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## INFORMATION SHEET

PRESIDING: Commissioner Clodfelter, Commissioner Beatty, and Commissioner Gray

PLACE: Johnston County Courthouse, Smithfield, NC

DATE: October 30, 2017

TIME: 6:30 p.m. – 8:05 p.m.

DOCKET NO.: E-2, Sub 1150

COMPANY: Duke Energy Progress, LLC

DESCRIPTION: Application for a Certificate of Environmental Compatibility and Public Convenience and Necessity to Construct Approximately 11.5 Miles of New 230-kV Transmission Line in Johnston County, NC

VOLUME: 1

## APPEARANCES

DUKE ENERGY PROGRESS, LLC:

Bo Somers, Esq.

FOR THE USING AND CONSUMING PUBLIC:

Heather D. Fennell, Esq., Public Staff

## WITNESSES

See Attached

## EXHIBITS

See Attached

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REPORTED BY: Kim Mitchell

TRANSCRIBED BY: Kim Mitchell

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**NORTH CAROLINA UTILITIES COMMISSION**  
**APPEARANCE SLIP**

DATE 10/30/17  
DOCKET #: 6-2, Sub 1150  
NAME OF ATTORNEY Lawrence B. Scales  
TITLE \_\_\_\_\_  
FIRM NAME \_\_\_\_\_  
ADDRESS \_\_\_\_\_  
CITY \_\_\_\_\_  
ZIP \_\_\_\_\_

**APPEARING FOR:** Duke Energy Progress

APPLICANT  COMPLAINANT \_\_\_\_\_ INTERVENOR \_\_\_\_\_  
PROTESTANT \_\_\_\_\_ RESPONDENT \_\_\_\_\_ DEFENDANT \_\_\_\_\_

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**NORTH CAROLINA UTILITIES COMMISSION**  
**PUBLIC STAFF - APPEARANCE SLIP**

DATE 10/30/17 DOCKET # : E-2, sub 1150

PUBLIC STAFF MEMBER Heather Fennell

ORDER FOR TRANSCRIPT OF TESTIMONY TO BE **EMAILED** TO THE PUBLIC STAFF - PLEASE INDICATE YOUR DIVISION AS WELL AS YOUR EMAIL ADDRESS BELOW:

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\_\_\_\_\_  
\_\_\_\_\_  
✓ Heather Fennell  
Signature of Public Staff Member  
By Kim M.

# Lassiter Family Homeplace - Elevation

## Factors Considered When Siting Transmission Lines

 Linda Lassiter Keen did not receive an invitation to the public meetings held in November 2016.

✓ **Community/Public Input**

 Linda Lassiter Keen - crop of loblolly pines in the Conservation Reserve Program for almost 30 years.

✓ **Land Use**  
Residential, commercial, industrial, major developments, schools, conservation lands and parks, existing linear facilities, airports and managed lands

 A  
L

✓ **Land Cover**  
Forest woodland, mixed forest, grassland/pasture, fresh water development and urban residential

 Lassiter Family Homeplace - settled by Elijah Lassiter (1762-1848)

✓ **Cultural Resources**  
Archaeological resources, historic resources, historic districts and cemeteries

 Have seen and often hear woodpeckers, possibly Red-Cockaded Woodpeckers \*

✓ **Natural Resources**  
State and federal rare, threatened and endangered species

 Line Road; Diagonal

✓ **Visual Resources**  
View shed analysis  
ir  
pr  
ru

 Area of lower elevation nearby that feeds into Gum Swamp. Line is very close to family pond. Concern w/ herbicides and fishing.

✓ **Water Resources**  
Wetlands, streams and floodplains

Approximate distances

 ① Parker 500-600 ft.  
② Canady 700-800 ft.  
③ Moore - close?

✓ **Occupied Buildings**  
Number of single-family residences in proximity of a proposed route

 Cancer incidence in relation to Linda L. Keen (partial list)

✓ **Safety/Reliability/Concerns**

- Mother - breast
- Father - prostate
- Brother - Kidney
- Son - Childhood leukemia
- maternal aunt - breast
- maternal 1<sup>st</sup> cousin - breast
- maternal 1<sup>st</sup> cousin
- daughter - breast
- niece - BRCA

\* per the USDA forest website [www.fs.fed.us](http://www.fs.fed.us) one of the preferred habitats of the Red Cockaded woodpecker are loblolly pine.

