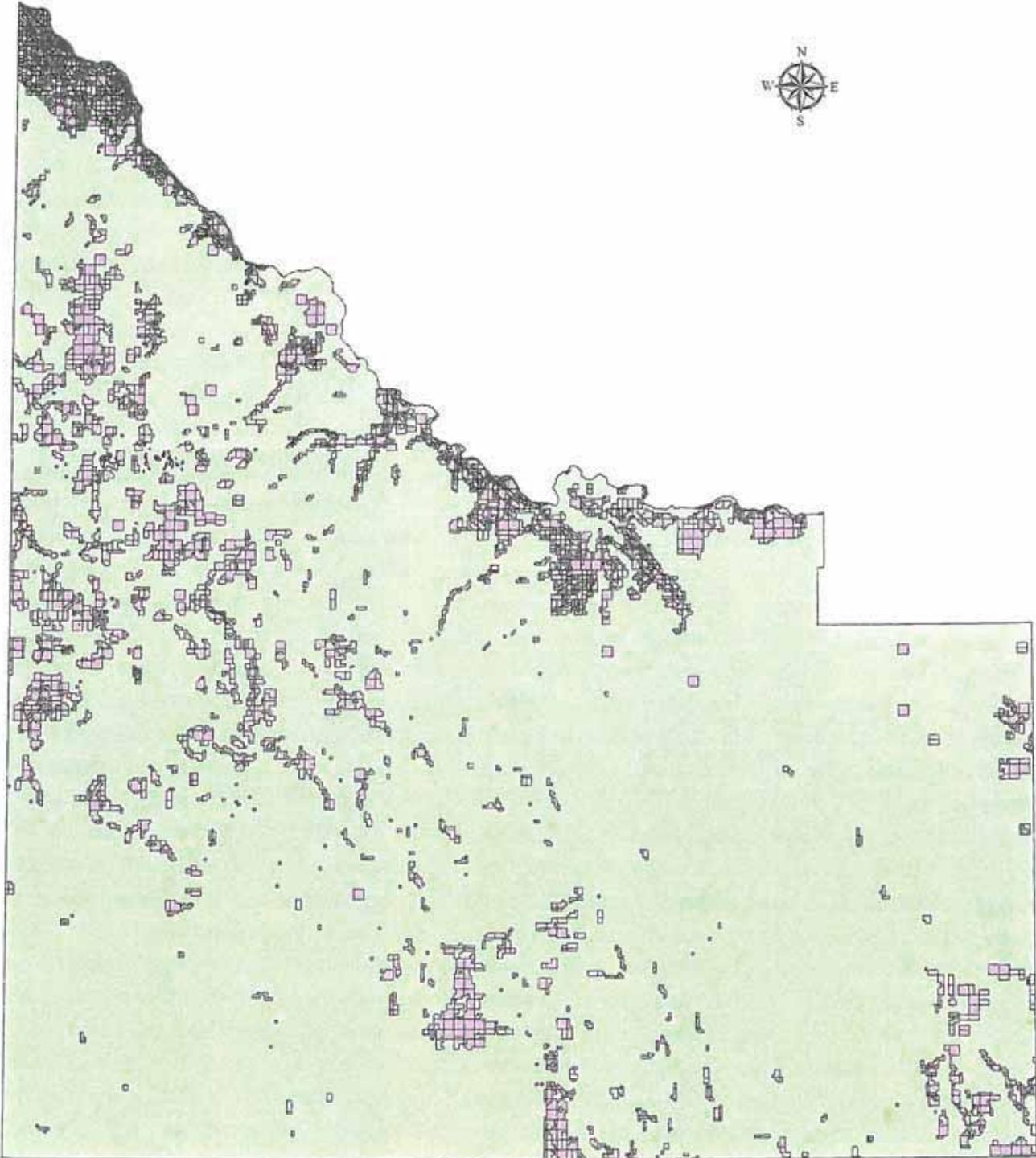
(e) SPECIFICATIONS OF CORRIDOR.—A corridor designated under this section shall, at a minimum, specify the centerline, width, and compatible uses of the corridor.

Owyhee County Private Property Parcels

100524

Legend

 Parcels



9/90

Comment:

Idaho Power's proposed route for segment 9 is 57.2 miles of which 18.4 miles is comprised of private land.

32.16% of the Proposed Route was not analyzed in the DEIS.

Alternative 9D is 58.4 miles long with only 3.3 mi. consisting of private land. 5.65% of 9D was not analyzed in the DEIS.

Comment: Cultural Resources

To quote pg 2.3-1 "Cultural resources that are of traditional religious and cultural importance or TCP's, are places that are valued by the human community and play an important role in that community's historically rooted beliefs, customs and practices."

In chapter 3 the only historical site listed in Oreana is Our Lady Queen of Heaven Catholic Church that is affected by the Proposed Route. Our community of Oreana is entirely comprised of old established family ranches from the 1800's - early 1900's. The Formans, Collets, Johnstons, Kings, Fugways, Jayes, Smith, Adcock and the old Hyde Ranch (next to the church). These families have been running these ranches for decades and their properties have old homesteads still standing surrounded by old

farm implements. The Proposed Route would traipse right through these historic properties. The same can be said for the communities of Brunson, Grand View, Little Valley and Murphy.

Paul Nettleton owns the Joyce Ranch est. 1865. Paul is a Joyce descendant. Paul's son Chad is a 5th generation Joyce working this family owned ranch. The Joyce Ranch is the oldest family owned ranch in the state of Idaho. The Proposed Route goes right through this ranch.

One trip to the Owyhee County Historical Museum will expose the hundreds of historical and cultural sites the proposed route traverses; one of which is the museum itself! The museum is located right behind the Owyhee County Courthouse - also located within the Proposed Route!

Comment : Section 2.5 Environmental
Justice

Prior to Sept 4, 2009 all counties had to have alternative proposals submitted to the BLM for analysis in the DEIS. Idaho Power changed their preferred route for segment 8. Originally Idaho Power's preferred route was what is now 8B affecting significant amounts of agricultural and residential properties. Idaho Power accommodated the Ada County Commissioners. Owyhee County Commissioners strongly urged Idaho Power to adapt seg 9D as their preferred route to no avail.

11.8% of Ada County = below poverty level

17.4% of Owyhee County = below poverty level

7.1% of Ada County = Hispanic

25.8% of Owyhee County = Hispanic

0.7% of Ada County = American Indian

4.3% of Owyhee County = American Indian

Selecting Alternative 9D would negate this issue.

100526

Data Sets

Search ERS:

[Print](#) | [E-mail](#) | [Bookmark/share](#) | [Translate](#) | [Text only](#) | [A](#) [A](#) [A](#)

Browse by Subject:

- Animal Products
- Countries & Regions
- Crops
- Diet, Health, & Safety
- Farm Economy
- Farm Practices & Management
- Food & Nutrition Assistance
- Food Sector
- Natural Resources & Environment
- Policy Topics
- Research & Productivity
- Rural Economy
- Trade & International Markets

2009 County-Level Poverty Rates for Idaho

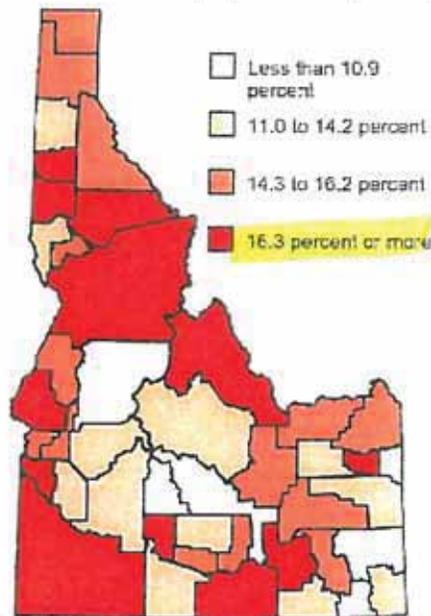
Idaho



Percent **Number**

[Go to the map to select a State](#)
[Go to Idaho State Fact Sheet](#)

Percent of total population in poverty, 2009



[Get this map as a JPG image or a PNG image.](#)

Click a column name to sort the table by that column.

				All people in poverty (2009)			Children ages 0-17 in poverty (2009)		
				90% confidence interval of estimate			90% confidence interval of estimate		
	FIPS*	Name	RUC Code ¹	Percent	Lower bound	Upper bound	Percent	Lower bound	Upper bound
1	16000	Idaho		14.4	13.9	14.9	18.5	17.5	19.5
2	16001	Ada County	2	11.8	10.7	12.9	13.5	11.4	15.6
3	16003	Adams County	8	14.9	11.4	18.4	20.9	16.0	25.9
4	16005	Bannock County	3	14.5	11.8	17.2	18.5	14.4	22.7

14/90

100526

5	16007	Bear Lake County	7	13.0	10.1	16.0	18.3	13.8	22.7
6	16009	Benewah County	6	16.3	12.8	19.8	21.9	16.6	27.2
7	16011	Bingham County	6	15.9	13.2	18.7	20.6	16.6	24.6
8	16013	Blaine County	7	8.0	6.1	9.8	10.7	8.0	13.3
9	16015	Boise County	2	12.8	9.9	15.8	18.7	14.2	23.2
10	16017	Bonner County	6	15.8	12.8	18.8	23.5	18.5	28.6
11	16019	Bonneville County	3	11.9	9.6	14.1	16.0	12.5	19.5
12	16021	Boundary County	7	15.9	12.2	19.5	23.2	17.9	28.6
13	16023	Butte County	8	14.4	10.9	18.0	18.0	13.6	22.4
14	16025	Camas County	9	10.3	8.0	12.6	15.7	12.3	19.6
15	16027	Canyon County	2	18.2	16.3	20.1	24.3	20.9	27.6
16	16029	Caribou County	6	10.9	8.3	13.5	14.7	11.3	18.2
17	16031	Cassia County	7	16.3	13.0	19.5	22.1	17.2	27.0
18	16033	Clark County	8	16.1	12.2	20.0	23.4	17.8	29.0
19	16035	Clearwater County	6	16.5	12.7	20.4	25.9	19.8	31.9
20	16037	Custer County	9	13.4	10.3	16.6	19.4	14.7	24.1
21	16039	Elmore County	4	13.8	10.5	17.1	18.6	14.2	23.1
22	16041	Franklin County	3	10.6	8.2	13.0	15.2	11.6	18.8
23	16043	Fremont County	6	14.9	11.6	18.2	21.3	16.3	26.2
24	16045	Gem County	2	14.8	11.3	18.3	22.2	17.0	27.4
25	16047	Gooding County	7	16.5	13.2	19.8	23.0	17.9	28.1
26	16049	Idaho County	6	21.0	17.2	24.8	28.6	22.8	34.3
27	16051	Jefferson County	3	11.1	8.5	13.7	16.1	12.3	19.9
28	16053	Jerome County	7	15.4	12.2	18.6	22.5	17.6	27.4
29	16055	Kootenai County	3	13.8	12.0	15.6	18.5	15.3	21.7
30	16057	Latah County	4	18.4	15.3	21.5	15.3	11.9	18.6
31	16059	Lemhi County	7	18.5	14.7	22.3	27.9	21.9	33.9
32	16061	Lewis County	8	15.2	11.6	18.9	26.0	19.7	32.5
33	16063	Lincoln County	9	12.2	9.3	15.1	17.3	13.2	21.5
34	16065	Madison County	6	28.9	25.6	32.2	18.2	14.3	22.2
35	16067	Minidoka County	7	14.9	11.4	18.3	21.7	16.7	26.8

15/90

100526

36	16069	Nez Perce County	3	13.7	11.1	16.2	19.1	15.3	23.0
37	16071	Oneida County	8	12.8	9.9	15.8	16.9	12.9	20.8
38	16073	Owyhee County	2	17.4	13.0	21.7	27.2	20.6	33.9
39	16075	Payette County	6	15.5	12.2	18.9	22.0	16.8	27.2
40	16077	Power County	3	16.6	12.6	20.5	23.9	18.3	29.5
41	16079	Shoshone County	6	14.8	11.0	18.6	25.3	19.2	31.5
42	16081	Teton County	9	10.3	8.0	12.7	15.3	11.7	19.0
43	16083	Twin Falls County	5	12.5	9.9	15.1	17.8	13.7	21.9
44	16085	Valley County	8	10.4	7.9	13.0	18.5	14.1	23.0
45	16087	Washington County	6	16.6	12.7	20.5	25.0	19.1	30.9

See the county-level poverty rates from the 1990 and 2000 Census of Population.

► Download the State- and county-level data in Excel format.

See important notes about intercensal model-based poverty estimates.

¹The 2003 rural-urban continuum codes classify metropolitan counties (codes 1 through 3) by size of the Metropolitan Statistical Area (MSA), and nonmetropolitan counties (codes 4 through 9) by degree of urbanization and proximity to metro areas. See rural-urban continuum codes for precise definitions of each code.

Source: Bureau of the Census, Small Area Income and Poverty Estimates.

*See the Census Bureau web site for a description of FIPS codes.

For more information, contact: Kathleen Kassel

Web administration: webadmin@ers.usda.gov

Updated date: December 11, 2009

16/90

0/7/2011 11:00 AM

100526

State & County QuickFacts

Owyhee County, Idaho

People QuickFacts	Owyhee County	Idaho
Population, 2010	11,526	1,567,582
Population, percent change, 2000 to 2010	8.3%	21.1%
Population, 2000	10,644	1,293,955
Persons under 5 years old, percent, 2009	7.7%	8.1%
Persons under 18 years old, percent, 2009	28.6%	27.1%
Persons 65 years old and over, percent, 2009	14.2%	12.1%
Female persons, percent, 2009	47.0%	49.8%
White persons, percent, 2010 (a)	76.0%	89.1%
Black persons, percent, 2010 (a)	0.2%	0.6%
American Indian and Alaska Native persons, percent, 2010 (a)	4.3%	1.4%
Asian persons, percent, 2010 (a)	0.5%	1.2%
Native Hawaiian and Other Pacific Islander, percent, 2010 (a)	0.0%	0.1%
Persons reporting two or more races, percent, 2010	2.4%	2.5%
Persons of Hispanic or Latino origin, percent, 2010 (b)	25.8%	11.2%
White persons not Hispanic, persons, 2010	68.3%	84.0%
Living in same house 1 year ago, pct 1 yr old & over, 2005-2009	84.4%	80.0%
Foreign born persons, percent, 2005-2009	15.1%	5.8%
Language other than English spoken at home, pct age 5+, 2005-2009	20.6%	10.0%
High school graduates, percent of persons age 25+, 2005-2009	74.2%	87.7%
Bachelor's degree or higher, pct of persons age 25+, 2005-2009	10.6%	23.7%
Veterans, 2005-2009	1,199	131,409
Mean travel time to work (minutes), workers age 16+, 2005-2009	25.5	20.0
Housing units, 2009	4,880	647,502
Homeownership rate, 2005-2009	71.9%	71.2%
Housing units in multi-unit structures, percent, 2005-2009	4.4%	14.6%
Median value of owner-occupied housing units, 2005-2009	\$127,800	\$166,700
Households, 2005-2009	4,010	552,726
Persons per household, 2005-2009	2.70	2.64
Per capita money income in past 12 months (2009 dollars) 2005-2009	\$18,049	\$22,262
Median household income, 2009	\$33,753	\$44,644
Persons below poverty level, percent, 2009	17.4%	14.4%
Business QuickFacts	Owyhee County	Idaho
Private nonfarm establishments, 2008	186	46,246 ²
Private nonfarm employment, 2008	1,774	537,952 ²
Private nonfarm employment, percent change 2000-2008	21.3%	19.3% ²
Nonemployer establishments, 2008	654	110,461
Total number of firms, 2007	999	151,671
Black-owned firms, percent, 2007	F	0.2%
American Indian and Alaska Native owned firms, percent, 2007	S	0.9%
Asian-owned firms, percent, 2002	F	0.9%

17/90

100526

	F	S
Native Hawaiian and Other Pacific Islander owned firms, percent, 2007		
Hispanic-owned firms, percent, 2007	4.0%	2.6%
Women-owned firms, percent, 2007	21.6%	23.5%
<hr/>		
Manufacturers shipments, 2007 (\$1000)	0 ¹	18,010,976
Merchant wholesaler sales, 2007 (\$1000)	34,609	14,286,715
Retail sales, 2007 (\$1000)	50,935	20,526,631
Retail sales per capita, 2007	\$4,653	\$13,691
Accommodation and food services sales, 2007 (\$1000)	4,150	2,415,951
Building permits, 2009	20	4,863
Federal spending, 2008	66,800	11,227,185 ²

Geography QuickFacts	Owyhee County	Idaho
Land area, 2000 (square miles)	7,677.98	82,747.21
Persons per square mile, 2010	1.5	18.9
FIPS Code	073	16
Metropolitan or Micropolitan Statistical Area	Boise City-Nampa, ID Metro Area	

1: Counties with 500 employees or less are excluded.
 2: Includes data not distributed by county.

(a) Includes persons reporting only one race.
 (b) Hispanics may be of any race, so also are included in applicable race categories.

D: Suppressed to avoid disclosure of confidential information
 F: Fewer than 100 firms
 FN: Footnote on this item for this area in place of data
 NA: Not available
 S: Suppressed; does not meet publication standards
 X: Not applicable
 Z: Value greater than zero but less than half unit of measure shown

Source U.S. Census Bureau: State and County QuickFacts. Data derived from Population Estimates, Census of Population and Housing, Small Area Income and Poverty Estimates, State and County Housing Unit Estimates, County Business Patterns, Nonemployer Statistics, Economic Census, Survey of Business Owners, Building Permits, Consolidated Federal Funds Report
 Last Revised: Friday, 03-Jun-2011 15:26:35 EDT

18/90

100526

State & County QuickFacts

Ada County, Idaho

People QuickFacts	Ada County	Idaho
Population, 2010	392,365	1,567,582
Population, percent change, 2000 to 2010	30.4%	21.1%
Population, 2000	300,906	1,293,955
Persons under 5 years old, percent, 2009	7.6%	8.1%
Persons under 18 years old, percent, 2009	25.9%	27.1%
Persons 65 years old and over, percent, 2009	10.2%	12.1%
Female persons, percent, 2009	49.7%	49.8%
White persons, percent, 2010 (a)	90.3%	89.1%
Black persons, percent, 2010 (a)	1.1%	0.6%
American Indian and Alaska Native persons, percent, 2010 (a)	0.7%	1.4%
Asian persons, percent, 2010 (a)	2.4%	1.2%
Native Hawaiian and Other Pacific Islander, percent, 2010 (a)	0.2%	0.1%
Persons reporting two or more races, percent, 2010	2.8%	2.5%
Persons of Hispanic or Latino origin, percent, 2010 (b)	7.1%	11.2%
White persons not Hispanic, persons, 2010	86.5%	84.0%
Living in same house 1 year ago, pct 1 yr old & over, 2005-2009	77.6%	80.0%
Foreign born persons, percent, 2005-2009	6.2%	5.8%
Language other than English spoken at home, pct age 5+, 2005-2009	9.3%	10.0%
High school graduates, percent of persons age 25+, 2005-2009	92.5%	87.7%
Bachelor's degree or higher, pct of persons age 25+, 2005-2009	34.2%	23.7%
Veterans, 2005-2009	32,975	131,409
Mean travel time to work (minutes), workers age 16+, 2005-2009	19.4	20.0
Housing units, 2009	157,178	647,502
Homeownership rate, 2005-2009	70.1%	71.2%
Housing units in multi-unit structures, percent, 2005-2009	18.3%	14.6%
Median value of owner-occupied housing units, 2005-2009	\$207,800	\$166,700
Households, 2005-2009	141,719	552,726
Persons per household, 2005-2009	2.54	2.64
Per capita money income in past 12 months (2009 dollars) 2005-2009	\$27,805	\$22,262
Median household income, 2009	\$53,828	\$44,644
Persons below poverty level, percent, 2009	11.8%	14.4%
Business QuickFacts	Ada County	Idaho
Private nonfarm establishments, 2008	12,971	46,246 ¹
Private nonfarm employment, 2008	179,549	537,952 ¹
Private nonfarm employment, percent change 2000-2008	17.2%	19.3% ¹
Nonemployer establishments, 2008	29,999	110,461
Total number of firms, 2007	42,344	151,671
Black-owned firms, percent, 2007	0.4%	0.2%
American Indian and Alaska Native owned firms, percent, 2007	1.0%	0.9%
Asian-owned firms, percent, 2002	1.2%	0.9%

19/90

100526

Native Hawaiian and Other Pacific Islander owned firms, percent, 2007	S	S
Hispanic-owned firms, percent, 2007	2.1%	2.6%
Women-owned firms, percent, 2007	25.4%	23.5%
<hr/>		
Manufacturers shipments, 2007 (\$1000)	4,942,388	18,010,976
Merchant wholesaler sales, 2007 (\$1000)	6,006,918	14,286,715
Retail sales, 2007 (\$1000)	5,855,102	20,526,631
Retail sales per capita, 2007	\$15,720	\$13,691
Accommodation and food services sales, 2007 (\$1000)	795,953	2,415,951
Building permits, 2009	1,438	4,863
Federal spending, 2008	2,259,639	11,227,185 ¹

Geography QuickFacts

	Ada County	Idaho
Land area, 2000 (square miles)	1,054.99	82,747.21
Persons per square mile, 2010	371.9	18.9
FIPS Code	001	16
Metropolitan or Micropolitan Statistical Area	Boise City-Nampa, ID Metro Area	

1: Includes data not distributed by county.

(a) Includes persons reporting only one race.

(b) Hispanics may be of any race, so also are included in applicable race categories.

D: Suppressed to avoid disclosure of confidential information

F: Fewer than 100 firms

FN: Footnote on this item for this area in place of data

NA: Not available

S: Suppressed; does not meet publication standards

X: Not applicable

Z: Value greater than zero but less than half unit of measure shown

Source U.S. Census Bureau: State and County QuickFacts. Data derived from Population Estimates, Census of Population and Housing, Small Area Income and Poverty Estimates, State and County Housing Unit Estimates, County Business Patterns, Nonemployer Statistics, Economic Census, Survey of Business Owners, Building Permits, Consolidated Federal Funds Report
Last Revised: Friday, 03-Jun-2011 15:26:26 EDT

26/90

Comment: ES-17

Ch. 3.21 Electrical Environment

pg 3.21-23 thru 25 Long-Term Effects

I have compiled some information and literature on the possible health effects from electromagnetic fields. This information was received from Idaho Department of Health and Welfare.

There may be an association between exposure to electromagnetic fields and adverse health effects.

The Proposed Route is sited where we live and work. Alternative 9D would have significantly less impact in this category.

After binding to the VDR, vitamin D is known to increase intestinal absorption of lead. It is possible that the influence of vitamin D on lead absorption from the gut differs by genotype, and that people with the VDR B allele have greater absorption of lead via the intestines and greater uptake and subsequent release of lead from bone. The scientists note, however, that tibia lead measurements by X-ray fluorescence were complicated by the fact that such analyses are standardized to bone mineral content. Thus, higher tibia lead readings could be attributed to higher lead content, lower calcium content, or both.

Subjects with the ALAD² allele showed higher blood lead concentrations but no differences in tibia or chelatable lead concentrations compared with subjects lacking this allele. The results reinforce observations that the ALAD² allele increases red blood cell binding of lead, and so probably decreases the relative deposition of lead in critical target organs, possibly protecting against the toxicity of lead by increasing the amount of lead excreted in urine.

All studies to date suggest that the ALAD¹ allele is more likely to confer health risks from lead exposure. The researchers also found that lead workers homozygous for the ALAD¹ allele were much less likely to have the VDR bb genotype; the two genes are apparently linked despite the fact that they are located on different chromosomes. Although the VDR gene may play a role in susceptibility to the health effects of lead, there are not enough data to indicate whether its polymorphisms will modify health risks, and if so, which allele brings about such risks. Compared with controls, lead workers seem to have a higher prevalence of ALAD² and VDR B. It may be that the ALAD² and VDR B alleles are protective, and there might be selection by genotype among lead workers—perhaps because workers who become symptomatic upon exposure to lead would choose to leave the occupation—but this speculation requires further study.
—Julian Josephson

A New View of ELF-EMFs Are They Linked with Cancer Promotion?

The debate over a possible link between cancer and extremely low frequency electromagnetic fields (ELF-EMFs) began with a 1979 study that found excess cancer in people who lived near large electrical wires. It has continued through subsequent *in vitro*, *in vivo*, and epidemiological studies that often produced conflicting results. In this issue, Gang Chen of the Department of Pediatrics and Human Development at Michigan State University and colleagues used an experimental model developed to test cancer-promoting chemicals to examine whether ELF-EMFs might play a role in cancer promotion [EHP 108:967–972].

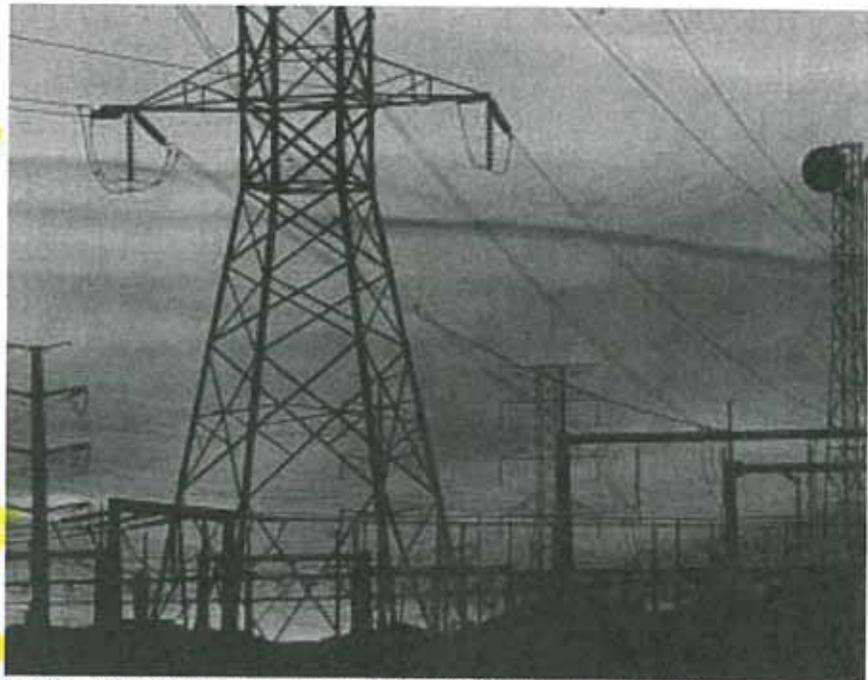
The development of cancer is a multistage process. During normal development, immature cells undergo a process called differentiation in which they become highly specialized (developing, for instance, into red blood cells) and are less able to continue proliferating. In the first stage of cancer, initiation, a cell's DNA is damaged through mutation, causing a differentiated cell to resemble an immature one, in effect reversing the process of differentiation. In the second stage, promotion, normal cellular controls go awry, and the mutated cell multiplies. ELF-EMFs are too weak to kill cells or (most scientists agree) to cause mutations and thus initiate cancer. However, they could play a role during the promotion stage of cancer, which involves so-called epigenetic mechanisms (those that affect gene expression rather than gene structure) and induce cancer in cells that have already mutated.

In the laboratory, differentiation—which can be stimulated by chemical treatment—can transform initiated cells into mature cells, converting cells that had started to become cancerous into normal-seeming adult cells. In this case, differentiation seems to be a healing process that nullifies the mutation. What the group was testing was whether ELF-EMFs could prevent differentiation in cells that had started down the road to cancer.

The research used mouse leukemia cells that, when treated with dimethyl sulfoxide (DMSO), differentiate into red blood cells. The researchers group compared control cells, cells treated with DMSO, and cells treated with DMSO and maintained inside a culture chamber exposed to a 60-hertz ELF-EMF at varying strengths. In a system used to investigate the epigenetics of cancer promotion, the scientists measured three end points. Proliferation, or cell growth, was determined by measuring DNA concentration. Differentiation was measured by detecting hemoglobin, a sign that the cell had developed into a red blood cell. Youthfulness was gauged by measuring telomerase, the enzyme that builds telomeres, which keep chromosomes "young" and able to divide.

Starting at a threshold dose of about 20 milligauss (a measure of the strength of the electrical field), the 60-hertz ELF-EMF caused a dose-dependent reduction of differentiation, as well as an increase in telomerase and proliferation. These effects resemble those of chemical cancer promoters. (Under a power line, fields measure roughly 300 milligauss, and near home appliances they can exceed 1 gauss.)

While the study showed that ELF-EMFs could conceivably play a biological role in carcinogenesis, cancer-promoting chemicals require a long exposure to promote cancer, and human exposures to ELF-EMFs are hard to gauge. Because electric fields change so radically from point to point, it's too early to say if typical exposures actually promote cancer. But by stressing the importance of promotion, the study could focus future research on the environmental health effects of ELF-EMFs. —David J. Tenenbaum



A different role for electromagnetic fields? A 1979 study found that children who lived near power lines (and consequently had higher ELF-EMF exposures) had a higher incidence of cancer. Although most scientists believe ELF-EMFs are too weak to initiate cancer, new research suggests they could play a role as cancer promoters.

Childhood leukemia and magnetic fields in Japan: A case-control study of childhood leukemia and residential power-frequency magnetic fields in Japan

Michinori Kabuto^{1*}, Hiroshi Nitta¹, Seiichiro Yamamoto², Naohito Yamaguchi³, Suminori Akiba⁴, Yasushi Honda⁵, Jun Hagihara⁶, Katsuo Isaka⁷, Tomohiro Saito⁸, Toshiyuki Ojima⁹, Yosikazu Nakamura⁹, Tetsuya Mizoue¹⁰, Satoko Ito¹¹, Akira Eboshida¹¹, Shin Yamazaki¹², Shigeru Sokejima¹², Yoshika Kurokawa¹ and Osami Kubo³

¹National Institute for Environmental Studies, Ibaraki, Japan

²National Cancer Center, Tokyo, Japan

³Tokyo Women's Medical University, Tokyo, Japan

⁴Kagoshima University, Kagoshima, Japan

⁵University of Tsukuba, Ibaraki, Japan

⁶Miyagi University, Miyagi, Japan

⁷Tokushima University, Tokushima, Japan

⁸National Research Institute for Child Health and Development, Tokyo, Japan

⁹Jichi Medical School, Tochigi, Japan

¹⁰University of Occupational and Environmental Health, Fukuoka, Japan

¹¹Hiroshima University, Hiroshima, Japan

¹²Kyoto University, Kyoto, Japan

Residential power-frequency magnetic fields (MFs) were labeled as a possible human carcinogen by the International Agency for Research on Cancer panel. In response to great public concern, the World Health Organization urged that further epidemiologic studies be conducted in high-exposure areas such as Japan. We conducted a population-based case-control study, which covered areas inhabited by 54% of Japanese children. We analyzed 312 case children (0–15 years old) newly diagnosed with acute lymphoblastic leukemia (ALL) or acute myelocytic leukemia (AML) in 1999–2001 (2.3 years) and 603 controls matched for gender, age and residential area. Weekly mean MF level was determined for the child's bedroom. MF measurements in each set of a case and controls were carried out as closely in time as possible to control for seasonal variation. We evaluated the association using conditional logistic regression models. The odds ratios for children whose bedrooms had MF levels of 0.4 μ T or higher compared with the reference category (MF levels below 0.1 μ T) was 2.6 (95% CI = 0.76–8.6) for AML + ALL and 4.7 (1.15–19.0) for ALL only. Controlling for some possible confounding factors did not alter the results appreciably. Even an analysis in which selection bias was maximized did not fully explain the association. Most of the leukemia cases in the highest exposure category had MF levels far above 0.4 μ T. Our results provided additional evidence that high MF exposure was associated with a higher risk of childhood leukemia, particularly of ALL.

© 2006 Wiley-Liss, Inc.

Key words: residential magnetic fields; childhood leukemia; population-based; case-control study; Japan

Exposure to residential power-frequency magnetic fields (MFs) has been suspected to increase the risk of childhood leukemia, although the risk suggested by the first report¹ has not consistently been supported by the following ones.^{2–10} Recently, however, pooled analyses conducted by Ahlbom *et al.*¹¹ used geometric means of MF levels and showed that the estimated summary relative risk was 2.00 (95% CI = 1.27–3.13) when 0.4+ μ T was compared with < 0.1 μ T. Another pooled analysis by Greenland *et al.*¹² used arithmetic means of MF levels and showed that the Mantel-Haenszel odds ratio comparing 0.3+ μ T with < 0.1 μ T was 1.7 (95% CI = 1.2–2.3).

Still, the small number of cases in high-dose ranges remains one of the limitations of these pooled analyses, and the causal inference remains tenuous because of little evidence from animal experiments and lack of appropriate biologic models. Thus, the World Health Organization recommended conducting one or more epidemiologic studies to evaluate the risk with more subjects exposed to high MF levels in 1999,¹³ although the International Agency for Research on Cancer (IARC) rated the power-fre-

quency MF as a possible human carcinogen in 2002¹⁴ mainly based on the above finding by the pooled analyses.

Thus, the present nationwide case-control study of childhood leukemia was conducted in Japan, where high MF exposures were expected to be more common than in the previously studied countries. This expectation was derived from Japan's high population density and the proximity of residences to electric power transmission lines (which refers not only to high-voltage power lines but also to distributing transmission lines to the residences) and other facilities as major sources of high residential MF levels, although detailed data on residential MF levels and exposures were not available when the present study was initiated in 1999.

Compared to the previous studies, this study is characterized by more precise week-long exposure measurements, shorter intervals between the diagnosis and the MF measurements both in cases and controls, as well as a rigorous selection bias assessment. We believe this study, the first elaborately conducted study from non-Western countries, will add substantial evidence to the body of scientific knowledge concerning this controversial issue.

Material and methods

The present study was approved by the Ethics Committee for Human Studies of the National Institute for Environmental Studies, Tsukuba, Japan.

Subjects

We identified newly diagnosed childhood leukemia cases through the following 5 major children's cancer study groups in Japan: Tokyo Children's Cancer Study Group (TCCSG), Children's Cancer and Leukemia Study Group (CCLSG), Tohoku Children's Leukemia Study Group, Japan Association of Childhood Leukemia Study (JACLS) and Kyushu/Yamaguchi Children's Cancer Study Group (KYCCSG). Participating hospitals in these groups numbered 245 in total. More than half of the hospi-

Grant sponsor: Special Coordination Funds for Promoting Science and Technology from the Ministry of Education, Culture, Sports, Science and Technology for 1999–2001.

*Correspondence to: National Institute for Environmental Studies, 16-2 Onogawa, Tsukuba, Ibaraki, 305-8506, Japan.

Fax: +81-29-850-2571. E-mail: kabuto@nies.go.jp

Received 28 September 2004; Accepted after revision 31 May 2005

DOI 10.1002/ijc.21374

Published online 22 February 2006 in Wiley InterScience (www.interscience.wiley.com).

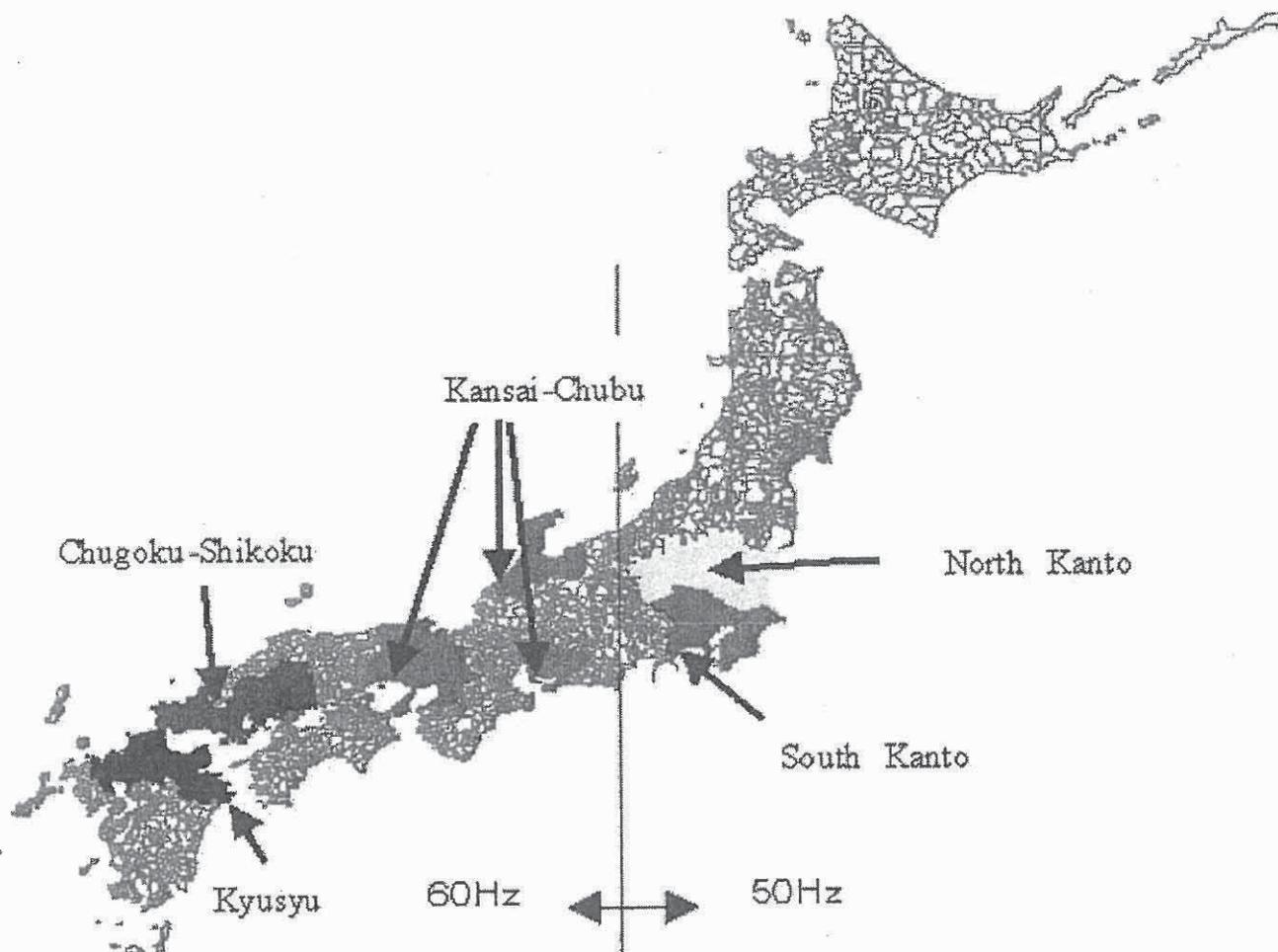


FIGURE 1 - Catchment area.

tals were located in the areas with megalopolis. Our cases were restricted to acute lymphoblastic leukemia (ALL) and acute myelocytic leukemia (AML) for the sake of comparability with the previous studies.^{6,8,11,12} The diagnoses of ALL and AML were made on the basis of morphologic, immunologic, as well as cytogenetic and molecular genetic features of the peripheral blood and bone marrow aspirate. Children with diseases or conditions known to increase the risk for leukemia were excluded from this study. These conditions included chromosomal abnormalities such as Down's syndrome, DNA fragility syndromes such as Fanconi's anemia and immunodeficiency syndromes such as Wiscot-Aldrich syndrome.

Although the case ascertainment through the 5 groups virtually covered the entire nation, we restricted our study area, or catchment area, to 5 regions, including Tokyo, Nagoya, Kyoto, Osaka and Kitakyushu metropolitan areas (Fig. 1). This was because other areas than the catchment area were mainly sparsely populated and we expected that the proportion of residents with high levels of MF exposure would be very low. The catchment area consisted of 18 prefectures in the 5 regions covering 10.7 million (53.5%) of the total 20.0 million children aged 0-15 years in Japan.¹⁵

Requests for participation in the interview survey were sent to 781 (ALL + AML) cases living in the catchment area through the children's physicians; 381 families agreed to participate. Thus, the participation rate for the overall cases was $381/781 = 0.49$. Sixty out of the 381 (AML + ALL) cases who initially agreed were excluded. Among the 60 cases, 25 were excluded due to moving

after diagnosis and 35 were excluded due to measurement failure. The reasons for the MF measurement failure were the following: instrument trouble occurred in the houses of 5 cases, and 30 cases changed their minds and stopped participating.