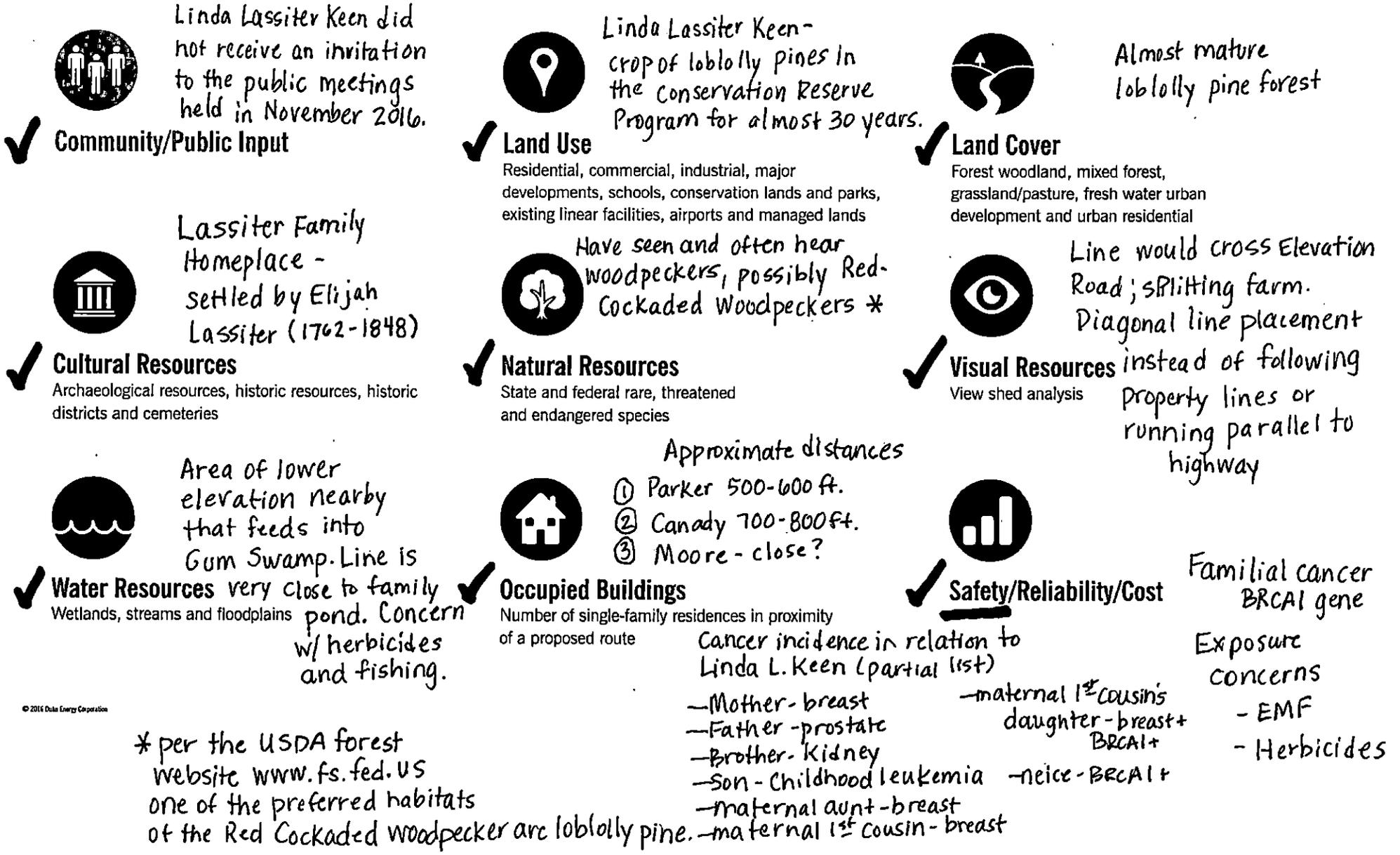
Kimberly Canady Exhibit 1

Cancer gene  
concerns  
MF  
Herbicides

Annex 1 - Data 10 of 14

# Lassiter Family Homeplace - Elevation Rd., Four Oaks

## Factors Considered When Siting Transmission Lines



\* per the USDA forest website [www.fs.fed.us](http://www.fs.fed.us) one of the preferred habitats of the Red Cockaded woodpecker are loblolly pine.

Hearing in regards to the State of North Carolina Utilities Commission Docket No. E-2, Sub 1150 (Cleveland Matthews Project)

1  
Good evening. I am Kimberly Lassiter Canady. I appreciate the opportunity to address the Commission tonight and discuss my concerns about this project. I am a property owner in segment 33 of the proposed route, in the Four Oaks area of Johnston County NOT the Cleveland area of Johnston County. Let me reiterate, the material that DEP has distributed states this is a "Cleveland area of Johnston County" project. I own parcel 07 H 09 028 jointly with my brother Marty R. Lassiter. I live with my husband of 23 years and two children in a home on Gum Swamp Road, (adjacent to the parcel with the proposed line). My home parcel is connected to the farm. I am 43 years old, and have lived on my family farm the entire time, other than being away at school for nine months. My brother and I inherited the land from my grandfather. My father died at the age of 44 from kidney cancer in 1992, prior to my grandfather's death in 1998. My brother lives in Benson, NC.

2  
You know the saying, you can't see the forest for the trees? I like that one since I am a tree farmer. But in this case, I think the opposite is true, Duke Energy Progress (DEP) did not see the trees for the forest. DEP wants this project in place because the banks of Raleigh are overflowing.....and in the process, has totally overlooked one of the most important parts, stakeholder input. If you live in a subdivision, then you might think proximity to houses is the most important part. If you live on a farm then, you may think the land and the environment is the most important. Everyone has an opinion. To evaluate human impact, a more personal study would have been necessary. I have included information about a Transource project in Harford, MD. And my opinion is that there could be lessons learned from the Harford project: a short route was chosen, it parallels roads and there were 10 events provided for the landowners and the community, as well as the company presenting two tower structures. Tensions run high about life, liberty and property, (so I am thinking most people would not want that assignment) I understand that it is a huge responsibility to find that balancing point and implement projects that are truly in the best interest of the people that we serve. I use we, because I am employed with the State of NC. When I swipe the card reader each morning, there is a sign above it that says "Do the right thing." I am a Registered Nurse, a Nurse Consultant, and I work for Department of Health and Human Services. I have the responsibility and privilege to work for a department that serves the most vulnerable in our population. I have actually worked on both sides, a Nurse with a previous vendor, now with the State. I realize that it is important to provide the oversight needed, so that the vendor acts with the same interests as the state. That responsibility may even be greater, when the entity is so large and powerful. However, I am not here as a professional, I am here to discuss how this project has impacted me on a personal level.

I did not understand the scope of this project until May 2017, and then that information was from a neighbor, so I would like you, the Utility Commission to carefully examine how "reasonable" the effort was to contact and inform the landowners. There are many comments from landowners in the Docket about lack of information. I have previously submitted a letter about my concerns of the Nov 4 letter not being written to address the general public. The average US resident

reads at an 8<sup>th</sup> grade level and the Nov4 letter appeared to be written to a Cleveland Community audience that needed to be familiar with electricity and utilities. (SMEs subject matter experts). The subject line was misleading, as I do not live in the Cleveland area of Johnston County. **The letter failed to notify me in a clear and concise way that they needed or would need an easement through my property to build 65-85 ft. H frame structures with high voltage power lines to carry electricity to the new substation at the intersection of Matthews Road and Polenta Road, PRIOR TO THE CLOSE OF COMMENT.** The previous sentence shows transparency. I actually take pride in the fact that I am able to read and summarize documents, because in November of 2016, I was reading numerous medical documents each day to summarize the important information to relay to the doctors. As I previously stated in my letter to Mr. Christopher J. Ayers, lack of transparency on the part of DEP is not doing the right thing. I had no personal contact with Duke Energy Progress employees regarding this project, prior to the determination of the proposed route, and therefore my comments and concerns were not addressed prior to the public deadline for comments. I initiated the first call to DEP on 5/17/17. Since that time, it has been confirmed to me more than once, that the route selection had been made and the period for public comment had ended. I am a stakeholder in this process, and my input has not been factored in at all.

Mr. Timothy J. Same was asked if Duke Energy Progress sought public input as part of the Cleveland-Matthew line siting process. He stated, "Yes," and his testimony further states that "all input was used to assess the values and attitudes of the residents and public officials regarding the project, which enabled the Project Team to identify the most appropriate factors to evaluate the routes and to develop routes that limited impacts to resources of primary concern to the environmental agencies and to the residents." Also, participants at the public meeting received a questionnaire to "gather important information to be considered during the route selection process." **I learned of this questionnaire in July 2017 when the Utilities Commission published Docket No. E-2, Sub 1150. (The formal public comment period ended December 31, 2016). This questionnaire was not mailed to me. I wasn't notified of its existence, prior to the close of comment, and the use of it in the selection process, appears discriminatory.**

And even after the close of comment and the decision was made, why didn't DEP make a "reasonable" effort to contact my family to address the extensive removal of forest land that they have proposed? A reason cited for selection of the preferred route was that it utilizes cropland acres when possible to avoid extensive removal of forested areas along the route. However, a little over a mile (estimated) of the proposed 11.5-mile project crosses my family's planted pine forest. If avoiding extensive removal of forested areas is important enough to be one of the 8 reasons (as cited in Docket No. E-2, Sub 1150) why this route is chosen, and one family owns so much forested land in the preferred route, why would Duke Energy Progress not make personal contact with the family to address this at any point; prior to close of public comment or after? They have our address. It is on their map. My family has 3 parcels of land involved, and Duke Energy Progress sends ONE letter to me alone in November and THAT IS IT. I used Google to do a quick search and found my brother's address in less than a minute. Our tax statement lists his name first. That is not doing the right thing. Duke Energy Progress also sends me emails

each and every month about conserving energy and various energy topics, but not once did they reach out to me by email prior to close of comment.