Once informed consent for participation was obtained from the patient's family, we randomly selected controls from the Japanese resident registration system in the catchment area by matching for gender, age ($\pm 25\%$ for age < 4 years; ± 1 year for age above 4 years), region and population size of the municipality (4 classes). We selected 10 control candidates for each case to achieve 3 matched controls, because the participation rate for mail surveys in Japan is usually less than 30%.¹⁶ Multiple letters requesting participation were sent until 3 controls were set for each case. In total, 3,833 candidates in the catchment area were requested to participate and 1,097 (28.6%) agreed. Among them, interview and MF measurements were completed for 634 subjects. We excluded 23 controls due to incomplete MF measurements. Further, we did not interview the remaining 440 control candidates since the number of controls to be interviewed was reduced to 2 for each case later in the study because of the difficulty of arranging interview schedules for a set of 1 case and 3 matched controls within a short study period.

Interviews

One or 2 trained interviewers were allocated for each of the 5 regions in the catchment area. The questionnaire was based on that used in the National Cancer Institute study⁶ and modified for this Japanese study. In brief, the modified questionnaire consisted of

TABLE 1 - RISK OF ALL + AML WITH COVARIATES LISTED BELOW

Bedroom MF level (μ T)	All subjects included	All subjects included (nighttime measurements)	Subjects lived in current residences for more than 6 months
Below 0.1	1.00	1.00	1.00
0.1-0.2	0.93 (0.51-1.71)	0.97 (0.52-1.79)	0.90 (0.47-1.72)
0.2-0.4	1.08 (0.51-2.31)	1.08 (0.47-2.47)	1.09 (0.50-2.38)
Above 0.4	2.77 (0.80-9.57)	2.87 (0.84-9.88)	3.20 (0.87-11.7)

Covariates: father's education and mother's education.

¹Time-weighted average for 1 week, except for the middle column, for which only nighttime (19:00-06:00) measurements were used.

demographic profiles, medical history of family members, history of moves (from conception to leukemia diagnosis), type of residence, mother's education, child's history of vaccinations, mother's and child's histories of electric appliance use, mother's history of X-ray examination during pregnancy, drug use, smoking, alcohol drinking, pesticide and other agricultural chemical uses, as well as mother's and father's employment history. We interviewed one of the parents of the cases/controls; the interviews were predominantly with mothers (97.8% for cases and 96.2% for controls).

MF and other measurements

Two types of MF measurements were conducted: 1-week-long continuous measurement at 30-sec intervals with the EMDEX-Lite (40 Hz to 1 kHz; EnerTech, Cambell, CA) in the child's bedroom and 5-min-long spot measurements with the EMDEX-II (40 to 800 Hz; EnerTech) at several points inside and outside of the house. The study by Friedman *et al.*¹⁷ as well as ours¹⁸ showed that a 24-hr bedroom measurement was a good surrogate for 24-hr personal exposure.

Thus, we selected the MF level in the child's bedroom as a measure of residential exposure. However, instead of 24-hr mean levels, we used weekly mean levels, since residential MF levels in many of the residences examined were lower during weekends compared with weekdays, especially among residences where indoor MF levels highly correlated with outdoor levels generated by nearby power lines. It was also apparent that those weekly variations were reflecting those in the power supply through the lines.

It has been suggested that MFs generated by transmission lines may show a seasonal variation, depending on the extent of air conditioning use.¹⁹ In order to reduce a possible bias due to seasonal variation of MF levels, the dates of MF measurements in controls and their cases were arranged as closely in time as possible; the mean difference between case and controls within each set was 2.6 ± 13.0 days.

Residential history

Based on the residential history of the families, we obtained the length of stay at the current residence where the MF levels were measured. This information allowed us to consider possible exposure misclassification due to moving.

Statistical analyses

Among the subjects with MF measurements and interviews conducted, 9 cases lost their matched controls and 31 controls lost their matched cases. We excluded these 40 subjects. The remaining 312 cases (251 ALLs, 61 AMLs) and 603 controls (495 ALLs, 108 AMLs) were subjected to conditional logistic regression analyses. The association between residential MF exposure and childhood leukemia was measured in odds ratio (OR). We used PHREG procedure of the PC-SAS (version 8.2; SAS Institute, Tokyo, Japan) and computed ORs and their 95% confidence intervals.

Because of the possibility of an inverse dose-response relation even in the low-exposure range,²⁰ we first evaluated the association between ALL risk and MF levels with 0.05 μ T intervals using

the method of Greenland.²¹ Since this evaluation did not indicate any necessity for finer categorization, the MF level was then categorized with cut-points of 0.1, 0.2 and 0.4 μ T for the sake of comparability with the pooled analyses.^{11,12}

A possible confounding effect was examined by adding the potential confounders (Table 1) to the logistic regression model as covariates. In some models, we restricted the subjects to those who had lived at their present residence for more than 6 months.

Evaluation of selection bias

Because we had expected low participation proportions for both cases and controls, evaluation of the selection bias was planned in the study protocol. We evaluated the selection bias in the following 4 different ways.

GIS-based evaluation. Differential participation, if any, probably comes from the suspicion of high MF exposure in case families. It is natural to assume that their suspicion of high exposure comes from living in close proximity to the high-voltage power lines, not from the actual MF measurements. Thus, for the cases and the controls, we evaluated the difference in the distribution of the distance from the closest power line among cases and controls by the participation status.

For controls, we estimated the distribution of the distance from the residence to the closest power line by the participation status in the study. We were able to calculate the distance for all the control candidates even if they did not agree to participate because their names and addresses in the Japanese registration system are open to the public. As for the cases, we could calculate the distribution of the distance for those who agreed to participate in the study. Also, we obtained maps indicating power lines from the 10 power companies in Japan. From these data, we calculated the distance between a subject's house and the closest power line (22-500 kV) using a geographic information system (GIS) software (ArcInfo; ESRI, Tokyo, Japan). If the maps showed that a power line was located within 100 m from the house, the distance was actually measured with a laser-beam distance meter (Yardage Pro Model 20-1000; Bushnell, Overland Park, KS) for ascertainment in the field.

Possible influence of selection bias on relative risk parameter. In addition to the above evaluation, we evaluated the possible influence of selection bias on the ORs due to differential participation by sensitivity analysis. Detailed explanation can be found in the Appendix. In brief, we evaluated whether or not the possible most extreme scenario, in which all nonparticipant cases have low exposure, could reduce the apparent positive risk to the null.

Subgroup investigation on participation among cases. For case candidates identified through TCCSG, the largest children's cancer study group among the 5 groups, we investigated the reasons for nonparticipation by asking the physicians. By this investigation, we could evaluate whether or not there was a systematic bias in participation among subjects by exposure levels.

Matching failure. Some subjects were excluded from the conditional logistic regression analyses due to matching failure, *i.e.*, the situation in which a case-control set loses the case or the entire controls due to insufficiency of necessary data. To evaluate the effect of this matching failure, we did unconditional logistic

TABLE II - SOME DESCRIPTIVE CHARACTERISTICS OF CASES AND CONTROLS

100524

Total number ¹	ALL				AML			
	Cases		Controls		Cases		Controls	
	n = 251	%	n = 495	%	n = 61	%	n = 108	%
Sex								
Male	146	58.2	287	58.0	32	52.5	56	51.9
Female	105	41.8	208	42.0	29	47.5	52	48.2
Age at diagnosis (years)								
< 2	28	11.2	63	12.7	10	16.4	16	14.8
2-3	69	27.5	133	26.9	12	19.7	17	15.7
4-5	43	17.1	85	17.2	10	16.4	22	20.4
6-9	58	23.1	115	23.2	14	23.0	30	27.8
≥ 10	53	21.1	99	20.0	15	24.6	23	21.3
Father's education								
< junior high school	18	7.3	24	4.9	6	9.8	2	1.9
Senior high school	88	35.8	154	31.2	27	44.3	26	24.1
≥ college/university	140	56.9	316	63.9	28	45.9	80	74.0
Mother's education								
< junior high school	18	7.2	21	4.2	4	6.6	1	0.9
Senior high school	102	40.6	180	36.4	26	42.6	40	37.0
≥ college/university	131	52.2	294	59.4	31	50.8	67	62.0
Mother's history during pregnancy								
Smoking								
Yes	33	13.2	42	8.5	7	11.5	9	8.3
No	217	86.8	452	91.5	54	88.5	99	91.7
Alcohol drinking								
Yes	67	26.8	152	30.8	16	26.2	36	33.3
No	183	73.2	342	69.2	45	73.8	72	66.7
Passive smoking								
Yes	109	43.4	189	38.3	31	50.8	40	37.0
No	142	56.6	305	61.7	30	49.2	68	63.0
Type of residence								
Single-family house	141	56.2	283	57.2	32	52.5	67	62.0
Apartment	106	42.2	205	41.4	27	44.3	41	38.0
Length of stay at current house (months)								
< 6	16	6.4	27	5.4	3	4.9	12	11.1
6-11	23	9.2	31	6.3	3	4.9	5	4.6
≥ 12	212	84.5	437	88.3	55	90.2	91	84.3

¹Individual variable totals may not equal the total number of cases and controls due to missing values.

regression analyses, including those who were excluded from the conditional analyses.

Results

Basic profiles of subjects

Selected basic profiles of the subjects are summarized in Table II. Mothers of the ALL cases had lower educational levels and a higher proportion of smokers (including passive smokers) during pregnancy than those of the controls. Mothers of AML cases had the same distributions of the attributes except for passive smoking, which was more prevalent among AML controls' mothers than among AML cases' mothers. The mean time between the diagnosis and the MF measurements was 13.3 ± 5.8 months for ALL + AML cases and 13.2 ± 5.6 months for controls. (Controls do not have dates of diagnosis; the case's diagnosis date was assigned to the corresponding controls.)

One-week MF measurements and spot measurements

Table III shows the correlations between 1-week MF measurements and spot measurements. The 1-week measurements had high correlations with the spot measurements of detached houses, whereas the correlations with the spot measurements of condominiums were a little lower.

Risk of MF levels for childhood leukemia

As shown in Table IV, leukemia risk was associated with the highest MF category, $0.4+ \mu\text{T}$. The OR for ALL + AML was 2.56 (95% CI = 0.76-8.58) and that for ALL was 4.67 (95% CI = 1.15-19.0) against the reference category (MF levels below $0.1 \mu\text{T}$). As shown in Table I, controlling for the potential con-

founding factors or using nighttime (from 19:00 to 06:00) measurement only did not alter the ORs substantially, and restricting the subjects to those who lived for more than 6 months in a given residence yielded an even higher OR for the highest MF category.

When the distance from the power lines was used as a surrogate for residential MF level, the ORs for ALL for 50-100 m and < 50 m were 1.61 (95% CI = 0.88-2.95) and 3.06 (95% CI = 1.31-7.13), respectively, against the reference category (100+ m). The corresponding ORs for AML were 3.11 (95% CI = 0.71-13.6) and 1.25 (95% CI = 0.11-14.9).

Variations in MF levels

The large hour-to-hour and day-to-day variations in MF levels were observed especially among those with high exposure levels as shown in Figure 2. Specifically, the weekend levels were lower than the weekday levels. This weekday-weekend difference was also observed for the subjects with lower exposure levels, although the difference was smaller; the average MF level for weekends was $0.051 \mu\text{T}$ and that for weekdays was $0.054 \mu\text{T}$.

Profiles of highly exposed subjects

There were only 6 out of 312 cases and 5 out of 603 controls exposed to more than $0.4 \mu\text{T}$ MF. All high-exposure cases and 3 controls are for ALL and no cases and 2 controls are for AML. Table V shows the sex and age at diagnosis, and Figure 2 shows bedroom MF level for the cases and the controls in the highest-exposure category. Most of ALL cases in this group had MF levels much higher than $0.4 \mu\text{T}$. The ages at diagnosis of the cases were mostly less than 7 years old.

26/90

TABLE III - CORRELATIONS BETWEEN 1-WEEK MF MEASUREMENTS AND SPOT MEASUREMENTS

	One-week measurement (all houses)	Spot measurement (detached houses)						Spot measurement (condominiums)	
		BR1	BR2	OC1	OC2	OC3	OC4	Entrance	Window
One-week	1.000	0.938	0.826	0.914	0.910	0.874	0.895	0.712	0.632
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
BR1	915	914	913	530	526	502	477	375	372
		0.938	1.000	0.894	0.891	0.880	0.863	0.891	0.735
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
BR2	914	914	913	530	526	502	477	374	371
		0.826	0.894	1.000	0.868	0.856	0.852	0.878	0.444
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
OC1	913	913	913	529	525	501	476	374	371
		0.914	0.891	0.868	1.000	0.937	0.842	0.886	
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	
OC2	530	530	529	530	519	492	470		
		0.910	0.880	0.856	0.937	1.000	0.894	0.839	
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	
OC3	526	526	525	519	526	492	463		
		0.874	0.863	0.852	0.842	0.894	1.000	0.882	
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	
OC4	502	502	501	492	492	502	470		
		0.895	0.891	0.878	0.886	0.839	0.882	1.000	
		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	
Entrance	477	477	476	470	463	470	477		
		0.712	0.735	0.444				1.000	0.558
		< 0.0001	< 0.0001	< 0.0001					< 0.0001
Window	375	374	374					375	372
		0.632	0.726	0.415				0.558	1.000
		< 0.0001	< 0.0001	< 0.0001				< 0.0001	
		372	371	371				372	372

The 3 numbers in each cell are correlation coefficient, *p*-value, and number of pairs. BR1 (bedroom 1) and BR2 are 2 places in the bedroom, the former being the center of the room and the latter being at the head of the sleeping child. OC1 (outside corner 1) through OC4 are 4 corners of the houses. Window is the one opposite to the entrance.

TABLE IV - RISK OF ALL + AML AND ALL AND CHILD'S BEDROOM MF MEASUREMENTS CONDITIONAL LOGISTIC REGRESSION ANALYSES WITH NO COVARIATE

	ALL + AML			ALL		
	Cases	Controls	OR (95% CI)	Cases	Controls	OR (95% CI)
Bedroom MF level (μT) ¹						
Below 0.1	276	542	1.00	223	447	1.00
0.1-0.2	18	36	0.91 (0.50-1.63)	14	29	0.87 (0.45-1.69)
0.2-0.4	12	20	1.12 (0.53-2.36)	8	16	1.03 (0.43-2.50)
Above 0.4	6	5	2.56 (0.76-8.58)	6	3	4.67 (1.15-19.0)
Total	312	603		251	495	

¹Time-weighted average for 1 week.

Evaluation of selection bias: GIS-based evaluation and sensitivity analysis

No association was found between participation status of controls and proximity to power lines; according to Appendix Table II, the participation proportion for < 50, 50-100, 100-300 and 300+ m were 0.15, 0.16, 0.16 and 0.16, respectively (*p* = 0.98).

Assuming the worst possible case scenario in which all highly exposed cases participated and cases not participating had low exposure levels (details are explained in the Appendix), we obtained 1.84 for the lowest possible odds ratio for subjects exposed to MF levels 0.4+ μT against levels < 0.1 μT .

Evaluation of selection bias: investigation of nonparticipation among cases

Among the letters sent to the cases identified through TCCSG, 40% were not delivered to patients' families, and 12% were delivered but the families did not agree to participate. Therefore, the participating proportion among requests delivered was 80%. The reasons for nondelivery included patients' serious medical conditions and failure to contact the families by the physicians due to various reasons, such as a long interval between our request to the physician and the patient's visit to the hospital.

Evaluation of selection bias: matching failure

The unconditional logistic regression analysis of ALL showed that the OR for the 0.4+ μT category was 4.45 (95% CI = 1.10-17.9) controlling for age, sex and population size of the city. Substantial difference was not observed for different combination of potential confounders. The value of OR was not substantially different from the results of the conditional logistic regression analysis.

Discussion

Our study found a statistically significant association between high residential MF levels (0.4+ μT) and childhood ALL in Japan. The dose-response pattern and magnitude of the ORs of ALL + AML in the present study were generally in good agreement with those of published pooled analyses.^{11,12} A meaningfully increased risk was observed in none of the studies up to the level of 0.4 μT , and the ORs for 0.4+ μT against < 0.1 μT were 2.00 (95% CI = 1.27-3.23) in the study by Ahlborn *et al.*¹¹ and 1.60 (95% CI = 1.03-2.48) in the study by Greenland *et al.*¹²

Our study has several methodologic advantages over the previous studies. First, MF levels were measured for an entire week. Although Friedman *et al.*¹⁷ reported that 24-hr bedroom spot MF

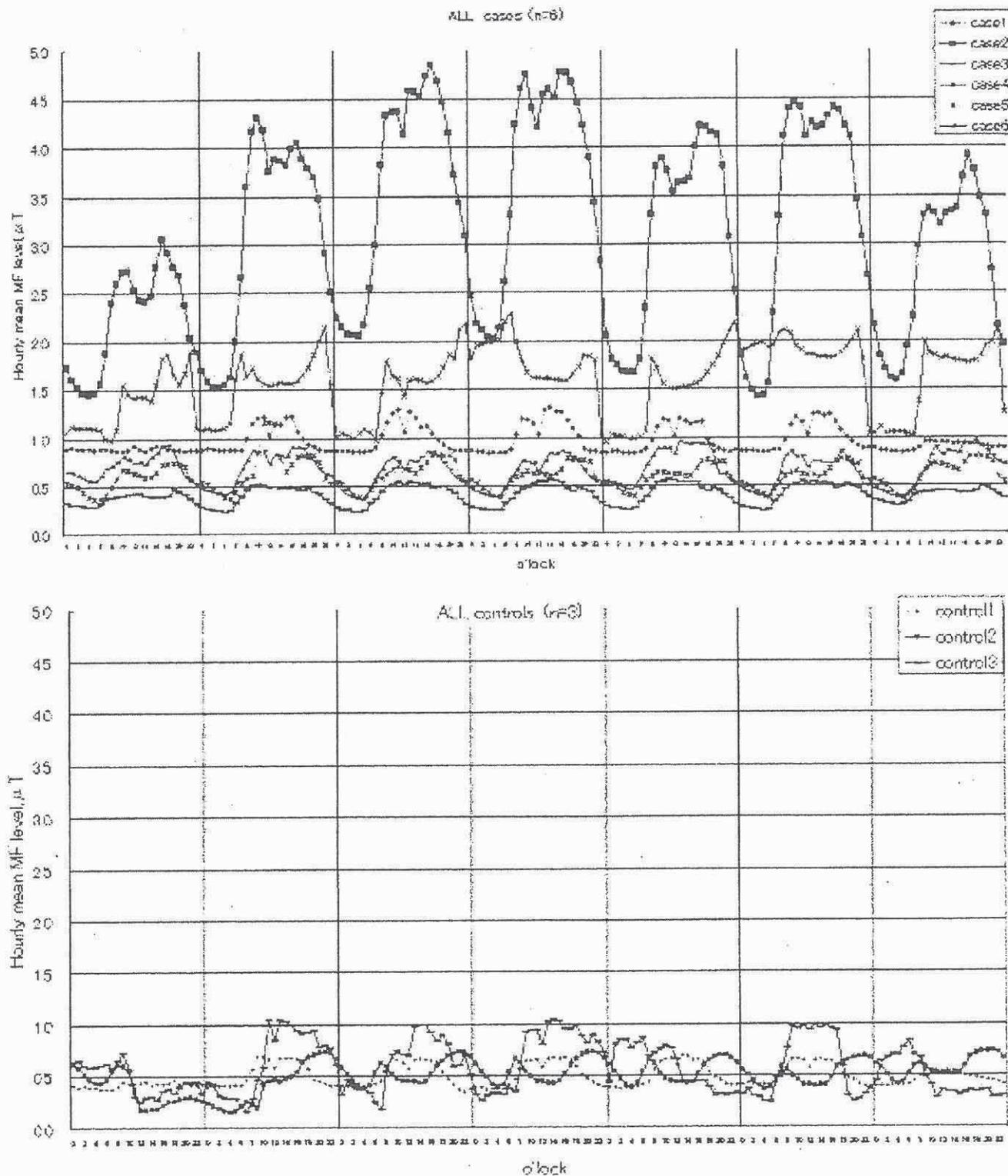


FIGURE 2 - Hourly mean bedroom MF level in the highest exposure category.

level used in the previous study⁶ was a useful predictor of personal dosimetry measurements, the hour-to-hour variation and day-to-day variation as demonstrated for the high bedroom MF level group may raise concern about measurement errors and possible biases. In this sense, our results using 1-week-long bedroom MF level as a measure of exposure are more precise and less biased.

To show this point, we evaluated the fluctuation of the OR estimates (adjusted for mother's and father's education) using 1-day MF level by restricting the MF measurements only to the first day, the second day, ..., or the seventh day of the weeklong measurements; the results showed that the point estimates of the OR comparing 0.4+ μT with < 0.1 μT ranged from 1.48 to 2.61. Although

28/90

TABLE V - SEX AND AGE AT DIAGNOSIS OF ALL CASES AND CONTROLS IN THE CATEGORY OF BEDROOM MF LEVEL > 0.4 μ T

Sex	Type	Age at diagnosis (years)
Controls		
Male	ALL	2.4
Female	ALL	3.1
Male	ALL	11.5
Female	AML	9.5
Male	AML	11.1
Cases		
Female	ALL	2.2
Male	ALL	5.0
Male	ALL	5.7
Male	ALL	6.2
Male	ALL	6.4
Male	ALL	12.0

the results are considered to be consistent, we might have obtained incidentally a lower or higher OR estimate if we had used 1-day MF level, instead of 1-week MF level. Second, the MF measurements for cases were taken close in time to those for matched controls. The short interval between the measurement dates for a case and matched controls should have reduced the seasonal variation in MF measurements between the case and controls. Third, the interval between the diagnosis and the interview was even shorter than the previous study that tried to reduce the interval.⁶ With a longer interval, we would expect a lower correlation between the MF levels measured in the study and the actual MF levels that might have caused the disease in the past.

As we expected at the study outset, the participation rate for the mail questionnaire survey for controls was low (28.6%), and this may be an important source of concern. In Japan, this level of response rate is not uncommon. Since people seldom encounter mail surveys for real scientific studies, people may be weary of commercial direct mails that pretend to be mail surveys. Whatever the reason for the poor participation rate, the key question is whether the differential participation led to significantly biased results regarding the relation with exposure, and hence the rigorous analysis of selection bias was performed in this study. We found no difference in participation proportion between those controls living close to the power line and those living not close to the power line by GIS-based analysis. Unfortunately, the same analysis could not be conducted for cases because the information was not available for cases that did not participate in the study. However, as shown in the results, low participation rate in cases (0.49) was due not to the cases' intentional rejection of the study, but to the low accessibility to patients through physicians. Although the low accessibility may still be related to exposure level, our sensitivity analysis showed the minimum true OR for subjects with

0.4+ μ T MF is 2.32 and the maximum true odds ratio under the null hypothesis is 2.04 if selection bias is taken into consideration. Although the actual OR suffers from random variation, this calculation shows that the selection bias *per se* cannot fully explain our positive finding.

In addition, our bias evaluation, such as controlling for potential confounding factors including socioeconomic factors like mothers' education, the subject restriction by residence history and the unconditional logistic regression analyses, revealed no substantial difference in the results.

Another limitation of our study is the lower than expected proportion of subjects who were exposed to high MF exposures. Possible reason for the lower proportion seems to be related to the following characteristics in Japan: towers for high-voltage overhead transmission lines are higher (taller) than other countries in general; the power lines have been paired such that the MFs of the lines cancel out (this system was adopted in the 1970s).

In conclusion, the present study, the first large-scale case-control study conducted outside of Europe and the United States, based on weekly measured MF levels in children's bedrooms, showed an increased risk of MF level above 0.4 μ T for ALL + AML or ALL only, but not for the lower MF levels. We consider the above results were not due to bias alone, although these may be due to chance.

Acknowledgements

The authors thank Dr. Martha Linet and Dr. Kiyohiko Mabuchi, Radiation Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute, for their helpful comments and are indebted to the advisory committee (Dr. Fumimaro Takaku, Jichi Medical University; Dr. Mutsuro Ohira, National Cancer Center; Dr. Sadanobu Kagamimori, Toyama Medical and Pharmaceutical University; Ms. Midori Kaneko, Consumer Union; Dr. Jun Komiya, University of Shinshu; Dr. Takeshi Shiga, Kinran Junior College; Dr. Kintomo Takakura, Tokyo Women's Medical University; Dr. Masao Taki, Tokyo Metropolitan University; Dr. Isamu Nagano, University of Kanazawa; Mr. Tadashi Negishi, Central Research Institute of Electric Power Industry; Dr. Kazuhiro Nomura, National Cancer Center; Dr. Masayoshi Yanagisawa, National Center for Child Health and Development; Dr. Takesumi Yoshimura, University of Occupational and Environmental Health) of the project for the constructive comments. They are deeply grateful to the children's families, data managers and responsible physicians in the member hospitals of the 5 children's cancer study groups, academic societies concerned and also all of the other staff members in the central and local offices for their kind collaborations and laborious efforts.

References

1. Wertheimer N, Leeper E. Electrical wiring configurations and childhood cancer. *Am J Epidemiol* 1979;109:273-84.
2. Feychting M, Ahlbom A. Magnetic fields and cancer in children residing near Swedish high voltage power lines. *Am J Epidemiol* 1993;138:467-81.
3. Olsen JH, Nielsen A, Schulgen C. Residence near high voltage facilities and risk of cancer in children. *Br Med J* 1993;307:891-5.
4. Verkasalo PK, Pukkala E, Hongisto MY, Valjus JE, Jürvinen PJ, Heikkilä PV, Koskenvuo M. Risk of cancer in Finnish children living close to power lines. *Br Med J* 1993;307:895-9.
5. Dockerty JD, Elwood JM, Skegg DCG, Herbison CP. Electromagnetic field exposures and childhood cancers in New Zealand. *Cancer Cause Control* 1998;9:299-309.
6. Linet MS, Hatch EE, Kleiner RA, Robinson LL, Kaune WT, Friedman DR, Severson RK, Haines CM, Hartsock CT, Niwa S, Wacholder S, Tarone RE. Residential exposure to magnetic fields and acute lymphoblastic leukemia in children. *N Engl J Med* 1997;337:1-7.
7. Michaelis J, Schuez J, Meinen R, Zmann E, Grigat JP, Kaatsch P, Kalesch U, Meisner A, Brinkman K, Kalkner W, Kärner H. Combined risk estimates for two German population-based case-control studies on residential magnetic fields and childhood leukemia. *Epidemiology* 1998;9:92-4.
8. UK Childhood Cancer Study Investigators. Exposures to power-frequency magnetic fields and the risk of childhood leukemia. *Lancet* 1999;354:1925-31.
9. McBride ML, Gallagher RP, Theriault G, Armstrong BG, Tamaro S, Spinelli JJ, Deadman JE, Fincham S, Robson D, Choi W. Power frequency electric and magnetic fields and risk of childhood leukemia in Canada. *Am J Epidemiol* 1999;149:831-42.
10. Tynes T, Haldorsen T. Electromagnetic fields and cancer in children residing near Norwegian high-voltage power lines. *Am J Epidemiol* 1999;145:219-26.
11. Ahlbom A, Day N, Feychting M, Roman E, Skinner J, Dockerty J, Linet M, McBride M, Michaelis J, Olsen JH, Tynes T, Verkasalo PK. A pooled analysis of magnetic fields and childhood leukemia. *Br J Cancer* 2000;83:692-8.
12. Greenland S, Sheppard AR, Kaune WT, Poole C, Kelsh MA. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. *Epidemiology* 2000;11:624-34.
13. Repacholi MH, Ahlbom A. Link between electromagnetic fields and childhood cancer unresolved. *Lancet* 1999;354:1918-9.

14. IARC. Monographs on the evaluation of carcinogenic risks to humans. Non-ionizing radiation: I, static and extremely low-frequency (ELF) electric and magnetic fields. vol.80. Lyon: IARC, 2002.
15. Statistics and Information Department, Ministry of Health and Welfare. The results of prompt sample tabulation of national census of 2000. In: Japanese statistical yearbook. Tokyo: Printing Bureau, Ministry of Finance, 2000.46-49.
16. Statistics Section, Department of Social Sciences, College of Arts and Sciences, University of Tokyo. Statistical methods for the social and behavioral sciences (basic statistics 2). Tokyo: University of Tokyo Press, 1994. 8-10.
17. Friedman DR, Hatch EE, Tarone R, Kaune WT, Kleinerman RA, Wacholder S, Boice JD Jr, Linet MS. Childhood exposure to magnetic fields: residential area measurements compared to personal dosimetry. *Epidemiology* 1996;7:151-5.
18. Kabuto M, Nitta H, eds. Health risk assessment of exposure to extremely-low-frequency electromagnetic fields: NIES SR report (SR-35-2001). Tsukuba: National Institute for Environmental Studies, 2001.
19. Swanson J. Residential power-frequency electric and magnetic fields: sources and exposures. *Radiat Prot Dosim* 1999;83:9-14.
20. Sokejima S, Kagamimori S, Tatumura T. Electric power consumption and leukemia death rate in Japan. *Lancet* 1996;348:821-2.
21. Greenland S. Analysis of polytomous exposures and outcomes. In: Rothman KJ, Greenland S, eds. *Modern epidemiology*, 2nd ed. Philadelphia: Lippincott Williams and Wilkins, 1998. 306-13.

Appendix

Sensitivity analysis of selection bias on relative risk parameter

The data layout for formulations for the bias evaluation is shown in Appendix Table I, which shows the counterfactual situation in which all the case and control candidates participate, as well as the actual situation in the present study. The true odds ratio estimate (OR_t) and observed odds ratio estimate (OR_a) in this instance would be calculated as

$$OR_t = \frac{AD}{BC} \tag{1}$$

$$OR_a = \frac{ad}{bc}$$

The OR_t would be rewritten as follows using Equation 1 and participation proportions:

$$OR_t = \frac{(a/P_{CaH})(d/P_{CoL})}{(b/P_{CoH})(c/P_{CaL})} \tag{2}$$

$$= OR_a \frac{P_{CaL}}{P_{CaH}} \frac{P_{CoH}}{P_{CoL}}$$

where P_{CaH} is the participation proportion for the cases with high exposure, P_{CaL} that for the cases with low exposure, P_{CoH} that for the controls with high exposure and P_{CoL} that for the controls with low exposure. That is, $\hat{P}_{CaH} = a/A$, $\hat{P}_{CoH} = b/B$, $\hat{P}_{CaL} = c/C$ and

APPENDIX TABLE I - DATA LAYOUT FOR FORMULATIONS OF THE BIAS EVALUATION

Exposure level	Cases	Controls
Situation in which all the eligible subjects are included		
High	A	B
Low	C	D
Observed situation in which part of the eligible subjects are included		
High	a	b
Low	c	d

APPENDIX TABLE II - DISTRIBUTION OF THE DISTANCE FROM THE CLOSEST POWER LINE

Distance from power line	Cases	Controls
Situation in which all the eligible subjects are included (column percent in parentheses)		
< 50 m		190 (5.0)
50-100 m		231 (6.1)
100-300 m		735 (19.4)
300+ m		2,626 (69.4)
Total	791 (100)	3,782 (100.0)
Observed situation (column percent in parentheses)		
< 50 m	20 (6.4)	29 (4.8)
50-100 m	25 (8.0)	37 (6.1)
100-300 m	55 (17.6)	115 (19.1)
300+ m	212 (67.9)	422 (70.0)
Total	312 (100.0)	603 (100.0)

Some subjects were excluded because their GIS information was not available.

APPENDIX TABLE III - SELECTION BIAS EVALUATION WITH SOME OBTAINED DATA: ALL AS AN EXAMPLE

Exposure level	Cases	Controls
Observed situation reconstructed from Table 2-1, ALL panel ¹		
High (0.4+ μT)	6	3
Low (0.4 μT <)	245	492
Total	251	495
Situation in which all the eligible subjects are included		
High (0.4+ μT)	A	B
Low (0.4 μT <)	C	D
Total	626	B + D
Situation with $P_{CaH} = 1$, i.e., $A = a = 6$		
High (0.4+ μT)	6	B
Low (0.4 μT <)	620	D
Total	626	B + D

¹ $OR_a = 4.02$.

$\hat{P}_{CoL} = d/D$. Therefore, sensitivity analysis can be conducted by evaluating OR_t with Equation 2 and some obtained results.

Appendix Table II shows the distribution of the distance from the closest power line by selection status. The controls studied had almost identical distance distribution as those who were requested to participate, i.e., there was no selection bias among the control candidates. This observation implies that $P_{CoH} = P_{CoL}$ can be assumed for controls, and Equation 2 can be reduced to

$$OR_t = OR_a \frac{P_{CaL}}{P_{CaH}} \tag{3}$$

We tried to evaluate the minimum value of OR_t in the sensitivity analysis in order to maximize the bias. The above formula says OR_t is the smallest when $P_{CaH} = 1$ (meaning that all the cases who were exposed to the high MF levels were included in the study). If this value is applied to the ALL panel of Table 2-1, then $a = A = 6$ for Appendix Table III. Since the total number of ALL cases we requested to participate was 626, then $C = 626 - 6 = 620$, as shown in Appendix Table III. From these data, $\hat{P}_{CaL} = c/C = 245/620 = 0.40$. Since OR_a for the ALL was 4.02 (using Appendix Table III), substituting 0.40 for \hat{P}_{CaL} into Equation 3 yields the minimum OR_t being 1.61. In the same line of reasoning, minimum observable OR_a 2.53 under the null hypothesis of OR_t being unity. Hence, OR_t should be greater than unity when OR_a 2.53. Although the above analysis neglects matching, doing so did not appear to influence the estimation of ORs in our study, as mentioned in the main body of the article.

Residential exposure to electromagnetic fields and childhood leukaemia: a meta-analysis

I.F. Angelillo¹ & P. Villari²

Although individual epidemiological investigations have suggested associations between residential exposure to electromagnetic fields (EMFs) and childhood leukaemia, overall the findings have been inconclusive. Several of these studies do, however, lend themselves to application of the meta-analysis technique. For this purpose we carried out searches using MEDLINE and other sources, and 14 case-control studies and one cohort study were identified and evaluated for epidemiological quality and included in the meta-analysis. Relative risk estimates were extracted from each of the studies and pooled. Separate meta-analyses were performed on the basis of the assessed EMF exposure (wiring configuration codes, distance to power distribution equipment, spot and 24-h measures of magnetic field strength (magnetic flux density) and calculated magnetic field). The meta-analysis based on wiring configuration codes yielded a pooled relative risk estimate of 1.46 (95% confidence interval (CI) = 1.05–2.04, $P = 0.024$) and for that for exposure to 24-h measurements of magnetic fields, 1.59 (95% CI = 1.14–2.22, $P = 0.006$), indicating a potential effect of residential EMF exposure on childhood leukaemia. In most cases, lower risk estimates were obtained by pooling high-quality studies than pooling low-quality studies. There appears to be a clear trend for more recent studies to be of higher quality. Enough evidence exists to conclude that dismissing concerns about residential EMFs and childhood leukaemia is unwarranted. Additional high-quality epidemiological studies incorporating comparable measures for both exposure and outcomes are, however, needed to confirm these findings and, should they prove to be true, the case options for minimizing exposure should be thoroughly investigated to provide definitive answers for policy-makers.

Keywords: electromagnetic fields; leukaemia, radiation induced; meta-analysis; risk factors.

Voir page 913 le résumé en français. En la página 913 figura un resumen en español.

Introduction

In 1979 Wertheimer & Leeper (1) published the first report that showed an increased risk of cancer mortality among children living near electrical wiring configurations and which were consistent with the presence of high currents. Although many studies linking exposure to electromagnetic fields (EMFs) with health effects have been conducted, there is still much debate over whether this exposure at the levels that occur in domestic settings can cause cancer, particularly childhood leukaemia. Some workers have recently called for an end to further research on exposure to magnetic fields (2), whereas others believe that abandoning research on this topic is premature (3, 4).

Meta-analysis is a quantitative approach for systematically combining the results of previous studies in order to arrive at conclusions that cannot be drawn from the results of any one study alone. Although it has been applied most often to combine

the results of randomized trials, use of meta-analysis is not confined to the synthesis of information from experimental studies. A large number of studies that involve meta-analysis of nonexperimental data have been published in recent years, although such use of the technique is less accepted than it is in the analysis of data from clinical trials (5). Meta-analyses of observational epidemiological studies have also previously been carried out to examine the relationship between residential EMF exposure and childhood leukaemia (6–11). In general, such analyses have shown a significant increased risk of childhood leukaemia when residential exposure is assessed through the use of wiring configuration codes (a categorical exposure rating scheme based on wire size and distance from the residence), whereas the association with other related markers of exposure, such as proximity to power lines and calculated magnetic fields from power lines, appears less evident.

Several well-conducted epidemiological studies on the association between EMFs and childhood leukaemia were published after the above-mentioned meta-analyses appeared. The purpose of the present investigation was to reassess the risk of childhood leukaemia associated with residential EMF exposure in the light of these more recent publications. In so doing we hoped to be able to provide answers to the following questions.

¹ Associate Professor, Chair of Hygiene, Medical School, University Magna Graecia, Via Tommaso Campanella, 88100 Catanzaro, Italy. Correspondence should be addressed to this author (e-mail: igiene@thebrain.net).

² Assistant Professor, Department of Health and Preventive Sciences, University Federico II, Naples, Italy.

- Could the association between wire codes and childhood leukaemia be confirmed?
- Is there an association between childhood leukaemia and other markers of residential EMF exposure?
- What is the overall quality of existing studies and is there a relationship between the quality of studies and the magnitude of risk?
- What recommendations can we make for further studies, if warranted?

Materials and methods

Identification of relevant studies

Studies pertaining to the relationship between EMFs and childhood leukaemia were identified using a MEDLINE search of the medical literature published in English over the period 1966–98. Copies of the relevant articles were obtained and reviewed to identify additional references.

To be eligible for inclusion in the meta-analyses, studies had to satisfy the following criteria: be primary studies, not reanalyses or reviews; be of case-control, cohort, or cross-sectional design; examine residential-based exposures through wiring configuration codes, distance to power distribution equipment, spot and 24-h measures of magnetic field strength (magnetic flux density), and calculated magnetic fields; examine childhood leukaemia; report an odds ratio and its variance or sufficient data to estimate them; be in English, and be published before January 1999.

Quality assessment

Each article was blinded with regard to authors, institution, and journal. The articles were read and scored for quality by two independent readers using a system that incorporates elements of methods developed by Chalmers et al. (12), Longnecker et al. (13), Morris et al. (14) and Villari et al. (15). The criteria employed are shown in the results section below. A quality score was calculated as the percentage of applicable criteria that were met in each study. Items concerned with efforts to minimize potential bias were given twice the weight of those evaluating data analysis.

Data extraction

A number of different methods were used to measure EMF exposure in the studies examined. In the absence of a unique criterion, not all studies examining EMF exposure and childhood leukaemia risk could be included in a single analysis. Exposure assessment methods were aggregated into the following categories: wiring configuration codes; distance to power distribution equipment; spot and 24-h measures of magnetic field strength; calculated indices of magnetic field strength using distance to power distribution equipment; and historic load data. To aggregate exposure categories across studies, we

dichotomized exposure strata using the cut-off points that were common to most of the studies. More specifically, in studies where the exposure status was assessed through the wiring code or distance criterion, subjects were considered exposed if they were living in homes with high current configurations or at a distance of less than 50 m from any electrical sources, respectively. Similarly, in studies where the assessment of exposure was made via spot, 24-h measurements or historical calculation of magnetic field strength, a cut-off point of 0.2 μ T was used.

For each blinded study, data were extracted in a contingency table format, and odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Data extraction was carried out independently by two readers. After completing data extraction, the two readers met to resolve any differences and arrive at a consensus.

Statistical pooling

The random effects model described by DerSimonian & Laird (16) was used to combine the collected values. This procedure yields a single estimate of the OR for leukaemia in children exposed to EMFs compared with nonexposed children. It also enables testing the homogeneity across the individual studies (using the Q statistics); the heterogeneity, if not zero, is then incorporated into the pooled variance estimate.

We avoided pooling results obtained through the use of different exposure criteria, and instead carried out separate meta-analyses based on exposure assessment methods, using the cut-off points specified above. Studies that used more than one method of EMF measurement were included in more than one meta-analysis. In order to test all decision rules that we used in extracting OR data, we performed additional meta-analyses using, for each exposure assessment method, data that in the single studies gave the lowest OR (best scenario) and the highest OR (worst scenario).

Finally, studies were also divided into two groups and analysed according to their quality score: the potential impact of the quality of studies on the results was assessed by comparing pooled results from studies with scores above the median to studies whose scores were equal to or below the median.

Results

Literature search

The literature search identified 14 case-control studies (1, 17–29) and one cohort study (30) that investigated the relationship between residential magnetic-field exposures and childhood leukaemia and which met our inclusion criteria. Of these studies, 10 employed only one method of EMF measurement (1, 17, 18, 20, 22, 24, 26, 27, 29, 30), three used two methods (19, 25, 28), and two studies used three methods (21, 23). Thus, six studies were

available for the meta-analysis evaluating exposure through wiring configuration codes (1, 17, 19, 21, 25, 27) and five studies for the meta-analysis in which the exposure was determined by spot measures (18, 19, 21, 23, 28), whereas meta-analyses based on distance from electrical sources (20, 23, 24, 26), 24-h measurements of magnetic fields (21, 25, 28, 29) and calculated magnetic fields (22, 23, 26, 30) comprised four studies each.

The inclusion of the studies by Tomenius (18) and Feychting & Ahlbom (23) in the meta-analysis based on spot measurements of magnetic fields was problematic, since a partial overlap of their data cannot be excluded. Therefore, we included in our main analysis only the Feychting & Ahlbom study, testing the impact of this choice in a sensitivity analysis.

Magnetic field strength was not dichotomized at the 0.2 μT level in four studies reporting spot or

24-h measures and calculated indices (Tomenius (18) = 0.3 μT ; London et al. (21) = 0.125 and 0.264 μT for spot and 24-h measurements, respectively; Olsen et al. (22) = 0.25 μT ; Tynes & Haldorsen (26) = 0.14 μT). In order to include in the meta-analyses all available data, we assumed that the exposure cut-off points used in these studies were comparable to the 0.2 μT exposure.

Quality assessment

Table 1 shows the results of the quality scoring procedure. The potential for selection bias may be a concern in the individual studies considered. Although 11 of the 14 case-control studies used population-based cancer registries to identify cases, there are a number of important concerns about the control selection. Only five studies used a population register to identify controls. If the ascertainment of

Table 1. Items used in quality scoring for studies of the association between exposure to residential electromagnetic fields (EMF) and childhood leukaemia

Quality scoring item	% of studies complying ^a
Case-control studies	
Cases either randomly selected or selected to include all cases in a specific population	79
Cases identified without knowledge of exposure status	100
Response rate for identified cases > 75%	64
Control drawn randomly from the same population of cases	36
No known association between control status and exposure	93
Response rate for identified controls > 75%	50
Cohort studies	
Initial response rate > 75%	100
Comparison of persons who did and did not participate	0
Follow-up rate > 75%	100
Comparison of who were and were not lost to follow-up	0
Exposed/nonexposed subjects identified without knowledge of disease status	100
No known association between nonexposed status and disease	100
All studies	
Subjects unaware of specific associations of interest insofar as possible	67
Exposure/disease assessment made blindly with respect to the case-control/exposure status of subjects	53
Specific disease criteria given	33
Disease validated by histology or other gold standard	53
Exposure evaluations made in relation to the time of diagnosis	67
Differential mobility among cases and controls (or among exposed and nonexposed) considered	80
Age considered as potential confounder	100
Sex considered as potential confounder	93
Socioeconomic status considered as potential confounder	87
Parental occupational exposure to EMF considered as potential confounder	20
Indicators of air quality (e.g. traffic density) considered as potential confounder	40
Competing carcinogenic exposures considered as potential confounder	40
Demographic data listed	53
Statistical analysis of demographic data	7
Power calculations performed	13
Precise <i>P</i> -values and/or confidence interval given	87
Test statistic specified	93
Appropriate statistical analysis	80

^a If compliance was not specifically indicated in the text, noncompliance was assumed.

cases as well as the population register is complete, these studies should be free of selection biases. In most studies, however, controls were selected in less desirable ways, such as using regional birth certificate files, random digit dialing or other cancer cases. Response rates for cases and controls were frequently less than 75%, particularly in studies requiring subject interviews or magnetic field measurements.

Misclassification bias cannot be considered negligible. Although few studies specified disease criteria or clearly stated that cancer diagnosis was validated by histology or some other gold standard, diseases like leukaemia are subject to relatively little misclassification (false negatives are unlikely given the severity of the disease, and false positives are unlikely given the medical scrutiny of suspected cases). By contrast, exposure misclassification is a pervasive concern in case-control studies of the effect of exposure to EMFs. Unfortunately, not all studies made serious efforts to collect as much of the exposure data as possible while being unaware of the case-control status of the subjects. Use of such a procedure does not ensure the absence of errors but makes it highly probable that errors would be independent of case or control status and therefore that the results would be biased towards the null.

Information bias associated with failure to consider confounding variables may have been more of a problem. With the exception of age (100% of studies), sex (93%) and socioeconomic status (87%), less than half of the studies considered potentially confounding variables such as traffic density (40%), parental occupational exposure to EMFs (20%) or other competing carcinogenic exposures (40%). Although attempts made to adjust for those variables have produced evidence against the presence of substantial confounding, such attempts are severely hampered by the scarcity of established or even strongly suspected causes of childhood leukaemia.

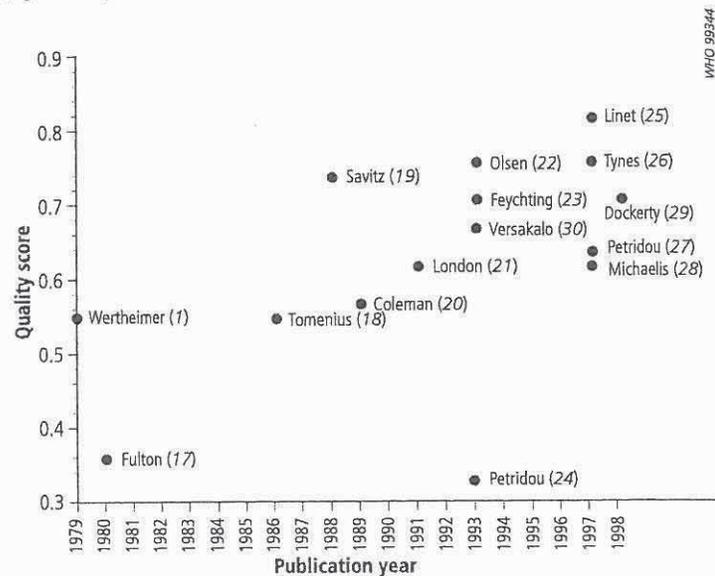
Statistical analysis was judged appropriate in most studies. Although the majority of studies listed *P* values and/or 95% CI, very few listed or analysed demographic data. Only two studies provided power calculations.

Quality scores for the individual studies ranged from 0.33 to 0.82, and there was a clear tendency for more recent studies to be of better quality (Fig. 1).

Statistical analysis

Table 2 lists the OR estimates with their respective 95% CIs extracted from each study and shows the results of pooling data from all studies according to the five different methods of exposure assessment. Although 17 of these 23 estimates (73.9%) had ORs greater than 1.00, only 5 (21.7%) were significantly greater than this ($P < 0.05$). The overall OR estimates of these meta-analyses were always greater than 1.00, indicating a potential effect of residential EMF exposure on childhood leukaemia risk. However, this effect is statistically significant only for meta-analyses based on wiring configuration codes (pooled OR = 1.46; 95% CI

Fig. 1. Relationship between year of publication and quality score of studies relating electromagnetic field (EMF) and childhood leukaemia. Individual studies are identified by the name of the first author (figures in parentheses are the literature references).



= 1.05–2.04; $P = 0.024$) and 24-h measurements of magnetic fields (pooled OR = 1.59; 95% CI = 1.14–2.22; $P = 0.006$). In the meta-analysis of studies in which the exposure assessment was made through spot measurements of magnetic field, the exclusion of the Feychting & Ahlbom study (23) and the inclusion of that by Tomenius (18) decreased the overall OR below 1.00 (pooled OR = 0.94; 95% CI = 0.50–1.77; $P = 0.850$). Individual studies included in the different meta-analyses appear heterogeneous ($P < 0.05$) only in the case of exposure assessment performed with wiring configuration codes or by distance.

Even in the presence of predefined and well-established criteria, we encountered in many studies more than one possibility for data extraction. For example, in the study by Wertheimer & Leeper (1), exposure assessment was performed both at birth and death addresses, which resulted in OR estimates of 2.28 and 2.97, respectively; Linnet et al. (25) calculated results using unmatched analysis as well as analysis of matched case-control pairs, with corresponding OR estimates of 1.19 and 1.39. To test the sensitivity of our results to the different choices of data extraction, we performed separate meta-analyses using, for each exposure assessment method, data that gave the lowest (best scenario) and the highest (worst scenario) OR estimates. The impact of the different choices of data extraction did not seem to be substantial (Table 3). The overall ORs resulting from studies based on wiring configuration codes and 24-h measurements of magnetic fields were always significantly different from 1.00, ranging from 1.33 to 1.58 and from 1.50 to 1.95, respectively. Interestingly, the heterogeneity of studies included in the meta-analysis based on wiring configuration codes, compared with the baseline analysis, tended to decrease, and in the best-scenario hypothesis,

Research

Table 2. Summary of studies included in the meta-analyses relating electromagnetic fields (EMFs) and childhood leukaemia according to EMF measurement methods

Study	Country	Age of study subjects (years)	No. of cases ^a	No. of controls ^a	OR ^b
Wiring configuration codes^c					
Wertheimer & Leeper (ref. 1, 1979)	USA	<19	155 (63, 40.6%)	155 (29, 18.7%)	2.98; 1.78–4.98
Fulton et al. (ref. 17, 1980)	USA	<21	198 (103, 52.0%)	225 (112, 50.0%)	1.09; 0.75–1.60
Savitz et al. (ref. 19, 1988)	USA	<15	97 (27, 27.8%)	259 (52, 20.1%)	1.54; 0.90–2.63
London et al. (ref. 21, 1991)	USA	<11	211 (122, 57.8%)	205 (92, 44.9%)	1.68; 1.14–2.48
Linet et al. (ref. 25, 1997)	USA	<15	402 (111, 27.6%)	402 (113, 28.1%)	0.98; 0.72–1.33
Petridou et al. (ref. 27, 1997)	Greece	<15	117 (11, 9.4%)	202 (14, 6.9%)	1.39; 0.61–3.18
<i>Overall</i>			1 180 (437, 37.0%)	1 448 (412, 28.4%)	1.46; 1.05–2.04 ^d
Distance from power distribution equipment^e					
Coleman et al. (ref. 20, 1989)	England	<18	84 (14, 16.7%)	141 (15, 10.6%)	1.68; 0.77–3.68
Feychting & Ahlbom (ref. 23, 1993)	Sweden	<16	38 (6, 15.8%)	554 (34, 6.1%)	2.87; 1.12–7.33
Petridou et al. (ref. 24, 1993)	Greece	<15	136 (96, 70.6%)	187 (132, 70.6%)	1.00; 0.62–1.62
Tynes & Haldorsen (ref. 26, 1997)	Norway	<15	148 (9, 6.1%)	579 (55, 9.5%)	0.62; 0.30–1.28
<i>Overall</i>			406 (125, 30.8%)	1 461 (236, 16.1%)	1.23; 0.70–2.18 ^f
Spot measurements of magnetic fields^g					
Tomenius (ref. 18, 1986)	Sweden	<19	243 (4, 1.6%)	212 (10, 4.7%)	0.34; 0.10–1.09
Savitz et al. (ref. 19, 1988)	USA	<15	36 (5, 13.9%)	207 (16, 7.7%)	1.93; 0.66–5.63
London et al. (ref. 21, 1991)	USA	<11	140 (16, 6.7%)	109 (11, 10.1%)	1.15; 0.51–2.59
Feychting & Ahlbom (ref. 23, 1993)	Sweden	<16	24 (4, 16.7%)	344 (70, 20.3%)	0.78; 0.26–2.36
Michaelis et al. (ref. 28, 1997)	Germany	<15	176 (6, 3.4%)	414 (16, 3.9%)	0.88; 0.34–2.28
<i>Overall</i>			376 (31, 8.2%)	1 074 (113, 10.5%)	1.11; 0.68–1.79 ^f
24-h measurements of magnetic fields^g					
London et al. (ref. 21, 1991)	USA	<11	164 (20, 12.2%)	144 (11, 7.6%)	1.68; 0.78–3.64
Linet et al. (ref. 25, 1997)	USA	<15	463 (58, 12.5%)	463 (44, 9.5%)	1.36; 0.90–2.07
Michaelis et al. (ref. 28, 1997)	Germany	<15	176 (9, 5.1%)	414 (8, 1.9%)	2.74; 1.04–7.21
Dockerty et al. (ref. 29, 1998)	New Zealand	<15	40 (7, 17.5%)	40 (3, 7.5%)	2.62; 0.63–10.95
<i>Overall</i>			843 (94, 11.1%)	1 061 (66, 6.2%)	1.59; 1.14–2.22 ^f
Calculated magnetic fields^g					
Feychting & Ahlbom (ref. 23, 1993)	Sweden	<16	37 (7, 18.9%)	554 (46, 8.3%)	2.58; 1.07–6.19
Olsen et al. (ref. 22, 1993)	Denmark	<15	833 (3, 0.4%)	1 666 (4, 0.2%)	1.50; 0.34–6.73
Verkasalo et al. (ref. 30, 1993)	Finland	<20	35 (3, 8.6%)	134 797 (7297, 5.4%)	1.64; 0.50–5.35
Tynes & Haldorsen (ref. 26, 1997)	Norway	<15	148 (1, 0.7%)	579 (14, 2.4%)	0.27; 0.04–2.10
<i>Overall</i>			1 053 (14, 1.3%)	137 596 (7 361, 5.3%)	1.55; 0.73–3.32 ^f

^a Figures in parentheses are the number and percentage of exposed subjects.

^b Figures in italics are 95% confidence intervals.

^c Exposure strata dichotomized in low current configuration vs high current configuration.

^d Random effects model, as described by DerSimonian & Laird (ref. 16).

^e Exposure strata dichotomized as distance <50 m vs ≥ 50 m.

^f Summary odds ratio (random effects model, as described by DerSimonian & Laird (ref. 16)).

^g Exposure strata dichotomized as magnetic field strength <0.2 μT vs ≥ 0.2 μT.

^h Calculation performed excluding the study by Tomenius (ref. 18), because of data overlapping with Feychting & Ahlbom's study (ref. 23).

became not significant ($P < 0.05$). In contrast, the heterogeneity of studies in which the exposure was assessed by 24-h EMF measures, although not significant, tended to increase slightly either in the best-case or worst-case scenario.

The quality of the studies does seem to have a substantial impact on our summary estimates, particularly in meta-analyses based on wiring configuration codes, 24-h measurements of magnetic fields,

and calculated magnetic fields, since high-quality-score studies have lower risk estimates than low-quality-score studies (Table 4).

Discussion

By the end of 1998, 15 studies had been published that provided relevant data on the association between

Table 3. Sensitivity of summary results of meta-analyses relating electromagnetic fields (EMFs) and childhood leukaemia, according to choices of data extraction

EMF measurement method	No. of studies	Summary OR ^a	P-value	Heterogeneity	
				χ^2 test	P-value
Wiring configuration codes					
Best scenario	6	1.33; <i>1.00-1.76</i> ^b	0.048	10.86 (5) ^c	0.054
Baseline analysis	6	1.46; <i>1.05-2.04</i>	0.024	16.00 (5)	0.007
Worst scenario	6	1.58; <i>1.14-2.21</i>	0.007	14.65 (5)	0.011
Distance from power distribution equipment					
Best scenario	—	—	—	—	—
Baseline analysis	4	1.23; <i>0.70-2.18</i>	0.47	8.46 (3)	0.037
Worst scenario	—	—	—	—	—
Spot measurements of magnetic fields					
Best scenario	4	1.03; <i>0.63-1.70</i>	0.9	0.81 (3)	0.847
Baseline analysis	4	1.11; <i>0.68-1.79</i>	0.68	1.73 (3)	0.63
Worst scenario	—	—	—	—	—
24-h measurements of magnetic fields					
Best scenario	4	1.50; <i>1.03-2.19</i>	0.034	3.96 (3)	0.266
Baseline analysis	4	1.59; <i>1.14-2.22</i>	0.006	2.55 (3)	0.466
Worst scenario	4	1.95; <i>1.11-3.40</i>	0.019	6.04 (3)	0.109
Calculated magnetic fields					
Best scenario	—	—	—	—	—
Baseline analysis	4	1.55; <i>0.73-3.32</i>	0.25	6.10 (3)	0.107
Worst scenario	—	—	—	—	—

^a Summary odds ratio (random effects model, using the method described by DerSimonian & Laird, ref. 16).

^b Figures in italics are 95% confidence intervals.

^c Figures in parentheses are degrees of freedom.

residential exposure to EMF and childhood leukaemia: nine in Europe, five in the USA, and one in New Zealand. The majority of these studies appeared between 1986 and 1993, and five studies were published in the period 1997-98. All but one were case-control studies, most of which were based on a comprehensive case ascertainment in a geographically defined population. Exposure assessment was based on a variety of methods, including wiring configuration codes, distance to power distribution equipment, spot and 24-h measures of magnetic field strength, and calculated indices using distance to power distribution equipment and historical load data. One-third of the studies employed more than one method of EMF measurement.

In the present investigation, pooling results arising from the use of different exposure criteria was avoided, and we performed separate meta-analyses for each method of exposure assessment. The meta-analysis based on wiring configuration codes confirmed the 1.5-fold statistically significant excess of childhood leukaemia already documented in previous studies (7-17). The exposure rates among control subjects in the individual studies varied from 6.9% to 50%, and the heterogeneity was found to be statistically significant. The ca. 1.5-fold risk of childhood leukaemia was also reported in meta-

analyses based on calculated magnetic fields and 24-h measurements of magnetic fields. This excess risk was significant only in the analysis of studies relying on 24-h measurements of magnetic fields, in which the exposure rate among controls varied from 1.9% to 9.5%, and the heterogeneity was not statistically significant. Meta-analyses based on distance and spot measurements of magnetic fields produced ORs of lower magnitude and not significantly different from unity.

Other meta-analyses of the association between exposure to residential electromagnetic fields and childhood leukaemia have been carried out. For example, Washburn et al. (6), combining results of studies published before 1992, found increased risks for leukaemia, lymphoma, and nervous system cancers, although the risk of lymphoma was not significant. Miller et al. (7), in separate meta-analyses of studies published before 1993 according to EMF measurement methods, documented statistically significant increased risks for childhood leukaemia for wiring configuration codes, and distance and calculated indices, whereas spot measures consistently showed non-significant odds ratios. On the basis of studies published before 1994, Meinert & Michaelis (8) confirmed the significant association between childhood leukaemia and residential EMF

36/90

Table 4. Sensitivity of summary results of meta-analyses relating electromagnetic fields (EMFs) and childhood leukaemia to quality scores of individual studies

EMF measurement method	No. of studies	Summary OR ^a	P-value	Heterogeneity	
				χ^2 test	P-value
Wiring configuration codes					
All studies	6	1.46; 1.05–2.04 ^b	0.024	16.00 (5) ^c	0.007
Low-quality studies	3	1.72; 1.01–2.93	0.045	9.29 (2)	0.009
High-quality studies	3	1.15; 0.85–1.55	0.37	2.42 (2)	0.298
Distance from power distribution equipment					
All studies	4	1.23; 0.70–2.18	0.47	8.46 (3)	0.037
Low-quality studies	2	1.18; 0.73–1.89	0.68	1.24 (1)	0.265
High-quality studies	2	1.29; 0.29–5.81	0.74	6.93 (1)	0.008
Spot measurements of magnetic fields					
All studies	4	1.11; 0.68–1.79	0.68	1.73 (3)	0.63
Low-quality studies	2	1.03; 0.55–1.91	0.93	0.18 (1)	0.671
High-quality studies	2	1.24; 0.51–2.99	0.63	1.47 (1)	0.225
24-h measurements of magnetic fields					
All studies	4	1.59; 1.14–2.22	0.006	2.55 (3)	0.466
Low-quality studies	2	2.03; 1.11–3.71	0.022	0.93 (1)	0.335
High-quality studies	2	1.43; 0.96–2.14	0.08	0.71 (1)	0.399
Calculated magnetic fields					
All studies	4	1.55; 0.73–3.32	0.25	6.10 (3)	0.107
Low-quality studies	2	2.21; 1.07–4.58	0.033	0.65 (1)	0.42
High-quality studies	2	0.74; 0.14–3.83	0.72	1.57 (1)	0.21

^a Summary odds ratio (random effects model, calculated using the method described by DerSimonian & Laird, ref. 16).

^b Figures in italics are 95% confidence intervals.

^c Figures in parentheses are degrees of freedom.

exposure measured through wiring configuration codes, whereas no association was found with distance; the meta-analysis of studies in which the EMF exposure was either measured directly or calculated did not show an increase of childhood leukaemia with higher cut-off points. More recently, Wartenberg (9, 11) documented that wiring codes and related markers of exposure, such as proximity to power lines and calculated magnetic fields from power lines, were associated with an approximate 1.5-fold excess risk of childhood leukaemia, whereas the evidence of an association with magnetic fields measured directly was not, in the aggregate, supported. None of these previous meta-analyses provided overall risk estimates from studies in which the exposure assessment was performed through 24-h measurements of magnetic fields.

The quality of the individual studies included in our meta-analyses was assessed on the basis of their statistical analyses and the efforts made to minimize potential for selection bias, misclassification bias related to exposure as well as disease, and information bias due to failure to consider potential confounding variables. Items concerned with efforts to minimize potential bias were given twice the weight of items evaluating data analysis. Since there appears to be no "gold standard" at present for EMF

measurement, we did not evaluate the operational definition of exposure (e.g., all EMF measurements methods were assumed to be equally valid). It is well known that all quality assessment systems have a subjective component, none have yet been validated, and efforts to correlate quality scores with direction or size of effect have had mixed findings (31, 32). Therefore, we did not use quality scores to determine studies to be included in the meta-analysis or to assign statistical weights.

Despite these limitations, assessment of the quality of the individual studies used in our meta-analysis allowed us to draw the following conclusions:

- there has been improvement in study design and reporting, since findings published more recently tended to receive a higher quality rating;
- the possibility of selection bias, misclassification bias related to exposure, and information bias related to failure to consider potential confounders cannot be ruled out;
- most importantly, pooling high-quality-score studies resulted in lower risk estimates than did pooling low-quality-score studies.

If high-quality studies are more likely to yield valid information than low-quality studies, we can con-

clude that currently available data do not permit exact quantification of the true excess risk of childhood leukaemia due to residential EMF exposure.

An important limitation of the meta-analytical approach is related to publication bias. This occurs if positive results are more likely to be published than negative ones (33–35). In the case of residential EMF exposure and childhood leukaemia, there is an important factor that may mitigate the tendency for negative findings to be excluded from the published literature. In view of the considerable interest in this topic, it seems unlikely that any investigator would have trouble in getting even a negative study published. Indeed, the great majority of the studies that we included in our meta-analyses reported risk estimates with *P* values greater than 0.05, suggesting that non-significant results are readily publishable.

Several conclusions may be drawn from the results of this study.

- First, an association between residential EMF exposure and childhood leukaemia may exist. This possibility is supported by the statistically significant risk estimates obtained by pooling results of studies in which the exposure was assessed not only indirectly with markers such as wiring

configuration codes but also through direct measurements of magnetic field for at least a 24-h period.

- Second, the magnitude of this excess risk, if any, is at present unknown, given the possibility of selection bias, exposure misclassification, and the existence of confounding variables in the individual studies.
- Third, there appears to be a clear trend for the more recent publications to be of better quality. If this trend continues, new good-quality studies can be expected in the future.
- Finally, enough evidence exists to lead us to conclude that dismissing concerns about EMF and childhood leukaemia is unwarranted. What is required is the publication of new state-of-the-art epidemiological studies that incorporate comparable measures for both exposure and outcomes in order to facilitate future meta-analyses. If this excess risk of childhood leukaemia is confirmed, we should thoroughly investigate, also from a cost-effectiveness point of view, possible options for minimizing exposure in order to provide definitive answers for policy-makers. ■

Résumé

Méta-analyse de la relation entre leucémie de l'enfant et exposition à des champs électromagnétiques du fait du lieu de résidence

Les études épidémiologiques consacrées à la relation entre leucémie de l'enfant et exposition à des champs électromagnétiques du fait du lieu de résidence sont suggestives sans toutefois être concluantes. Un certain nombre d'entre elles se prêtent cependant à la technique de la méta-analyse. Nous avons effectué une recherche bibliographique au moyen de MEDLINE et d'autres sources de données pour retenir 14 études cas-témoins et une étude de cohorte dont nous avons évalué la qualité épidémiologique et que nous avons soumises ensuite à une méta-analyse. Nous avons tiré de chacune d'entre elles une estimation du risque relatif et nous avons réuni ces valeurs. Une méta-analyse distincte a été effectuée selon le mode d'évaluation du champ électromagnétique (code de configuration du bobinage, distance au centre de distribution, mesures ponctuelles ou sur 24 h de l'intensité du champ magnétique (densité de flux magnétique) ou détermination de ce champ par le calcul). La méta-analyse basée sur les codes de configuration a donné une estimation combinée du risque relatif égale à 1,46 (intervalle de confiance (IC) à 95% = 1,05–2,04, *p* = 0,024); celle qui prenait en

compte la mesure du champ sur 24 h donnant une valeur de 1,59 (IC à 95% = 1,14–2,22, *p* = 0,006) et indiquant donc la possibilité d'une relation entre exposition au champ magnétique et leucémie chez l'enfant). Dans la plupart des cas, le regroupement des résultats des études de très bonne qualité a donné une estimation du risque plus faible que dans le cas des études de qualité médiocre. On constate une nette tendance à l'amélioration de la qualité dans les études récentes. On a en tout cas suffisamment de preuves pour pouvoir conclure qu'il n'est pas justifié de faire bon marché des craintes qui se sont exprimées au sujet du risque de leucémie chez les enfants exposés à des champs électromagnétiques du fait de leur lieu de résidence. Il est nécessaire d'effectuer d'autres études épidémiologiques de très bonne qualité basées sur des mesures comparables de l'exposition et de son résultat pour pouvoir confirmer ces observations. Dans l'éventualité d'une confirmation de cet excès de risque, il faudrait étudier minutieusement les possibilités de réduction de l'exposition afin que les décideurs puissent disposer de conclusions définitives.

Resumen

Exposición a campos electromagnéticos en zonas de residencia y leucemia infantil: metanálisis

Las diversas investigaciones epidemiológicas realizadas sobre la relación entre la exposición a campos electromagnéticos (CEM) en zonas de residencia y la leucemia

infantil se han saldado con indicios no concluyentes. Varios de esos trabajos, sin embargo, se prestan a ser estudiados mediante técnicas de metanálisis. En el

presente metanálisis se incluyeron 14 estudios de casos y testigos y un estudio de cohortes identificados a través de MEDLINE y de otras fuentes, previa evaluación de su calidad epidemiológica. Se combinaron las estimaciones del riesgo relativo obtenidas en cada uno de los estudios y se realizaron diversos metanálisis basados en las distintas evaluaciones de la exposición a los CEM (códigos de configuración del cableado, distancia a las instalaciones de distribución de la energía, mediciones puntuales y de 24 horas de la potencia de los campos electromagnéticos (densidad de flujo magnético), campos magnéticos calculados). El metanálisis basado en los códigos de configuración del cableado arrojó una estimación del riesgo relativo de 1,46 (IC95%: 1,05 - 2,04, $p = 0,024$), y en el caso de las mediciones de 24 horas de los campos electromagnéticos se obtuvo un valor de 1,59 (IC95%: 1,14-2,22, $p = 0,006$), lo que

indica una posible relación entre la exposición a CEM en zonas de residencia y la aparición de leucemia infantil. En la mayoría de los casos las combinaciones de estudios de alta calidad dieron estimaciones del riesgo más bajas que las combinaciones de estudios de baja calidad. Se observa una clara tendencia a una mayor calidad en los estudios más recientes. Existen datos suficientes para concluir que no puede descartarse una relación entre la influencia de los campos electromagnéticos en la zona de residencia y la leucemia infantil. Es necesario realizar nuevos estudios epidemiológicos de gran calidad, con mediciones comparables tanto de la exposición como de los resultados, para corroborar estos resultados. Si se confirmara el posible exceso de riesgo, habría que investigar a fondo las alternativas para reducir al mínimo la exposición y proporcionar respuestas definitivas a los formuladores de políticas.

References

1. Wertheimer N, Leeper E. Electrical wiring configurations and childhood cancer. *American journal of epidemiology*, 1979, **109**: 273–284.
2. Campion EW. Power lines, cancer, and fear. *New England journal of medicine*, 1997, **337**: 44–46.
3. ELF-EMF European Feasibility Study Group. Need for a European approach to the effects of extremely low-frequency electromagnetic fields on cancer. *Scandinavian journal of work, environment and health*, 1997, **14**: 23–25.
4. Wartenberg D. Leukaemia and exposure to magnetic field. *New England journal of medicine*, 1997, **337**: 1471.
5. Blettner M et al. Traditional reviews, meta-analyses, and pooled analyses in epidemiology. *International journal of epidemiology*, 1999, **28**: 1–9.
6. Washburn EP et al. Residential proximity to electricity transmission and distribution equipment and risk of childhood leukaemia, childhood lymphoma, and childhood nervous system tumors: systematic review, evaluation, and meta-analysis. *Cancer causes and control*, 1994, **5**: 299–309.
7. Miller MA et al. Variation in cancer risk estimates for exposure to powerline frequency electromagnetic fields: a meta-analysis comparing EMF measurement methods. *Risk analysis*, 1995, **15**: 281–287.
8. Meinert R, Michaelis J. Meta-analyses of studies on the association between electromagnetic fields and childhood cancer. *Radiation and environmental biophysics*, 1996, **35**: 11–18.
9. National Research Council. *Possible health effects of exposure to residential electric and magnetic fields*. Washington, DC, National Academy Press, 1997.
10. Angelillo IF, Ferrera G, Pavia M [Exposure to electromagnetic fields and effects on human health: a meta-analysis]. Proceedings of the 38th National Meeting of the Italian Public Health Association (Fiuggi, Italy, 27–30 September 1998). *Annali di igiene, medicina preventiva e di comunità*, 1998, **10** (Suppl.1): 115–141 (in Italian, summary in English).
11. Wartenberg D. Residential magnetic fields and childhood leukaemia: a meta-analysis. *American journal of public health*, 1998, **88**: 1787–1794.
12. Chalmers TC et al. A method for assessing the quality of a randomized trial. *Controlled clinical trials*, 1981, **2**: 31–49.
13. Longnecker MP et al. A meta-analysis of alcohol consumption in relation to risk of breast cancer. *Journal of the American Medical Association*, 1988, **260**: 652–656.
14. Morris RD et al. Chlorination, chlorination by-products and cancer: a meta-analysis. *American journal of public health*, 1992, **82**: 955–963.
15. Villari P et al. Cesarean section to reduce perinatal transmission of human immunodeficiency virus: a meta-analysis. *The online journal of current clinical trials*, 1993, Jul 8: Doc. No. 74.
16. DerSimonian R, Laird N. Meta-analysis in clinical trials. *Controlled clinical trials*, 1986, **7**: 177–188.
17. Fulton JP et al. Electrical wiring configurations and childhood leukaemia in Rhode Island. *American journal of epidemiology*, 1980, **111**: 292–296.
18. Tomenius L. 50-Hz electromagnetic environment and the incidence of childhood tumors in Stockholm County. *Bioelectromagnetics*, 1986, **7**: 191–207.
19. Savitz DA et al. Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. *American journal of epidemiology*, 1988, **128**: 21–38.
20. Coleman MP et al. Leukaemia and residence near electricity transmission equipment: a case-control study. *British journal of cancer*, 1989, **60**: 793–798.
21. London SJ et al. Exposure to residential electric and magnetic fields and risk of childhood leukaemia. *American journal of epidemiology*, 1991, **134**: 923–937.
22. Olsen JH, Nielsen A, Schulgen G. Residence near high voltage facilities and risk of cancer in children. *British medical journal*, 1993, **307**: 891–895.
23. Feychting M, Ahlbom A. Magnetic fields and cancer in children residing near Swedish high-voltage power line. *American journal of epidemiology*, 1993, **138**: 467–481.
24. Petridou E et al. Age of exposure to infections and risk of childhood leukaemia. *British medical journal*, 1993, **307**: 774.
25. Linet MS et al. Residential exposure to magnetic fields and acute lymphoblastic leukaemia in children. *New England journal of medicine*, 1997, **337**: 1–7.
26. Tynes T, Haldorsen T. Electromagnetic fields and cancer in children residing near Norwegian high-voltage power lines. *American journal of epidemiology*, 1997, **145**: 219–226.
27. Petridou E et al. Electrical power lines and childhood leukaemia: a study from Greece. *International journal of cancer*, 1997, **73**: 345–348.
28. Michaelis J et al. Combined risk estimates for two German population-based case-control studies on residential magnetic fields and childhood acute leukaemia. *Epidemiology*, 1997, **9**: 92–94.
29. Dockerty JD et al. Electromagnetic field exposures and childhood cancers in New Zealand. *Cancer causes and control*, 1998, **9**: 299–309.
30. Verkasalo PK et al. Risk of cancer in Finnish children living close to power lines. *British medical journal*, 1993, **307**: 895–899.

Residential exposure to electromagnetic fields and childhood leukaemia

31. Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. *American journal of epidemiology*, 1990, **132**: 612-628.

32. Emerson JD et al. An empirical study of the possible relation of treatment differences to quality scores in controlled randomized clinical trials. *Controlled clinical trials*, 1990, **11**: 339-352.

33. Begg CB, Berlin JA. Publication bias and dissemination of clinical research. *Journal of National Cancer Institute*, 1989, **81**: 107-115.

34. Easterbrook PJ et al. Publication bias in clinical research. *Lancet*, 1991, **337**: 867-872.

35. Dickersin K, Min YI, Meinert CL. Factors influencing publication of research results: follow-up of applications submitted to two institutional review boards. *Journal of American Medical Association*, 1992, **267**: 374-378.

Magnetic Fields and Cancer: Animal and Cellular Evidence—an Overview

Bo Holmberg

National Institute of Occupational Health, Solna, Sweden

A few animal studies on the possible carcinogenic effect of magnetic fields have been published. They have been designed to reveal a possible tumor promotion obtained by applying continuous or pulsed alternating fields at flux densities varying between 0.5 μ T and 30 mT on mice or rats initiated with different initiators. One study with 2 mT applied on DMBA-initiated mice may suggest a copromotive effect together with the promoter TPA. Another study on rats suggests an inhibitory effect by a magnetic field on rat liver foci formation, induced with DENA. Cell studies show that magnetic fields at some frequencies, amplitudes, and wave forms interact with biological systems. Thus effects have been seen, e.g., on enzymes related to growth regulation, on calcium balance in the cell, on gene expression, and on pineal metabolism and its excretion of the oncostatic melatonin. Cellular and physiologic studies thus suggest effects that may be related to cell multiplication and tumor promotion. — Environ Health Perspect 103(Suppl 2):63–67 (1995)

Key words: magnetic fields, carcinogenicity, mechanisms

Introduction

Carcinogenesis is a multistep and multifactorial phenomenon (1). Early animal experimental studies demonstrating the multistep character of tumorigenesis (2, 3) defined the two major steps as initiation and promotion by observing the development of tumors in mouse skin after local treatment with two types of carcinogens. The term promotion was originally an operational definition, associated with the particular two-phase experimental protocol used. Initiation has since been largely associated with genotoxic effects due to direct or indirect interactions between the carcinogen, or its metabolites, and DNA. The promotion step is responsible for the conversion of initiated cells to transformed cells. Promotion is associated with a number of cellular events, largely nongenotoxic in nature. Some animal studies also form the basis for a further division of the promotion stage into stage I (conversion) and stage II (propagation). A continuing cellular evolution into a fully invasive and metastasizing tumor cell tissue is termed progression. A chemical or physical factor capable to be effective in all biological steps is termed a complete carcinogen. It may be possible in series of animal experiments to

define the activity of a factor for one or more of these (often overlapping) biological steps. The multistep nature of tumor development is assumed to be a general phenomenon and not restricted to certain tumors.

Two-phase experimental protocols have been established for mouse skin tumors and for rat liver tumors. In the mouse, the occurrence of benign tumors, papillomas, in the skin is analyzed and in the rat the formation of preneoplastic cell areas (foci) in the liver is determined. Two-phase protocols for other types of tumors, such as leukemia and brain tumors, have been less commonly used in animal models in the past. Such studies with magnetic fields are planned or ongoing with different initiator regimens.

Animal studies are essential to define the exposure parameters of magnetic fields responsible for an effect. The flux density, the frequency, the exposure duration, and exposure profile are among the important critical variables. Animal studies, as well as cell studies, are also essential in order to get an indication of the carcinogenic mechanism. The identification of the carcinogenic mechanism is important for a solid risk assessment.

So far no full-scale, long-term animal studies have been performed studying the possibility of magnetic fields acting as a complete carcinogen. Such studies are planned or already started in the United States (the NIEHS National Toxicology Program) and Italy. Data from these studies will appear within the next few years.

In some animal experiments designed primarily for studying possible tumor

promotion by magnetic fields (summarized below), parallel series of animals have been included, which attempt to give an indication on whether magnetic fields also act as a complete carcinogen. As such parallel series consisted generally of small group numbers, only limited conclusions can be drawn from those observations in this context. With one exception (4), the other studies (5,6) seem not to reveal an effect of magnetic fields as a complete carcinogen. The Georgian study (4) showed around 30% mammary tumors in rats exposed to 20 μ T 50 Hz daily for lifetime. Control animals had no tumors. Details of design, exposure regimen, and tumor observations are, however, lacking.

Animal Experiments

Tumor Promotion Studies

In a 2-year tumor promotion study with continuous 50-Hz magnetic fields, using 0.5-mT and 50- μ T exposure on female NMRI mice (5), the skin tumors and other neoplastic lesions were observed after topical application of DMBA. The animals were exposed to a magnetic field for 19 or 21 hr a day. TPA was used as a positive control for skin tumor promotion. There was no difference in skin tumor development among DMBA and magnetic field-exposed animals compared to the DMBA-treated animals. This analysis was made with correction for survival and made for both cumulated tumor-bearing animals and cumulated number of skin tumors. Skin hyperplasia analyses did not reveal skin hyperplasia among DMBA + magnetic

This article was presented at the Fifth International Conference of the International Society for Environmental Epidemiology held 15–18 August 1993 in Stockholm, Sweden.

Address correspondence to Dr. Bo Holmberg, Department of Toxicology, National Institute of Occupational Health, S-17184 Solna, Sweden. Telephone 46 8 7309759. Fax 46 8 7303312.

field-exposed mice related to magnetic field exposure.

A study (6) using female CBA/S mice and pulsed magnetic fields at 20 kHz with a saw-tooth shape and a flux density of 15 μ T (peak to peak) was designed to observe a possible enhancing effect of X-ray-induced lymphomas or of spontaneous lymphomas in this particular strain. Four X-ray doses of 1.31 Gy each were administered with a 4-day interval and the subsequent magnetic field exposure was performed 24 hr/day over the lifetime. In the X-ray-treated series a high lymphoma frequency was obtained. There was no statistical difference in lymphoma appearance between the X-irradiated animals and those also exposed to magnetic fields.

A large German study (7) using Sprague-Dawley rats involved four experiments and exposures to 15-mT DC fields or a gradient field of 0.31–1 μ T, as well as a homogeneous field of 30-mT (continuous) 50-Hz AC fields, after initiation of mammary tumors with repeated (4 \times 5 mg) oral administrations of DMBA. The exposure was performed 24 hr/day for 91 days. Magnetic fields did not alter the induced tumor frequency except in one experiment, where 30-mT AC fields increased the number of tumors per tumor-bearing animal. Fifteen-millitesla DC fields increased tumor weight but not tumor frequency in one experiment. The number of animals in the groups was small (18–36), limiting the sensitivity of the study. Also, the exposure and observation period was short. The experiment with 30-mT AC fields was repeated without the second time showing an increase in the number of tumors per tumor-bearing animal.

In a Georgian study (4) female rats (strain not specified) were initiated with NMU at a dose of 50 mg/kg bw administered iv to induce mammary tumors, which could be enhanced by subsequent 50 Hz magnetic field exposure. Two types of magnetic fields were used, variable (AC) fields and static (DC) fields with an intensity of 20 μ T. The magnetic field exposure was 0.5 hr or 3 hr daily starting 2 days after NMU administration and possibly ongoing for the animal's lifetime (exposure details are not specified). Three hours of daily exposure to AC or DC fields increased the incidence of NMU-induced mammary tumors, while 0.5 hr of daily exposure did not. Also, the time-to-tumor appearance was shorter among the animals exposed for 3 hr daily. In the 3-hr groups, malignant tumors dominated among the histologic types, compared to the control

group. The results obtained in these experimental series need to be confirmed.