In most decisions, if you apply the "golden rule," the process of elimination is usually clear. In this case, how would you feel if you were left out of an important/life changing decision? How would you like to be told, "we regret any inconvenience," if you didn't receive notice of the public workshops? (I am referring to the rebuttal testimony of Timothy J. Same for Duke Energy Progress, LLC on 10/25/17)/How would you feel, as a stakeholder in a project that used an elaborate systematic evaluation of impact, that included public comment, and the results were ignored? I am referring to the z score for Route 4, -32.02 vs. the z score of Route 31 at -11.64 in section 4.4.4 of the docket/If the results are being ignored because of potential litigation about greenspace, then someone in the planning/siting process dropped the ball. Why should the people along our route suffer from someone else's oversight? If the results are being ignored because of "minimal input from concerned landowners along our route," I'd like to point out that it is hard to show concern, when you aren't aware. And I want to touch on one more thing.

I ran into a landowner a couple of days ago and asked her about the project. She told me that the line went straight through the middle of her property (Kim Guignard). I asked if she were planning on attending the public meeting and she replied, "No, because, Duke is going to do what they want anyway." I have heard this comment many times. I think that the public perception is that Duke is a giant and we are defenseless against them. That is much more accurate than the statement that DEP included in the Docket that stated minimal input from concerned landowners equates to a more positive public perception of the project. And finally, back to my questions about the golden rule, how would you feel if you were not able to preserve your family homeplace and feel safe at home?

The impact of this project to me personally is huge. If the notification that we received would have been written more clearly or the public involvement activities more thorough, the following concerns could have been addressed: As I stated, my family heritage in the area dates back to the 1700's. I have submitted information about Elijah Lassiter. My children will be the 8<sup>th</sup> generation that lives in the area he settled. This is of great importance to me, but due to time constraints, my aunt will appear and discuss that information. Another major impact is the line placement on my farm, as it does not allow for movement within the property. If you move the line one way, it would be closer to the property that my brother owns, if I request to move it the other way, it would result in a longer segment on my Aunt's land. The line placement also cuts the property diagonally and would essentially decrease the value to a fraction of what it currently is. We have another special circumstance, because I own the property with a sibling (Marty R. Lassiter), instead of a spouse. Splitting the property with a high voltage power line going through it would be extremely difficult. To try to find equal value, it would have to be cut up like a checkerboard. The proposed line is directly over the highest elevation, cuts across close to Elevation Road and then very close to Gum Swamp Road; close enough to take future homesites with road frontage on both, yet not directly parallel following the right of way. So, the three areas that would be the most valuable is what DEP has proposed to for the easement. When you are on the ground and can see the lay of the land, you can see how impactful it would be. But the greatest potential impact to me and my family is health concerns.

I realize that DEP does not acknowledge EMF as pollution. I have read the EMF information that was included in the Docket. It states on the title page, compliments of Duke Energy. Also, note it has a copyright of 2013 and there has been a lot of developing science since 2013. I appreciated the analogy that was included to help me understand association, "A rooster crowing in the morning will cause the air temperature to rise." It explained that there was a strong statistical association between the two because it occurs a very high percentage of the time.

Appendix C: Page 38 of 51 states that, "some researchers believe that if EMF's are shown to cause health effects, the risk of these effects will probably be comparatively small." I asked myself....Small as in maybe 0.2-0.3%? That is how prevalent having a BRCA gene mutation is among the general population. I think this raises additional questions that were not addressed in DEP's EMF brochure. I think there is a potential for risk and how to best minimize the risk should be of concern to everyone. In my case, I have a condition that predisposes me to a higher than average risk of cancer; a BRCA1 gene mutation that is associated with cancer clusters in families. The BRCA1 gene mutation could have been passed to my children. By installing high voltage power lines near my place of residence, it will potentially increase the risk of disease in myself and my family. I have included a statement from my family physician and additional information from InformedDNA for verification. According to the (NIH)National Institutes of Health, (refer to attached article BRCA1 gene,DNA Associated, US National Library of Medicine), "The BRCA1 protein is involved in repairing damaged DNA. In the nucleus of many types of normal cells, the BRCA1 protein interacts with several other proteins to mend breaks in DNA. These breaks can be caused by natural and medical radiation or other environmental exposures, and they also occur when chromosomes exchange genetic material in preparation for cell division. By helping to repair DNA, the BRCA1 protein plays a critical role in maintaining the stability of a cell's genetic information." It further states that, "these mutations are present in every cell in the body and can be passed from one generation to the next. As a result, they are associated with cancers that cluster in families." In the EMF brochure contained in Docket E-2 Sub 1150 (Appendix C: Page 37 of 51), it states, "Some studies thus far have tied a slight association to EMF and cancer." It further states, "No common cause has been directly related to the effect." The literature states none of the researchers found a direct link between actual EMF exposure and cancer incidence. That is because there are a number of behavioral and environmental triggers that cause changes in the body's cells to push them into a cancerous state, but I firmly believe that EMF exposure could be a trigger based on evolving science, most especially in people that have limitations in the DNA repair process. IARC **International Agency for Research on Cancer (IARC)** has classified ELF *magnetic* fields as "possibly carcinogenic to humans." In 1999, the US **National Institute of Environmental Health Sciences (NIEHS)** described the scientific evidence suggesting that ELF exposure poses a health risk as "weak," but noted that it cannot be recognized as entirely safe, and considered it to be a "possible" human carcinogen.

I have included articles that discuss DNA strand breaks, and as I stated previously, BRCA1 protein interacts with several other proteins to mend breaks in DNA. One such article is, extremely low-frequency electromagnetic fields cause DNA strand breaks in normal cells <http://www.ijehse.com/content/12/1/15> The conclusion stated, "The analysis of the registered comet indices and of cell cycle showed that extremely low frequency electromagnetic field of 100 Hz and 5.6 mT had a genotoxic impact on Vero cells." Genotoxin meaning a substance that can cause damage to or mutation in DNA. Science that supports an indirect link is included in the

article Signal transduction of the melatonin receptor MT1 is disrupted in breast cancer cells by electromagnetic fields [www.ncbi.nlm.nih.gov/pubmed/19882681](http://www.ncbi.nlm.nih.gov/pubmed/19882681) that states, “these results *convincingly* prove the negative effect of EMF on the antiestrogenic effect of melatonin in breast cancer cells.” “Among the general population, prevalence of having a BRCA mutation is as follows: General population 0.2 to 0.3%” according to <https://www.knowbrca.org/Provider/FNA>. Another source, Cancer and the Environment-What You Need to Know and What You Can Do, published by the US Department of Health and Human Services, National Institutes of Health, National Cancer Institute and National Institute of Environmental Health Sciences states that only a very small percentage of people in the general population have abnormal copies of these genes. Cancers caused by these genes, known as familial cancers, account for only two to five percent of all cancers, per [https://www.niehs.nih.gov/health/materials/cancerand\\_the\\_environment\\_508.pdf](https://www.niehs.nih.gov/health/materials/cancerand_the_environment_508.pdf). (Per the National Cancer Institute familial cancer is defined as cancer that occurs in families more often than would be expected by chance. These cancers often occur at an early age, and may indicate the presence of a gene mutation that increases the risk of cancer. They may also be a sign of shared environmental and lifestyle factors). The alarmingly high percentage to remember is that when I was diagnosed last fall, Wendy Garlitz at InformedDNA told me over the phone that my lifetime chance of getting ovarian cancer is up to 44%, and my chance of getting breast cancer was up to 87% without risk reduction measures. (Included a June 2017 JAMA article that supports that also, however 72% for breast). Another article at [www.ncbi.nlm.nih.gov/pubmed/25688995](http://www.ncbi.nlm.nih.gov/pubmed/25688995) Comparison of the genotoxic effects induced by 50 Hz extremely low-frequency electromagnetic fields and 1800 MHz radiofrequency electromagnetic fields in GC-2 cells, states, “Our results suggest that both ELF-EMF and RF-EMF under the same experimental conditions may produce genotoxicity at relative high intensities, but they create different patterns of DNA damage.” Again, the BRCA1 protein is responsible for mending breaks in DNA. The next one is found at [www.ncbi.nlm.nih.gov/pubmed/24984538](http://www.ncbi.nlm.nih.gov/pubmed/24984538), Relationship between exposure to extremely low-frequency electromagnetic fields and breast cancer risk: a meta-analysis, with the following conclusion: “The authors found that ELF-EMFs may increase the risk of human breast cancer. The women’s exposure to ELF-EMF may be the risk factor of breast cancer when they are non-menopausal.” This one goes with the next article found at [www.ncbi.nlm.nih.gov/pubmed/8196082](http://www.ncbi.nlm.nih.gov/pubmed/8196082), Breast cancer mortality among female electrical workers in the United States. Results state electrical workers had excess mortality from breast cancer relative to other employed women. There was no excess of breast cancer, however, in seven other occupations held more frequently by women and also involving potentially elevated electrical exposures, including telephone operators, data keyers, and computer operators and programmers.” I have also included the 2012 BioInitiative Report for review. According to the article Electric and Magnetic Fields (EMF): Health Concerns from the Connecticut Department of Public Health, Environmental Health Section Environmental & Occupational Health Assessment Program [http://www.ct.gov/dph/lib/dph/environmental\\_health/eoha/pdf/emf\\_fact\\_sheet\\_-\\_2008.pdf](http://www.ct.gov/dph/lib/dph/environmental_health/eoha/pdf/emf_fact_sheet_-_2008.pdf) it states, “In a study that measured EMF in almost 1000 homes in the United States, 50% had average EMF levels of 0.6 mG or less, and 95% had average EMF levels below 3 mG. Keep in mind that these are average EMF levels within a home.” It further

states, "The high voltage lines can have EMF levels of 30 to 90 mG underneath the wires, depending on the voltage, height, and placement of the lines."

The 1994 article <https://www.ncbi.nlm.nih.gov/pubmed/7731404> Carcinogenic risk of extremely-low-frequency electromagnetic fields: state of the art states that, "On the basis of several epidemiological studies on occupational exposure, an **increased risk of leukemia**, brain cancer and male breast cancer is apparent; the literature on residential exposure provides some evidence of an effect on childhood cancer, especially leukemia;" This caught my attention because **I have a second-degree relative. (A second-degree relative (SDR) is someone who shares 25% of a person's genes. It includes uncles, aunts, nephews, nieces, grandparents, grandchildren, half-siblings, and double cousins), who has had a child with childhood leukemia.**

Another potential carcinogenic concern is the use of herbicides. In section 2.2 of the Docket, Construction Operation and Maintenance, it states that herbicides are applied in individual woody stems using a low volume backpack sprayer. Duke Energy Progress uses herbicides approved by the U.S. EPA for use on terrestrial and wetland transmission line ROWs. At [www.epa.gov](http://www.epa.gov), there is an article dated 12/15/17, entitled EPA takes action to Prevent Poisonings from the herbicide Paraquat (attached). To summarize, "the measures included a new closed-system packaging, special training and changes to the pesticide label. Other names for this chemical are Paraquat Dichloride and it is often referred to as Gramoxone (a popular end-use product)," according to the EPA website. Therefore, it appears Paraquat is an approved herbicide by the U.S. EPA. See the attached letter from the Journal of Toxicological Sciences, Vol.38 No.3, The Correlations between BRCA1 defect and environmental factors in the risk of breast cancer. This study assessed the risks of various environmental factors for increase in ROS production or ROS induced DNA damage. Reactive oxygen species (ROS) play a critical role in cellular physiopathology. The results suggested that, "the concurrent exposure to environmental factors increases the risk of breast cancer carrying genetic factors such as BRCA1 defect." Paraquat was used as an environmental factor in the study. There is a stream that runs parallel to my house and a pond is beside the proposed line on my property. The docket states that Johnston County receives a total of 47 inches of rainfall per year. The runoff containing the herbicides could lead to contamination of the pond and the stream that my son especially enjoys.

**There is evidence to suggest EMF exposure induces DNA damage. Note enclosed 2006 article [www.ncbi.nlm.nih.gov/pubmed/16836873](http://www.ncbi.nlm.nih.gov/pubmed/16836873) , Effects of GSM 1800 MHz radiofrequency electromagnetic fields on DNA damage in Chinese hamster lung cells by the National Institutes of Health, which I will just refer to as NIH as I continue. The conclusion states, "1800 MHz RF EMF (SAR, 3.0 W/kg) for 24 hours might induce DNA damage in CHL cells."** I personally have a higher cancer risk than average, and my children have potentially inherited the same risk. It has not been clinically indicated at their age, to have them tested, but publicly disclosing my status to try to protect them, could have future ramifications for them too. The land that I own will surround the easement containing the high voltage power line, I should be free to enjoy all of it, and pass it to my children. This land is our sanctuary, and we especially need to keep potentially harmful pollutants out, for our protection. Again, BRCA1 gene mutations are rare in

the general population and protecting us on our land is no different than protecting any other endangered species. Merriam-Webster *broadly* defines it as anyone or anything whose continued existence is threatened, (<https://www.merriam-webster.com/dictionary/endangered%20species>) and is even sometimes a result of a genetic mutation. The protection of the Dwarfwedge mussel was mentioned several times in the Docket. I think the question could be raised about how much protection that my family and I should be afforded given my health status. Publicly revealing my diagnosis, (Protected Health Information (PHI)), as a factor of concern during this public process, has already put me at risk, as the GINA (Genetic Information Nondiscrimination Act) does not give full protection (per Susan G. Komen site <https://ww5.komen.org/>). What if my land is also devalued in the process?

In the rebuttal testimony of Timothy J. Same, he states “the expected EMF readings would essentially be the same along any alternative route for the Cleveland- Matthews Transmission Line and, therefore, it would have no impact on the relative rankings of the alternative routes had it been considered.” I feel that if a cancer cluster exists along a route, potential to cause harm should be considered. The Docket states in Appendix C, Page 39 of 51, referring to EMF, it states, “additional research on this complex subject is needed.”