The rat liver foci model has been used in a series of experiments (8,9). The exposure lasts for 12 weeks, when animals are sacrificed and liver samples are taken for staining for the enzyme markers, GSTp and GGT. In one study (8), 0.5-, 5-, 50- μ T, and 0.5-mT continuous magnetic fields at 50 Hz were used in a tumor promotion protocol using Sprague-Dawley rats and DENA as initiator. This study did not show an enhancing effect of continuous magnetic fields on the development of preneoplastic foci induced by DENA.

Interaction Studies

In three papers on skin tumor promotion in SENCAR mice (10–12) 2 mT and 60 Hz continuous magnetic field-exposure was used during 6 hr/day, 5 days/week up to 21 to 23 weeks. In the investigation by McLean et al. (11) both promotion (above) and co-promotion of skin papillomas were studied in female SENCAR mice exposed to 2-mT, 60-Hz magnetic field. Using an initiating treatment of 2.56 μ g DMBA (10 nmole) and a subsequent exposure to 1 μ g TPA applied to the dorsal skin weekly, they tested whether a 21-week magnetic field exposure could modify tumor development. They found a slightly earlier development of tumors in the magnetic field-exposed animals, the difference in time to appearance of tumors was, however, not statistically significant. In a follow-up study (12) the same strain and sex was exposed under the same field conditions using a weekly dose of 0.3 mg of TPA. The rate of tumor development was found to be increased in the magnetic field-exposed group compared to a sham-exposed TPA group. A difference in the cumulated number of mice with tumors was observed, but this difference did not reach statistical significance at the end of the observation period (23 weeks). Splenomegaly (11) was observed among the TPA+magnetic field-exposed mice, and there was a greater number of mononuclear cells in the spleens of animals with combined exposure. There was also a depressed NK cell activity. This is suggestive of a suppression of the immune system, possibly related to a development of leukemia/lymphomas.

In one liver foci experiment (9), 50-Hz continuous magnetic field exposure at 0.5 μ T and 0.5 mT was applied during both the hepatectomy and the initiation phase (with DENA), as well as during the promotion phase in combination with a

phenobarbital (PB) treatment. The formation of preneoplastic foci was slightly inhibited in terms of foci area, frequency, and volume for both enzyme markers. This inhibition was statistically significant for 0.5 μ T in terms of number of GGT-positive foci per cubic centimeter liver and for 0.5 mT in terms of mean area and percentage foci volume as estimated by the GSTp marker.

Hitherto published animal studies, using continuous 50- or 60-Hz magnetic fields, do not support the hypothesis of a tumor promotive effect of magnetic fields, as studied in mice and rats, with different protocols and initiators. Only one study reports a (surprisingly) high incidence of mammary tumors induced by NMU after 3 hr of daily exposure to variable or static magnetic fields. One other study in mice suggests a copromotional effect of 2-mT and 60-Hz magnetic fields obtained with the skin-specific control promoter. The suggestive information on mammary tumors and copromotion of skin tumors needs to be confirmed. Several animal experimental studies on tumor promotion with different magnetic field exposure regimens, initiators, tumor types under observation, and animal strains are ongoing or planned in Canada, Italy, Japan, Sweden, and the United States.

Physiologic Studies

One hypothesis, which has been forwarded (13) for magnetic field-related cancer development, is derived from physiologic studies on the hormone melatonin. Melatonin is excreted nightly from the pineal gland, and its formation and secretion are inhibited by periods of light or magnetic field exposure. Melatonin is oncostatic (14) via inhibition of the mitogenic activity of, e.g., estrogen, or by acting directly by blocking cell proliferation (15). Disturbances in the melatonin rhythm appear also to affect the function of the immune system (16). Pinealectomy increases chemically induced melanomas in Syrian hamsters (17) and mammary tumors in the rat (14,18). Injections of melatonin into rats induced with DMBA reduced tumor growth and incidence in some studies (18–21). Whether melatonin plays a regulating role for the development of other tumor types is not known.

Short, repeated exposures to 40- μ T magnetic fields inhibit the conversion of serotonin to melatonin in the pineal gland in the rat (22), probably by eddy currents (23,24) induced by rapid alterations in the instantaneous inversion of the horizontal component of the geomagnetic field.

Cell Experiments

A few studies have been made on the possible genotoxicity of magnetic fields on cell systems (25), without finding mutations in *Salmonella* (26,27), strand breaks (28), or effects on DNA repair in human fibroblasts *in vitro* (29). In some of these studies there have been exposures to both electric and magnetic fields. Pulsed magnetic fields in some pulse widths, flux densities, and frequencies, but not in others, seem to increase the rate of DNA synthesis (30) of V79 cells and human fibroblasts (31) *in vitro*.

There seems to be general agreement (25,32,33) among reviewers on the issue of possible DNA injury related to magnetic field exposure that no such effects can be ascribed to magnetic fields.

Studies with cellular systems using different exposure setups, exposure durations, amplitudes, frequencies, and wave forms indicate that biological effects of magnetic fields on cellular systems are at hand (34-44), which may be related to cell multiplication or even tumor promotion.

Exposing human leukemia cells (45-47) or normal rat lymphocytes (48) to electromagnetic fields at various frequencies increases the transcripts of *c-myc* or histone. Transcriptional changes on the RNA level have also been observed with magnetic field in *Drosophila* and *Sciara* salivary gland cells (49,50) and in *Sacharomyces* of specific genes, genes responsible for the production of heat shock proteins (51).

In addition to effects on *c-myc* transcription, the expression of *c-fos*, *c-jun*, and protein kinase C in a lymphoblastoid cell line has been found to be altered by a magnetic

field exposure (52) of short duration and of *c-fos* in a HeLa subline by a static magnetic field (53). Those genes are involved in the regulation of cell growth, and a change in the production of proteins resulting from a changed expression of those genes may influence the cellular proliferation in the tissue. Should such cells already be initiated, a tumor may result.

Effects on gene expression seem to depend on frequency, field strength, and exposure time (39,46). Thus the concept of exposure or dose "windows," where biological effects occur—like effects on calcium metabolism (35,36,54), or increases in the activity of certain enzymes (55) involved in cell growth—has been forwarded.

It seems to be well established that electromagnetic fields induce changes in the calcium metabolism of exposed cells. The calcium efflux has been increased (37, 54-57) in a number of studies; also, an increase in cytosolic calcium from extracellular sources in stimulated rat thymocytes exposed to 60-Hz sinusoidal magnetic field (44) and 3-Hz pulsed fields has been observed (58). Magnetic fields from an MRI unit (59) increase cytosolic calcium in human leukemia cells. Calcium is involved, e.g., in cellular processes (signal transduction) leading to mitogenesis; and the investigation of the magnetic field interaction with the calcium balance may be of great importance for the understanding of the molecular mechanism of magnetic field effects on different cell types. Transmembrane calcium signaling events may also be of importance (57) for the mediation of magnetic field effects on cells of the immune system. The effect of magnetic fields on calcium metabolism seems to be optimized when the ratio of the

electromagnetic frequency to the DC field intensity equals the charge-to-mass ratio (cyclotron resonance theory) of nonhydrated ions such as calcium, lithium, potassium, and magnesium (60).

The cell growth-related enzyme ornithine decarboxylase has been found to be increased (61) in mammalian tumor cells after exposure to electromagnetic fields. This increase was optimized (62) in mouse fibroblasts when the coherence of the time-varying magnetic field was maintained for a certain minimum period.

Exposure of mouse embryo cells *in vitro* to repeated 60-Hz electromagnetic fields enhances the colony formation of TPA-treated cells (63). This suggests that magnetic fields may act as a growth stimulator via membrane-related events. Also, studies using 72-Hz pulse trains and 15-Hz recurrent bursts (64) on osteoblasts *in vitro* indicate that magnetic fields affect membrane receptor function as observed by an inhibition of responses of cells to parathyroid hormone.

Thus studies on a variety of cellular systems, including mammalian and human cells, seem to indicate that biological effects on the cellular and subcellular level can be related to electromagnetic fields. The effects studied have some relevance to cell function and growth and, perhaps, tumor promotion. The exposure parameters are far from well characterized or uniform over the cellular systems studied. Furthermore, the possible magnetic field-related effects on cellular and subcellular phenomena need to be demonstrated as to their validity for whole animals (41) at long-term exposure conditions that can be related to human risk.

REFERENCES

1. Hermo H. Chemical carcinogenesis: tumor initiation and promotion. In: Occupational Cancer and Carcinogenesis (Brandt-Rauf PW, ed). Occupational Medicine. State of the Art Reviews Vol 2, No 1. Philadelphia: Hanley & Belfus Inc., 1987;1-25.
2. Mottram JC. A developing factor in experimental blastogenesis. J Pathol Bacteriol 56:181-187 (1944).
3. Berenblum I, Shubik P. The role of croton oil applications, associated with a single painting of a carcinogen, in tumour induction of the mouse's skin. Br J Cancer 1:379-382 (1947).
4. Beniashvili DSh, Bilanishvili VG, Menabde MZ. Low-frequency electromagnetic radiation enhances the induction of rat mammary tumors by nitrosomethyl urea. Cancer Lett 61:75-79 (1991).
5. Rannug A, Ekström T, Hansson Mild K, Holmberg B, Gimenez-Conti I, Slaga TJ. A study on skin tumour formation in mice with 50 Hz magnetic field exposure. Carcinogenesis 14:573-578 (1993).
6. Svedenstål BM, Holmberg B. Lymphoma development among mice exposed to X-rays and pulsed magnetic fields. Int J Radiat Biol 64:119-125 (1993).
7. Mevissen M, Stamm A, Buntenkötter S, Zwingelberg R, Wahnschaffe U, Löscher W. Effects of magnetic fields on mammary tumor development induced by 7,12-dimethylbenz(a)anthracene in rats. Bioelectromagnetics 14:131-143 (1993).
8. Rannug A, Holmberg B, Hansson Mild K. A rat liver foci promotion study with 50 Hz alternating magnetic fields. Environ Res 62:223-229 (1993).
9. Rannug A, Holmberg B, Ekström T, Hansson Mild K. Rat liver foci study on coexposure with 50 Hz magnetic fields and known carcinogens. Bioelectromagnetics 14:17-27 (1993).
10. Stuchly MA, Lecuyer DW, McLean J. Cancer promotion in a mouse-skin model by 60 Hz magnetic field: 1. Experimental design and exposure system. Bioelectromagnetics 12:261-271 (1991).

11. McLean JRN, Stuchly MA, Mitchel REJ, Wilkinson D, Yang H, Goddard M, Lecuyer DW, Schunk M, Callary E, Morrison SD. Cancer promotion in a mouse-skin model by a 60 Hz magnetic field. 2. Tumor development and immune response. *Bioelectromagnetics* 12:273-287 (1991).
12. Stuchly MA, McLean JRN, Burnett R, Goddard M, Lecuyer DW, Mitchel REJ. Modification of tumor promotion in the mouse skin by exposure to an alternating magnetic field. *Cancer Lett* 65:1-7 (1992).
13. Stevens R. Electric power use and breast cancer: a hypothesis. *Am J Epidemiol* 125:556-561 (1987).
14. Blask DE. The emerging role of the pineal gland and melatonin in oncogenesis. In: *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer* (Wilson BW, Stevens RG, Anderson LE, eds). Columbus, OH: Battelle Press, 1990; 319-335.
15. Narita T. Effect of melatonin on B16 melanoma growth. In: *The Pineal Gland and Cancer* (Gupta D, Attanasio A, Reiter RJ, eds). London: Brain Research Promotion, 1988; 345-354.
16. Angeli A, Gatti G, Sartori D, Del Ponte D, Carignola R. Effects of Exogenous Melatonin on the Human Natural Killer (NK) Cell Activity. An Approach to the Immunomodulatory Role of the Pineal Gland (Gupta D, Attanasio A, Reiter RJ, eds). London: Brain Research Promotion, 1988; 145-156.
17. Aubert C, Prade M, Bouhon C. Effet de la pinealectomie sur les tumeurs melaniques du hamster dore induites par l'administration (*per os*) d'une seule dose 9-10-dimethyl-1-2-benz(a)anthracene. *CR Acad Sci* 272:2465-2468 (1970).
18. Tamarkin L, Cohen M, Rosell D, Reichert C, Lippman M, Chabner B. Melatonin inhibition and pinealectomy enhancement of 7,12-dimethyl-benz(a)anthracene-induced mammary tumours in the rat. *Cancer Res* 41:4432-4436 (1981).
19. Hamilton T. Influence of environmental light and melatonin upon mammary tumor induction. *Br J Surg* 56:764-766 (1969).
20. Aubert C, Janiaud P, Lecalvez J. Effect of pinealectomy and melatonin on mammary tumor growth in Sprague-Dawley rats under conditions of lighting. *J Neural Transm* 47:121-130 (1980).
21. Shah PN, Mhatre MC, Kothari LS. Effect of melatonin on mammary carcinogenesis in intact and pinealectomized rats in varying photoperiods. *Cancer Res* 44:3403-3407 (1984).
22. Reiter RJ, Richardson BA. Magnetic field effects on pineal indoleamine metabolism and possible biological consequences. *FASEB J* 6:2283-2287 (1992).
23. Lerchl A, Nonaka KO, Stokkan K-A, Reiter RJ. Marked rapid alterations in nocturnal pineal serotonin metabolism in mice and rats exposed to weak intermittent magnetic fields. *Biochem Biophys Res Commun* 169:102-108 (1990).
24. Lerchl A, Honaka KO, Reiter RJ. Pineal gland magnetosensitivity to static magnetic fields is a consequence of induced electric currents (eddy currents). *J Pineal Res* 10:109-116 (1991).
25. Murphy JC, Kaden DA, Warren J, Sivak A. Power frequency electric and magnetic fields: a review of genetic toxicology. *Mutat Res* 296:221-240 (1993).
26. Juutilainen J, Liimatainen A. Mutation frequency in *Salmonella* exposed to weak 100-Hz magnetic fields. *Hereditas* 104:145-147 (1986).
27. Moore RL. Biological effects of magnetic fields: studies with microorganisms. *Can J Microbiol* 25:1145-1151 (1979).
28. Reese JA, Jostes RF, Frazier ME. Exposure of mammalian cells to 60 Hz magnetic or electric fields: analysis for DNA single-strand breaks. *Bioelectromagnetics* 9:237-247 (1981).
29. Whitson GL, Carrier WL, Francis AA, Shih CC, Georghiu S, Regan JD. Effects of extremely low frequency electric fields on cell growth and DNA repair in human skin fibroblasts. *Cell Tissue Kinet* 19:39-47 (1986).
30. Takahashi KI, Kaneko I, Date M, Fukada E. Effect of pulsing electromagnetic fields on DNA-synthesis in mammalian cells in culture. *Experientia* 42:185-186 (1986).
31. Liboff AR, Williams T, Strong DM, Wistar R. Time-varying magnetic fields: effect on DNA synthesis. *Science* 223:818-820 (1984).
32. Sagan L. Epidemiological and laboratory studies of power frequency electric and magnetic fields. *JAMA* 268:625-629 (1992).
33. Easterly CE. Cancer link to magnetic field exposure: a hypothesis. *Am J Epidemiol* 114:169-174 (1981).
34. Blackman CF. ELF effects on calcium homeostasis. In: *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer* (Wilson BW, Stevens RG, Anderson LE, eds). Columbus, OH: Battelle Press, 1990; 187-208.
35. Blackman CF. Calcium release from neural tissue: experimental results and possible mechanisms. In: *Interaction Mechanisms of Low-Level Electromagnetic Fields in Living Systems* (Norden B, Ramel C, eds). Oxford: Oxford Science Publ, 1992; 107-129.
36. Adey WR. Joint actions of environmental nonionizing electromagnetic fields and chemical pollution in cancer promotion. *Environ Health Perspect* 86:297-305 (1990).
37. Adey WR. ELF magnetic fields and promotion of cancer. In: *Interaction Mechanisms of Low-Level Electromagnetic Fields in Living Systems* (Norden B, Ramel C, eds). Oxford: Oxford Science Publ, 1992; 23-46.
38. Goldberg RB, Creasy WA. A review of cancer induction by extremely low frequency electromagnetic fields. Is there a plausible mechanism? *Med Hypoth* 35:265-274 (1991).
39. Goodman R, Shirley-Henderson A. Transcription and translation in cells exposed to extremely low frequency electromagnetic fields. *Bioelectrochem Bioenerg* 25:335-355 (1991).
40. Walleczek J. Electromagnetic field effects on cells of the immune system: the role of calcium signaling. *FASEB J* 6:3177-3185 (1992).
41. Szmigielski S. Electromagnetic fields and neoplasms with reference to extremely low frequencies. *Bioelectrochem Bioenerg* 30:253-264 (1993).
42. Tenforde TS. Interaction of ELF magnetic fields with living matter. In: *Handbook of Biological Effects of Electromagnetic Fields* (Polk C, Postow E, eds). Boca Raton, FL: CRC Press, 1988; 197-225.
43. Liburdy RP. ELF fields and the immune system: signal transduction, calcium metabolism, and mitogenesis in lymphocytes with relevance to carcinogenesis. In: *Interaction Mechanisms of Low-Level Electromagnetic Fields in Living Systems* (Norden B, Ramel C, eds). Oxford: Oxford Science Publ, 1992; 217-239.
44. Liburdy RP. Biological interactions of cellular systems with time-varying magnetic fields. *Ann NY Acad Sci* 649:74-95 (1992).
45. Goodman R, Wei LX, Bumann J, Shirley-Henderson A. Exposure to electric and magnetic (EM) fields increases transcripts in HL-60 cells—does adaptation to EM fields occur? *Bioelectrochem Bioenerg* 29:185-192 (1992).
46. Wei L-X, Goodman R, Henderson A. Changes in levels of *c-myc* and histone H2B following exposure of cells to low-frequency sinusoidal electromagnetic fields: evidence for a window effect. *Bioelectromagnetics* 11:269-272 (1990).
47. Goodman R, Wei L-X, Xu L-C, Henderson A. Exposure of human cells to low-frequency electromagnetic fields results in quantitative changes in transcripts. *BBA* 1009:216-220 (1989).
48. Liburdy RP, Callahan DE, Sloma TR, Yaswen P. Intracellular calcium, calcium transport, and *c-myc* RNA induction in lymphocytes exposed to 60 Hz magnetic fields: the cell membrane and the signal transduction pathway. In: *Electricity and Magnetism in Biology and Medicine* (Blank M, ed). San Francisco: San Francisco Press, 1993; 311-318.
49. Goodman R, Henderson A. Some biological effects of electromagnetic fields. *Bioelectrochem Bioenerg* 15:39-55 (1986).
50. Goodman R, Bassett CAL, Henderson AS. Pulsing electromagnetic fields induce cellular transcription. *Science* 220:1283-1285 (1983).
51. Weisbrot D, Khorkova O, Henderson A, Goodman R. Transcript levels for some genes, including a heat shock gene, are increased in *Sacharomyces cerevisiae* cells exposed to ELF-EMF fields. Abstract C-2-2. 15th Annual Meeting,

- Bioelectromagnetic Society, Los Angeles, 13-17 June, 1993.
52. Phillips JL, Haggren W, Thomas WJ, Ishida-Jones T, Adey WR. Magnetic field-induced changes in specific gene transcription. *Biochim Biophys Acta* 1132:140-144 (1992).
 53. Hiraoka M, Miyakoshi J, Li YP, Shung B, Takebe H, Abe M. Induction of *c-fos* gene expression by exposure to a static magnetic field in HeLaS3 cells. *Cancer Res* 52:6522-6524 (1992).
 54. Liboff AR, McLeod BR, Smith SD. Ion cyclotron resonance effects of ELF fields in biological systems. In: *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer* (Wilson BW, Stevens RG, Anderson LE, eds). Columbus, OH: Battelle Press, 1990;251-289.
 55. Adey WR. Electromagnetic fields, cell membrane amplification, and cancer promotion. In: *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer* (Wilson BW, Stevens RG, Anderson LE, eds). Columbus, OH: Battelle Press, 1990;211-249.
 56. Blackman CF, Benane SG, Rabinowitz JR, House DE, Joines WT. A role for the magnetic field in the radiation-induced efflux of calcium ions from brain tissue *in vitro*. *Bioelectromagnetics* 6:327-337 (1985).
 57. Walleczek J. Electromagnetic field effects on cells of the immune system: the role of calcium signaling. *FASEB J* 6:3177-3185 (1992).
 58. Walleczek J, Budinger TF. Pulsed magnetic field effects on calcium signaling in lymphocytes: dependence on cell status and field intensity. *FEBS Lett* 314:351-355 (1992).
 59. Carson JJJ, Prato FS, Drost DJ, Diesbourg LD, Dixon SJ. Time-varying magnetic fields increase cytosolic free Ca^{2+} in HL-60 cells. *Am J Physiol* 259:C687-C692 (1990).
 60. Liboff AR. The "cyclotron resonance" hypothesis: experimental evidence and theoretical constraints. In: *Interaction Mechanisms of Low-Level Electromagnetic Fields in Living Systems* (Norden B, Ramel C, eds). Oxford: Oxford Science Publ, 1992;130-147.
 61. Byus CV, Pieper SE, Adey WR. The effects of low-energy 60 Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase. *Carcinogenesis* 8:1385-1389 (1987).
 62. Litovitz TA, Krause D, Mullins JM. Effect of coherence time of the applied magnetic field on ornithine decarboxylase activity. *Biochem Biophys Res Commun* 178:862-865 (1991).
 63. Cain CD, Thomas DL, Adey WR. 60 Hz magnetic field acts as co-promoter in focus formation of C3H/10T1/2 cells. *Carcinogenesis* 14:955-960 (1993).
 64. Luben RA, Cain CD, Chen MC-Y, Rosen DM, Adey WR. Effects of electromagnetic stimuli on bone and bone cells *in vitro*: inhibition of responses to parathyroid hormone by low-energy low-frequency fields. *Proc Natl Acad Sci USA* 79:4180-4184 (1982).

Increased exposure to pollutant aerosols under high voltage power lines

A. P. FEWS, D. L. HENSHAW*, P. A. KEITCH, J. J. CLOSE and R. J. WILDING

(Received 23 April 1999; accepted 8 August 1999)

Abstract.

Purpose: To assess increased exposure to airborne pollutants near power lines by investigating theoretically and experimentally the behaviour of ^{222}Rn decay product marker aerosols in the 50 Hz electric field under power lines.

Materials and methods: The behaviour of aerosols in outdoor air including those carrying ^{222}Rn decay products was modelled theoretically in the presence of an AC field. TASTRAK α -particle spectroscopy was used to characterize ^{218}Po and ^{214}Po aerosols outdoors. Sampling points were chosen along a line at right angles up to 200 m from a number of high voltage power (transmission) lines. Each sampling point comprised an arrangement of mutually orthogonal TASTRAK detectors. Exposures were carried out at different power line locations in various weather conditions.

Results: The model predicts a two- to three-fold increase in deposition of aerosols on spherical surfaces mimicking the human head under high voltage power lines. Experimental measurements using detectors mounted on grounded metal spheres showed an enhanced deposition of both ^{218}Po and ^{214}Po aerosols. Enhanced ^{218}Po deposition on 400 kV lines ranged from 1.96 ± 0.15 to 2.86 ± 0.32 . Enhanced ^{214}Po deposition on 275 kV and 132 kV lines were 1.43 ± 0.07 and 1.11 ± 0.21 , respectively, where the latter value was not significant.

Conclusions: The observations demonstrate a mode of increased exposure to pollutant aerosols under high voltage power lines by increased deposition on the body. The total (indoor + outdoor) ^{218}Po and ^{214}Po dose to the basal layer of facial skin is estimated to be increased by between 1.2 and 2.0 for 10% of time spent outdoors under high voltage power lines.

1. Introduction

Epidemiological studies have demonstrated an association between exposure to power frequency electromagnetic fields (EMF) and increased incidence of childhood leukaemia. The associations are strongest for exposures under high voltage power lines (Ahlbom *et al.* 1993, Feychting and Ahlbom 1993, Olsen *et al.* 1993, Verkasalo *et al.* 1993, NRPB 1994, NIEHS 1998). No causal mechanism to explain these associations has yet been convincingly identified. However, on the strength of the epidemiological data, the US National Institute of Environmental Sciences Working Group has recently stated that

electric and magnetic fields like those surrounding electric power lines should be regarded as a possible human carcinogen (NIEHS 1998), and the full report to the US Congress recommends that the power industry continue its current US practice of siting power lines to reduce exposures (NIEHS 1999).

The search for a causal mechanism by which exposure to power frequency EMF may affect the process of carcinogenesis has tended to concentrate on direct effects from the magnetic field component. The authors are, however, studying a number of ways in which the electric, E-field component interacts with airborne pollutant aerosols, each suggesting a mechanism of increased exposure to these pollutants under high voltage power lines. The relevance of this approach is that both childhood and adult leukaemia are known to be associated with traffic density and its associated exhaust pollution (Savitz and Feingold 1989, Lindquist *et al.* 1991, Robinson 1991, Nordlinder and Järholm 1997).

Henshaw *et al.* (1996) described two possible mechanisms of increased exposure to pollutants near power frequency E-field sources. In indoor experiments using ^{222}Rn decay product marker aerosols, an excess deposition of these aerosols on surfaces carrying a power frequency electric potential and an increased concentration in air near the electric field source were observed.

Excess deposition of ^{222}Rn decay product marker aerosols on surfaces mimicking the human head has now been observed outdoors under 400, 275 and 132 kV high voltage overhead transmission lines (here referred to as power lines). Results of these deposition measurements are reported in this paper.

2. Materials and methods

2.1. Theoretical modelling

2.1.1. Theoretical analysis of atmospheric ^{222}Rn decay product aerosols in AC fields. The processes that govern the production and loss of aerosols in outdoor air have been summarized by Hinds (1982). Positive or negative ions present in the air act as nucleation sites,

*Author for correspondence.

H. H. Wills Physics Laboratory, University of Bristol, Tyndall Avenue, Bristol BS8 1TL, UK.

which attract polar molecules forming a cluster of molecules 1–4 nm in diameter. Such a molecular cluster constitutes a so-called ultrafine aerosol. The high diffusivity of these aerosols $(4-9) \times 10^{-6} \text{ m}^2 \text{ s}^{-1}$ results in frequent collisions, leading to rapid growth by coagulation up to $\sim 0.1 \mu\text{m}$ in size. The diffusivity of these larger aerosols is around four orders of magnitude lower: $\sim 2 \times 10^{-10} \text{ m}^2 \text{ s}^{-1}$.

The experimental measurements described below employ ^{222}Rn decay product aerosols as markers of general aerosol behaviour. The principle of this marker is the different behaviour in air of the ultrafine and aerosol-attached ^{222}Rn decay products (Porstendörfer 1984, 1994, Raes 1985). The attachment rate in outdoor air of ultrafine particles to the ambient aerosol is typically in the range $30-100 \text{ h}^{-1}$.

The growth of ^{222}Rn decay product aerosols is shown schematically in figure 1. A key feature is that 90% of recoil ^{218}Po from ^{222}Rn is positively charged allowing the rapid nucleation of an ultrafine aerosol as described above (see Hopke 1989 and references therein). A similar ^{214}Pb recoil occurs following the decay of ^{218}Po , resulting in a 0.8 probability of detachment from an attached aerosol (Mercer 1976). This process, however, is not repeated for the subsequent β -decays due to the low recoil energy. Two other features should be noted. As explained below, the present authors are interested in detecting the deposition of these aerosols by recording α -emission from ^{218}Po and ^{214}Po using α -sensitive TASTRAK plastic track detectors (based on poly allyl diglycol carbonate plastic; supplied by Track Analysis Systems Ltd, H. H. Willis Physics Laboratory, Briston, UK). The mean lifetime of ^{218}Po is 4.4 min, so that in the absence of other loss mechanisms, the above rate of aerosol attachment suggests that between 14% and 48% of ^{218}Po in air is in ultrafine form. The majority (95–99%) of ^{218}Po deposition recorded on TASTRAK detectors will be from ultrafine aerosols due to their high diffusivity. Conversely, the combined lifetimes of the aerosols feeding the subsequent ^{214}Po decay is sufficiently large that essentially all ^{214}Po decays in air are in attached form. However, those ^{214}Po deposition decays recorded on TASTRAK result from earlier deposition, which may be in either ultrafine or attached aerosol form.

The present work was aimed at measuring excess deposition of aerosols under power lines, and in modelling the expected increased deposition on spherical surfaces, similar to that which occurs around the human head. In AC fields the excess deposition occurs by the oscillation, coupled with turbulent diffusion, of charged aerosol particles along the E-field line vectors. The drift velocity v (m s^{-1}) of a charged aerosol in an E-field of strength E (V m^{-1}) is given

by $v = \mu E$, where μ is the particle mobility, typically $1.5 \times 10^{-4} \text{ m}^2 \text{ s}^{-1} \text{ V}^{-1}$ (Tyndall 1938).

Of crucial importance to the transport of ^{218}Po in E-fields is the recoil charge neutralization time (nominally $\sim 1 \text{ s}$ in figure 1). This has been investigated by several authors in laboratory measurements (Hopke 1989, Shi and Hopke 1991, Howard and Strange 1992). The published data, however, do not readily extrapolate to conditions outdoors so that there is uncertainty in the actual neutralization time.

Under high voltage power lines the maximum recommended E-field is 10 kV m^{-1} (ICNIRP 1998). In practice such E-fields are usually in the range $1-6 \text{ kV m}^{-1}$. Because the human body is a good conductor, the field will be enhanced by a factor of approximately 18 around the head.

2.1.2. Description of an aerosol transport model in AC fields.

Aerosol transport in the atmosphere and surface deposition is modelled using a one dimensional algorithm. Figure 2 summarizes the transport pathways. ^{214}Po is not shown because of its short half-life. It is assumed to decay instantaneously following the decay of ^{214}Bi . The various constants are defined below. The model divides the atmosphere into n cells vertically. The first cell accommodates the source term for ^{222}Rn , which emanates from the ground, and the deposition onto the ground of the decay product aerosols. The remaining cells are identical with the n th cell coupled to neighbouring cells by atmospheric diffusion and the following quantities: (1) oscillation in the AC field; (2) the drift in the Earth's natural DC field; (3) gravitational settling; and (4) the deposition under the action of the mirror charge close to a surface. The aerosol-attached particles are assumed to have a Boltzmann charge distribution. Typically, 100 cells are used, the sizes of which increase exponentially from an initial size of 10^{-8} m in the first cell. The model treats radioactive decay and includes the aerosol attachment rates.

The pathways within a given cell can be described by a series of steady state equations, which can be solved for the activity concentration of the surface deposited ^{218}Po and ^{214}Po aerosols. These measurable quantities allow the model to be tested experimentally. The following are employed as standard values:

$$\begin{aligned} \lambda_{\text{Rn}} &= ^{222}\text{Rn decay constant} = 2.111 \times 10^{-6} \text{ s}^{-1} \\ \lambda_{\text{Po}} &= ^{218}\text{Po decay constant} = 3.787 \times 10^{-3} \text{ s}^{-1} \\ \lambda_{\text{Pb}} &= ^{214}\text{Pb decay constant} = 4.31 \times 10^{-4} \text{ s}^{-1} \\ \lambda_{\text{Bi}} &= ^{214}\text{Bi decay constant} = 5.86 \times 10^{-4} \text{ s}^{-1} \\ \lambda_a &= \text{attachment rate of ultrafine aerosols to} \\ &\quad \text{larger aerosols} = 0.01 \text{ s}^{-1} \text{ for a 200 nm} \\ &\quad \text{aerosol at a nominal aerosol density of} \\ &\quad 7000 \text{ cm}^{-3} \text{ (Porstendörfer 1994)} \end{aligned}$$

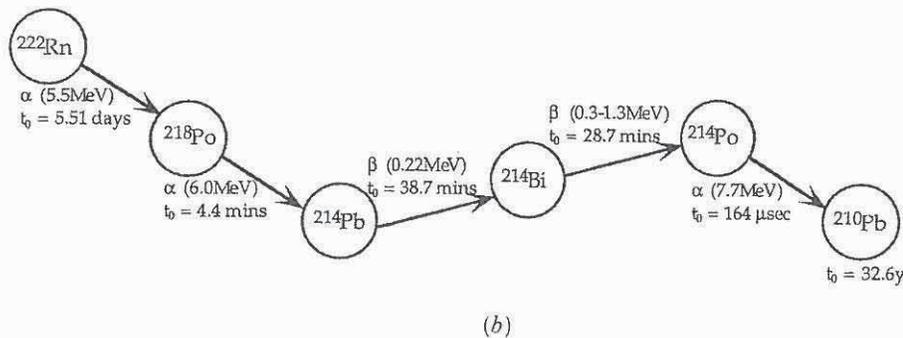
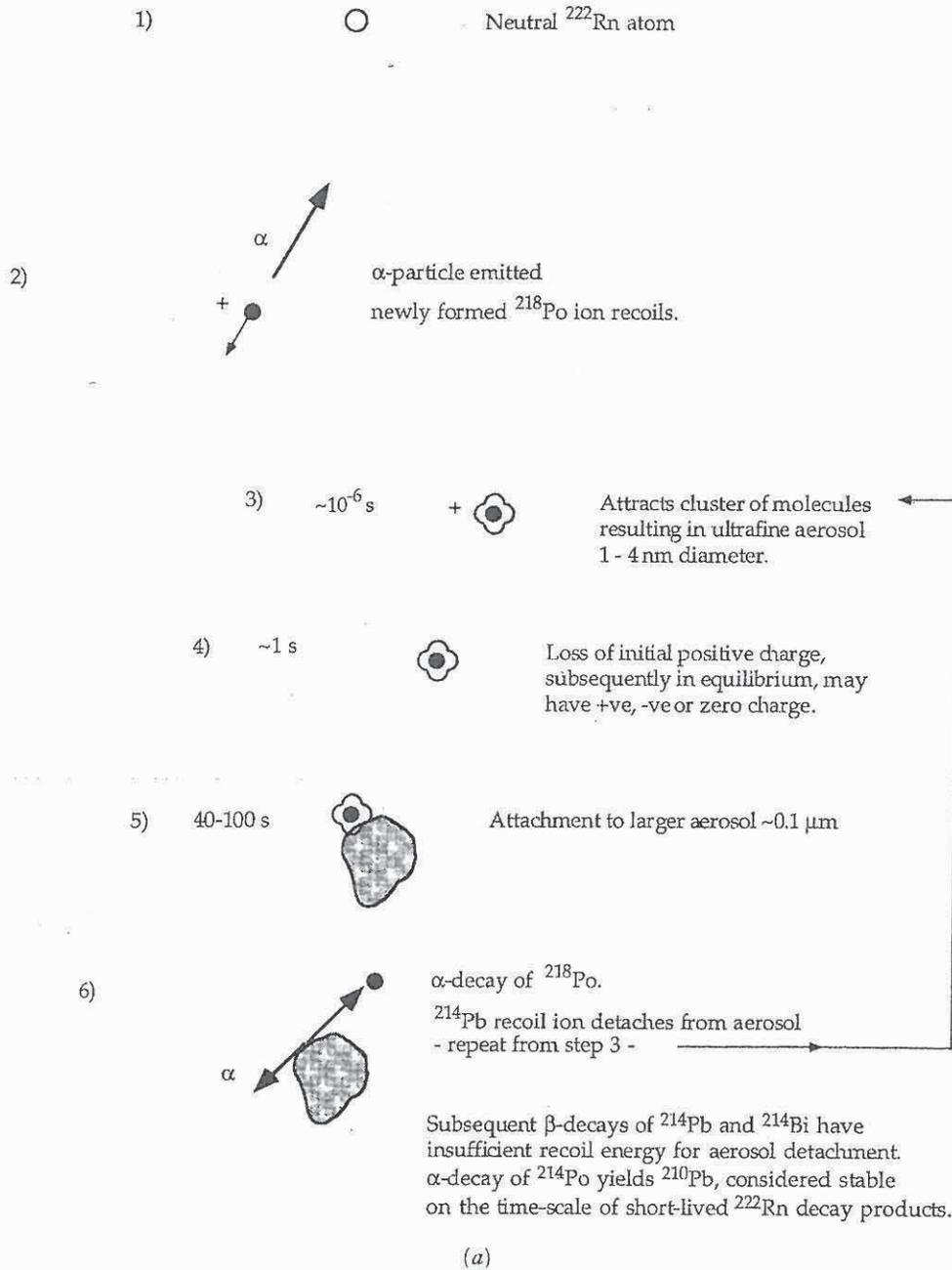


Figure 1. (a) Growth of ^{222}Rn decay product aerosols; (b) ^{222}Rn short-lived radionuclide decay chain.

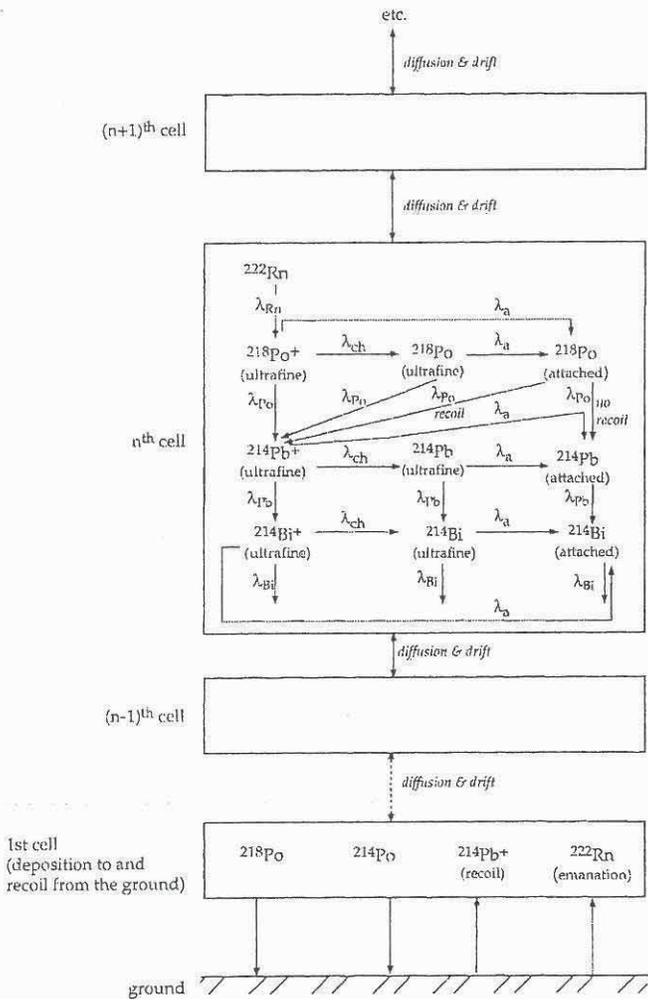


Figure 2. Schematic outline of the pathways used to model the behaviour of ^{222}Rn decay product aerosols in the atmosphere. The particle drift is made up of contributions from gravity, the Earth's natural DC field and the AC field.