I charge that Duke Energy Progress’ proposal does not meet the criteria set forth in General Statute 62-105 (NC General Statute 62-105 the Commission shall grant a certificate for the construction, operation, and maintenance of the proposed transmission line if it finds that, when compared with reasonable alternative courses of action, construction of the transmission line in the proposed location is reasonable, preferred, and in the public interest, among conditions), because public interest is not served when DEP disregards the quantifiable method of impact used for route selection, and does not have a thorough process for including stakeholders. First, according to the direct testimony, contained in the application, of Timothy J. Same (in the October 9, 2017 Verified Responses to Commission Order it states, “Page 17, Timothy Swane’s testimony.” There appears to be a typographical error, so I am not sure who testified), Duke Energy Progress, LLC a study area was established. Then, collected data were grouped into one of ten categories: cultural resources, flood zones, land cover, community amenities and public infrastructure, natural resources, occupied buildings, prime and important farmland, public visibility, water features and current zoning. Each category was further divided into individual criteria and assigned a weight from 1 to 5 according to each criterion’s potential sensitivity to a transmission line, as determined by members of DEP’s team and feedback obtained from public comments. The weight scale of 5 representing the highest consideration during the evaluation. For example, Residential Proximity Score has a weight scale of 5.

According to Docket No. E-2, Sub 1150, ultimately, 32 distinct routes were developed, using a combination of 39 segments. Criteria totals for each potential route were summed, and a Z-score was calculated for each criterion for each route. To streamline the analysis, approximately 20 percent of the lowest-scoring (least-impacting) routes in the Z-score analysis were retained for additional evaluation and comparison. The lowest-scoring 20 percent included seven route alternatives. Route 4 was the lowest scoring route and was the shortest overall alignment. This

route was not chosen. Route 31, the third overall lowest-scoring and the longest route, was chosen. After all Z-scores were calculated, Burns & McDonnell applied a weight factor to each criterion to give greater consideration in the evaluation process to those criteria that are considered to have a greater impact on the overall Project evaluation (see Table 4-2). **If weight factors were not applied, all criteria would be assumed to have the same level of impact on the evaluation process. Although all criteria need to be considered during the routing process because they have the capacity to influence potential impacts, design, and cost, certain criteria have the capacity to influence the Project in a greater manner. Therefore, all criteria are not equal in terms of importance to the Project, and thus are weighted accordingly.** The Docket states DEP used prior siting experience and direct feedback from the public during the comment period to help determine the weights used.

“Routes 4 and 1 also impact acres of designated open space (1.1 and 4.6 acres, respectively), which are areas attributed to subdivisions in the area and generally serve as non-developed greenspace as part of the subdivision. The two southern routes do not cross any designated open space acres as part of their alignments.” Designated is the key word here. The Docket states in section 4.4.1, **“Open Space/Green Areas Crossed (i.e., parks, wildlife areas, nature preserves, etc.) was used to determine potential impacts the proposed routes would have on any greenspace or open lands within the study area. This criterion was measured but not included in the evaluation because only a handful of segments crossed any public lands, which were open space areas associated with residential subdivisions.”** (The max acreage in Routes 1-4 was 7.2 in Route 2, per table 4-4 in the Docket). On page 4-24 the Docket states, “Upon further investigation by the Project Team, it was discovered that the potential condemnation of open space/green space areas owned by a subdivision homeowner association could require the condemnation of all property owners within that subdivision, based on a precedent from a previous legal case. This knowledge, along with the proximity to residences and subdivisions, potential environmental impacts to sensitive streams and floodplains, and construction and maintenance concerns associated with the western routes, resulted in the elimination of these two routes (Route 4 and Route 1) from further consideration. All criteria that I just mentioned has already been factored into the impact analysis, so, **it seems that it just boiled down to a potential lawsuit, not total comprehensive impact.**”

Why were the quantitative results not used? Also, if the weight scale was “trumped” by public comment and all the landowners were not involved, then the process for selection was unjust. Sending one non-certified letter that may or may not have reached the landowner is not acceptable when you use public comment as criteria for route selection. DEP could afford to pay someone minimum wage to ride the routes and leave a notice on your door, place yard signs in the ROW in the areas of interest to notify people of the project, use the e-mail addresses that they have on file or maybe even place a call. Not everyone is checking the newspaper on a weekly basis to see if their land is set to be taken by a government entity, and one would expect that to be even less likely in a rural area. If you are a homeowner and you get behind on your mortgage, how many layers of protection do you have before you are foreclosed on? If your performance is failing at work, how many layers of protection do you have from being fired? This is the United States. People are supposed to have rights and protection. I was given 45 days to respond to a

letter that I did not understand. (I specifically address the concerns I had with the letter in a previous letter to Christopher J. Ayers that I have included). (I am using 45 days, because that was the time allowed after the distribution of questionnaires at the public hearing and the close of comment. Keep in my that the time period was between the two major holidays that the State recognizes). THAT IS IT. No second notice was given, no public meeting was called when DEP narrowed the route choices from the 277 square mile study area. DEP may have checked the box for minimum requirements, but that is not doing the right thing. I am respectfully asking the Utility Commission to provide the necessary oversight to ensure complete transparency.

I am asking that if that if DEP does not intend to use the data that was gathered beyond the 45-day window of public comment, that certainly exemplifies further impact, then adhere to the quantifiable measure that the consulting agency, Burns & McDonnell, has provided. Keep in mind that the docket states, "Proximity to residences, businesses, and public facilities was considered for the route analysis." I think adhering to the quantifiable method used in route impact analysis, which should have equated to route selection, would exemplify impartiality on the part of the State of North Carolina to all citizens.

In the Docket, section 4.4.1 states "The evaluation of the proposed routes included a **systematic comparison of the alternatives based on the social, environmental, and engineering factors that represent the potential adverse effects on resources in the study area.**" It also states "After further desktop and field reviews of these seven routes, combined with additional meetings with the Project team, it was determined that any one of these routes would be feasible and constructible." The people, the environment and the generations that will follow, deserve the least impactful route. The application is for a Certificate of Environmental Compatibility and Public Convenience, NOT Duke Energy Progress' Convenience.

Cleveland Residents live in close proximity to each other, they are Facebook friends and see each other in passing in the subdivisions. Word tends to travel faster when you are in close proximity. The Four Oaks area is mainly agricultural/rural. There are not as many of us in the southern end, and it doesn't help when invitations to the public meetings don't make it to everyone. Our land in the southern route is being offered up to help ease the utility burden that has been created from the commercialization of the Cleveland area of Johnston County. Cleveland may not be incorporated, but it is the largest "town" in Johnston County. I am sure that the residents have pushed back on that too because that would mean city taxes. The building of subdivisions and stores has become much more lucrative than maintaining family farms. The Cleveland area wants to benefit from all the incoming businesses and subdivisions, avoid higher taxes and push their utility problems to the southern end of the county. I think there should be one take away here. The lowest scoring route was deemed least impactful by Duke Energy Progress' own measurements. Not following through with these findings only suggests NON-TRANSPARENCY. That is not doing the right thing.

Is the criteria that is not published simply cost? Duke Energy Progress is the business of making money. In the 2017 article from the Charlotte Observer (<http://www.charlotteobserver.com/news/business/article133059044>) it states that the 2016

earnings, though down, were \$2.1 billion. The impact is higher here, by DEP's own measure. This leads me to believe that it is either cost or their convenience that resulted in this decision. My land is just as valuable as any other in the study area. Where is the comparison for the estimate to buy easements? Was that quantifiable impact measure not published? Was this route chosen because it would be easier for the construction crew? Their preferences should not be included unless the impact analysis deems two routes equal in score. DEP states in the Docket, design issues are relatively easy to address when crossing streams and measures can be taken to mitigate impacts. The docket states in section 4.4.1 potential impacts would be more likely to occur where a route would be built away from existing corridors, so length not along existing infrastructure was measured; however, Length Not Along Existing Infrastructure was not included. This makes no common sense at all. The overall least impactful way to implement this project should be used.

Please note also, the Docket states in 6.2.3 Federally Listed Species Communication has been initiated with the USFWS and NCWRC regarding potential impacts concerning State and federally protected species. State or federally protected species known to occur within the study Cleveland-Matthews Road 230kV Tap Line Project Mitigation Measures Duke Energy 6-3 Burns & McDonnell area or near the preferred route ROW are not expected to be adversely impacted. Further consultation with the USFWS and NCWRC will be initiated once a route has been approved to comply with the Endangered Species Act. Duke will hire a contractor to conduct a review of the preferred route to determine whether potential habitat for protected species is likely to be impacted by the route. **DEP hire the contractor? Conflict of interest?**

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## Signal transduction of the melatonin receptor MT1 is disrupted in breast cancer cells by electromagnetic fields.

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### Author information

#### Abstract

The growth of estrogen-receptor positive breast cancer cells is inhibited by the pineal gland hormone, melatonin. Concern has been raised that power-line frequency and microwave electromagnetic fields (EMFs) could reduce the efficiency of melatonin on breast cancer cells. In this study we investigated the impact of EMFs on the signal transduction of the high-affinity receptor MT1 in parental MCF-7 cells and MCF-7 cells transfected with the MT1 gene. The binding of the cAMP-responsive element binding (CREB) protein to a promoter sequence of BRCA-1 after stimulation with melatonin was analyzed by a gel-shift assay and the expression of four estrogen-responsive genes was measured in sham-exposed breast cancer cells and cells exposed to a sinusoidal 50 Hz EMF of 1.2 microT for 48 h. In sham-exposed cells, binding of CREB to the promoter of BRCA-1 was increased by estradiol and subsequently diminished by treatment with melatonin. In cells exposed to 1.2 microT, 50 Hz EMF, binding of CREB was almost completely omitted. Expression of BRCA-1, p53, p21(WAF), and c-myc was increased by estradiol stimulation and subsequently decreased by melatonin treatment in both cell lines, except for p53 expression in the transfected cell line, thereby proving the antiestrogenic effect of melatonin at molecular level. In contrast, in breast cancer cells transfected with MT1 exposed to 1.2 microT of the 50 Hz EMF, the expression of p53 and c-myc increased significantly after melatonin treatment but for p21(WAF) the increase was not significant. **These results convincingly prove the negative effect of EMF on the antiestrogenic effect of melatonin in breast cancer cells.**

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## About BRCA1, BRCA2, and Hereditary Breast and Ovarian Cancers

1. How do *BRCA1* and *BRCA2* gene mutations function?
2. What are the population estimates of the likelihood of having a *BRCA1* or *BRCA2* gene mutation?
3. How do *BRCA1* and *BRCA2* gene mutations affect risk of cancer?
4. What are the clinical recommendations for *BRCA* risk assessment and referral to genetic counseling and testing?
5. What are the benefits of *BRCA* risk assessment and genetic counseling and testing (when appropriate)?
6. What are the clinical recommendations for women assessed as being NOT at increased risk for a *BRCA1* or *BRCA2* gene mutations?
7. What clinical options are available to reduce cancer risk in *BRCA* mutation carriers (confirmed through genetic counseling and testing)?

### HOW DO *BRCA1* AND *BRCA2* GENE MUTATIONS FUNCTION?

Most *BRCA1* and *BRCA2* mutations are predicted to produce a truncated protein product, and thus loss of protein function, although some missense mutations (<http://www.cancer.gov/Common/PopUps/popDefinition.aspx?id=460164&version=HealthProfessional&language=English>) cause loss of function without truncation. Because inherited breast/ovarian cancer is an autosomal dominant condition, persons with a *BRCA1* or *BRCA2* mutation on one copy of chromosome 17 or 13 also carry a normal allele on the other paired chromosome. In most breast and ovarian cancers that have been studied from mutation carriers, deletion (<http://www.cancer.gov/Common/PopUps/popDefinition.aspx?id=460141&version=HealthProfessional&language=English>) of the normal allele results in loss of all function, leading to the classification of *BRCA1* and *BRCA2* as tumor suppressor genes. In addition to, and as part of, their roles as tumor suppressor genes, *BRCA1* and *BRCA2* are involved in myriad functions within cells, including homologous DNA (<http://www.cancer.gov/Common/PopUps/popDefinition.aspx?id=45671&version=HealthProfessional&language=English>) repair, genomic stability, transcriptional regulation, protein ubiquitination, chromatin remodeling, and cell cycle control.<sup>17</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.17](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.17))<sup>18</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.18](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.18))

Nearly 2,000 distinct mutations and sequence variations in *BRCA1* and *BRCA2* have already been described.<sup>19</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.19](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.19)) *BRCA1* or *BRCA2* prevalence varies by population - 0.2 to 0.3 percent in the general population - <http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestes101.pdf> (<http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestes101.pdf>). The mutations that have been associated with increased risk of cancer result in missing or nonfunctional proteins, supporting the hypothesis that *BRCA1* and *BRCA2* are tumor suppressor genes. While a small number of these mutations have been found repeatedly in unrelated families (e.g. founder mutations in Ashkenazi Jewish families), most have not been reported in more than a few families.