λ_{ch} = rate constant for neutralization of the charged ultrafine ions = 2 s^{-1}
 X_{218} = re-suspension source term (in $\text{Bq m}^{-3} \text{ s}^{-1}$) of ^{214}Pb from decay and recoil of deposited ^{218}Po , into $50 \mu\text{m}$ air layer (Jacobi 1972). These ^{214}Pb recoil particles are distributed uniformly with distance over the cells that are within the recoil range.
 r_a = recoil fraction of ^{214}Po in air = 0.8 (Mercer 1976)
 r_p = recoil fraction of ^{214}Po following decay of plated-out ^{218}Po = 0.5
 Earth's DC field = 100 V m^{-1}
 Gravity is assumed active unless otherwise stated.
 The general transport equation for species j in

spherical geometry with distance r and time t is given by:

$$\frac{\partial C_j(r, t)}{\partial t} = \frac{1}{r^2} \frac{\partial}{\partial r} \left[\left((D_{jB} + D_T(r)) \frac{\partial C_j(r, t)}{\partial r} - V_j(r, t) \cdot C_j(r, t) \right) r^2 \right] - \lambda_j C_j(r, t) + S_j(r, t) \quad (1)$$

where C_j is the concentration of species j , and S_j is a species-dependent additional term representing the pathways shown in figure 2 as follows:

- $S_1 = 0$ (^{222}Rn atoms)
- $S_2(r, t) = \lambda_1 C_1(r, t) - (\lambda_{ch} + \lambda_a) C_2(r, t)$ (charged ultrafine ^{218}Po ions)
- $S_3(r, t) = \lambda_{ch} C_2(r, t) - \lambda_a C_3(r, t)$ (neutral ultrafine ^{218}Po atoms)
- $S_4(r, t) = \lambda_a [C_2(r, t) + C_3(r, t)]$ (aerosol-attached ^{218}Po atoms)
- $S_5(r, t) = \lambda_2 C_2(r, t) + \lambda_3 C_3(r, t) - (\lambda_{ch} + \lambda_a) C_5(r, t) + \lambda_4 \cdot r_a \cdot C_4(r, t) + r_p \cdot X_{218}$ (charged ultrafine ^{214}Pb ions)
- $S_6(r, t) = \lambda_{ch} C_5(r, t) - \lambda_a C_6(r, t)$ (neutral ultrafine ^{214}Pb atoms)
- $S_7(r, t) = \lambda_a [C_5(r, t) + C_6(r, t)] + \lambda_4 (1 - r_a) C_4(r, t)$ (aerosol-attached ^{214}Pb atoms)
- $S_8(r, t) = \lambda_a [C_5(r, t) + C_6(r, t)] + \lambda_4 (1 - r_a) C_4(r, t)$ (charged ultrafine ^{214}Bi ions)
- $S_9(r, t) = \lambda_a [C_5(r, t) + C_6(r, t)] + \lambda_4 (1 - r_a) C_4(r, t)$ (neutral ultrafine ^{214}Bi ions)
- $S_{10}(r, t) = \lambda_a [C_5(r, t) + C_6(r, t)] + \lambda_4 (1 - r_a) C_4(r, t)$ (aerosol-attached ^{214}Bi atoms)

- V_j = drift velocity of species j due to E-fields and gravity
- n_j = number of charges on species j particles
- K_1 = atmospheric diffusion constant
- K_2 = diffusion constant near deposition surface

The Brownian diffusion and the particle drift velocity are as follows:

$$D_{jB} = \mu_j \cdot \frac{kT}{e} \quad (2)$$

$$V_j = -n_j \cdot \mu_j \cdot \left[\frac{mg}{e} + E_{DC} + \sqrt{2} \cdot E_{rms}(r) \cdot \sin(2\pi 50t) \right] \quad (3)$$

where e is the electronic charge, k the Boltzmann's constant and T the absolute temperature. In a 50 Hz E-field, the aerosol will oscillate with a peak-to-peak amplitude of $(\sqrt{2} \mu E_{rms}) / (50\pi)$. $E(r)$ has the variation with distance $E(r) = E_0 / r^2$, and gravity is assumed to be invariant with distance. At the boundary where

49/90

$r = 0$, C_{ij} will fall to zero. The aerosol-attached ^{218}Po and ^{214}Po are assumed to have a Boltzmann distribution of charge, which has been found to be a good empirical description of the charge state for particles of 200 nm diameter being modelled here (Hinds 1982). It is possible that the attached ^{214}Pb and ^{214}Bi might have an excess positive charge from the earlier radioactive decays (Clement and Harrison 1992). Any initial excess charge will tend to increase the enhanced (E-field) deposition. However, the model does not allow for this because we consider that neutralization by ambient air ions will generally be fast enough to ensure that the charge distributions are close to the Boltzmann distribution. Because each charge state will drift differently in E-fields, equations for C_4 , C_7 and C_{10} are themselves subdivided into five components, representing the five most probable charge states. Each component is treated separately for diffusion and drift and then recombined to give the total concentration of attached ^{218}Po and ^{214}Po . This system of linked equations is written in a finite difference form and solved sequentially by the Crank–Nicholson algorithm.

The mobility of ultrafine aerosols is taken as $1.5 \times 10^{-4} \text{ m}^2 \text{ V}^{-1} \text{ s}^{-1}$, the average of reported values. The attached aerosols have an assumed diameter of 200 nm, leading to a mobility of $8.3 \times 10^{-9} \text{ m}^2 \text{ V}^{-1} \text{ s}^{-1}$, using the data of Cheng *et al.* (1992). The calculation can use either an exact treatment for particle motion where all the charged particles oscillate with the AC phase, or the dividing surface approximation similar to that used by Mayya and Sapra (1997). The latter is used (e.g. for indoor simulation) where it is a good approximation. For outdoor conditions of high deposition velocity (and short turbulence time scales compared with the AC period), this approximation is poor and the exact method is used instead.

2.1.3. Model parameters. The calculation is run in two stages. The first sets up the vertical concentration profile of the particles, by including transport to the open atmosphere. The second estimates the deposition on the test surface (here a 20 cm diameter sphere represents the human head) at a specified height by using the calculated profile from the first stage and letting turbulent diffusion transport these particles to the surface. This allows a perturbed E-field deposition to be calculated, which will locally affect deposition but will not change the overall atmospheric profile.

The first stage simply seeds the main calculation with appropriate particle concentrations giving reasonable values of F and f (radon decay product equilibrium fraction and ultrafine fraction), by using

a simple form for the turbulent diffusion away from the ground, $D\tau = K_1 r$. This linear form is similar to that used by Porstendörfer (1994) and Jacobi and André (1963), and can represent a form of flow-induced turbulence studied by Sehmel (1973). Values of the constant K_1 between 10^{-3} and 1 were tested to represent different weather conditions, but were found to have a negligible effect on the deposition, which was determined as discussed below by the form of turbulent diffusion close to the test surface. In other words, uncertainty in atmospheric transport has little effect on deposition close to the ground, and only the diffusion coefficient close to the ground is relevant. Note that this is almost irrelevant for ^{218}Po deposition because this is determined almost totally from the ultrafine particle deposition.

Deposition close to the test surface was modelled using different forms of $D\tau$, in order to overcome the uncertainty of which form to use. Thus, the form $D\tau = K_2 r^n$ was used, with $n = 1.5, 2.0$ and 3.0 . The quadratic form is the same as that used by Crump and Seinfeld (1981) and Mayya and Sapra (1997). This form enables the simulation to reproduce the experimental observation by Porstendörfer (1994) that the ultrafine ^{222}Rn decay product aerosols have a deposition velocity ~ 100 times that of an attached aerosol. The values of the constant K_2 completely determine the deposition velocity, and were set to give a nominal ultrafine deposition velocity of 0.01 m s^{-1} outdoors.

For the ^{218}Po charge neutralization time, the published data indicate that while a value around 1 s is reasonable, at low ^{222}Rn concentrations in outdoor conditions it could rise to tens of seconds. Alternatively, in wet conditions the value could fall below 1 s. In the diffusion model, the time has been varied between 0.02 and 20 s ($0.05 \leq \lambda_{ch} \leq 50$).

2.2. Experimental methods

2.2.1. Principles of α -particle track detection in TASTRAK plastic. The development of TASTRAK plastic for α -particle detection and spectroscopy has been described in a number of previous publications (Fews 1992a,b, Henshaw *et al.* 1994, 1995, 1996).

In the present application, TASTRAK, typically $7 \times 5 \text{ cm}$, is held in air, sampling two categories of aerosol. As described above, ^{222}Rn decay product aerosols on production undergo diffusive dispersion in room air. If during their lifetime they come into contact with a surface, they may adhere to it—a process commonly known as ‘plateout’ but referred to in this paper as (*surface*) *deposition*. A proportion of ^{222}Rn decay product aerosols will therefore deposit

on the TASTRAK surface. On radioactive decay, these aerosols emit α -particles of the characteristic energy of the decay products ^{218}Po and ^{214}Po . Those α -emissions in the direction of the plastic will be recorded at their characteristic full energy of respectively 6.0 and 7.7 MeV and ranges in TASTRAK of 40.2 and 60.5 μm .

The ^{218}Po and ^{214}Po α -particles have respective ranges in air of 5.1 and 7.6 cm. Therefore, a proportion of the ^{222}Rn decay product aerosols in the air in front of the detector as well as ^{222}Rn itself may also be recorded if their α -emissions occur both within range and in the direction of the detector surface. (Note that TASTRAK is not sensitive to the β -particles emitted from the decay products ^{214}Pb and ^{214}Bi , so that the detection of the extremely short half-life ^{214}Po is a signature of these preceding β -emitting decay products.)

In the present work, cone-like etch tracks were revealed by etching in 6.25 M NaOH at 75°C for 4.5 h. The detectors were analysed using an image analysis system in which up to 14 parameters of the shape and size of each recorded α -particle track are measured (Fewes 1992a, b). These measurements, combined with track geometry data from calibration exposures, enable the α -particle energies to be directly determined.

Figure 3 illustrates how recorded track size varies with α -particle energy and angle of incidence. The projected track length (the major axis of the etch track opening mouth on steep tracks, and the track length in projection on shallow tracks) is plotted against the minor axis diameter. The data fall within an envelope characteristic of the α -particle track response for the etch conditions employed. Bands are seen which originate from α -emissions from ^{218}Po and ^{214}Po deposited on the plastic surface. These arise from the deposited α -radionuclides observed at constant energy over a range of incident angles. Airborne emission will give a variation of both energy and angle, and discrete bands would not be observed. Therefore, a separate measurement of both the deposited and airborne components is made from the same TASTRAK detector.

2.2.2. *Detection of ^{222}Rn decay product aerosols outdoors.* Use of TASTRAK outdoors requires comparatively long exposure periods to acquire enough counting statistics, although exposures of several days have the advantage of integrating over short-term variations in aerosol concentration. In practice TASTRAK exposed outdoors suffers solar UV damage which limits its exposure period to around 6 days. Under these conditions typical count densities range from a

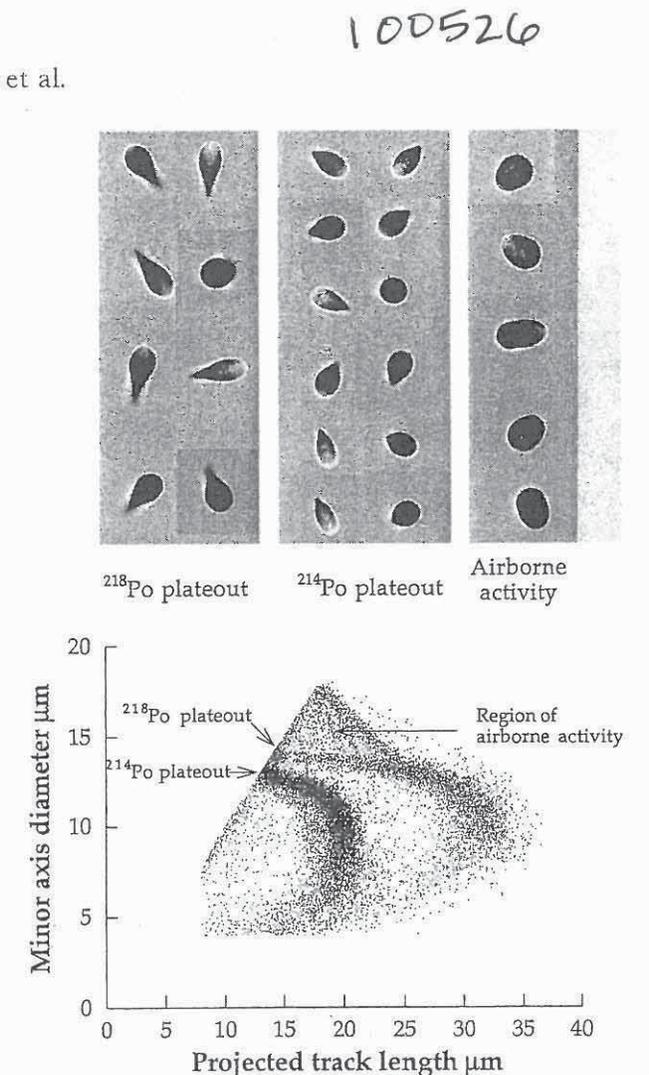


Figure 3. Photomontage of etched tracks and scatter plot of track minor axis versus projected length, showing ^{218}Po and ^{214}Po plateout and the region of airborne activity.

few hundred tracks per cm^2 from deposited activity, to < 30 per cm^2 from the air activity.

Outdoor exposures often result in solid particles landing on the TASTRAK surface. These can introduce microscopic scratches onto the detector surface which when etched grow into defects that can mimic real etch tracks. Such defects are recognized as an extra-Poissonian number of closely separated tracks, taking account of genuine close α -emissions that may be recorded from sequential decays of ^{218}Po and ^{214}Po on the detector surface. Details of this procedure will be described in another publication.

2.2.3. *Layout of detectors under power lines.* Measurements were made at several locations under high voltage transmission lines in south Gloucestershire, UK: (1) 400 kV at OSGB map reference 672830; (2) and (3) 400 kV at two separate locations at OSGB 774775;

(4) 400 kV at OSGB 644838; (5) 275 kV at OSGB 666868; and (6) 132 kV at OSGB 713854.

Two configurations of TASTRAK detectors were employed. One configuration used detectors placed at 30 sampling points 1 m above the ground at right angles to the line. Each sampling point comprised five detectors (the 'pizza box' assembly) mounted on a varnished wooden stake (figure 4a). Three detectors were mounted orthogonally, in the horizontal plane and in parallel and perpendicular planes with respect to the power line; two further detectors were mounted horizontally, looking respectively upwards and downwards. The downwards-facing detector was mounted in the bottom of an inverted plastic pizza box, to protect it from rainfall. In all, including controls, 180 detectors were employed.

This configuration enables the deposition of aerosols to be studied in various directions with respect

to the power line E-field vector, but suffers a difficulty in that the field distortion around each detecting element is uncertain due to uncertainties in the moisture content of the stake. The E-field distortions around the stake are always uncertain and are likely to be substantial when the stakes are wet. For this reason, and the fact that the detectors are not in the highest field-enhanced region but a few centimetres away from it, the 'pizza box' results are considered to be difficult to interpret.

Therefore another configuration was also employed using a grounded 20 cm diameter metal sphere which created a field perturbation similar to that expected around the human head. Apart from underneath the sphere, field lines would terminate on the sphere at right angles (figure 4b). The sphere was mounted 1 m above the ground with five TASTRAK detectors attached: one horizontally on the top surface and the remainder vertically in north, east, south and west-facing directions. Spheres were placed in the high-field region under the power line and in the low-field region about 100 m away.

Table 1 summarizes the exposures carried out. Weather conditions throughout were recorded. Aerosol concentrations were not measured contemporaneously but background monitoring using a TSI-3010 condensation particle counter found a diurnal variation in the range > 7000 to $< 70\,000$ cm^{-3} . A typical mean value was $16\,000$ cm^{-3} .

After each exposure the TASTRAK detectors were processed and measurements were carried out by automated image analysis as described in §2.2.1. These were blind in the sense that the person carrying out the image analysis and subsequent data processing had no knowledge of the position of each TASTRAK detector with respect to the power line.

2.2.4. Calibration. The raw track densities are measured in counts per cm^2 . For a given exposure time they can be expressed in Bq m^{-2} of recorded tracks. The detection efficiency for the deposited activity is calculated using the known track response of TASTRAK, by calculating a cut-off angle. Typical detection efficiencies are 0.268 for ^{218}Po deposition and 0.209 for ^{214}Po depositions.

The airborne activity concentration in Bq m^{-3} is calibrated by simulating emission as a function of height above the TASTRAK surface (the activity concentration is assumed to be uniform with height), and integrating the calculated detection efficiency. This results in an equivalent sampling thickness by the plastic and hence an absolute conversion to activity concentration.

The particle tracks are resolved by energy and therefore a proportion of the airborne activity may

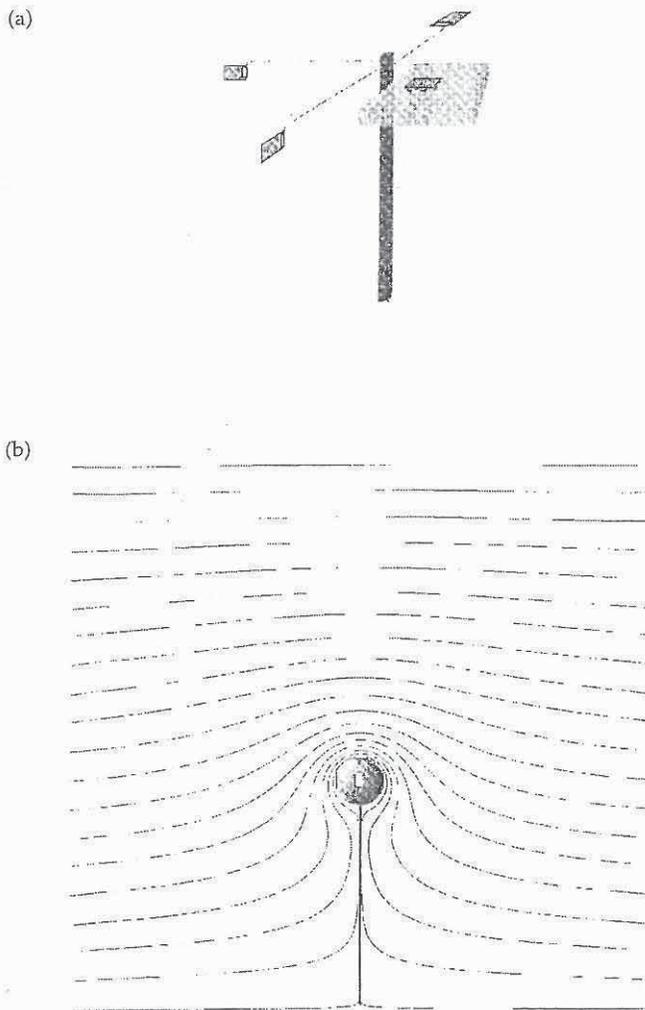


Figure 4. Configuration of TASTRAK detectors under power lines: (a) a perspective view of the pizza box assembly and (b) a sectional view of the sphere assembly showing equipotential contours.

Table 1. Summary of exposure parameters.

Experiment	Location UK grid ref. (OSGB)	Dates	Powerline voltage (kV)	E-field at 1 m height (kV m ⁻¹)	Fraction of time dry	Rain (mm per day)	Average wind speed (m s ⁻¹) ^a
1 ^b	672830	1–9 May 1997	400	4.0	0.86	3.6	3.2
2 ^c	774775	27 Aug–2 Sep 1998	400	3.8	0.97	0.4	1.0
3 ^c	774775	20–26 Jan 1999	400	6.3	0.83	5.3	3.0
4	644838	16–22 Feb 1999	400	7.0	0.86	1.5	3.2
5	666868	5–11 Mar 1999	275	3.4	0.95	0.1	2.6
6	713854	24 Feb–2 Mar 1999	132	1.9	0.25	6.0	5.4

^a 1 m s⁻¹ = 3.6 kilometres per hour = 2.2 miles per hour).

^b Experiment 1 was a 'pizza box' run and the others were sphere experiments. ^c Note that the locations of experiments 2 and 3 were not identical.

Sphere exposures were not carried out in experiment 1.

interfere with the deposition bands. For example, an airborne ²¹⁴Po decay of 7.7 MeV may be recorded with a degraded energy of 6.0 MeV, mimicking a 6 MeV ²¹⁸Po deposition. The calibration for the airborne region allows for the presence of the various ²²²Rn-derived species, including those lost into the deposition bands. Simulations suggest that ~5–10% of the airborne tracks are superimposed on each deposition band. Since the ratios of airborne to deposition track densities is ~0.1 for outdoor exposures, then it follows that up to ~0.5–1% of deposition tracks may be wrongly categorized. This will apply nearly equally to ²¹⁸Po and ²¹⁴Po deposition, and therefore the resulting error on both deposition and the ²¹⁴Po/²¹⁸Po ratio is inconsequential. Thoron progeny would reveal additional deposition bands, but were not observed in these experiments. Illustrative track counts on each detector without an E-field are 300–1500 counts for ²¹⁴Po, 100–500 for ²¹⁸Po and 10–50 for the airborne region, where each sphere or pizza box employed five detectors. In general, the errors from counting statistics are negligible compared with the intrinsic variation between adjacent detectors. This intrinsic variation is the basis for the quoted errors in the tables of results. The authors believe this intrinsic variability is generally due to patches of wet deposition, and is worst on the wettest experiments (experiment 6).

3. Results

The model used the standard set of conditions indicated by the constants given in §2.1.2, from which particular parameters were varied.

3.1. Theoretical modelling

Figure 5 shows the variation in ²¹⁸Po-enhanced deposition factor, EF and ²¹⁴Po/²¹⁸Po deposition

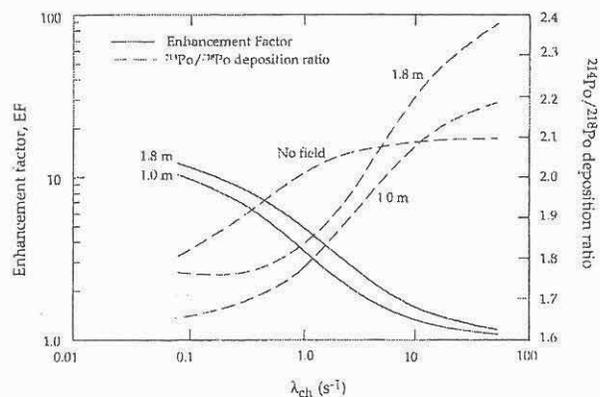


Figure 5. Predicted variation in ²¹⁸Po enhancement factor and ²¹⁴Po/²¹⁸Po deposition ratio in a 5 kV m⁻¹ perturbed AC field as a function of λ_{ch} .

ratio in a 5 kV m⁻¹ perturbed AC field as a function of λ_{ch} . The heights of 1.0 and 1.8 m correspond with a child and adult respectively. The ²¹⁸Po deposition shows an EF of ~4 at $\lambda_{ch} = 1$, increasing to 10–15 for low λ_{ch} (long charged lifetimes). EF values greater than 2 are seen for $\lambda_{ch} \leq 3$ s⁻¹, and therefore significant enhancements are expected for the range of λ_{ch} , which could occur outdoors. The variation in the ²¹⁴Po/²¹⁸Po deposition ratio spans a comparatively small range, between 1.7 and 2. As will be seen later, measured ratios can be larger than this because the ²¹⁴Po deposition is increased due to wet deposition, which is not included in this model. For most values of λ_{ch} the action of the perturbed field is to reduce the ²¹⁴Po/²¹⁸Po deposition ratio, in other words, the enhancements for ²¹⁴Po are somewhat smaller because of a higher proportion of deposition from attached particles.

Figure 6 shows the variation in ²¹⁸Po EF and the ²¹⁴Po/²¹⁸Po deposition ratio with λ_a in a perturbed 5 kV m⁻¹ AC field. The equivalent aerosol concentration is based on an aerosol attachment coefficient of

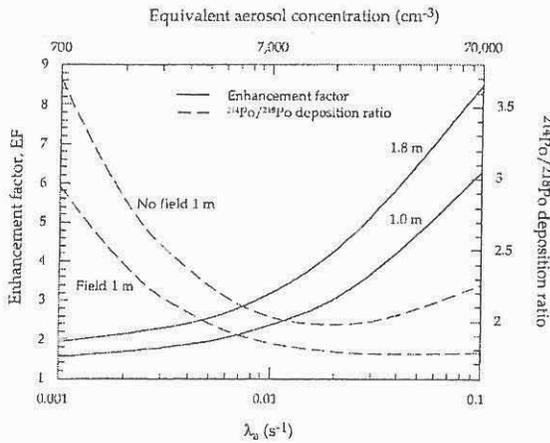


Figure 6. Predicted variation in ^{218}Po enhancement factor and $^{214}\text{Po}/^{218}\text{Po}$ deposition ratio with λ_a and equivalent aerosol concentration in a perturbed 5 kV m^{-1} AC field.

$1.4 \times 10^{-6} \text{ cm}^3 \text{ s}^{-1}$ (table 5 in Porstendörfer 1994). The actual aerosol concentrations may differ by a small factor because variations in the attachment coefficient may occur. As indicated in §2.2.3, the range of interest is where $0.01 < \lambda_a < 0.1$, corresponding to an aerosol concentration in the approximate range 7000 to 70 000 cm^{-3} . In this range EF is predicted to lie between 3 and 9 for ^{218}Po .

The form of the variation in the $^{214}\text{Po}/^{218}\text{Po}$ deposition ratio with λ_a and aerosol concentration can be understood qualitatively. In the limit of very low aerosol concentration all ^{222}Rn decay product aerosols are in ultrafine form because there are insufficient aerosols present to allow attachment to larger aerosols. Here (not shown) the deposition ratio is simply equal to the ratio of the mean lifetimes $(^{214}\text{Pb} + ^{214}\text{Bi} + ^{214}\text{Po})/^{218}\text{Po} = 15.2$. Similarly, at very high aerosol concentration (again not shown) it may be considered that all aerosols are in attached form, again giving a deposition ratio of 15.2. In the range of the aerosol concentration of practical interest, where deposition occurs for a combination of ultrafine and attached aerosols, the ratio is less than 15.2 because the ^{218}Po deposition is mainly from ultrafine particles but the ^{214}Po is from a mixture of ultrafine and attached aerosol particles. Therefore the attached deposition of ^{214}Pb and ^{214}Bi is not negligible, and it is this attached deposition that drives the $^{214}\text{Po}/^{218}\text{Po}$ ratio, which is why it can be used as an indicator of aerosol concentration. With no applied field it passes through a minimum of around 2.0 at an attachment rate of around 0.02 to 0.03 s^{-1} , corresponding to a nominal aerosol concentration between 14 000 and 20 000 cm^{-3} . The effect of the perturbed AC field is to decrease the ratio at normal aerosol concentrations because with increas-

ing attachment rate the ultrafine particles spend an increasing proportion of their time in the charged state. As a result the ^{218}Po and ^{214}Po behave more similarly and the effect of neutral ultrafine aerosols is small in this region. This results in the ratio being essentially flat throughout the range of aerosol concentration of interest.

Figure 7 shows the variation in EF and deposition velocity as a function of aerosol size at 1.8 m height with and without a 5 kV m^{-1} perturbed AC field. Increased deposition is seen throughout the aerosol size range up to $10\ \mu\text{m}$. A typical 200 nm aerosol shows an enhanced deposition factor of ~ 2 . The EF values at smaller sizes differ from ^{218}Po ultrafine aerosols in that the particles are assumed to have a Boltzmann charge state, which includes an uncharged component. In the presence of gravity the deposition velocity shows the characteristic minimum in the range 0.2 to 0.3 μm (Porstendörfer 1994). The velocity increase above 0.3 μm is due to the effect of gravitational settling.

Figure 8 shows the variation in ^{218}Po EF with the field-free deposition velocity at 1.8 m height in a perturbed 5 kV m^{-1} AC field and a nominal Earth's DC field of 100 V m^{-1} . Values of $n = 1.5, 2$ and 3 in the turbulent diffusion expression $D\tau = K_2 r^n$ have been used. This also yields (not shown) values for the ultrafine/attached deposition ratio of 32, 155 and 756, respectively. Of these, the value for $n = 2$ is within the normal observed range of 100–200 (Porstendörfer 1994). This suggests that $n = 2$, as used by Mayya and Sapra (1997), is appropriate.

Figure 9 shows the variation of EF outdoors and indoors in AC and DC (static) fields. The outdoor and indoor situations were represented by adjusting the constant K_2 to give ultrafine deposition velocities

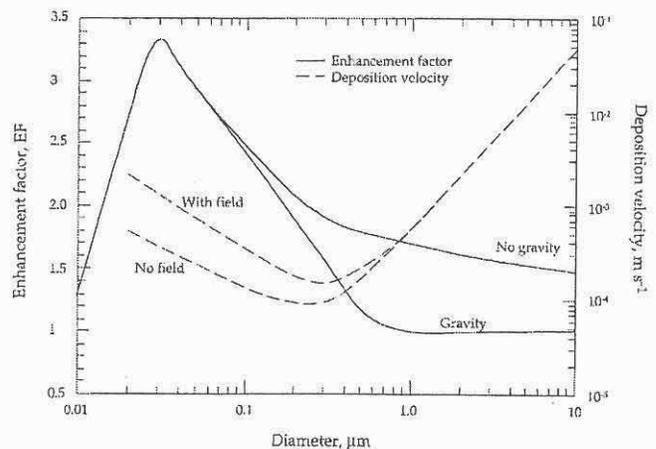


Figure 7. Variation in enhancement factor and deposition velocity as a function of aerosol size with and without a 5 kV m^{-1} perturbed AC field.

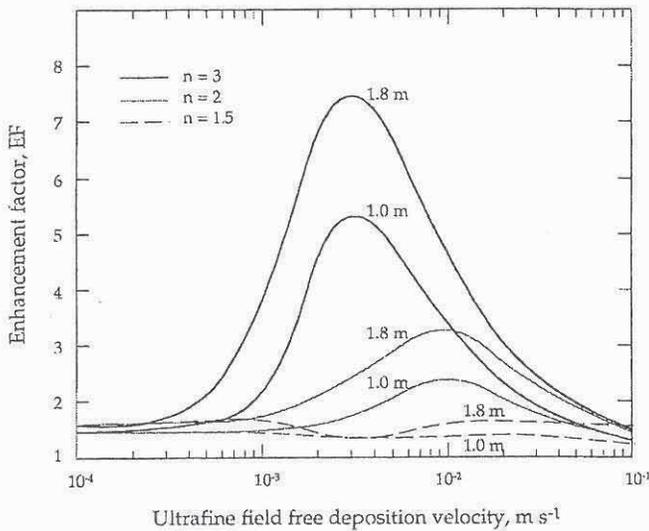


Figure 8. Variation in ^{218}Po enhancement factor with the field-free deposition velocity at 1.0 and 1.8 m heights for $n = 1.5, 2$ and 3 in the turbulent diffusion expression, in a 5 kV m^{-1} perturbed AC field.

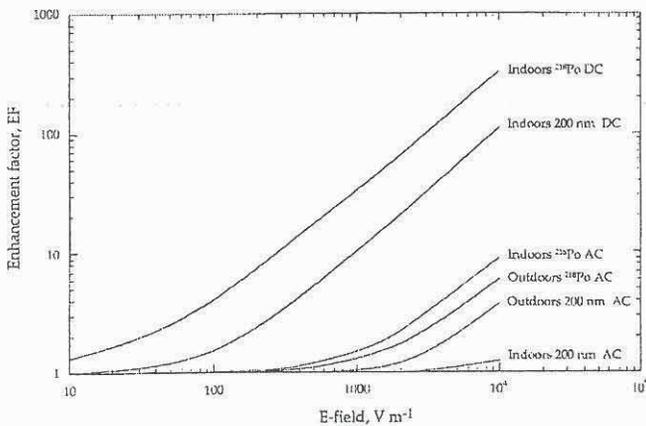


Figure 9. Predicted variation in enhancement factor for ^{218}Po deposition in AC and DC perturbed fields.

in the absence of fields of 0.01 m s^{-1} and 0.0055 m s^{-1} (2 m h^{-1}), respectively. These values are typical of those determined from measurements in the present study and those given by Porstendörfer (1994). Data are shown for the EF in the perturbed field of a 20 cm sphere at a height of 1.8 m, for ^{218}Po and 200 nm aerosol deposition. Enhanced deposition of 200 nm particles is not predicted indoors—this is expected because the deposition boundary layer thickness is much greater than the peak-to-peak oscillation amplitude. Indoors, the effects of turbulent diffusion and wind are depressed as are the effects of the Earth's DC field. Note, however, that EF would rise further for departures from the standard aerosol conditions, i.e. for $\lambda_a > 0.01 \text{ s}^{-1}$ (> 7000 aerosols cm^{-3}) and $\lambda_{ch} < 1 \text{ s}^{-1}$. Significant enhanced depos-

ition is expected indoors for DC fields as low as 10 V m^{-1} for both ^{218}Po and 200 nm particles.

In figure 8, note that since the simulation includes the Earth's DC field, the EF at a deposition velocity of $5.5 \times 10^{-4} \text{ m s}^{-1}$ (the nominal indoor value) is not comparable to the indoor EF value in figure 9 because the AC result here does not include any DC field.

The model calculations were checked by comparing the predictions with known analytical solutions for particular configurations. This test was made using a uniform initial concentration profile in an enclosed system, which has a known analytic solution for the concentration profile as a function of time. The model showed good agreement with the expected concentration profile. The deposition velocity also matched the analytic value ($2/\pi \sqrt{K_2 D_0}$) for the K_2 values used (Mayya and Sapra 1997).

The reliability of the model in predicting the measured data has to be considered because it is a one dimensional representation of an essentially three dimensional air flow and diffusion pattern around the detectors. The model allows particles to flow freely towards the spheres, so depletion in the downwind direction is not modelled. However, the authors are particularly interested in relative deposition, and not the absolute values, and this depends only very weakly on the K_1 and K_2 turbulent diffusion coefficients. The absolute deposition velocity was set at $K_2 = 62 \text{ s}^{-1}$, giving a typical deposition velocity for ultrafine aerosols of 0.01 m s^{-1} , comparable with the deposition velocities measured from the detectors, which were generally in the range $0.005\text{--}0.02 \text{ m s}^{-1}$. The atmospheric profile of ^{222}Rn and its decay products depends on the value of K_1 used, but has no effect on either the deposition velocity to the ground (because this depends on K_2), or on the EF. Therefore, uncertainties in K_1 , and the details of atmospheric transport have a negligible effect on the EF. It is therefore unimportant that transport into the atmosphere can only be an approximation, although it will have some impact on the predicted $^{214}\text{Po}/^{218}\text{Po}$ deposition ratio. Therefore, the model should match the experiment reasonably closely in terms of EF values.

Table 2 gives expected amplitude of oscillation and velocity at deposition for ultrafine aerosols for various field strengths. In the perturbed field at 1.8 m height, values between 2.7 and 27 m s^{-1} or $10\text{--}100 \text{ km h}^{-1}$ are predicted for applied fields between 1 and 10 kV m^{-1} .

3.2. Experimental measurements

Results for the pizza box detectors in experiment 1 are shown in figure 10. Figure 10a shows the

Table 2. Estimated range of amplitude of oscillation of ultrafine aerosol of mobility $1.5 \times 10^{-4} \text{ m}^2 \text{ V}^{-1} \text{ s}^{-1}$ for various 50 Hz field strengths and conditions.

Type of E-field	Applied E-field (kV m^{-1})	E-field at deposition surface (kV m^{-1})	E_{rms} peak-to-peak amplitude from deposition surface (cm)	Particle velocity at surface (m s^{-1})
(a) Unperturbed (uniform field)	1	1	0.14	0.15
	5	5	0.70	0.75
	10	10	1.40	1.5
(b) Perturbed by 20 cm Sphere, at 1 m height	1	10	1.2	1.5
	5	50	4.6	7.5
	10	100	7.4	15
(c) Perturbed by 20 cm Sphere, at 1.8 m height	1	18	2.1	2.7
	5	90	6.9	14
	10	180	10.5	27

measured deposition velocity of ^{218}Po on detectors mounted vertically facing parallel and perpendicular to the line. Note that the deposition velocity shown is for the total ^{218}Po deposition, and is not the ultrafine deposition velocity. The deposition velocity was calculated using the ratio of the deposited activity to the airborne activity, using the standard definition of deposition velocity. For the parallel facing detectors excess deposition is seen within $\pm 25 \text{ m}$ of the line where the E-field exceeds 1.5 kV m^{-1} up to a maximum of 4 kV m^{-1} . No excess deposition is seen in the perpendicular facing detectors, indeed these show an essentially uniform profile of deposition with distance from the line. Figure 10b and c, respectively, show the data plotted as the ratio of deposition in the parallel and perpendicular directions for ^{218}Po and ^{214}Po .

Results for the 20 cm diameter sphere experiments 2 to 6 are shown in figure 11 and table 3. The EF values range from 1.1 to 2.9. The statistical significance was tested using a *t*-test and all values except that at 132 kV were significant at the 95% confidence level. The latter, however, was carried out in particularly adverse weather conditions. Table 1 shows that there was rain 75% of the time for this exposure, indeed this is the only exposure that was predominantly wet. The results from the corresponding pizza box detectors for all of the sphere exposures are given in table 4, where each value is the average of all detector directions.

Table 5 shows a breakdown of the deposition values with respect to direction for experiment 4. The average wind direction was north-westerly. There was remarkable uniformity in the corresponding values on each of the four spheres for both power lines and controls, as indicated by the small spread in each value quoted. The control deposition values are higher on the upwind sides. However, under the

power line, the deposition on the upwind side is particularly enhanced and there is depletion on the downwind side. The observation suggests a coupling of the AC oscillation to turbulent diffusion onto the sphere on the upwind face. The depletion downwind is assumed to be due to the excess upwind deposition. The resulting EF values are higher upwind with an overall excess deposition of 2.86 ± 0.32 and 2.44 ± 0.23 for ^{218}Po and ^{214}Po , respectively.

4. Discussion

4.1. Modelling results

An important feature from figure 9 is the difference in factors governing deposition outdoors compared with indoors. Outdoors, the deposition in power line AC fields is little affected by the Earth's DC field of $\sim 100 \text{ V m}^{-1}$. Apart from field strength, the sensitive parameters are λ_{ch} and λ_a , the latter being sensitive to aerosol size and concentration. As seen in table 2, the velocity of ions at deposition in the perturbed field is in the range $2.7\text{--}27 \text{ m s}^{-1}$ or $10\text{--}100 \text{ km h}^{-1}$, which is faster than typical wind speeds. These velocities underline the nature of the increased exposure on the human head and face. In particular, they will overcome any thermophoretic effects, which have sometimes been invoked to argue against excess deposition of aerosols on the human body.

Indoors, the effects of DC fields are much greater than for AC. The human body can readily pick up static potentials of up to 10 kV in the office and home environment. Under these conditions EF values in excess of 300 are predicted. Such dramatic effects of DC have been proposed as a method of reducing ^{222}Rn decay product concentration indoors (Jonassen 1983). The concern here is with excess deposition on the human body and in this situation even transient

static body potentials could lead to an overall significant excess deposition. Viruses in the relatively small size range of 40–300 nm would be attracted to the body with particular efficiency. The importance of such DC effects indoors are well known and a recent study by Hatch *et al.* (1998) suggested an association between childhood leukaemia and use of TV sets and video games, which might be associated with exposure to static. Future case-control studies of childhood leukaemia in relation to time-varying EMF exposure could usefully be extended to include measurements of exposure to static fields.

The EF predicted for 200 nm aerosols in AC fields is negligible indoors because the particle oscillation amplitude is much smaller than the deposition boundary layer.

The field-free deposition velocity and the form of the turbulent diffusion coefficient were varied to test different turbulence conditions. However, to reproduce a deposition velocity of 0.01 ms^{-1} and an ultrafine/attached deposition velocity ratio of ~ 100 required the use of $D_T = 62.5r^2$. The authors therefore feel this is an appropriate form to represent the conditions experienced on the spheres in outdoor conditions.

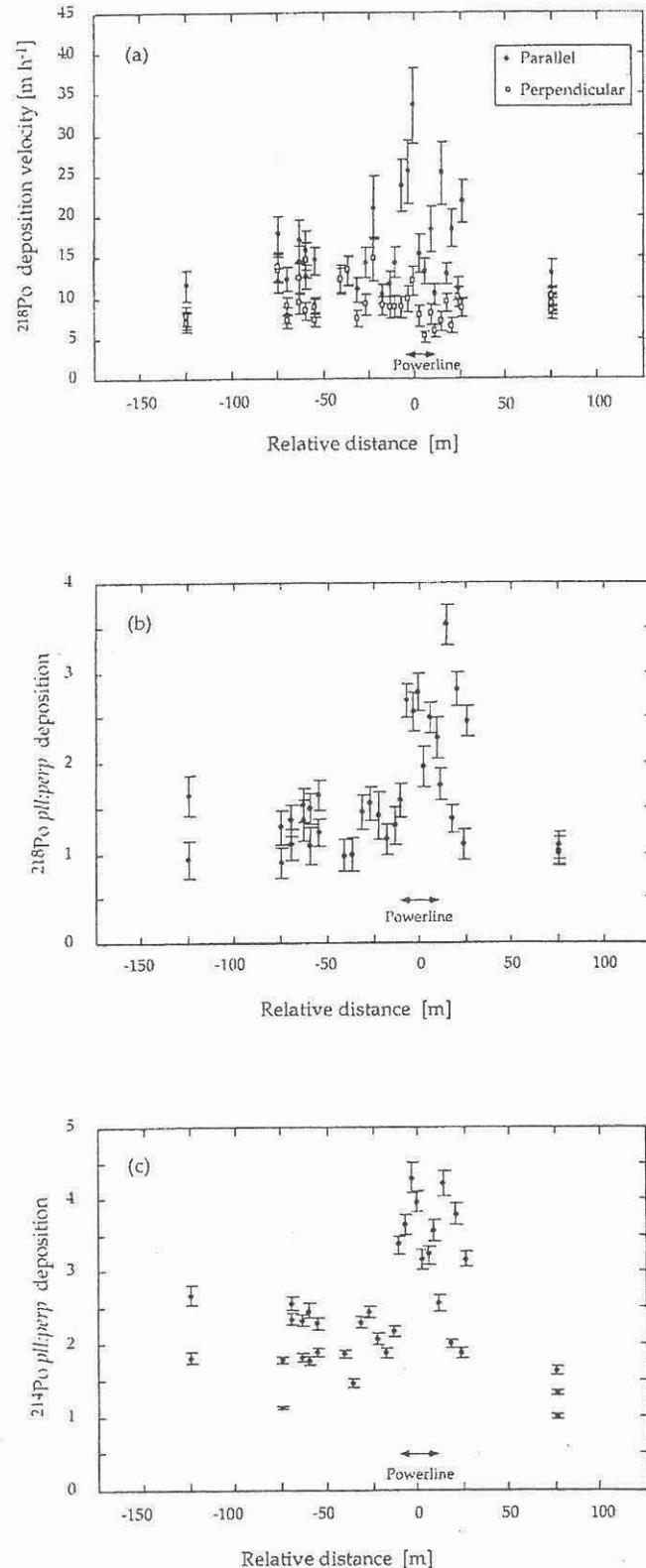
4.2. Experimental measurements

The results from the pizza box assembly for experiment 1 demonstrate the principle that excess deposition of aerosols occurs by oscillation along the E-field lines. This is underlined by the fact that no excess deposition was seen on detectors mounted vertically facing a direction perpendicular to the line (figure 10). Uniformity in deposition in this direction was found for both ^{218}Po and ^{214}Po aerosols. This further illustrates that the ^{222}Rn emission from the ground was uniform across the exposure site. This feature is important because small-scale spatial and temporal variations in ^{222}Rn emissions from the ground are known to occur (Robé *et al.* 1992) and if not corrected for could lead to false changes in measured deposition under power lines.

The metal spheres were permanently grounded and therefore the field perturbation was the same

Figure 10. Measured deposition velocity for total deposition (ultrafine and attached particles) for the 'pizza box' detector arrangement shown in figure 4a for the experiment 1 power line exposure. (a) Data points for ^{218}Po deposition on detectors mounted vertically parallel and perpendicular to the line are shown separately. Measured deposition data plotted as the ratio of deposition on detectors mounted parallel versus perpendicular to the line: (b) ^{218}Po deposition versus distance and (c) ^{214}Po deposition versus distance.

both in all directions and in all weather conditions. The data from table 3 show typical EF values from 1.1 to 2.9. Comparing the values with the prediction



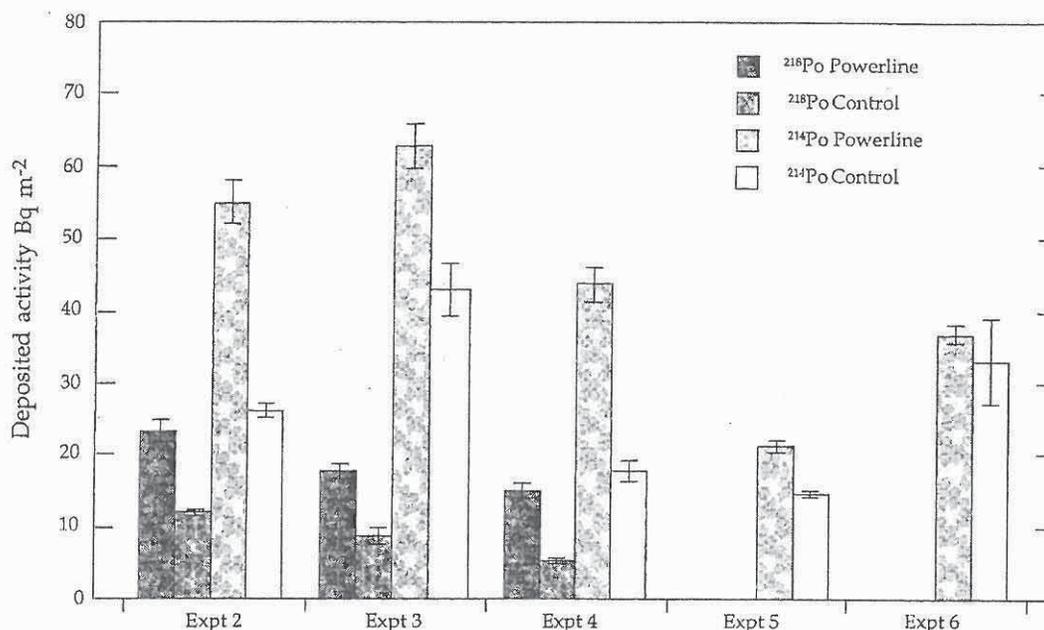


Figure 11. Sphere deposition of ^{218}Po and ^{214}Po aerosols under high voltage power lines and distant controls: experiments 2, 3 and 4, 400 kV; experiment 5, 275 kV and experiment 6, 132 kV.

Table 3. Summary of the sphere results giving the mean enhancement factor under the powerline.

Experiment	No of spheres expt, controls	^{218}Po			^{214}Po		
		Powerline Bq m^{-2}	Control Bq m^{-2}	Enhancement factor	Powerline Bq m^{-2}	Control Bq m^{-2}	Enhancement factor
2	2, 2	23.32 ± 1.57	11.92 ± 0.42	1.96 ± 0.15 (0.042)	54.96 ± 2.89	26.15 ± 1.10	2.10 ± 0.14 (0.033)
3	4, 4	17.80 ± 0.99	8.91 ± 1.12	2.00 ± 0.27 (0.001)	62.90 ± 3.12	43.05 ± 3.61	1.46 ± 0.14 (0.019)
4	4, 4	15.07 ± 0.97	5.27 ± 0.48	2.86 ± 0.32 ($< 10^{-4}$)	43.70 ± 2.29	17.92 ± 1.44	2.44 ± 0.23 ($< 10^{-4}$)
5	2, 2	-	-	-	21.10 ± 0.83	14.76 ± 0.40	1.43 ± 0.07 (0.028)
6	2, 2	-	-	-	36.90 ± 1.26	33.10 ± 5.99	1.11 ± 0.21 (0.39)*

*Not significant.
 p -value given in brackets.

in figure 9 suggests that λ_{ch} is likely to be in the region $1-2 \text{ s}^{-1}$, giving a neutralization time somewhat less than 1 s. In these conditions, enhancement is expected on the spheres but not on the pizza boxes with an unperturbed field.

4.3. General aerosol behaviour

Significant enhanced body deposition in the region 1.5–2.0 is expected for general aerosol particles in the entire size range 10 nm to $10 \mu\text{m}$, as shown in figure 7. This result is entirely independent of radon decay product behaviour, although experimental data for this enhancement is not available. For a

given aerosol size spectrum, the theoretical prediction could be used to estimate the increased mass deposition of pollutant aerosols under power lines. This could include the expected increased deposition of viruses and bacteria in air. It should also be noted that detectable excess deposition on the head would be expected for all common transmission line voltages. This is borne out in the measurements at 400 and 275 kV. The EF value at 132 kV in the current measurements was not statistically significant, although as stated above, the measurements were carried out in particularly wet and windy conditions.

The existence of meaningful excess deposition in terms of exposure to non- ^{222}Rn pollutants is essen-

Table 4. Summary of pizza box total deposition values in Bq m^{-2} for sphere exposures.

Exposure	Powerline		Control	
	^{210}Po	^{214}Po	^{210}Po	^{214}Po
2	7.6 ± 0.3 (15)	16.9 ± 0.7 (15)	8.2 ± 1.2 (3)	17.6 ± 3.4 (3)
3	7.9 ± 0.5 (14)	34.9 ± 1.6 (14)	7.8 ± 0.5 (12)	27.3 ± 1.7 (12)
4	3.92 ± 0.19 (1)	13.9 ± 0.4 (1)	3.10 ± 0.40 (2)	14.2 ± 1.3 (2)
5	—	12.7 ± 0.3 (1)	—	10.7 ± 0.3 (1)
6	—	17.9 ± 0.4 (1)	—	21.2 ± 0.4 (1)

Standard errors are shown. Number of detectors is given in brackets; where only one detector is available the error is based on the counting statistics.

Table 5. Directionality of ^{210}Po -enhanced deposition around the spheres for experiment 4.

Direction	^{210}Po powerline	^{210}Po control	EF
Top	16.20 ± 1.81	6.73 ± 0.71	2.41 ± 0.37
North-west	31.35 ± 0.57	6.00 ± 0.32	5.23 ± 0.29
South-east	1.80 ± 0.43	3.30 ± 0.16	0.55 ± 0.13
North-east	3.35 ± 0.58	4.10 ± 0.58	0.82 ± 0.18
South-west	22.65 ± 1.35	6.23 ± 0.70	3.64 ± 0.46

tially due to the perturbation of the E-field by the human body. In particular, no detectable effect is expected in the unperturbed field. This is in broad agreement with the overall pizza box results in table 4 and suggests that the increased deposition on pizza box assemblies in figure 10 (experiment 1) may indeed be attributed to uncertain field enhancement around individual detectors due to the wet conditions.

The deposition model suggests that the aerosol oscillation mechanism, coupled to turbulent diffusion, does not affect the air concentration of aerosols. Miles and Algar (1997) used air sampling to measure ^{222}Rn decay product activity in air and found no difference in values near compared with away from a 400 kV power line. McLaughlin and Gath (1999) also found no difference in ^{222}Rn decay product activity either in air or on surfaces near compared with away from a 400 kV power line. Their results, however, appear to be based on only four data points in the unperturbed field. Also, there is no indication that the detectors were aligned at right angles to the power line E-field vector. In any case, both these results and those of Miles and Algar (1996) are entirely consistent with the modelling presented here, which predicts that neither the airborne activity nor

the deposition in the unperturbed field will show any enhancement under power lines.

4.4. Implications for dose

The observed increased deposition on spheres representing the human head under high voltage power lines is in good agreement with the theoretical model which demonstrates a mode of increased exposure to ^{222}Rn marker aerosols under high voltage power lines. The model suggests that aerosols in general, including viruses and bacteria, will be subject to increased deposition and this could be tested experimentally in further work.

There is current interest in the radiation dose to the basal layer from ^{218}Po and ^{214}Po deposition on the skin and possible links with skin cancer. For indoor exposure at the average UK ^{222}Rn concentration of 20 Bq m^{-3} the annual equivalent dose to the skin basal layer on the face and neck has been estimated as 2.5 mSv y^{-1} (range $1.7\text{--}17 \text{ mSv y}^{-1}$) (Eatough 1997). Outdoors, the deposition velocity is ~ 20 times greater than indoors so that even accounting for lower ^{222}Rn concentration outdoors, the dose conversion per unit time is much greater outdoors than indoors.

It is possible to estimate the indoor and outdoor doses from the authors' own experimental measurements, assuming that deposition occurs at a constant rate throughout the experiments. Indoors, the unpublished measurements give average respective ^{218}Po and ^{214}Po deposition rates of 1.03 and 2.18 Bq m^{-2} at 20 Bq m^{-3} . This is consistent with results from modelling using a deposition velocity of $5 \times 10^{-4} \text{ m s}^{-1}$, similar to that reported by Porstendörfer (1994). Using equivalent dose conversion factors for the face of 0.7 and $1.1 \mu\text{Sv y}^{-1}$ per decay per cm^2 for ^{218}Po and ^{214}Po respectively, estimated by Eatough (1997), this yields a dose rate of 9.8 mSv y^{-1} . By comparison, the deposition rates outdoors away from power lines reported above are 7.5 Bq m^{-2} for ^{218}Po and 22.6 Bq m^{-2} for ^{214}Po . These values are consistent with an outdoor ^{222}Rn concentration of 7 Bq m^{-3} and an ultrafine deposition velocity of 0.01 m s^{-1} . This deposited activity corresponds to a dose rate of 95 mSv y^{-1} for continuous exposure outdoors.

If a person spends 10% of the time outdoors then half a person's skin dose arises outdoors, and typical skin doses are twice those arising from indoor exposure alone. Using these figures, the total dose rate (outdoors + indoors) away from power lines is 18.3 mSv y^{-1} . This mean value is consistent with recently reported values found on personal skin dosimeters (Eatough *et al.* 1999). Under power lines,

however, measurements suggest that the outdoor dose is increased in the range 1.4–2.9 so that the total skin dose rate becomes 22.1–36.4 mSv $^{-1}$, an increase in total exposure in the range 1.2–2.0. These surprising results have consequences for the skin cancer risk associated with natural ^{222}Rn exposure, especially for those living under high voltage power lines.

Quantitative estimates of dose to internal organs are difficult to make from the present work. Table 2 suggests that the velocity at deposition of ultrafine aerosols onto the human head is around 14 m s $^{-1}$ (50 km h $^{-1}$) in an AC field of 5 kV m $^{-1}$. Aerosols landing near the nose and mouth might deposit with even higher velocities owing to the higher field distortion in these regions where they might be susceptible to increased inhalation. In the case of some chemical species, there may be the possibility of increased absorption through the skin.

Further mechanisms of increased exposure to aerosols near power lines are under investigation. The authors are particularly interested in the effect of corona losses, which lead to the emission of large fluxes of ions into the atmosphere. The resulting atmospheric space charge can reverse the direction of the Earth's natural DC field up to several hundred metres from the line. In some circumstances, corona ions will have the effect of increasing the steady state charge on aerosols near power lines, which in turn will increase their lung deposition probability on inhalation (Cohen *et al.* 1998). This subject will be discussed in a separate paper.

4.5. Links with childhood leukaemia and traffic density

The authors suggest the results presented here are relevant to the reported epidemiological associations between high voltage power lines and childhood and adult leukaemia, given that it is only one of several mechanisms of increased exposure to pollutants near power lines. The body of evidence relating traffic pollution with childhood leukaemia is such that as a working hypothesis the link can be considered causal. The authors point out again that this link concerns exposure outdoors as does the increased exposure near power lines. Some studies have shown increased incidence of lung cancer in relation to power line exposures (Erren 1996, Henshaw 1997). Given that environmental pollution contains polycyclic aromatic hydrocarbons, including known lung carcinogens such as benzo[α]pyrene, it may be assumed that there is increased exposure to these agents under power lines.

5. Summary

1. Taking into account the special features of ^{222}Rn decay product aerosols, notably their initial positive charge state and ultrafine form, such aerosols are a useful marker of general aerosol behaviour in AC fields outdoors near high voltage power lines.
2. A theoretical model predicts enhanced aerosol deposition on the human head in typical power line fields.
3. Repeated measurements under high voltage power lines of ^{222}Rn decay product aerosol deposition on spheres mimicking the human head show EF values in the range 1.4–2.9 in a variety of weather conditions, in good agreement with the theoretical model.
4. The results suggest that the total (indoor + outdoor) dose to the basal layer of facial skin from deposited ^{218}Po and ^{214}Po is increased by between 1.2 and 2.0 for 10% of time spent outdoors under high voltage power lines.
5. The model suggests that, outdoors, the aerosol deposition in AC fields is only weakly affected by the Earth's natural DC field, but indoors the effects of DC (static) fields are likely to be significantly greater than for AC fields.
6. Overall, the present results lend support to the working hypothesis that the associations between childhood cancer and power (transmission) lines are causal and are due to increased exposure to environmental pollutants near power lines, notably from vehicle exhausts.

It is hoped that the findings in this paper will be of value in designing future case-control studies of childhood leukaemia and other cancers in relation to high voltage power (transmission) line exposures, as well as re-assessing the results of existing studies. The overall aim in future work is to establish risk factors for living under power lines based on increased exposure to environmental pollution.

Acknowledgements

The authors thank Nicola Holden and Adrian Castelino-Prabhu for assistance in detector assembly. This work was supported by the UK Medical Research Council, the Department of Health, the Foundation for Children with Leukaemia and the Spandex Foundation, UK.

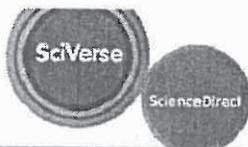
References

- AHLBOM, A., FEYCHTING, M., KOSKENVUO, M., OLSEN, J. H., PUKKALA, E., SCHULGEN, G. and VERKASALO, P., 1993,

- Electromagnetic fields and childhood cancer. *Lancet*, **342**, 1295–1296.
- CHENG, Y. S., SU, Y. F., NEWTON, G. J. and YEH, H. C., 1992, Use of a graded diffusion battery in measuring the activity size distributions of thoron progeny. *Journal of Aerosol Science*, **23**, 361–372.
- CLEMENT, C. F. and HARRISON, R. G., 1992, The charging of radioactive aerosols. *Journal of Aerosol Science*, **23**, 481–504.
- COHEN, B. S., XIONG, J. Q., FANG, C. P. and LI, W., 1998, Deposition of charged particles on lung airways. *Health Physics*, **74**, 554–560.
- CRUMP, J. G. and SEINFELD, J. H., 1981, Turbulent deposition and gravitational sedimentation of an aerosol in a vessel of arbitrary shape. *Journal of Aerosol Science*, **12**, 405–415.
- EATOUGH, J. P., 1997, Alpha-particle dosimetry for the basal layer of the skin and the radon progeny ^{218}Po and ^{214}Po . *Physics in Medicine and Biology*, **42**, 1899–1911.
- EATOUGH, J. P., WORLEY, A. and MOSS, G. R., 1999, Personal monitoring of ^{218}Po and ^{214}Po radionuclide deposition onto individuals under normal environmental exposure conditions. *Physics in Medicine and Biology*, **44**, 2227–2239.
- ERREN, T. C., 1996, Re: Association between exposure to pulsed electromagnetic fields and cancer in electric utility workers in Quebec, Canada, and France. *American Journal of Epidemiology*, **143**, 841.
- FEWS, A. P., 1992a, Fully automated image analysis of etched tracks. *Nuclear Instruments and Methods in Physics Research*, **B72**, 465–478.
- FEWS, A. P., 1992b, Flexible analysis of etched nuclear particle tracks. *Nuclear Instruments and Methods in Physics Research*, **B72**, 91–103.
- FEYCHTING, M. and AHLBOM, A., 1993, Magnetic fields and cancer in children residing near Swedish high-voltage power lines. *American Journal of Epidemiology*, **138**, 467–481.
- HATCH, E. E., LINET, M. S., KLEINERMAN, R. A., TARONE, R. E., SEVERSON, R. K., HARTSOCK, C. T., HAINES, C., KAUNE, W. T., FRIEDMAN, D., ROBISON, L. L. and WACHOLDER, S., 1998, Association between childhood acute lymphoblastic leukaemia and use of electrical appliances during pregnancy and childhood. *Epidemiology*, **9**, 234–245.
- HENSHAW, D. L., 1997, Electromagnetic field exposure and lung cancer. *American Journal of Epidemiology*, **146**, 366.
- HENSHAW, D. L., ALLEN, J. E., KEITCH, P. A. and RANDLE, P. H., 1994, The spatial distribution of naturally occurring ^{210}Po and ^{226}Ra in children's teeth. *International Journal of Radiation Biology*, **66**, 815–826.
- HENSHAW, D. L., KEITCH, P. A. and JAMES, P. R., 1995, Lead-210, polonium-210 and vehicle exhaust pollution. *Lancet*, **345**, 324–325.
- HENSHAW, D. L., ROSS, A. N., FEWS, A. P. and PREECE, A. W., 1996, Enhanced deposition of radon daughter nuclei in the vicinity of power frequency electromagnetic fields. *International Journal of Radiation Biology*, **69**, 25–38.
- HINDS, W. C., 1982, *Aerosol Technology—Properties, Behaviour and Measurement of Airborne Particles* (New York: John Wiley & Sons).
- HOPKE, P. K., 1989, Use of electrostatic collection of ^{218}Po for measuring Rn. *Health Physics*, **57**, 39–42.
- HOWARD, A. J. and STRANGE, W. P., 1992, Measurement of $^{218}\text{Po}^+$ neutralisation rates in gases. *Nuclear Instruments and Methods in Physics Research*, **A311**, 378–385.
- INTERNATIONAL COMMISSION ON NON-IONISING RADIATION PROTECTION (ICNIRP), 1998, Guidelines for limiting exposure to time varying electric, magnetic and electromagnetic fields (up to 300 GHz). *Health Physics*, **74**, 494–512.
- JACOBI, W., 1972, Activity and potential α -energy of ^{222}Rn and ^{222}Rn daughters in different air atmospheres. *Health Physics*, **22**, 441–450.
- JACOBI, W. and ANDRÉ, K., 1963, The vertical distribution of radon-222 and radon-220 and their decay products in the atmosphere. *Journal of Geophysical Research*, **68**, 3799–3814.
- JONASSEN, N., 1983, The effect of electric fields on ^{222}Rn daughter products in indoor air. *Health Physics*, **45**, 487–491.
- LINDQUIST, R., NILSSON, B., EKLUND, G. and GAHRTON, G., 1991, Acute leukaemia in professional drivers exposed to gasoline and diesel. *European Journal of Haematology*, **47**, 98–103.
- MAYYA, Y. S. and SAPRA, B. K., 1997, Radon daughter deposition on surfaces carrying alternating electric fields. *International Journal of Radiation Biology*, **71**, 69–74.
- MCLAUGHLIN, J. P. and GATH, G., 1999, Radon progeny activities in the vicinity of high voltage power lines. *Radiation Protection Dosimetry*, **4**, 257–262.
- MERCER, T. T., 1976, The effect of particle size on the escape of recoiling RaB atoms from particulate surfaces. *Health Physics*, **31**, 175–176.
- MILES, J. C. H. and ALGAR, R. A., 1997, Measurements of radon decay product concentrations under power lines. *Radiation Protection Dosimetry*, **74**, 193–194.
- NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES, 1998, Working Group Report: Assessment of health effects from exposure to power-line frequency electric and magnetic fields, edited by C. J. Portier and M. S. Wolfe. US DoE/NIEHS/NIH (Research Triangle Park: NIEHS).
- NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES, 1999, NIEHS Report on health effects from exposure to power-line frequency electric and magnetic fields. NIH Publication No. 99-4493 (Research Triangle Park: NIEHS).
- NATIONAL RADIOLOGICAL PROTECTION BOARD (NRPB), 1994, Supplementary report by the advisory group on non-ionising radiation, of 12 April 1994, Chairman: Sir Richard Doll. *Radiological Protection Bulletin*, No. 154, June 1994.
- NORDLINDER, R. and JÄRVHOLM, B., 1997, Environmental exposure to gasoline and leukaemia in children and young adults—an ecological study. *International Archives of Occupational and Environmental Health*, **70**, 57–60.
- OLSEN, J. H., NIELSEN, A. and SCHULGEN, O., 1993, Residence near high-voltage facilities and risk of cancer in children. *British Medical Journal*, **307**, 891–895.
- PORSTENDÖRFER, J., 1984, Behaviour of radon daughter products in indoor air. *Radiation Protection Dosimetry*, **7**, 107–113.
- PORSTENDÖRFER, J., 1994, Properties and behaviour of radon and thoron and their decay products in the air. *Journal of Aerosol Science*, **25**, 219–263.
- RAES, F., 1985, Description of the properties of unattached ^{218}Po and ^{212}Pb particles by means of the classical theory of cluster formation. *Health Physics*, **49**, 1177–1187.
- ROBÉ, M. C., RANNOU, A. and LE BRONEC, J., 1992, Radon measurement in the environment in France. *Radiation Protection Dosimetry*, **45**, 455–457.
- ROBINSON, A. A., 1991, Cancer deaths due to all causes, its relationship with vehicle travel in Australia, Japan and European Countries. *Medical Hypotheses*, **36**, 166–171.
- SAVITZ, D. A. and FEINGOLD, L., 1989, Association of childhood cancer with residential traffic density. *Scandinavian Journal of Work, Environmental Health*, **15**, 360–363.

- SEHMEL, G. A., 1973, Particle eddy diffusivities and deposition velocities for isothermal flow and smooth surfaces. *Journal of Aerosol Science*, **4**, 125-138.
- SHI, B. and HOPKE, P. K., 1991, Study of neutralisation of ^{218}Po by small recombination in O_2 , Ar and N_2 . *Health Physics*, **61**, 209-214.
- TYNDALL, A. M., 1938, *The Mobility of Positive Ions in Gases* (Cambridge, UK: Cambridge University Press).
- VERKASALO, P. K., PUKKAL, E., HONGISTO, M. Y., VALJUS, J. E., JÄRVINDEN, P. J., HEIKKILÄ, K. V. and KOSKENVUO, M., 1993, Risk of cancer in Finnish children living close to power lines. *British Medical Journal*, **307**, 895-899.

100526



Brought to you by:
Boise State University Albertsons
Library

Articles All fields Author
 Images Journal/Book title Volume Issue Page Advanced search Search tips

Download SciVerse Scopus mobile apps today
Available for iPhone, Blackberry and android devices

PDF (240 K) Export citation E-mail article Highlight keywords on

Article Figures/Tables (4) References (77) Thumbnails | Full-Size images

Science of The Total Environment
Volume 408, Issue 15, 15 July 2010, Pages 3062-3069

doi:10.1016/j.scitotenv.2010.03.039 | How to Cite or Link Using DOI
Permissions & Reprints

Review

Exposure to electromagnetic fields (non-ionizing radiation) and its relationship with childhood leukemia: A systematic review

I. Calvente^{a,b}, M.F. Fernandez^{a,b}, J. Villalba^b, N. Olea^{a,b}, M.L. Nuñez^b

^a Laboratory of Medical Investigations, San Cecilio University Hospital, CIBER de Epidemiología y Salud Pública (CIBERESP), Spain

^b Department of Radiology, University of Granada, Granada, Spain

Received 16 November 2009; revised 17 March 2010; Accepted 24 March 2010. Available online 7 May 2010.

Abstract

Childhood exposure to physical contamination, including non-ionizing radiation, has been implicated in numerous diseases, raising concerns about the widespread and increasing sources of exposure to this type of radiation. The primary objective of this review was to analyze the current state of knowledge on the association between environmental exposure to non-ionizing radiation and the risk of childhood leukemia. Scientific publications between 1979 and 2008 that include examination of this association have been reviewed using the MEDLINE/PubMed database. Studies to date have not convincingly confirmed or ruled out an association between non-ionizing radiation and the risk of childhood leukemia. Discrepancies among the conclusions of the studies may also be influenced by confounding factors, selection bias, and misclassification. Childhood defects can result from genetic or epigenetic damage and from effects on the embryo or fetus, which may both be related to environmental exposure of the parent before conception or during the pregnancy. It is therefore critical for researchers to define *a priori* the type and "window" of exposure to be assessed. Methodological problems to be solved include the proper diagnostic classification of individuals and the estimated exposure to non-ionizing radiation, which may act through various mechanisms of action. There appears to be an urgent need to reconsider exposure limits for low frequency and static magnetic fields, based on combined experimental and epidemiological research into the relationship between exposure to non-ionizing radiation and adverse human health effects.

Abbreviations: AML, acute myeloid leukemia; ALL, acute lymphoid leukemia; EMR, electromagnetic radiation; ELF-EMR, extremely low-frequency electromagnetic radiation; ICNIRP, International Council of Non-ionizing Radiation Protection; NIR, non-ionizing radiation; LF-EMR, low-frequency electromagnetic radiation; RF, radio frequencies

Keywords: Childhood leukemia; Non-ionizing radiation

Related Articles

- Electromagnetic field exposure during pregnancy and chi... *The Lancet*
- Compute extremely low-frequency electromagnetic field e... *The Journal of China Universities of Posts and Telecomm...*
- Occupational exposure to non-ionizing radiation: Hazard... *Chemical Health and Safety*
- Spectroscopic evidence of the effects induced by non-io... *Vibrational Spectroscopy*

[View more related articles](#)

My Applications

Add

Table Download

Find HTML data tables from the current article to download.

[About Table Download](#)

Most Downloaded

Most downloaded articles from the last three months in this journal:

- Combination of Advanced Oxidation Processes and biological treatments for wastewater decontamination-A review
- Treatment of pulp and paper mill wastewater-a review
- Exposure and effects assessment of persistent organohalogen contaminants in arctic wildlife and fish
- [View the most downloaded articles for Science of The Total Environment](#)

Provided by ScienceDirect Top25

Cited by (1)

- Some practical considerations in electromagnetic biocom... *Environmental Engineering and Management Journal*

[View details of all 1 citing articles in Scopus](#)

Provided by Scopus

Related reference work articles e.g. encyclopedias

63/90

100526

Article Outline

- 1. Introduction
- 2. Mechanisms
- 3. Methods
- 4. Results
 - 4.1. Non-ionizing radiation: low and extremely low-frequency electromagnetic fields
 - 4.1.1. First period (1979–2000)
 - 4.1.2. Second period (2001–2008)
 - 4.2. Non-ionizing radiation: radio frequency electromagnetic fields
- 5. Discussion
- 6. Possible future actions
- Acknowledgments
- References

1. Introduction

Humans have been constantly exposed to electromagnetic radiation, including sunlight, cosmic rays, and terrestrial radiations. However, a substantial increase in exposure, especially to low-frequency electromagnetic radiation (EMR), started in the early 20th century with the generation of artificial electromagnetic fields and continued with the development of power stations, radio, radar, television, computers, mobile phones, microwave ovens, and numerous devices used in medicine and industry. These technological advances have aroused concerns about the potential health risks associated with unprecedented levels of EMR exposure.

The amount of energy deposited by EMR and the form of its absorption is determined by the frequency and type of incident radiation and by the nature of the tissue that absorbs it. According to its effects on the organism, EMR can be divided between ionizing radiation (including high-frequency radiation such as gamma rays and X-rays) and non-ionizing radiation (NIR; low to very low frequency). Ionizing radiation causes biological effects by directly or indirectly damaging the DNA molecule; the effects of this type of radiation are not addressed in the present review.

Exposure to the multiple sources of NIR (Table 1), including residential exposure to high-voltage power lines, transformers, and domestic electrical installations, varies in duration and according to the distance from the source. Exposure is usually to low-frequency (LF-EMR) or extremely low-frequency (ELF-EMR) radiation and is continuous and rising among populations in the industrialized world. Besides LF-EMR and ELF-EMR radiation, individuals are increasingly exposed to radio frequencies (RF) from television (TV) towers, radio stations, mobile phone/wi-fi systems, and personal computers. Table 1 summarizes the different types, frequency ranges, and sources of NIR. Nevertheless, the average magnetic flux density is generally considered to be below the maximum exposure limits established by different organizations such as the International Council of Non-Ionizing Radiation Protection (ICNIRP) or the Spanish Experts' Committee of Electromagnetic Fields and Public Health in our setting (2001, 2003).

Table 1. Frequencies and sources of non-ionizing radiation.

Frequency	Type of radiation	Sources
0 Hz–300 kHz	Low frequency to extremely low frequency (LF–ELF) electromagnetic radiation	Electrical fields of devices, conventional electrical network, video monitors, sections of AM radio
3 kHz–300 MHz	Radio frequencies (RF)	Sections of AM radio, FM radio, medical short-wave, nuclear magnetic resonance (NMR)
300 MHz–300 GHz	Microwave (MW)	Domestic microwave devices, mobile telephones, microwave for medical physical therapy, radar and other microwave communications
300 GHz–780 nm	Infrared (IR)	Solar light, heat and laser therapy devices
780 nm–400 nm	Visible light	Solar light, phototherapy, laser
400 nm–100 nm	Ultraviolet (UV)	Solar light, fluorescent tubes, food/air sterilization, radiotherapy, etc.

Hz: Hertz (kHz: kilohertz = 10³ Hz; MHz: Megahertz = 10⁶ Hz; GHz: Gigahertz = 10⁹ Hz). Ultraviolet (UV) within the range 280–185 nm is considered as ionizing radiation.

In 1998, guidelines on reference values, exposure limits, and restrictions were issued by the ICNIRP and other with the aim of protecting citizens against the possible harmful effects of acute exposure to this type of radiation (Table 2). The exposure limit for the general public is currently 50 Hz at 100 μT and higher frequencies (Table 2) (ICNIRP, 1998). These limits had previously been established by the IEEE (Institute of Electric and Electronics Engineers, Inc, 1992), also based on protection from immediate short-term

- Electromagnetic Fields: Environmental Exposure
Encyclopedia of Environmental Health
- Electromagnetic Fields
Encyclopedia of Toxicology
- Magnetic Fields: Possible Environmental Health Effects
Encyclopedia of Environmental Health
- Microwaves: Exposure and Potential Health Consequences
Encyclopedia of Environmental Health
- Risk of Radiation Exposure to Children and Their Mother...
Encyclopedia of Environmental Health

More related reference work articles

Relevant terms from this article

Click for **Data Correlations**, **Clinical Trials** and more

10 Diseases

- leukemia
 - acute lymphoid leukemia
 - acute myeloid leukemia
 - dna damage
- ➔ View more...

8 Tissues & Cells

- embryo
 - bone-marrow
 - fetus
 - fetuses
- ➔ View more...

3 Organisms

- human
- rats
- arabi

Powered by NextBio What is this?

Find it

View Record in Scopus

12th **ISBGMO**
St. Louis | USA 2012

**The 12th
International Symposium
on Biosafety of Genetically
Modified Organisms**
16-20 September 2012

64/90

100526

effects. Although there is experimental evidence of biological responses at non-thermal NIH levels, it is not considered sufficiently robust or relevant to establish their potential impact on health (Kundi et al., 2009).

Table 2. Protection limits for exposure to electrical, magnetic, and electromagnetic fields.

Frequency range	Field intensity E (V/m)	Field B (μ T)	Power density (W/m^2)
0–1 Hz	–	4×10^4	–
1–8 Hz	10,000	$4 \times 10^4 / f$	–
8–25 Hz	10,000	$5000 / f$	–
0.025–0.8 kHz	$250 / f$	$5 / f$	–
0.8–3 kHz	$250 / f$	6.25	–
3–150 kHz	87	6.25	–
0.15–1 MHz	87	$0.92 / f$	–
1–10 MHz	$87 / f^{1/2}$	$0.92 / f$	–
10–400 MHz	28	0.09	2
400–2000 MHz	$1.375 \times f^{1/2}$	$0.0046 \times f^{1/2} / 200$	
2–300 GHz	61	0.2	10

f : frequencies as indicated in the column of frequency range.

Among the few studies on magnetic flux density (magnetic flux density mapping), some show that the established limits were exceeded in a number of areas of Eastern Europe and North America ([Kheifetz et al., 2006], [Maslianyj et al., 2007] and [Straume et al., 2008]). In Spain, exposure to EMR has been measured in schools in the cities of Oviedo and Barcelona and in the region of Extremadura ([Tardón et al., 2002] and [Paniagua et al., 2002]), and magnetic flux density has been mapped in Extremadura (Paniagua et al., 2004).

Children are considered more vulnerable than adults to environmental exposure and deserve special research attention ([Fernández et al., 2007] and [Ramón et al., 2005]). Childhood exposure to physical contamination, including NIR, has been implicated in numerous diseases, raising concerns about their widespread and increasing use of mobile phones and other sources of NIR. The first authors to report a possible link between leukemia and environmental factors were Ager et al. (1965). Since then, a significant increase in the incidence of childhood leukemia has been reported in Europe and the United States, identifying clusters of cases associated with potential environmental etiological components ([Draper et al., 2005] and [McNally and Parker, 2006]). A study in 2001 reported that the incidence of leukemia among 2 to 4-year-olds was higher in industrialized countries than in developing countries, especially for the common subtype of acute lymphoblastic leukemia (ALL) (Milham and Ossiander, 2001). They investigated the advance of electrification between 1920 and 1960 and detected a peak in childhood leukemia incidence rates in 1930 in all states that had introduced electricity in >75% of residences. By 1950, an elevated incidence was recorded in all states but was more pronounced in those with a higher percentage of households connected to mains electricity. According to the authors, the association of leukemia with urbanization, modernization, and industrialization may be explained by the increase in electrification.

The annual incidence of childhood leukemia was estimated to be 4/100,000 by the WHO and 5/100,000 by the ICNIRP, in 2003. In our setting, 107 new cases (under 15 years old) were recorded in the Cancer Registry of Granada province (Spain) between 1985 and 2004, representing a mean annual incidence of 3.25/100,000 children, lower than in any other Spanish cancer registry. Nevertheless, according to the estimated cumulative rate (0.5%) and if this trend continues, 1 out of every 2000 children living in Granada will develop leukemia before the age of 15 years (WHO: www.who.int/emf).

The primary objective of this review was to analyze the current state of knowledge on the association between environmental exposure to NIR and the risk of childhood leukemia.

2. Mechanisms

Our working hypothesis was that NIR may be an environmental risk factor for developing childhood leukemia. The greatest obstacle to testing this hypothesis is the absence of a consensus on the effects of LF-EMR or ELF-EMR on the organism at organ, tissue, cell, or molecular level (Ruiz-Gómez et al., 2009).

Koifman (1993) suggested that EMR might be carcinogenic, and this proposition was subsequently supported by the findings of the "UK Childhood Cancer Study Investigators" (UKCCS, 1999). However, the International Agency for Research on Cancer (IARC) only began to consider LF and ELF electromagnetic fields as possible carcinogens (category 2B) after publication of the pooled analysis by Ahlbom et al. (2000) and Greenland et al. (2000). No classification of RF electromagnetic fields has been proposed (Schüz and Ahlbom, 2008).

Research has especially focused on the extremely low frequencies used in electrical power lines (50–60 Hz) ([Carrubba and Marino, 2008], [Coulton et al., 2004], [Girgert et al., 2005] and [IARC and Working Group on the Evaluation of Carcinogenic Risks to Humans, 2002]), and on radiofrequencies (RF: 3 kHz to 300 MHz) and microwaves (MW: 300 MHz to 3 GHz) typically used for cell phone transmissions (Valentini

65/90

100526

et al., 2007). The thermal effects of EMH via direct energy transfer are well established, while possible non-thermal effects are controversial ([Carrubba and Marino, 2008], [Coulton et al., 2004], [de Pomerai et al., 2003], [del Vecchio et al., 2009], [Diem et al., 2005] and [Girgert et al., 2005]). In fact, microwaves have been reported to exert non-thermal effects in biological systems, at least partially arising from alterations in the conformation of cellular proteins (de Pomerai et al., 2003).

It has also been suggested that ELF-EMR influences proliferation and DNA damage in both normal and tumor cells through the action of free radical species, with no significant temperature difference between culture media of exposed and unexposed cells (Wolf et al., 2005). Other studies revealed an increase in DNA single-strand breaks in cells of rats exposed to 2.45 GHz (Paulraj and Behari, 2006). However, it was also reported that oxidative DNA damage does not significantly contribute to the DNA fragmentation observed in human fibroblasts after ELF-EMR exposure (Focke et al., 2010). It was recently demonstrated that the DNA molecule can be adversely affected by intermittent exposure to ELF-EMR, which may explain the relationship between LF-EMR and childhood leukemia and would support the classification of EMR as a potential genotoxin (Ivancsits et al., 2002). In this context, Binhi (2008) reported that magnetic nanoparticles found in some organisms played a role in increasing the concentration of free radicals within cells, which may explain the genotoxic action exerted by LF-EMR on the DNA molecule of hematopoietic stem cells.

There have been recent reports of other types of EMR exposure, especially through contact currents or voltages, e.g., from the voltage between water pipes and earth. Contact currents can also result from induction caused by magnetic fields from nearby heavily loaded power lines. This form of exposure is more likely in residences with high magnetic fields, in which contact current from metallic fixtures during the bathing of children may lead to elevated bone-marrow doses of induced currents ([Kavet and Zaffanella, 2002] and [Brain et al., 2003]). Unfortunately, there are few data on the role of contact voltages, and further research is required in this area.

Various *in vitro* studies and animal experiments found that simultaneous exposure to ELF radiation enhanced the effects of physical or chemical carcinogens, showing positive associations with a non-linear dose-response curve for energy fields between 1 and 3 mT (Juulilainen et al., 2006).

In considering the relationship with childhood leukemia, the timing of exposure is an important issue, as is the history of exposure, including occupational exposure of the parents of children, especially of the mothers during pregnancy. Some authors have linked exposure to NIR with the risk of abortion or a moderate increase in the risk of ALL ([Lee et al., 2002], [Li et al., 2002] and [Infante-Rivard and Deadman, 2003]). There have also been reports of overexposed fetuses in women receiving EMR doses within the established limits ([Wu et al., 2007], [Cech et al., 2007] and [Leitgeb and Cech, 2008]).

3. Methods

We reviewed the scientific publications between 1979 and 2008 that include examination of the association between human exposure to NIR (LF-EMR, ELF-ERM, and/or RF) and childhood leukemia. The MEDLINE/PubMed database was first searched for papers written in English or Spanish, using the key words electromagnetic fields and childhood leukemia. References cited in these papers were also examined for any additional publications. Studies were then selected according to the following criteria: 1) the type of design and the study population are reported; 2) the type and timing of exposure are defined *a priori*, and 3) the statistical methods are described.

Issues of major interest were the methods (direct or indirect) used to measure exposure and data on the variables influencing the exposure of parents. Direct assessments of EMR exposure included spot measurements of the strength of electromagnetic fields (magnetic flux density) at 24 and 48 h and at 1 week.