Source: National Cancer Institute Genetics of Breast and Ovarian Cancer PDQ - <http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq> (<http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq>)

### WHAT ARE THE POPULATION ESTIMATES OF THE LIKELIHOOD OF HAVING A *BRCA1* OR *BRCA2* GENE MUTATION?

Among the general population, prevalence of having a *BRCA* mutation is as follows:

- \* General population: 0.2 to 0.3%
- Women with breast cancer: 3%
- Women with breast cancer onset before age 40 years: 6%
- Women with ovarian cancer: 10%
- High-risk families: 20%

Source: <http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestfinalrs.htm> (<http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestfinalrs.htm>)

Among Ashkenazi Jewish individuals, the prevalence of having any *BRCA* mutation is as follows:

- General Ashkenazi Jewish population (2.5%)<sup>60</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.60](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.60))
- Women with breast cancer (any age) (10%)<sup>61</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.61](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.61))
- Women with breast cancer (younger than 40 years) (30%-35%)<sup>61-63</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.61](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.61))
- Men with breast cancer (any age) (19%)<sup>64</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.64](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.64))
- Women with ovarian cancer or primary peritoneal cancer (all ages) (36%-41%)<sup>65-67</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.65](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.65))

Two large U.S. population-based studies of breast cancer patients younger than age 65 years examined the prevalence of *BRCA1*<sup>54</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.54](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.54))<sup>68</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.68](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.68)) and *BRCA2*<sup>54</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.54](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.54)) mutations in various ethnic groups. The prevalence of

*BRCA1* mutations in breast cancer patients by ethnic group was 3.5% in Hispanics, 1.3% to 1.4% in African Americans, 0.5% in Asian Americans, 2.2% to 2.9% in non-Ashkenazi Caucasians, and 8.3% to 10.2% in Ashkenazi Jewish individuals.<sup>54</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.54](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.54)) 68 ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.68](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.68)) The prevalence of *BRCA2* mutations by ethnic group was 2.6% in African Americans and 2.1% in Caucasians.<sup>54</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.54](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.54))

A study of Hispanic patients with a personal or family history of breast cancer and/or ovarian cancer, who were enrolled through multiple clinics in the southwestern United States, examined the prevalence of *BRCA1* and *BRCA2* mutations. Deleterious *BRCA* mutations were identified in 189 of 746 patients (25%) (124 *BRCA1*, 65 *BRCA2*);<sup>69</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.69](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.69)) 21 of the 189 (11%) deleterious *BRCA* mutations identified were large rearrangements, of which 13 (62%) were *BRCA1* ex9-12 deletions. In another population-based cohort of 492 Hispanic women with breast cancer, the *BRCA1* ex9-12 deletion was found in three patients, suggesting that this mutation may be a Mexican founder mutation and may represent 10% to 12% of all *BRCA1* mutations in similar clinic- and population-based cohorts in the United States. Within the clinic-based cohort, there were nine recurrent mutations, which accounted for 53% of all mutations observed in this cohort, suggesting the existence of additional founder mutations in this population.

A retrospective review of 29 Ashkenazi Jewish patients with primary fallopian tube tumors identified germline (<http://www.cancer.gov/Common/PopUps/popDefinition.aspx?id=460154&version=HealthProfessional&language=English>) *BRCA* mutations in 17%.<sup>67</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.67](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.67)) Another study of 108 women with fallopian tube cancer identified mutations in 55.6% of the Jewish women and 26.4% of non-Jewish women (30.6% overall).<sup>70</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.70](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.70)) Estimates of the frequency of fallopian tube cancer in *BRCA* mutation carriers are limited by the lack of precision in the assignment of site of origin for high-grade, metastatic, serous carcinomas at initial presentation.<sup>65</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.65](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.65)) <sup>67</sup>, ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.67](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.67)) <sup>70</sup>, ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.70](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.70)) <sup>71</sup> ([http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section\\_2.71](http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq#cit/section_2.71))

Source: National Cancer Institute Genetics of Breast and Ovarian Cancer PDQ - <http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq> (<http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq>)

## HOW DO *BRCA1* AND *BRCA2* GENE MUTATIONS AFFECT RISK OF CANCER?

Clinically significant mutations in the *BRCA1* and *BRCA2* genes are associated with an increased risk of breast, ovarian, tubal, peritoneal, and other cancers. *BRCA* testing looks for these variations, which can help patients and health care providers understand a person's risk for these cancers. For women who have a *BRCA* mutation, the risk of developing breast or ovarian cancer is greatly increased, with current cumulative risk estimates ranging from 45 - 65% for breast cancer and 10 - 39% for ovarian cancer by age 70.

(<http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestfinalrs.pdf>) Men with *BRCA* mutations, especially *BRCA2* mutations, are also at increased risk for breast cancer. Certain other cancers may be more frequently seen in mutation carriers of either sex. *BRCA* gene mutations may increase risk of fallopian tube, peritoneal (lining in the abdomen), and pancreatic cancer in women. *BRCA* gene mutations may increase risk of pancreatic, prostate, and breast cancer in men.

## FACTS ABOUT *BRCA1* AND *BRCA2*

- *BRCA* mutations account for 5%-10% of breast cancer and 10%-15% of ovarian cancer cases in the U.S. each year (<http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestes101.pdf>)
- Genetic tests are available to check for *BRCA1* and *BRCA2* mutations. Genetic counseling is recommended before and after the tests, to make sure that the risks and benefits are understood, that informed consent is achieved, and that the appropriate test is being ordered.
- If a harmful *BRCA1* or *BRCA2* mutation is found, several options are available to help reduce cancer risk. Different screening regimens may be recommended. For more information on clinical management, see the National Cancer Institute Genetics of Breast and Ovarian Cancer PDQ - <http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq> (<http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq>)
- Many research studies are being conducted to find newer and better ways of detecting, treating, and preventing cancer in *BRCA1* and *BRCA2* mutation carriers.

Source: CDC Public Health Genomics - Genomics Implementation - Detailed Information on Tier 1 Applications - Hereditary Breast and Ovarian Cancer - [http://www.cdc.gov/genomics/implementation/toolkit/HBOC\\_1.htm](http://www.cdc.gov/genomics/implementation/toolkit/HBOC_1.htm) ([http://www.cdc.gov/genomics/implementation/toolkit/HBOC\\_1.htm](http://www.cdc.gov/genomics/implementation/toolkit/HBOC_1.htm))

## WHAT ARE THE CLINICAL RECOMMENDATIONS FOR *BRCA* RISK ASSESSMENT AND GENETIC COUNSELING AND TESTING?

In 2013, the U.S. Preventive Services Task Force (USPSTF) updated and reaffirmed its 2005 recommendations for *BRCA* testing (<http://www.uspreventiveservicestaskforce.org/uspstf/uspstfbrgen.htm>) stating: "The USPSTF recommends that primary care providers screen women who have family members with breast, ovarian, tubal, or peritoneal cancer with 1 of several screening tools designed to identify a family history that may be associated with an increased risk for potentially harmful mutations in breast cancer susceptibility genes (*BRCA1* or *BRCA2*). Women with positive screening results should receive genetic counseling and, if indicated after counseling, *BRCA* testing (B recommendation)."

The USPSTF (<http://www.uspreventiveservicestaskforce.org/>) is an independent panel of non-Federal experts in prevention and evidence-based medicine. The USPSTF conducts scientific evidence reviews of a broad range of clinical preventive health care services and develops recommendations for primary care clinicians and health systems.

The USPSTF guidelines focus on family history in unaffected individuals in the general population. The Tier 1 application, which follows the USPSTF guidelines, therefore focuses on this public health approach as well.