4. Results

Selected studies were classified according to the type of NIR (LF-EMR, ELF-ERM or RF) as summarized below.

4.1. Non-ionizing radiation: low and extremely low-frequency electromagnetic fields

Table 3 lists studies on the association between LF/ELF NIR and childhood leukemia. They are divided between two time periods: 1979–2000 and 2001–2008, because the meta-analyses by Ahlbom et al. (2000) and by Greenland et al. (2000) represented a turning point in exposure assessment and results.

Table 3. Childhood leukemia epidemiologic studies: low and extremely low-frequency electromagnetic fields.

Authors	Country, study period, design	Exposure assessment	Exposure category	Outcome [95% CI]
Wertheimer and Leeper (1979)	USA (Colorado) 1950–1973 Case-control: 344 cancer deaths and controls from birth registry	Wire codes	LCC ^a (birth address) HCC	OR: 2.28 [1.34–3.91]

66/90

100526

Authors	Country, study period, design	Exposure assessment	Exposure category	Outcome [95% CI]
Ahlbom et al. (2000)	Pooled analyses of 9 studies	24/48-hour magnetic field measurements or calculated magnetic fields	<0.1 μ T 0.1–0.2 μ T 0.2–0.4 μ T \geq 0.4 μ T	RR: 1.08 [0.89–1.31] RR: 1.11 [0.84–1.47]RR: 2.00 [1.27–3.13]
Greenland et al. (2000)	Meta-analyses with 15 studies	Wire code, 24/48-hour magnetic field measurements or calculated magnetic fields	\leq 0.1 μ T 0.1–0.2 μ T 0.2–0.3 μ T >0.3 μ T	OR: 1.00 [0.81–1.22] OR: 1.13 [0.92–1.39]OR: 1.65 [1.15–2.36]
Feychting et al. (2000)	SwedenChildren born between 1976–1977, and 1981–1982Cohort of 235,635 children	Parental occupational exposure before conception	Father occupational exposure \geq 0.3 μ T Mother occupational exposure \geq 0.3 μ T Parental occupational exposure \geq 0.3 μ T	RR: 2.0 [1.1–3.5] RR: 1.2 [0.5–2.4] RR: 4.7 [1.2–18.2]
Schüz et al. (2001)	West Germany1993 (90)–1997(94)Case-control: 514 cases from cancer registry and 1301 controls from population registry	24-h measurements in child's bedroom, living room and perimeter measurements	<0.1 μ T (MD 24 h) ^a 0.1–0.2 μ T 0.2–0.4 μ T \geq 0.4 μ T 0.1–0.2 μ T (MD night-time) ^b 0.1–0.2 μ T 0.2–0.4 μ T \geq 0.4 μ T	OR: 1.15 [0.73–1.81]OR: 1.16 [0.43–3.11]OR: 5.81 [0.78–43.2]OR: 1.42 [0.90–2.23]OR: 2.53 [0.86–7.46]OR: 5.53 [1.15–26.6]
Li et al. (2002)	USA (San Francisco) Prospective cohort of pregnant women: 969 cases	24-hour personal exposure and miscarriage	<1.6 μ T \geq 1.6 μ T \geq 1.6 μ T (miscarriages — 10 weeks of gestation)	RR: 1.80 [1.20–2.07]RR: 3.1 [1.3–7.7]
Infante-Rivard and Deadman (2003)	Canada (Québec) 1980–1993Case-control (ALL)—control (2003)	Cumulative maternal occupational exposure throughout pregnancyThree measurements: cumulative, average and maximum levels	\geq 0.4 μ T Only working women All studied women	OR: 2.2 [1.2–4.2] ^b OR: 2.3 [1.3–4.0] ^b OR: 2.3 [1.2–4.3] ^c OR: 2.3 [1.3–4.0] ^c
Draper et al. (2005)	England and Wales1962–1995Case-control: 29,081 cases and 29,081 controls from registries	Distance of residence to the nearest overhead power line	\geq 600 m (from power line) ^b 200–600 m <200 m	RR: 1.22 [1.01–1.47]RR: 1.68 [1.12–2.52]
Kabuto et al. (2006)	Japan (metropolitan areas)1999–2001Case-control: 321 cases from several registries and 634 controls from residential registry	Measurement of 7-day measurements in child's bedroom, Spot measurements inside and outside the house	(ALL + AML) <0.1 μ T (1 week TWA) ^a 0.1–0.2 μ T 0.2–0.4 μ T <0.1 μ T (1 week night-time) ^b 0.1–0.2 μ T 0.2–0.4 μ T \geq 0.4 μ T (ALL) <0.1 μ T (1 week TWA) ^c 0.1–0.2 μ T 0.2–0.4 μ T \geq 0.4 μ T	OR: 0.93 [0.51–1.71]OR: 1.08 [0.51–2.31]OR: 2.77 [0.80–9.57]OR: 0.97 [0.52–1.79]OR: 1.08 [0.47–2.47]OR: 2.87 [0.84–9.88]OR: 0.87 [0.45–1.69]OR: 1.03 [0.43–2.50]OR: 4.67 [1.15–19.0]
Feizi and Arabi (2007)	IranCase-control: 60 cases and 59 controls	Distance of residence to high-voltage overhead power lines (\leq 500 m)Intensities of magnetic fields calculated by mean intensity of electrical current and other line characteristics	>0.45 μ T 0.6 μ T 0.35 μ T	OR: 8.67 [1.74–58.40]OR: 3.60 [1.11–12.39]

67/90

100926

Authors	Country, study period, design	Exposure assessment	Exposure category	Outcome [95% CI]
Mejia-Arangure et al. (2007)	Mexico (Mexico-City) 1995–2003 Case-control Children with Down's syndrome and acute leukemia	Spot measurements at front door; wire coding.	<0.1 μ T (spot) ^a 0.1–0.4 μ T 0.4–0.6 μ T \geq 0.6 μ T Low according to Kaune-Savitz ^b Medium-High	OR: 0.94 [0.37–2.4] OR: 0.88 [0.15–5.1] OR: 3.70 [1.05–13] OR: 5.8 [0.92–37] OR: 4.1 [0.66–25]

AML: acute myeloid leukemia, ALL: acute lymphoblastic leukemia, HCC: high-current code, LCC: low-current code, MD: median, OR: odds-ratio, RR: relative risk, TWA: time-weighted average.

^a Reference category.

^b Adjusted (child age and sex).

^c Adjusted: child age and sex, and maternal age.

4.1.1. First period (1979–2000)

Before 2000, exposure to ELF radiation was estimated according to the distance from normal-to-high-voltage overhead power lines, using the wire code method. This method offers an indirect measurement of residential exposure to magnetic fields produced by electrical currents as a function of the distance from the residence to the electric power line, also taking into account the characteristics (e.g., wire-size) and location of the line.

We highlight the epidemiological study by Wertheimer and Leeper (1979) with a case-control design. It included patients with childhood leukemia diagnosed between 1976 and 1977 and living in Colorado (USA) and compared them with controls from 1950 to 1973. Children highly exposed to ELF radiation were found to have a two-fold higher risk of developing leukemia versus children with lower exposure (OR 2.28; 95% CI 1.34–3.91). This study was the first to propose and use the wire code approach, which was later considered to introduce a bias that invalidated results to some degree ([Jones et al., 1993], [Savitz and Poole, 2001] and [Schüz, 2007]).

Greenland et al. (2000) analyzed 15 available studies and described a higher overall risk of leukemia with an OR of 1.65 (95% CI 1.15–2.36) in children exposed to magnetic fields above 0.3 μ T in comparison to children exposed to fields <0.1 μ T. The pooled analysis by Ahlbom et al. (2000) considered the results of 9 studies, involving a total of 3203 children with leukemia and ten thousand controls, and calculated a higher risk (RR 2.0; 95% CI 1.27–3.13) for children exposed to \geq 0.4 μ T radiation versus those exposed to <0.1 μ T. Therefore, both pooled analyses described a similar increase in risk with higher levels of magnetic field.

4.1.2. Second period (2001–2008)

2001 saw the first publication of studies using a direct method to estimate exposure to EMR. It is based on spot measurements in rooms, especially bedrooms, determining mean exposure values for 24 h, 48 h, and 1 week, while also considering the distance from high-voltage power lines. Account also began to be taken of maternal exposure during pregnancy and the occupational exposure of the parents. However, these improved measurement methods yielded similar outcomes and conclusions to those obtained in the first period (1979–2000).

Schüz et al. (2001) and Kabuto et al. (2006) conducted studies of exposure to NIR for 24 h and one week, respectively, using spot measurements of magnetic fields. No statistically significant associations with childhood leukemia were obtained. However, when exposure exceeded 0.4 μ T, the risk was estimated to be five-fold higher (OR 5.81; 95% CI 0.78–43.2) by Schüz et al. (2001) and more than two-fold higher (OR 2.77; 95% CI 0.80–9.57) by Kabuto et al. (2006), using 0.1 μ T as reference category. Kabuto et al. also found a statistically significant risk (OR 4.7; 95% CI 1.15–19) for ALL.

One of the largest investigations into magnetic fields and childhood cancer was conducted by Draper et al. (2005) in England and Wales. In their epidemiological case-control study, the distance from the residence to high-voltage power lines was related to the relative risk of childhood leukemia, finding a significant risk for distances of 200–600 m (RR 1.22; 95% CI 1.01–1.47) and for distances less than 200 m (RR 1.68; 95% CI 1.12–2.52). Feizi and Arabi used the same approach in a residential area in northwest Iran, comparing between residences less and more than 500 m from high-voltage power lines ($>$ 4.5 μ T) and obtaining an OR of 3.6 for the former (95% CI 1.11–12.39) (Feizi and Arabi, 2007). The authors concluded that the presence of high-voltage overhead power lines within 500 m of residential areas should be considered a risk factor for acute childhood leukemia. Mejia-Arangure et al. (2007) examined the effect of exposure to EMR in children with Down's syndrome and diagnosed with acute leukemia. They found a higher risk of leukemia (OR 3.7; 95% CI 1.05–13) for magnetic fields \geq 0.6 μ T.

Li et al. (2002) studied 969 cases of childhood leukemia in San Francisco, where they collected information on reproductive health. They studied women at \leq 10 weeks of gestation and found no risk of miscarriage in those exposed to <1.6 μ T but a high risk (RR 1.8; 95% CI 1.2–2.07) in those exposed to >1.6 μ T. The association was stronger for women with a history of previous miscarriage or sub-fertility (RR 3.1; 95% CI 1.3–7.7).

68/90

100526

Infante-Rivard and Deadman (2003) investigated the association between childhood leukemia and cumulative exposure to NIR during pregnancy in an epidemiological case-control study of 491 children (0-9 years) diagnosed with leukemia in Quebec between 1980 and 1993. Estimation of maternal exposure during pregnancy took account of: a) the cumulative exposure in $\mu\text{T-days}$, b) mean exposure, and c) maximum exposure. The results suggest that children of mothers exposed to electromagnetic fields $\geq 0.4 \mu\text{T}$ during pregnancy had an increased risk of leukemia (OR 2.2; 95% CI 1.2-4.2). Results were similar (OR 2.3; 95% CI 1.2-4.3) when potential confounding variables were included in the model (age and sex of children and age of mothers). Other studies have also reported an association between residential exposure to $>0.4 \mu\text{T}$ NIR and childhood leukemia ([Ahlborn et al., 2000] and [Feychting et al., 2000]).

A cohort study conducted in Sweden by Feychting et al. (2000) showed an increased risk of leukemia in 10-year-old children whose parents had been exposed to $\geq 0.3 \mu\text{T}$ (RR 4.7; 95% CI 1.2-18.2), although this increase only reached significance in the boys (RR 2.2; 95% CI 1.0-4.5). Occupational exposure studies have in general suggested that children of mothers exposed during pregnancy to high levels of electromagnetic fields ($\geq 0.4 \mu\text{T}$) have a moderately increased risk of ALL ([Feychting et al., 2000], [Infante-Rivard and Deadman, 2003] and [Pearce et al., 2007]).

No study of this type has yet been carried out in Spain. However, exposure to ELF-EMR in Spanish schools was assessed by Tardón et al. (2002) in the cities of Oviedo and Barcelona and by Paniagua et al. (2002) in the region of Extremadura. Both groups measured 24-h exposure levels (in classrooms, playground, etc.) and determined the distance from power lines. Mean exposure levels were similar in both cities (0.016 μT for Barcelona and 0.015 μT for Oviedo), with higher maximum exposure levels in Barcelona (0.057 μT) than in Oviedo (0.017 μT). Values never exceeded 0.3 μT in Oviedo but were much higher in three sites in Barcelona (0.62 μT , 0.49 μT , and 0.43 μT). With regard to sources of exposure, no transmission lines were located within the study area in four of the schools selected, whereas lines ran along the walls of classrooms in two other schools and were underground (at depth of 50 cm) in others. Paniagua et al. (2002) found generally low values in classrooms, offices, and leisure areas, but reported a mean value of 1.17 μT and maximum value of 37 μT in laboratories, which may be attributable to the presence of computers. Similar findings were described by Tardón et al. (2002). EMR levels found in the laboratories were similar to values estimated for professionally exposed workers (Merchant et al., 1994). These authors did not measure the daily total exposure and were therefore unable to estimate the proportion of exposure received by children during school hours. The authors concluded that proximity to power lines did not appear to be the main source of exposure to EMR and that the distribution of power transmission lines, underground cables, and other sources (e.g., transformers and electrical equipment) in the school may have significantly contributed to the children's exposure.

4.2. Non-ionizing radiation: radio frequency electromagnetic fields

Very few publications have investigated the effects on human health of exposure to NIR in the range of radio frequencies and microwaves, and most of these epidemiological studies had an ecological design, comparing leukemia incidence rates in different populations using aggregate data on exposure and disease, not individual data (Table 4).

Table 4. Radio frequency electromagnetic fields studies.

Authors	Country, study period, design	Exposure assessment	Exposure category	Outcome [95% CI]
Micholozzi et al. (2002)	Italy (Rome/Vatican) 1987-1999 Ecologic: 8 cases out of 49,656 inhabitants	Measurements of all transmissions (5-600 kW) Cumulative areas around the radio station.	RFDistances (0-2) kmDistances (0-4) kmDistances (0-6) kmDistances (0-8) kmDistances (0-10) km	SMR: 6.1 [0.40-27.5] SMR: 2.9 [0.7-7.6] SMR: 2.2 [1.0-4.1] SMR: 1.5 [0.7-2.7] SMR: 1.2 [0.6-2.3]
Hocking and Gordon (2003)	Australia (Sydney) Ecologic: 123 ALL cases	Measurements of RF (TV towers): In an inner ring (radius of 4 km) of 3 municipalities surrounding TV towers, compared with outer ring (radius between 4-12 km) of 6 municipalities surrounding TV towers	AMFMortality ratio inner ring compared with outer ring.	RR: 2.1 [1.1-4.0]
Ha et al. (2007)	South Korea 1993-1999 Case-control: 1928 cases and 3082 controls from the national medical insurance data system	31 towers of ≥ 20 kW power, operating 24 h/day	AMFDistance ≥ 20 km Distance ≤ 2 km All leukemia Lymphatic leukemia (continuous) Lymphatic leukemia (categorical) Second quartile Third quartile	OR: 2.15 [1.00-4.67] OR: 1.60 [0.69-3.72] OR: 1.39 [1.04-1.86] OR: 1.59 [1.19-2.11]

69/90

100526

Authors	Country, study period, design	Exposure assessment	Exposure category	Outcome [95% CI]
Merzenich et al. (2008)	West Germany 2005–2007 Case-control: 1959 cases from the German childhood cancer registry, and 5848 controls	High-power radio and TV broadcast towers, 1 AM and 8 FM/TV transmitters	Distances (10–15) km ALL Distances (0–<2) km Distances (2–6) km Distances (6–10) km AML Distances (0–<2) km Distances (2–6) km Distances (6–10) km ALL + AML Distances (0–<2) km Distances (2–6) km Distances (6–10) km	OR: 1.31 [0.80–2.15] OR: 0.82 [0.66–1.03] OR: 0.76 [0.63–0.91] OR: 0.19 [0.02–1.47] OR: 0.75 [0.45–1.24] OR: 1.00 [0.68–1.47] OR: 1.04 [0.65–1.67] OR: 0.81 [0.66–0.99] OR: 0.79 [0.67–0.93]

AMF: frequency modulated amplitude; AML: acute myeloid leukemia; ALL: acute lymphoblastic leukemia; SMR: standardized mortality ratio; RF: radio frequency.

In 2002, Michelozzi et al. analyzed the incidence of leukemia in the Vatican (Rome, Italy) between 1987 and 1999 and its relation to the Radio Vatican transmitter (Michelozzi et al., 2002). According to the 1991 census, 49,656 people lived within 10 km of the transmitter. The risk of childhood leukemia was higher for children living within 6 km (standardized incidence rate, SMR 2.2; 95% CI 1.0–4.1), observing a significant decline in risk with increasing distance ($p = 0.036$). The authors also reported an association between survival and the distance from the transmitter. However, the results of this study were limited by the small number of cases included ($n = 8$) and the absence of other exposure data.

Hocking and Gordon (2003) studied the incidence of childhood leukemia as a function of the distance from TV transmitters, finding an increased risk of leukemia in children living within a radius of less than 4 km compared with those living in a radius of 4 to 12 km. The mortality rate ratio between the inner and outer ring was 2.1 (95% CI 1.1–4.0), very similar to the findings reported by Michelozzi.

A case-control study in South Korea (Ha et al., 2007) examined exposure to amplitude modulated (AM) radio frequency from 31 towers and 49 antennas, transmitting with a power ≥ 20 kW and operating 24 h/day. The study included 1928 cases of leukemia diagnosed between 1993 and 1999 and 3082 controls. The estimated cancer risk was adjusted for socioeconomic status, area of residence, and population density. The risk of leukemia was higher in communities with low population density or lower economic status, but the differences were not statistically significant. The risk was significantly greater for children living within 2 km of AM source in comparison to those residing at distances >20 km (OR: 2.15; 95% CI 1.00–4.67). However, they did not find an increased risk of leukemia with shorter distance from the source, a decrease in risk with increasing inverted quadratic distance, or the linear dose-response relationship suggested by Michelozzi et al. (2002). When the analysis was conducted separately for ALL and acute myeloid leukemia (AML), the total exposure to RF showed only a significant association with ALL ($p = 0.06$) but not with AML. Children were classified according to the exposure received, and those receiving a higher level of exposure had an increased risk of ALL, with an OR of 1.39 (95% CI 1.04–1.86) for the second quartile and of 1.59 (95% CI 1.19–2.11) for the third quartile in comparison to the first quartile.

Between 2005 and 2007, Merzenich et al. performed a case-control study in Germany, including 1959 cases (diagnosed between 1984 and 2003) and 5848 matched controls. The most frequent diagnosis among the cases was ALL (81.0%) (Merzenich et al., 2008). They investigated the distribution of RF (dB [microvolts/m]) from all radio/TV stations a year before diagnosis and the distance between the residence of study subjects and the nearest station. They compared individuals residing ≤ 2 km distant and at 30 km from a radio/TV station and found that the distance was an important determinant of exposure to RF EMR, with a considerable variation in exposure from 85 dB to >120 dB (microvolts/m) at intermediate distances (20 km). However, the authors found no significant difference in the risk for any type of leukemia as a function of the distance from either AM or FM/TV transmitters.

5. Discussion

Most of the studies reviewed here found an association between exposure to EMR and the risk of childhood leukemia, although statistical significance was not always reached. Taking into account exposure values of $\geq 0.3 \mu T$, the risk of cancer is significant and greater than 1 in the majority of datasets analyzed in our review (Table 3), despite the wide interval ranges in some of them. In fact, half ($n = 20$) of these datasets show statistically significant increases and none yields a statistically significant decrease. Hence, according to this epidemiological evidence, there is an increased risk of leukemia in children exposed to low electromagnetic fields of $\geq 0.3 \mu T$. However, the authors that detected an association generally attributed it to confounding factors, potential biases, misclassifications of exposure, or simple chance. For this reason, no definitive conclusions can be drawn at this point in time.

Given the uncertainty on this issue, it seems appropriate to clarify some key aspects of the research to date. Different methods have been used to estimate exposure. Some authors measured exposure indirectly, by using the original wire code method developed by Wertheimer and Leeper (1979) or a modification ([Leeper et al., 1991] and [Savitz and Kaune, 1993]). Their results have not significantly

70/90

100526

differed from those obtained by direct methods, e.g., 24-h spot measurements ([Thomas et al., 1999] and [Angelillo and Villari, 1999]).

Numerous researchers have claimed that indirect measurements are susceptible to error and are not valid for the quantification of exposure to NIR ([Jones et al., 1993] and [Savitz and Poole, 2001]). Besides, the data used in many of the studies are from registers and censuses or are approximations derived from epidemiological questionnaires that are not always designed to establish the timing and duration of exposure or to collect data on potentially confounding variables (e.g., place of residence, occupation, and living habits). The use of questionnaires often leaves researchers at the mercy of the quality and validity of the questions and faces them with problems of legibility or absence of annotations. These drawbacks limit the possibility of obtaining reliable information and can produce misclassifications of exposure (Delgado Rodríguez and Palma Pérez, 2006). Variability in study populations and in epidemiological design may also contribute to the disparity in results to date (Delgado Rodríguez and Palma Pérez, 2006).

Hence, discrepancies among the conclusions of the studies in this review may also be influenced by confounding factors, selection bias, and misclassification ([ICNIRP (International Commission for Non-ionizing Radiation Protection), 2001], [Wartenberg, 2001], [Greenland, 2003], [Kheifets and Shimkhada, 2005], [43], [Kheifets and Oksuzyan, 2008] and [Schüz and Ahlbom, 2008]). It is difficult to identify confounding factors when knowledge of the etiology of the disease is incomplete, as in the case of childhood leukemia. Speculative hypotheses are usually proposed to explain away observations of an association between exposure to magnetic fields and childhood leukemia (Kheifets and Shimkhada, 2005). The fact that we live in a complex EMR environment with a multiplicity of exposures makes it especially complicated to interpret population studies on EMR. We are now exposed to all types of electrical pollution from various sources and at myriad frequencies, confounding our ability to assess the contribution of a single determinant. This adds to the value of research performed before the current ubiquitous electrical excess.

Misclassification of exposure may mask association with the disease or understate its magnitude ([ICNIRP (International Commission for Non-ionizing Radiation Protection), 2001] and [Greenland and Kheifets, 2006]). However, it is unlikely that only one design flaw has a consistent effect across studies and represents the sole explanation for the alleged association ([Wartenberg, 2001], [Kheifets and Oksuzyan, 2008] and [Schüz and Ahlbom, 2008]). Selection bias would be another factor to consider in the interpretation of these results. Most of the data suggesting an increased risk of childhood leukemia are usually based on relatively small numbers of exposed children, and some social levels have been underrepresented in the reviewed studies.

Investigations into the damage produced during the first years of life should consider exposure before and during pregnancy. Birth defects can result from genetic or epigenetic damage as well as from effects on the embryo or fetus, and both may be related to environmental exposure of the parent before conception or during the pregnancy ([Wu et al., 2007], [Cech et al., 2007] and [Leitgeb and Cech, 2008]). It is therefore critical for researchers to define *a priori* the type and "window" of exposure to be assessed. For example, maternal exposure to NIR during organogenesis has been proposed as a cause of defects observed in early life ([Feychting et al., 2000], [Lee et al., 2002], [Li et al., 2002], [Infante-Rivard and Deadman, 2003] and [Pearce et al., 2007]). It has also been suggested that exposure of the father may play a role in some congenital defects through epigenetic or genetic damage to germ cells (Jensen et al., 2004).

According to the WHO Experts' Committee of Electromagnetic Fields and Public Health (WHO: www.who.int/emf), between 1% and 4% of children in the world are exposed to magnetic fields above 0.4 μ T. Current exposure limits for the general public are based on known thermal effects, in the range of 100 μ T range at 50 Hz and higher frequencies (Table 2) (ICNIRP, 1998). Moreover, current safety levels are based on short-term or immediate effects, and cancers and other diseases can have a long latency period. However, the Spanish "Declaration of Alcalá" (2002) called for safety levels to be reviewed in the light of growing evidence of biological effects at lower levels that are not associated with an increase in temperature (Havas, 2002). The majority of the evidence comes from *in vitro* laboratory and animal studies, and is of very limited use for determining health risk (Ruiz-Gomez and Martinez-Morillo, 2009). Nevertheless, epidemiological evidence to date and the severity of the potential harm, especially to children, would argue in favor of application of the precautionary principle. Several reports have recommended use of the precautionary principle for these exposures ([Kundi et al., 2009] and [77]; International Commission for Electromagnetic Safety (ICEMS) 2008; Committee on Non-Ionizing Radiation Protection 2008; Sage et al. 2007). It is essential to achieve an international standardization of regulatory levels, supporting the adoption of preventive measures to reduce exposures and facilitating comparisons among countries.

Studies to date have not convincingly confirmed or ruled out an association between NIR and the risk of childhood leukemia. Methodological problems to be solved include the proper diagnostic classification of individuals and the estimated exposure to non-ionizing radiation, which may act through various mechanisms of action. There appears to be an urgent need to reconsider exposure limits for low frequency and static magnetic fields, based on sound epidemiological research into the relationship between exposure to NIR and adverse human health effects. In the meantime, it would be advisable to adopt a precautionary approach to NIR (RF, MW), limiting body exposure whenever possible and feasible. Further research on the effects of this radiation is required to improve the basis and reliability of the safety standards.

71/90

100526

In summary, the epidemiological evidence reviewed in this article reveals a consistent pattern of increased leukemia incidence in children exposed to low electromagnetic fields. This increase is pronounced in children exposed to fields greater than 0.3 μT but can also be observed in weaker fields. However, all of the studies in this area are affected by various confounding variables that make it difficult to conclusively establish a causal relationship at this juncture.

6. Possible future actions

- 1) Combined laboratory and epidemiological research is warranted to analyze DNA damage in exposed individuals and their offspring as a function of their exposure level, e.g., using COMET or micronucleus assays to study lymphocytes from these populations.
- 2) It would be useful to establish exposure profiles for different child populations by considering the timing of exposure, particularly during special windows of susceptibility (e.g. pregnancy) region, and the occupational exposure of parents. It would also be of interest to analyze the contribution of different sources to total NIR exposure and to examine differences in exposure for different days of the week, among other variables. Personal exposimeters have been recommended for the estimation of the exposure of populations.
- 3) It is important to investigate adverse effects to a lower level of exposure than is classically used for risk estimations ($\geq 0.3 \mu\text{T}$), comparing between groups with low and high exposure to NIR.
- 4) It is desirable to consider exposure to electromagnetic fields as a whole, simultaneously measuring electrical and magnetic fields. Most of the studies in the present review solely investigate magnetic fields, which may lead to an underestimation of exposure levels.

Acknowledgments

The authors are grateful to Richard Davies for editorial assistance. This research was supported by grants from the Spanish Ministry of Health (FIS PI080728), and the Spanish Ministry of Science and Innovation (Ramon y Cajal Program — for MFFC).

References

- Ager et al., 1965 E.A. Ager, L.M. Schuman, H.M. Wallace, A.B. Rosenfield and W.H. Gullen, An epidemiological study of childhood leukemia. *J Chronic Dis*, 18 (1965), pp. 113–132.
- Ahlborn et al., 2000 A. Ahlbom, N. Day, M. Feychting, E. Roman, J. Skinner and J. Dockerty, et al. A pooled analysis of magnetic fields and childhood leukaemia. *Br J Cancer*, 83 (2000), pp. 692–698.
- Angelillo and Villari, 1999 I.F. Angelillo and P. Villari, Residential exposure to electromagnetic fields and childhood leukaemia: a meta-analysis. *Bull World Health Organ*, 77 11 (1999), pp. 906–915.
- Binhi, 2008 V. Binhi, Do naturally occurring magnetic nanoparticles in the human body mediate increased risk of childhood leukaemia with EMF exposure?. *Int J Radiat Biol*, 84 7 (2008), pp. 569–579.
- Brain et al., 2003 J.D. Brain, R. Kavet, D.L. McCormick, C. Poole, L.B. Silverman and T.J. Smith, et al. Childhood leukemia: electric and magnetic fields as possible risk factors. *Environ Health Perspect*, 111 7 (2003), pp. 962–970.
- Carrubba and Marino, 2006 S. Carrubba and A.A. Marino, The effects of low-frequency environmental-strength electromagnetic fields on brain electrical activity: a critical review of the literature. *Electromagn Biol Med*, 27 (2008), pp. 83–101.
- Cech et al., 2007 R. Cech, N. Leitgeb and M. Padiaditis, Fetal exposure to low frequency electric and magnetic fields. *Phys Med Biol*, 52 (2007), pp. 879–888.
- Coulton et al., 2004 L.A. Coulton, P.A. Harris, A.T. Barker and A.G. Pockley, Effect of 50 Hz electromagnetic fields on the induction of heat-shock protein gene expression in human leukocytes. *Radiat Res*, 161 (2004), pp. 430–434.
- Declaración de Alcalá, 2002 Declaración de Alcalá. Contaminación electromagnética y salud. Alcalá de Henares, 2002.
- de Pomerai et al., 2003 D.I. de Pomerai, B. Smith, A. Dawe, K. North, T. Smith and D.B. Archer, et al. Microwave radiation can alter protein conformation without bulk heating. *FEBS Lett*, 543 (2003), pp. 93–97.
- del Vecchio et al., 2009 G. del Vecchio, A. Giuliani, M. Fernandez, P. Mesirca, F. Bersani and R. Pinto, et al. Continuous exposure to 900 MHz GSM-modulated EMF alters morphological maturation of neural cells. *Neurosci Lett*, 455 (2009), pp. 173–177.
- Delgado Rodríguez and Palma Pérez, 2006 M. Delgado Rodríguez and S. Palma Pérez, Aportaciones de la revisión sistemática y del metaanálisis a la salud pública. *Rev Esp Salud Pública*, 80 (2006), pp. 483–489.
- Diem et al., 2005 E. Diem, C. Schwarz, F. Adlkofer, O. Jahn and H. Rudiger, Non-thermal DANN breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro. *Mutat Res*, 583 (2005), pp. 178–183.

72/90

100526

Draper et al., 2005 G. Draper, I. Vincent, M.E. Kroll and J. Swanson, Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study. *Br Med J*, **330** (2005), pp. 1290–1294. 

Experts' Committee of Electromagnetic Fields and Public Health in our setting, 2001 Experts' Committee of Electromagnetic Fields and Public Health in our setting (2001 and 2003). Ministry of Health. Spain. 

Feizi and Arabi, 2007 A.A. Feizi and M.A. Arabi, Acute childhood leukemias and exposure to magnetic fields generated by high voltage overhead power lines: a risk factor in Iran. *Asian Pac J Cancer Prev*, **8** 1 (2007), pp. 69–72. 

Fernández et al., 2007 M.F. Fernández, B. Olmos, A. Granada, M.J. López-Espínosa, J.M. Molina-Molina and J.M. Fernández, et al. Human exposure to endocrine disrupting chemicals and prenatal risk factors for cryptorchidism and hypospadias: a nested case-control study. *Environ Health Perspect*, **115** Suppl 1 (2007), pp. 8–14. 

Feychting et al., 2000 M. Feychting, B. Floderus and A. Ahlbom, Parental occupational exposure to magnetic fields and childhood cancer (Sweden). *Cancer Causes Control*, **11** (2000), pp. 151–156. 

Focke et al., 2010 F. Focke, D. Schuermann, N. Kuster and P. Schar, DNA fragmentation in human fibroblasts under extremely low frequency electromagnetic field exposure. *Mutat Res*, **683** 1–2 (2010), pp. 74–83. 

Girgert et al., 2005 R. Girgert, H. Schimming, W. Korner, C. Grundker and V. Hanf, Induction of tamoxifene resistance in breast cancer by ELF electromagnetic fields. *Biochem Biophys Res Commun*, **336** (2005), pp. 1144–1149. 

Greenland and Kheifets, 2006 S. Greenland and L. Kheifets, Leukemia attributable to residential magnetic fields: results from analyses allowing for study biases. *Risk Anal*, **26** 2 (2006), pp. 471–482. 

Greenland, 2003 S. Greenland, The impact of prior distributions for uncontrolled confounding and response bias: a case study of the relation of wire codes and magnetic fields to childhood leukemia. *J Am Stat Assoc*, **98** (2003), pp. 47–54. 

Greenland et al., 2000 S. Greenland, A.R. Sheppard, W.T. Kaune, C. Poole and M.A. Kelsh, A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Childhood Leukemia—EMF Study Group. *Epidemiology*, **11** (2000), pp. 624–634. 

Ha et al., 2007 M. Ha, H. Im, M. Lee, B.C. Kim, Y.M. Gimm and J.K. Pack, Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. *Am J Epidemiol*, **166** (2007), pp. 270–279. 

Havas, 2002 M. Havas, Intensity of electric and magnetic fields from power lines within the business district of 60 Ontario communities. *Sci Total Environ*, **298** 1–3 (2002), pp. 183–206 21. 

Herberman, 2008 Herberman RB. Memorandum: Important Precautionary Advice Regarding Cell Phone Use, 2008. Available: http://www.post-gazette.com/downloads/20080722upci_cellphone_memo.pdf. 

Hocking and Gordon, 2003 B. Hocking and I. Gordon, Decreased survival for childhood leukemia in proximity to television towers. *Arch Environ Health*, **58** 9 (2003), pp. 560–564. 

IARC and Working Group on the Evaluation of Carcinogenic Risks to Humans, 2002 IARC and Working Group on the Evaluation of Carcinogenic Risks to Humans, Non ionizing radiation, Part 1: static and extremely low-frequency (ELF) electric and magnetic fields, IARC Press, Lyon (2002). 

ICNIRP (International Commission for Non- Ionizing Radiation Protection), 2001 ICNIRP (International Commission for Non- Ionizing Radiation Protection), Review of the epidemiologic literature on EMF and health, (2001). 

ICNIRP, 1998 ICNIRP, Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). *Health Phys*, **74** (1998), pp. 494–522. 

IEEE (Institute of Electrical & Electronics Engineers), 1992 IEEE (Institute of Electrical and Electronics Engineers), Inc. Section 4.2 of "IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz," ANSI/IEEE C95.1-1992. New York, NY 10017. 

Infante-Rivard and Deadman, 2003 C. Infante-Rivard and J.E. Deadman, Maternal occupational exposure to extremely low frequency magnetic fields during pregnancy and childhood leukemia. *Epidemiology*, **14** (2003), pp. 437–441. 

Ivancsits et al., 2002 S. Ivancsits, E. Diem, A. Pilger, H.W. Rüdiger and J. Oswald, Induction of DNA strand breaks by intermittent exposure to extremely-low-frequency electromagnetic fields in human diploid fibroblasts. *Mutat Res*, **519** (2002), pp. 1–13. 

Jensen et al., 2004 M. Jensen, H. Leffers, J.H. Petersen, A. Nyboe Andersen, N. Jørgensen and E. Carlsen, et al. Frequent polymorphism of the mitochondrial DNA polymerase gamma gene (POLG) in patients with normal spermograms and unexplained subfertility. *Hum Reprod*, **19** (2004), pp. 65–70. 

73/90

100526

Jones et al., 1993 I.L. Jones, C.H. Shih, D.H. Hurston, B.J. Ware and P. Cole, Selection bias from differential residential mobility as an explanation for associations of wire codes with childhood cancer. *J Clin Epidemiol*, 46 6 (1993), pp. 545–548. 

Juutilainen et al., 2006 J. Juutilainen, T. Kumlin and J. Naarala, Do extremely low frequency magnetic fields enhance the effects of environmental carcinogens? A meta-analysis of experimental studies. *Int J Radiat Biol*, 82 (2006), pp. 1–12. 

Kabuto et al., 2006 M. Kabuto, H. Nitta, S. Yamamoto, N. Yamaguchi, S. Akiba and Y. Honda, et al. Childhood leukemia and magnetic fields in Japan: a case-control study of childhood leukemia and residential power-frequency magnetic fields in Japan. *Int J Cancer*, 119 (2006), pp. 643–650. 

Kavel and Zaffanella, 2002 R. Kavel and L.E. Zaffanella, Contact voltage measured in residences: implications to the association between magnetic fields and childhood leukemia. *Bioelectromagnetics*, 23 (2002), pp. 464–474. 

Kheifets and Oksuzyan, 2008 L. Kheifets and S. Oksuzyan, Exposure assessment and other challenges in nonionizing radiation studies of childhood leukaemia. *Radiat Prot Dosim*, 132 2 (2008), pp. 139–147. 

Kheifets et al., 2006 L. Kheifets, A.A. Afifi and R. Shimkhada, Public health impact of extremely low-frequency electromagnetic fields. *Environ Health Perspect*, 114 (2006), pp. 1532–1537. 

Kheifets and Shimkhada, 2005 L. Kheifets and R. Shimkhada, Childhood leukemia and EMF: review of the epidemiologic evidence. *Bioelectromagnetics*, 7 (2005), pp. S51–S59. 

Koifman, 1993 S. Koifman, Electromagnetic fields: a cancer promoter?. *Med Hypotheses*, 41 (1993), pp. 23–27. 

Kundi et al., 2009 M. Kundi, L. Hardell, C. Sage and E. Sobel, Electromagnetic fields and the precautionary principle. *Environ Health Perspect*, 117 11 (2009), pp. A484–A485. 

Kundi, 2007 Kundi M. Evidence for childhood cancer (leukemia). Prepared for the BioInitiative Working Group. 2007. www.bioinitiative.org/report/docs/section_10.pdf. 

Lee et al., 2002 G.M. Lee, R.R. Neutra, L. Hristova, M. Yost and R.A. Hiatt, A nested case-control study of residential and personal magnetic field measures and miscarriages. *Epidemiology*, 13 1 (2002), pp. 21–31. 

Leeper et al., 1991 E. Leeper, N. Wertheimer, D.A. Savitz, F.A. Barnes and H. Wachtel, Modification of the 1979 Denver wire code for different wire or plumbing types. *Bioelectromagnetics*, 12 (1991), pp. 314–318. 

Leitgeb and Cech, 2008 N. Leitgeb and R. Cech, Dosimetric assessment of simultaneous exposure to ELF electric and magnetic fields. *IEEE Trans Biomed Eng*, 55 (2008), pp. 671–674 2Pt 1. 

Li et al., 2002 D.K. Li, R. Odouli, S. Wi, T. Janevic, I. Golditch and D.T. Bracken, et al. A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of miscarriage. *Epidemiology*, 13 1 (2002), pp. 9–20. 

Maslanyj et al., 2007 M.P. Maslanyj, T.J. Mee, D.C. Renew, J. Simpson, P. Ansell and S.G. Allen, et al. Investigation of the sources of residential power frequency magnetic field exposure in the UK Childhood Cancer Study. *J Radiol Prot*, 27 (2007), pp. 41–58. 

McNally and Parker, 2006 R.J. McNally and L. Parker, Environmental factors and childhood acute leukemias and lymphomas. *Leuk Lymphoma*, 47 4 (2006), pp. 583–598. 

Mejia-Arangure et al., 2007 J.M. Mejia-Arangure, A. Fajardo-Gutierrez, M.L. Perez-Saldivar, C. Gorodezky, A. Martinez-Avalos and L. Romero-Guzman, et al. Magnetic fields and acute leukemia in children with Down syndrome. *Epidemiology*, 18 (2007), pp. 158–161. 

Merchant et al., 1994 C.J. Merchant, D.C. Renew and J. Swanson, Occupational exposures to power-frequency magnetic fields in the electricity supply industry. *J Radiol Prot*, 14 2 (1994), pp. 155–164. 

Merzenich et al., 2008 H. Merzenich, S. Schmiedel, S. Bennack, H. Brüggemeyer, J. Philipp and M. Blettner, et al. Childhood leukemia in relation to radio frequency electromagnetic fields in the vicinity of television and radio broadcast transmitters. *Am J Epidemiol*, 168 10 (2008), pp. 1169–1178. 

Michelozzi et al., 2002 P. Michelozzi, A. Capon, U. Kirchmayer, F. Forastiere, A. Biggeri and A. Barca, et al. Adult and childhood leukemia near a high-power radio station in Rome, Italy. *Am J Epidemiol*, 155 (2002), pp. 1096–1103. 

Milham and Ossiander, 2001 S. Milham and E.M. Ossiander, Historical evidence that residential electrification caused the emergence of the childhood leukemia peak. *Med Hypotheses*, 56 (2001), pp. 290–295. 

Paulraj and Behari, 2006 R. Paulraj and J. Behari, Single strand DNA breaks in rat brain cells exposed to microwave radiation. *Mutat Res*, 596 1–2 (2006), pp. 76–80. 

74/90

100526

- Paniagua et al., 2004 J.M. Paniagua, A. Jimenez, M. Huto and A. Antolin, Exposure assessment of ELF-magnetic fields in urban environments in Extremadura (Spain). *Bioelectromagnetics*, 25 (2004), pp. 58–62. ✎
- Paniagua et al., 2002 J.M. Paniagua, A. Jiménez and M. Rufo, Exposición a campos magnéticos en el ambiente educativo. *Rev Esp Fis*, 16 4 (2002), pp. 40–43. ✎
- Pearce et al., 2007 M.S. Pearce, D.M. Hammal, D. Tevfik, J.Q. Richard and Parker L. McNally, Paternal occupational exposure to electro-magnetic fields as a risk factor for cancer in children and young adults: a case-control study from the north of England. *Pediatr Blood Cancer*, 49 (2007), pp. 280–286. ✎
- Ramón et al., 2005 R. Ramón, F. Ballester, M. Rebagliato, N. Ribas, M. Torrent and M. Fernández, et al. Red INMA. The environment and childhood research network ("INMA" network): study protocol. *Rev Esp Salud Publica*, 79 2 (2005), pp. 203–220. ✎
- Ruiz-Gómez and Martínez-Morillo, 2009 M.J. Ruiz-Gómez, M. Martínez and Morillo, Electromagnetic fields and the induction of DNA strand breaks, . *Electromagn Biol Med*, 28 2 (2009), pp. 201–214 Review. ✎
- Sage et al., 2007 Sage C, Carpenter D, BioInitiative Working Group. BioInitiative Report: A Rationale for a Biological-based Public Exposure Standard for Electromagnetic Fields (ELF and RF), 2007. Available: <http://www.bioinitiative.org>. ✎
- Savitz and Kaune, 1993 D.A. Savitz and W.T. Kaune, Childhood cancer in relation to a modified residential wire code. *Environ Health Perspect*, 101 (1993), pp. 76–80. ✎
- Savitz and Poole, 2001 D.A. Savitz and C. Poole, Do studies of wire code and childhood leukemia point towards or away from magnetic fields as the causal agent?. *Bioelectromagnetics*, 5 (2001), pp. S69–S85. ✎
- Schüz and Ahlbom, 2008 J. Schüz and A. Ahlbom, Exposure to electromagnetic fields and the risk of childhood leukaemia: a review. *Radiat Prot Dosim*, (2008), pp. 1–10. ✎
- Schüz, 2007 J. Schüz, Implications from epidemiologic studies on magnetic fields and the risk of childhood leukemia on protection guidelines. *Health Phys*, 92 (2007), pp. 642–648. ✎
- Schüz et al., 2001 J. Schüz, J.P. Grigat, K. Brinkmann and J. Michaelis, Residential magnetic fields as a risk factor for acute childhood leukemia: results from a German population-based case-control study. *Int J Cancer*, 91 (2001), pp. 728–735. ✎
- Straume et al., 2008 A. Straume, A. Johnsson and G. Oftedal, ELF-magnetic flux densities measured in a city environment in summer and winter. *Bioelectromagnetics*, 29 (2008), pp. 20–28. ✎
- Tardón et al., 2002 A. Tardón, H. Velarde, P. Rodríguez, S. Moreno, M. Raton and J. Muñoz, et al. Exposure to extremely low frequency magnetic fields among primary school children in Spain. *J Epidemiol Community Health*, 56 (2002), pp. 432–433. ✎
- Thomas et al., 1999 D.C. Thomas, J.D. Bowman, L. Jiang, F. Jiang and J.M. Peters, Residential magnetic fields predicted from wiring configurations: II. Relationships to childhood leukemia. *Bioelectromagnetics*, 20 (1999), pp. 414–422. ✎
- UKCCS (UK Childhood Cancer Study Investigators), 1999 UKCCS (UK Childhood Cancer Study Investigators), Exposure to power-frequency magnetic fields and the risk of childhood cancer. *Lancet*, 354 (1999), pp. 1925–1931. ✎
- Valentini et al., 2007 E. Valentini, G. Curcio, F. Moroni, M. Ferrara, L. De Gennaro and M. Bertini, Neurophysiological effects of mobile phone electromagnetic fields on humans: a comprehensive review. *Bioelectromagnetics*, 28 (2007), pp. 415–432. ✎
- Wartenberg, 2001 D. Wartenberg, The potential impact of bias in studies of residential exposure to magnetic fields and childhood leukemia. *Bioelectromagnetics*, 5 (2001), pp. S32–S47. ✎
- Wertheimer and Leeper, 1979 N. Wertheimer and E. Leeper, Electrical wiring configurations and childhood cancer. *Am J Epidemiol*, 109 (1979), pp. 273–284. ✎
- WHO WHO (World Health Organization). www.who.int/erf. ✎
- Wolf et al., 2005 F.I. Wolf, A. Torsello, B. Tedesco, S. Fasanella, A. Boninsegna, M. D'Ascenzo, C. Grassi, GB Azzena and A. Cittadini, 50-Hz extremely low frequency electromagnetic fields enhance cell proliferation and DNA damage: possible involvement of a redox mechanism. *Biochim Biophys Acta*, 1743 1–2 (2005 Mar 22), pp. 120–129. ✎
- Wu et al., 2007 D. Wu, R. Qiang, J. Chen, S. Seidman, D. Witters and W. Kainz, Possible overexposure of pregnant women to emissions from a walk through metal detector. *Phys Med Biol*, 52 (2007), pp. 5735–5748. ✎

✎ Corresponding author. Department of Radiology. University of Granada, 18071-Granada, Spain. Tel.: +34 958 24 2077; fax: +34 958 249953.

75/90

100526

Copyright © 2010 Elsevier B.V. All rights reserved.

Science of The Total Environment
Volume 408, Issue 16, 15 July 2010, Pages 3062-3069

[Home](#) [Browse](#) [Search](#) [My settings](#) [My alerts](#)

[Help](#)

[About ScienceDirect](#)
[What is ScienceDirect](#)
[Content details](#)
[Set up](#)
[How to use](#)
[Subscriptions](#)
[Developers](#)

[Contact and Support](#)
[Contact and Support](#)

[About Elsevier](#)
[About Elsevier](#)
[About SciVerse](#)
[About SciVal](#)
[Terms and Conditions](#)
[Privacy policy](#)
[Information for advertisers](#)



Copyright © 2011 Elsevier B.V. All rights reserved. SciVerse® is a registered trademark of Elsevier Properties S.A., used under license. ScienceDirect® is a registered trademark of Elsevier B.V.

76/90



RESEARCH

Open Access

Risk of hematological malignancies associated with magnetic fields exposure from power lines: a case-control study in two municipalities of northern Italy

Carlotta Malagoli¹, Sara Fabbi², Sergio Teggi², Mariagiulia Calzari³, Maurizio Poli⁴, Elena Ballotti⁴, Barbara Notari⁵, Maurizio Bruni⁵, Giovanni Palazzi⁶, Paolo Paolucci⁶, Marco Vinceti^{1*}

Abstract

Background: Some epidemiologic studies have suggested an association between electromagnetic field exposure induced by high voltage power lines and childhood leukemia, but null results have also been yielded and the possibility of bias due to unmeasured confounders has been suggested.

Methods: We studied this relation in the Modena and Reggio Emilia municipalities of northern Italy, identifying the corridors along high voltage power lines with calculated magnetic field intensity in the 0.1- $<$ 0.2, 0.2- $<$ 0.4, and \geq 0.4 microTesla ranges. We identified 64 cases of newly-diagnosed hematological malignancies in children aged $<$ 14 within these municipalities from 1986 to 2007, and we sampled four matched controls for each case, collecting information on historical residence and parental socioeconomic status of these subjects.

Results: Relative risk of leukemia associated with antecedent residence in the area with exposure \geq 0.1 microTesla was 3.2 (6.7 adjusting for socioeconomic status), but this estimate was statistically very unstable, its 95% confidence interval being 0.4-23.4, and no indication of a dose-response relation emerged. Relative risk for acute lymphoblastic leukemia was 5.3 (95% confidence interval 0.7-43.5), while there was no increased risk for the other hematological malignancies.

Conclusions: Though the number of exposed children in this study was too low to allow firm conclusions, results were more suggestive of an excess risk of leukemia among exposed children than of a null relation.

Background

Since the original observation by Wertheimer and Leeper in 1979 [1], several epidemiologic studies have suggested an association between magnetic fields exposure [2,3], such as that induced by residence near high voltage power lines, and childhood leukemia, but some investigations yielded null results and the possibility of bias induced by unmeasured confounders or inappropriate exposure assessment in the former studies has been suggested [4-6]. Moreover, no consensus exists about the cutpoints of magnetic field exposure above which

leukemia risk might actually increase, and little evidence is available on a relation with risk of other hematological malignancies in childhood, suggesting the need for new methodological approaches to this issue of primary public health importance [7,8].

We investigated the possible association between magnetic fields exposure and risk of leukemia and other hematological cancers in the pediatric population of two municipalities in northern Italy, taking into consideration parental socioeconomic status as a potential confounder.

Methods

Study population

We attempted to identify all cases of hematological cancers newly-diagnosed during the 1986-2007 period in

* Correspondence: marco.vinceti@unimore.it

¹CREAGEN - Environmental, Genetic and Nutritional Epidemiology Research Center, Department of Public Health Sciences, University of Modena and Reggio Emilia, via Campi 287, 41125 Modena, Italy

100526

children while residing in the two northern Italy municipalities of Modena e Reggio Emilia (around 180,000 and 160,000 inhabitants in 2007, respectively). To do so, we used the nation-wide hospital-based registry of childhood malignancies managed by the Associazione Italiana Ematologia Oncologia Pediatrica (AIEOP) [9]. The AIEOP Registry, operating since 1985 and based on collaboration with 54 specialized hospital centers in Italy belonging to this network, has to date registered 35,256 newly diagnosed patients up to the age of 14. However, the physicians were aware that patients aged 14 were likely to be referred to the Hematological Departments usually taking care of adults: we therefore decided to limit the analysis to the subjects aged 0-13 to warrant a population-based design in patients' selection, removing two subjects from the AIEOP database (one with acute acute lymphoblastic leukemia and the other with non-Hodgkin's lymphoma) and consequently from further analysis. We randomly selected for each case four controls from the general population, by extracting these referents from the municipal databases of residents matched for year of birth, sex and municipality of residence to each case, and using historical databases of residents according to the year of diagnosis. We then reconstructed with the directories of the General Registry Offices the residential history of cases and controls, starting from date of birth, if the child had always resided in the municipality, or from the date of immigration into the municipality when appropriate. In case of uncertain data, we contacted family doctors, or when necessary the families directly. Information about paternal and maternal educational attainment level and paternal income was also collected from the General Registry Offices and through web-available resources.

Exposure assessment

We identified the high voltage power lines (≥ 132 kilovolts) located during the 1986-2007 period in the municipal territories of Modena and Reggio Emilia, whose code, average current intensity and voltage are reported in table 1. We then calculated magnetic field induction in the proximity of these lines using two models, CAMPI <http://www.ifac.cnr.it/pcemni/manuale.pdf> and EFC400 <http://www.fgeu.de/html/demos.htm>, to define the distance at which, at a height of 8 m, the magnetic fields intensity cutpoints of 0.1, 0.2 and 0.4 microTesla (μT) occurred. CAMPI is a freeware software simulation package developed by Andreuccetti at the Florence National Research Council Applied Physics Institute, to predict the intensity of the magnetic flux density (B) generated by power lines. The program is based on a 2D model, where the power line is represented with a set of straight, horizontal, infinite and parallel conductors. The ground is considered as not affecting the

magnetic field and interactions with obstacles (towers, trees or buildings) placed along the line are also neglected. Calculation of the B of each conductor is based on the application of the Biot-Savart formula and therefore, resulting directly proportional to the intensity of the current flowing in the conductor and inversely proportional to the distance between the conductor and the point where the field is computed. In the few cases of vicinity between two or more power lines, we used the alternative model generated by commercial EFC400 software to estimate magnetic fields distribution in these complex situations. EFC-400 is a commercial software simulation package developed by FGEU mbH (ForschungsGesellschaft für Energie und Umwelttechnologie mbH, Berlin) to predict the intensity of the magnetic flux density generated by power lines in a 3D model. The algorithm used is the same as the CAMPI model, but the conductors are divided into a certain numbers of portions (up to 20) which are considered as single sources of magnetic field, to calculate overall resulting B in prediction points. In this way EFC-400 is able to simulate complex situations, like closing, crossing, curved and finite electric lines. Both models have been validated in a survey which confirmed that both models carefully predicted the magnetic field distribution detected through on-site measurements around a high voltage (132 kilovolts) power line in the bordering Bologna province [10]. For both CAMPI and EFC400 models, we considered the characteristics and the disposition of conductors of each power line, and we used the average current run in the power lines during 2001, which is the earliest year datum made available by the regional environmental protection agency, ARPA, due to mandatory reporting of currents by the electric companies in compliance with the Emilia-Romagna Region rule 'Direttiva 197/2001'. We thus identified three 'exposed' corridors ($0.1 \leq B < 0.2$, $0.2 \leq B < 0.4$, $B \geq 0.4 \mu\text{T}$) surrounding the power lines, whose distance from the conductors is reported in table 1 along which the main technical characteristics of the power lines crossing the two study municipalities. Historical analysis of current consumption and requirements over the 1986-2007 period according to national electric company data http://www.terna.it/default/home/sistema_elettrico/statistiche.aspx indicated that the currents run in power lines in the Modena and Reggio Emilia provinces increased over time of about 3% on a yearly basis, and that the 2001 values were respectively 13.5% and 13.3% higher than the average values of the whole period.

We included the exposure corridors in a Geographical Information System (GIS), using ARC-GIS software (version 9.2, ESRI, Redlands, CA 2006), and we geocoded the historical residences of all study subjects in this GIS database. To do so, we identified the centroid

78/90

Table 1 Distance of exposure corridors from conductors (at calculated magnetic fields cutpoints of 0.1, 0.2 and 0.4 μ T along the high voltage power lines in Modena and Reggio Emilia municipalities

Modena municipality						
Line Code	Voltage (kilovolts)	Electric current (amperes, average at 2001)	Distance from power line (m)			
			0.1 μ T	0.2 μ T	0.4 μ T	
175	132	415	94	67	48	
176	132	128	52	37	27	
176/686	132	238	88	62	44	
686	132	238	59	42	30	
600	132	378	74	53	38	
175/600	132	415	108	77	54	
614	132	110	61	43	30	
615	132	95	40	28	20	
625	132	80	35	25	18	
634	132	121	45	32	22	
638	132	31	23	16	11	
688	132	360	88	63	45	
631/632	132	94	52	36	26	
300	380	273	84	60	43	
320	380	414	104	73	52	
395	380	285	86	61	44	
300/320	380	414	89	67	51	
BO 017	132	10	14	10	7	
BO 018	132	14	15	11	8	
Reggio Emilia municipality						
Line Code	Voltage (kilovolts)	Electric current (amperes, average at 2001)	Distance from power line (m)			
			0.1 μ T	0.2 μ T	0.4 μ T	
103	132	21	18	13	9	
660	132	88	35	25	18	
698	132	158	45	32	23	
315	380	404	99	72	51	
683	132	107	39	28	20	
104	132	91	44	32	23	
642	132	63	29	20	14	
656	132	41	30	22	16	
659	132	170	60	42	30	
668	132	34	20	14	10	
677	132	60	29	20	15	
685	132	233	85	60	43	
RFI-BO PR	132	25	20	14	10	

of the exact address of each subject's residence on the ARC-GIS municipal maps: when the building was not found in the files of Modena and Reggio Emilia municipalities, as occurred in 23 locations, we directly measured the coordinates on site using a global positioning system device (Garmin GPSmap 60CSx, Garmin Int. Corp., Olathe, KS). We eventually defined study subjects as 'exposed' if they had resided for at least six months in one of the exposed corridors, providing that the power lines were actually in operation. However, the six months prior to the date of diagnosis for cases and the

corresponding period for their matched controls were excluded from this analysis.

Data analysis

We calculated the relative risks (RR) with their 95% confidence interval (CI) of hematological cancers and more specifically of all leukemias, acute lymphoblastic leukemia and non-leukemic malignancies associated with antecedent exposure to magnetic fields by calculating the odds ratio of the disease in conditional and unconditional logistic regression models, using the

STATA statistical package (Stata version 10.1 for Windows, Stata Corporation, TX 2009). We also repeated the analysis adjusting for three potential confounders, paternal educational attainment level, maternal educational attainment level, and paternal income. Statistical precision of the risk estimates was evaluated by computing their 95% confidence limits.

Results

We identified 64 cases of childhood hematological malignancies newly-diagnosed during the study period (table 2), to whom we matched 256 population controls randomly drawn from the general populations. Of these diseases, 46 were cases of leukemia, 36 of which were of the acute lymphoblastic leukemia subtype, and the remaining 18 cases were included in three different nosological entities. Data about parental socioeconomic status of case and control children are reported in table 3.

Two cases (all affected by acute lymphoblastic leukemia), diagnosed in 2003 and in 2006, and five controls had been exposed to electromagnetic fields from power lines, since they had resided for more than six months in one of the exposed corridors. None of these subjects had resided for less than six months in one of the three exposed corridors before diagnosis, nor had been residing in more than one of these corridors. All of the exposed children had

been continuously residing in one of these corridors for more than one year, starting from 13 months up to 6.5 years. One case and three control children had been residing in the corridor with the 0.1- $<$ 0.2 μ T range of exposure, while the other case and the remaining two controls resided in the corridor with exposure \geq 0.4 μ T.

The risk of hematological malignancies associated with antecedent exposure to magnetic fields from power lines was 1.7 (95% CI 0.3-9.4) in the unadjusted analysis and 2.4 (95% CI 0.4-15.0) after adjusting for socioeconomic status (table 4). Limiting the analysis to leukemia cases and matched controls, the relative risk associated to magnetic field exposure was 3.2 (95% CI 0.4-23.4), which further increased to 6.7 (95% CI 0.6-78.3) after adjusting for socioeconomic status indicators. Corresponding figures for the acute lymphoblastic leukemia were 6.0 (95% CI 0.5-69.5) for crude analysis and 5.3 (95% CI 0.7-43.5) for the multivariate estimate, which, however, could only be computed in an unconditional logistic regression model. Conversely, we could not obtain a valid RR estimate for the hematological malignancies other than leukemia overall considered, a category for which we did not identify any exposed case.

Limiting the analysis of exposed children to those with highest exposure, i.e. \geq 0.4 μ T, RR, overall risk for the malignancies considered showed a slight increase

Table 2 Classification of childhood hematological malignancies diagnosed in the Modena and Reggio Emilia municipal populations from 1986 to 2007

Diagnosis	ICD-9 ^a	n	(%)
All malignant neoplasms of lymphatic and hematopoietic tissue	200-208	64	(100.0)
Lymphosarcoma and reticulosarcoma and other specified malignant tumors of lymphatic tissue			
Lymphosarcoma	200.1	6	(9.3)
Burkitt's tumor or lymphoma	200.2	2	(3.1)
Hodgkin's disease			
Lymphocytic-histiocytic predominance	201.4	1	(1.6)
Nodular sclerosis	201.5	1	(1.6)
Mixed cellularity	201.6	1	(1.6)
Hodgkin's disease, unspecified	201.9	2	(3.1)
Other malignant neoplasms of lymphoid and histiocytic tissue			
Letterer-Siwe disease	202.5	5	(7.8)
Lymphoid leukemia			
Acute lymphoid leukemia	204.0	36	(56.2)
Chronic lymphoid leukemia	204.1	1	(1.6)
Unspecified lymphoid leukemia	204.9	1	(1.6)
Myeloid leukemia			
Acute myeloid leukemia	205.0	3	(4.7)
Chronic myeloid leukemia	205.1	1	(1.6)
Unspecified myeloid leukemia	205.9	1	(1.6)
Monocytic leukemia			
Acute monocytic leukemia	206.0	3	(4.7)

^a International Classification of Diseases, 9th edition

80/90

100526

Table 3 Parental socioeconomic status of children with newly-diagnosed hematological disease during the 1986-2007 period and their matched controls, Modena and Reggio Emilia municipalities, northern Italy

	All hematological malignancies		All leukemias		Acute lymphoblastic leukemia	
	Cases (n = 64) n (%)	Controls (n = 256) n (%)	Cases (n = 46) n (%)	Controls (n = 184) n (%)	Cases (n = 36) n (%)	Controls (n = 144) n (%)
<i>Residence</i>						
Modena	30(46.9)	120(46.9)	21(45.7)	84(45.7)	17(47.2)	68(47.2)
Reggio Emilia	34(53.1)	136(53.1)	25(54.3)	100(54.3)	19(52.8)	76(52.8)
<i>Gender</i>						
Male	35(54.7)	140(54.7)	24(52.2)	96(52.2)	19(52.8)	76(52.8)
Female	29(45.3)	116(45.3)	22(47.8)	88(47.8)	17(47.2)	68(47.2)
<i>Paternal income^a</i>						
0	0(0.0)	11(4.3)	0(0.0)	8(4.4)	0(0.0)	7(4.9)
>0-8	7(10.9)	23(9.0)	5(10.9)	17(9.2)	5(13.9)	13(9.0)
9-15	4(6.2)	26(10.2)	4(8.7)	18(9.8)	3(8.3)	14(9.7)
16-25	14(21.9)	50(19.5)	9(19.6)	35(19.0)	6(16.7)	27(18.7)
26-40	8(12.5)	45(17.6)	7(15.2)	31(16.9)	4(11.1)	27(18.7)
41-100	10(15.6)	47(18.4)	7(15.2)	32(17.4)	6(16.7)	26(18.1)
>100	1(1.6)	5(1.9)	1(2.2)	5(2.7)	1(2.8)	5(3.5)
Unknown	20(31.2)	49(19.1)	13(28.2)	38(20.6)	11(30.6)	25(17.4)
<i>Paternal educational attainment</i>						
<Primary school	0(0.0)	3(1.2)	0(0.0)	2(1.0)	0(0.0)	2(1.4)
Primary school	6(9.4)	20(7.8)	3(6.5)	16(8.7)	3(8.3)	14(9.7)
Middle school	21(32.8)	81(31.6)	14(30.4)	53(28.8)	11(30.6)	38(26.4)
High school	12(18.7)	86(33.6)	11(24.0)	63(34.3)	9(25.0)	51(35.4)
University	11(17.2)	40(15.6)	8(17.4)	30(16.3)	5(13.9)	25(17.4)
Unknown	14(21.9)	26(10.2)	10(21.7)	20(10.9)	8(22.2)	14(9.7)
<i>Maternal educational attainment</i>						
<Primary school	0(0.0)	2(1.8)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Primary school	4(6.2)	26(10.2)	3(6.5)	19(10.3)	3(8.3)	16(11.1)
Middle school	23(35.9)	98(38.3)	17(37.0)	73(39.7)	17(47.2)	55(38.3)
High school	17(26.6)	80(31.2)	13(28.3)	59(32.0)	7(19.4)	45(31.2)
University	8(12.5)	30(11.7)	7(15.2)	18(9.8)	4(11.1)	16(11.1)
Unknown	12(18.7)	20(7.8)	6(13.0)	15(8.2)	5(13.9)	12(8.3)

^a Thousands of euros/year 2005

compared with the previous analysis, but this was not true for leukemia or for acute lymphoblastic leukemia alone.

Discussion

We must acknowledge a major limitation of this study and which limits interpretation of our data, i.e. the low number of cases and in particular the limited number of exposed cases, thus yielding statistically unstable risk estimates and hampering the prospect of detecting small changes in risk and limiting the chance to assess possible dose-response relations. Another limitation of the study is the use of calculated magnetic fields based on power lines configuration and not on direct measurements of exposure. However, previous studies have shown that, particularly for children, this methodology tends to be substantially adequate in reflecting actual exposure to power lines-induced magnetic fields, and

that this source of exposure tends to exceed the contribution of other sources such as home appliances [11], particularly in the case of high voltage transmission lines [12]. Moreover, we collected in the present study the entire residential history of the study subjects, and therefore we were able to estimate their historical exposure status, while assessments based on single measurements (generally for cases of children's residence at diagnosis) might have led to substantial misclassification of exposure [13]. Finally, we obtained information from the Modena and Reggio Emilia municipalities about location of primary schools with reference to magnetic fields from power lines, to investigate this potential source of confounding. None of the schools were located in an area with magnetic field intensity $\geq 0.1 \mu\text{T}$ in Modena or $\geq 0.2 \mu\text{T}$ in Reggio Emilia (data were unavailable for the 0.1- <0.2 range in Reggio Emilia).

81/90

Table 4 Relative risk (RR) of childhood hematological disease associated with magnetic field exposure from power lines $\geq 0.1 \mu\text{T}$ and $\geq 0.4 \mu\text{T}$ in Modena and Reggio Emilia municipalities, 1986-2007^a

Disease		< 0.1 μT		$\geq 0.1 \mu\text{T}$			$\geq 0.4 \mu\text{T}$		
		cases/controls	RR	cases/controls	RR	95% CI ^b	cases/controls	RR	95% CI ^b
All hematological malignancies	Crude	62/251	1.00	2/5	1.7	0.3-9.4	1/2	2.4	0.1-42.6
	Adjusted ^c	36/184	1.00	2/5	2.4	0.4-15.0	1/2	2.8 ^d	0.2-34.3 ^d
All leukemias	Crude	44/181	1.00	2/3	3.2	0.4-23.4	1/2	2.4	0.1-42.6
	Adjusted ^c	27/129	1.00	2/3	6.7	0.6-78.3	1/2	2.1 ^d	0.2-26.2 ^d
Acute lymphoblastic leukemia	Crude	34/142	1.00	2/2	6.0	0.5-69.5	1/2	2.4	0.1-42.6
	Adjusted ^c	20/106	1.00	2/2	5.3 ^d	0.7-43.5 ^d	1/2	2.3 ^d	0.2-29.1 ^d
Hematological malignancies other than leukemia	Crude	18/70	1.00	0/2	- ^e	- ^e	0/0	- ^e	- ^e
	Adjusted ^c	9/55	1.00	0/2	- ^e	- ^e	0/0	- ^e	- ^e

^aRelative risk estimates calculated with conditional logistic regression, using exposure category < 0.1 μT as reference category

^b95% confidence interval

^cAdjusted for paternal educational attainment and income and for maternal educational attainment

^dRisk estimates computed with unconditional logistic regression, adjusting for age, sex, municipality, paternal educational attainment and income, and maternal educational attainment

^eNo statistically valid estimates yielded by both conditional and unconditional logistic regression

In this study we used a disease registry (the AIEOP Registry) which was hospital-based and not population-based, but we consider it unlikely that this feature of the study biased the study results. In fact, all children affected by hematological malignancies are expected to be referred to hospital and to highly-specialized centers in particular, all of which are included in the AIEOP network. Moreover, we verified the completeness of this registry for hematological cancers in the Reggio Emilia municipality by comparing its database with information collected in loco from several data sources (hospital discharge registry, drug prescriptions, family doctors, death certificates) [14]. All but one case was reported to the study registry, which in turn was an independent source for only one additional case.

To assess exposure of study subjects through the period 1986-2007 in this investigation, we defined exposure corridors through calculation of magnetic fields based on average currents run in the year 2001, the earliest available, which were nearly 13% higher than the average current in the whole study period, thus raising the risk of some misclassification of exposure. However, in this investigation, the two cases found to be exposed received their diagnosis after 2001, thus suggesting that the methodology we used did not overestimate exposure status of these children and did not spuriously increase the RR estimates in exposed subjects.

In our study population, we found a prevalence of exposure to magnetic fields $\geq 0.4 \mu\text{T}$ of 0.94%, higher than the available figures for the Italian population of 0.20-0.35% [8], but this is likely due not only to our limited sample size and the uncertainties of the national estimates (which were calculated in the

1990s when currents run in power lines were still not officially available), but also to the urban, densely populated setting examined in the present investigation.

This study has some strengths compared with other investigations carried out in this field. First, the entire residential history of study subjects was available, a characteristic which made it possible to investigate the lifetime exposure and to account for adequate induction and latent periods from exposure to the disease. Moreover, we could acquire all study data (residential histories and socioeconomic information) without directly contacting the families, thus avoiding the risk of recall and selection bias due to refusal to participate, and consequently the likelihood of generating inaccurate information. We were also able to adjust the analysis for three indicators of socioeconomic status including paternal income, thus reducing the risk of substantial confounding, though no obvious relation between socioeconomic status and leukemia risk appears to exist [15], and in the present study risk estimates did not substantially change after adjusting for these indicators. Finally, we selected the controls on the basis of the historical databases of municipal residents matched to years of diagnosis of cases, different to previous case-control studies where the referents were sampled from a single, recently available directory of residents, despite the long period of time during which disease diagnoses occurred in cases (even in the order of 15-20 years). In such investigations, general population residential distribution might have substantially changed over time with reference to proximity to power lines, thus biasing exposure status of controls.

Overall, study results showed an excess risk of leukemia, but not of other hematological malignancies, in children exposed to magnetic fields generated from power lines, and this finding appears in line with most studies carried out on this topic [6,8], though the statistical instability of risk estimates and the lack of dose-response relations do not allow for the alternative hypothesis of no effect on disease risk of magnetic fields exposure to be entirely ruled out. In fact, an increased childhood leukemia risk in the proximity of high voltage power lines was detected not only in a recent large case-control investigation [16] but also in two smaller studies carried out in Italy [17] and in Tasmania (courtesy of Ray Lowenthal, Deirdre Tuck and Isabelle Bray, unpublished data), suggesting a homogeneous trend across different populations. It should also be noted that recent laboratory studies have increased the biological plausibility of adverse health effects of extremely low frequency magnetic fields [18,19].

Conclusions

Findings of the present study, which are consistent with those of recent case-control investigations independently of their characteristics and sample size [6-8], appear to support the hypothesis that magnetic fields exposure increases the risk of childhood leukemia, though likely representing a risk factor for a large minority of cases, as expected on the basis of epidemiologic evidence [20].

Abbreviations

GIS: Geographical Information System; ARPA: Agenzia Regionale per la Protezione Ambientale; μ T: microTesla; B: magnetic flux density; AIEOP: Associazione Italiana Ematologia Oncologia Pediatrica; ICD-9: International Classification of Diseases, 9th Edition; RR: relative risk; CI: confidence interval.

Acknowledgements

We acknowledge the cooperation in the study of Carlo Lucenti of ENIA Reggio Emilia, Giulia Paltrinieri, Sonia Fiorini, Giovanni Bigi, Clara Fiandri, Walter Gheduzzi and Giuseppe Zini of Modena Municipality, Simona Poli, Simona Maggi and Elena Infanti of Reggio Emilia Municipality, Daniele Andreuccetti of the Italian National Research Council Institute of Applied Physics "Nello Carrara". This study was supported by Associazione Sostegno Ematologia Oncologia Pediatrica (ASEOP) onlus in Modena and by the Department of the Environment of Reggio Emilia Municipality.

Author details

¹CREAGEN - Environmental, Genetic and Nutritional Epidemiology Research Center, Department of Public Health Sciences, University of Modena and Reggio Emilia, via Campi 287, 41125 Modena, Italy. ²LARMA - Laboratory of Environmental Analysis, Surveying and Environmental Monitoring, Department of Mechanical and Civil Engineering, University of Modena and Reggio Emilia, via Vignolese 905, 41125 Modena, Italy. ³Local Health Unit of Reggio Emilia, via Amendola 2, 42122 Reggio Emilia, Italy. ⁴ARPA - Emilia Romagna Environmental Protection Agency, section of Reggio Emilia, via Amendola 2, 42122 Reggio Emilia, Italy. ⁵ARPA - Emilia Romagna Environmental Protection Agency, section of Modena, via Fontanelli 23, 41121 Modena, Italy. ⁶Department of Mother and Child, University of Modena and Reggio Emilia, via del Pozzo 71, 41124 Modena, Italy.

Authors' contributions

CM and MV conceived and coordinated the study, performed data analysis and drafted the manuscript; SF and ST designed and coordinated the GIS database; MP, EB, BN and MB collected information about power lines configuration and calculated magnetic fields around the lines; MC, GP and PP identified the incident cases. All authors have read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

Received: 16 December 2009 Accepted: 30 March 2010
Published: 30 March 2010

References

1. Wertheimer N, Leeper E: Electrical wiring configurations and childhood cancer. *Am J Epidemiol* 1979, **109**:273-284.
2. World Health Organization-International Agency for Research on Cancer: Non-Ionizing Radiation, Part 1: static and extremely low-frequency (ELF) electric and magnetic fields. Lyon: IARC Press 2002, **80**.
3. World Health Organization: Extremely low frequency fields. Geneva: World Health Organization 2007, **238**.
4. Greenland S, Sheppard AR, Kaune WT, Poole C, Kelsch MA: A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Childhood Leukemia-EMF Study Group. *Epidemiology* 2000, **11**:624-634.
5. Ahlbom A, Day N, Feychting M, Roman E, Skinner J, Dockerty J, Linet M, McBride M, Michaelis J, Olsen JH, et al: A pooled analysis of magnetic fields and childhood leukaemia. *Br J Cancer* 2000, **83**:692-698.
6. Schuz J, Svendsen AL, Linet MS, McBride ML, Roman E, Feychting M, Kheifets L, Lightfoot T, Mezei G, Simpson J, et al: Nighttime exposure to electromagnetic fields and childhood leukemia: an extended pooled analysis. *Am J Epidemiol* 2007, **166**:263-269.
7. Schuz J, Lagorio S, Bersani F: Electromagnetic fields and epidemiology: an overview inspired by the fourth course at the International School of Bioelectromagnetics. *Bioelectromagnetics* 2009, **30**:511-524.
8. Comba P, Fazzo L: Health effects of magnetic fields generated from power lines: new clues for an old puzzle. *Ann Ist Super Sanita* 2009, **45**:233-237.
9. Pession A, Dama E, Rondelli R, Magnani C, De Rosa M, Locatelli F, Fagioli F, Haupt R, Jankovic M, Terracini B, et al: Survival of children with cancer in Italy, 1989-98. A report from the hospital based registry of the Italian Association of Paediatric Haematology and Oncology (AIEOP). *Eur J Cancer* 2008, **44**:1282-1289.
10. Violanti S: EMF in urban areas, a study to assess low frequency fields in Bologna. *ARPA rivista* 2003, **1**:30-31 [http://www.arpa.emr.it/pubblicazioni/arpa_rivista/contenuto_riviste_140.asp].
11. Vistnes AI, Ramberg GB, Bjornevik LR, Tynes T, Haldorsen T: Exposure of children to residential magnetic fields in Norway: is proximity to power lines an adequate predictor of exposure? *Bioelectromagnetics* 1997, **18**:47-57.
12. Li CY, Mezei G, Sung FC, Silva M, Chen PC, Lee PC, Chen LM: Survey of residential extremely-low-frequency magnetic field exposure among children in Taiwan. *Environ Int* 2007, **33**:233-238.
13. Baris D, Linet MS, Tarone RE, Kleinerman RA, Hatch EE, Kaune WT, Robison LL, Lubin J, Wacholder S: Residential exposure to magnetic fields: an empirical examination of alternative measurement strategies. *Occup Environ Med* 1999, **56**:562-566.
14. Borciani N, Vinceti M, Avanzini P, Ilariucci F, Mangone L, Martini M, Merlin M, Ferretti A, Frassinetti M, Rodolfi R, et al: Health databases to assess the incidence of lymphoid malignancies in Italian population. *Epidemiol Prev* 2000, **24**:81-84.
15. Adam M, Rebholz CE, Egger M, Zwahlen M, Kuehni CE: Childhood leukaemia and socioeconomic status: what is the evidence? *Radiat Prot Dosimetry* 2008, **132**:246-254.
16. Draper G, Vincent T, Kroll ME, Swanson J: Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study. *BMJ* 2005, **330**:1290.
17. Bianchi N, Crosignani P, Rovelli A, Tittarelli A, Carnelli CA, Rossitto F, Vanelli U, Porro E, Berrino F: Overhead electricity power lines and

- childhood leukemia: a registry-based, case-control study. *Tumori* 2000, 86:195-198.
18. Yokus B, Akdag MZ, Dasdag S, Cakir DU, Kizil M: Extremely low frequency magnetic fields cause oxidative DNA damage in rats. *Int J Radiat Biol* 2008, 84:789-795.
 19. Gobba F, Bargellini A, Scaringi M, Bravo G, Borella P: Extremely low frequency-magnetic fields (ELF-EMF) occupational exposure and natural killer activity in peripheral blood lymphocytes. *Sci Total Environ* 2009, 407:1218-1223.
 20. Kheifets L, Swanson J, Greenland S: Childhood leukemia, electric and magnetic fields, and temporal trends. *Bioelectromagnetics* 2006, 27:545-552.

doi:10.1186/1476-069X-9-16

Cite this article as: Malagoli et al.: Risk of hematological malignancies associated with magnetic fields exposure from power lines: a case-control study in two municipalities of northern Italy. *Environmental Health* 2010 9:16.

Submit your next manuscript to BioMed Central
and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at
www.biomedcentral.com/submit

 BioMed Central

84/90

Comment : Table 2.2-8 pg 2-202

According to this table the Proposed Route has 6 residencies within 300 feet of the center line, 9 residencies within 1,000 feet. This is woefully inaccurate. Owyhee County has 13 homes within 300 feet of the center line, 40 homes within 1,000 feet. This information was deduced off of a 911 map obtained from the Owyhee County Assessors Office. The Owyhee County Commissioners will have possession of this map.

Alternative 9.D does not have any homes within the center line.

There are 197 homes within the 2 mile corridor of WWE.

Comment : Socioeconomics

The economy of Owyhee County is fragile.

Table 3.4-4 documents net migration 2000 to 2009 @ -204.

Table 3.4-7 lists Owyhee Co. as farming dependant.

Table 3.4-8 documents Owyhee Co. @ 74% of total land area dedicated to farming; the highest percentage in the state of Idaho.

Table 3.4-9 indicates Owyhee Co. agricultural employment in 2008 @ 17% with a high location quotient @ 4.1.

Pages 3.4-38 thru 3.4-43 document the negative impacts of the Proposed Route to our agricultural industry.

It is imperative Alternate 9D is chosen for this reason alone.

Comment : Socioeconomics

Pgs 3.4-55 thru 3.4-58

document the negative impacts of a new transmission line on property values.

I purchased my home in 2004, paid \$125,000⁰⁰ and have spent another \$10,000⁰⁰ in materials (windows, doors, roof, paint not including our labor).

The Owyhee County assessor is now assessing my home @ \$89,000⁰⁰ down from \$150,000⁰⁰.

The Proposed Route would negatively affect all of our property values with an average decrease of 10% (Delaney and Timmons 1992). We can not personally or collectively afford to take this hit. This would markedly decrease our county's tax revenues. This is one of the many reasons the Owyhee County Task Force has worked so hard to submit Alternative 9D.

Comment : Visual Resources

Pg. 3.4-56 states " The placement of the transmission line across a property also affects the visual quality. Each individual landowner has their own perception of what is visually acceptable or unacceptable. "

Pages 3.2-3 and 4 explain VRM classification. I must point out this is a system, it is not a science. This classification is subjective not objective thus should yield according to additional input or data gathering. An earlier comment addressed the fact that the Proposed Route is comprised of 18.4 miles of unanalyzed private property which incorporates some positively breathtaking creeks, ponds, canyons and old homesteads. These scenes are the ingredients of the Owyhee Avalanche's annual calendar, and many photographs, paintings and greeting cards on sale by our local artisans. There

are an incredible number of KOP's excluded from this DEIS due to the fact they are on private property.

I must point out the analysis of the KOP's on the Proposed Route lists many impacts as low to moderate due to "human made alterations".

There is a 10 mile stretch of BLM land between my house and Paul Nettleton's ranch which does not so much as have a service line in it! The Proposed Route would be a huge new environmental impact to all of Owyhee County.

Alternative 9D for the most part follows an existing 138 kv line. The environmental impact is already there. The visual impact is already there.

Summary :

The Proposed Route is a significant new environmental impact to Owyhee County affecting where we live, work and recreate. The Proposed Route impacts our agriculture, home values, land values, tax base, health and severely mars the beauty of our beloved Owyhee County.

In the state of Idaho counties have siting authority. Alternative 9D was developed by the citizenry of Owyhee County; concerned individuals who donated time, labor and money to responsibly site this massive 500 kv line. Alternative 9D follows an existing 138 kv line. Owyhee County is near and dear to our hearts, it is our chosen home, therefore the voice of its citizens should carry the most weight in the siting of this line. Please select Alternative 9D for the Gateway West Transmission Line Project.

Respectfully,

Roxana Thompson

90/90