Women might be at increased risk of having *BRCA* mutations (<http://www.medscape.com/viewarticle/749018>) if their family history includes one or more of the following in their first- or second-degree relatives (maternal and paternal sides of the family are equally important):

- Multiple relatives with either breast or ovarian cancer;
- Breast cancer at a young age (under 50 years);
- Presence of breast and ovarian cancer among relatives;
- A relative with primary cancers of both breasts;
- One or more family members with two primary types of *BRCA*-related cancer;
- Presence of breast cancer in one or more male relatives;
- Ashkenazi Jewish ancestry;
- A relative with a known *BRCA* mutation.

The USPSTF recommendation notes that "several familial risk stratification tools are available to determine the need for in-depth genetic counseling, such as the Ontario Family History Assessment Tool, Manchester Scoring System, Referral Screening Tool, Pedigree Assessment Tool, and FHS-7 (<http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestfinalrs.pdf>) ."

Source: CDC Public Health Genomics - Genomics Implementation - Detailed Information on Tier 1 Applications - Hereditary Breast and Ovarian Cancer - [http://www.cdc.gov/genomics/implementation/toolkit/HBOC\\_1.htm](http://www.cdc.gov/genomics/implementation/toolkit/HBOC_1.htm) ([http://www.cdc.gov/genomics/implementation/toolkit/HBOC\\_1.htm](http://www.cdc.gov/genomics/implementation/toolkit/HBOC_1.htm))

The National Comprehensive Cancer Network also provides guidelines for Genetic/Familial High-Risk Assessment for Breast and Ovarian Cancer. These guidelines can be viewed after registering for a free account: [http://www.nccn.org/professionals/physician\\_gls/pdf/genetics\\_screening.pdf](http://www.nccn.org/professionals/physician_gls/pdf/genetics_screening.pdf) ([http://www.nccn.org/professionals/physician\\_gls/pdf/genetics\\_screening.pdf](http://www.nccn.org/professionals/physician_gls/pdf/genetics_screening.pdf)) .

## WHAT ARE THE BENEFITS OF *BRCA* RISK ASSESSMENT AND GENETIC COUNSELING AND TESTING (WHEN APPROPRIATE)?

Genetic counseling and appropriate genetic testing for *BRCA1* and *BRCA2* based on the USPSTF guidelines, followed by appropriate management, are expected to reduce morbidity and mortality due to breast and ovarian cancer.

For women with *BRCA* mutations, interventions that might reduce the risk of cancer or reduce mortality include "earlier, more frequent, or intensive cancer screening; risk-reducing medications (i.e., tamoxifen or raloxifene); and risk-reducing surgery (e.g., mastectomy or salpingo-oophorectomy) (<http://www.uspreventiveservicestaskforce.org/uspstf12/brcatest/brcatestfinalrs.pdf>) "; however, the USPSTF found that the strength of evidence varies across the types of interventions. In studies cited in the USPSTF recommendation, prophylactic bilateral mastectomy reduced breast cancer risk by 85% or more, and prophylactic oophorectomy reduced ovarian cancer risk by 85% or more and breast cancer risk by 53% or more. The USPSTF found the evidence lacking regarding the effect of intensive screening on clinical outcomes in women who are *BRCA* mutation carriers. The USPSTF cited randomized controlled trials finding that the medications tamoxifen and raloxifene reduced the incidence of invasive breast cancer among women at increased risk, but also noted that clinical trials of these medications have not been conducted specifically in women who are *BRCA* mutation carriers.

2010 National Health Interview Survey data is used to monitor the Healthy People 2020 objective (<http://www.healthypeople.gov/2020/topics-objectives/topic/genomics/objectives>) to "increase the proportion of women with a family health history of breast and/or ovarian cancer who receive genetic counseling." Survey data reveal that approximately 47% of women with relevant first-degree family histories have not had genetic counseling for HBOC. It is not known to what degree this is due to their providers not alerting them to their increased risk status and offering them genetic counseling and/or testing. Extrapolating from the National Health Interview Survey one can assume that in the United States a very large number of women with relevant family histories who might benefit from genetic counseling and possible genetic testing for HBOC have not utilized these services. Genetic counseling and evaluation for *BRCA* genetic testing based on the USPSTF recommendation is a preventive service now covered under the Affordable Care Act (ACA). Both genetic counseling and testing, if appropriate, are included in the ACA-covered preventive service for women whose family histories are consistent with USPSTF guidelines.

**Note:** Some but not all health insurers cover genetic counseling and testing for *BRCA1* and *BRCA2* when recommended by a provider. Some financial assistance may be available through testing providers or advocacy organizations. The Affordable Care Act (ACA) requires coverage of genetic counseling and testing as described in the USPSTF recommendation.

Source: CDC Public Health Genomics - Genomics Implementation - Detailed Information on Tier 1 Applications - Hereditary Breast and Ovarian Cancer - [http://www.cdc.gov/genomics/implementation/toolkit/HBOC\\_1.htm](http://www.cdc.gov/genomics/implementation/toolkit/HBOC_1.htm) ([http://www.cdc.gov/genomics/implementation/toolkit/HBOC\\_1.htm](http://www.cdc.gov/genomics/implementation/toolkit/HBOC_1.htm))

## WHAT ARE THE CLINICAL RECOMMENDATIONS FOR WOMEN ASSESSED AS BEING NOT AT INCREASED RISK FOR A *BRCA1* OR *BRCA2* GENE MUTATIONS?

It is important to confirm family history of breast and ovarian cancer with all patients. In addition, it is good practice to confirm that the patient does not have a strong family history of other cancers that may be linked to *BRCA1* and *BRCA2* gene mutations including fallopian tube, peritoneal, prostate, and pancreatic cancers.

The U.S. Preventive Services Task Force (USPSTF) (<http://www.uspreventiveservicestaskforce.org/uspstf/uspbsrgen.htm>) recommends against routine genetic counseling or *BRCA* testing for women whose family history is not associated with an increased risk for potentially harmful mutations in the *BRCA1* or *BRCA2* genes. (D recommendation).

Women assessed as "not at increased risk for a *BRCA* gene mutation" may still have a family history and other risk factors that increase their risk for developing cancer and thus impact their screening recommendations.

The following guidelines provide information on recommended screening for patients at increased risk for developing breast and ovarian cancers:

- National Comprehensive Cancer Network Genetic/Familial High-Risk Assessment: Breast and Ovarian Cancers Guidelines  
[http://www.nccn.org/professionals/physician\\_gls/PDF/genetics\\_screening.pdf](http://www.nccn.org/professionals/physician_gls/PDF/genetics_screening.pdf)  
[http://www.nccn.org/professionals/physician\\_gls/PDF/genetics\\_screening.pdf](http://www.nccn.org/professionals/physician_gls/PDF/genetics_screening.pdf)
- American Cancer Society Recommendations for Early Breast Cancer Detection in Women without Breast Symptoms  
 (scroll to page bottom for Recommendations for Women at High Risk for Breast Cancer)  
<http://www.cancer.org/cancer/breastcancer/moreinformation/breastcancerearlydetection/breast-cancer-early-detection-ac-s-recs>  
<http://www.cancer.org/cancer/breastcancer/moreinformation/breastcancerearlydetection/breast-cancer-early-detection-ac-s-recs>

The following guidelines provide information on recommended screening for patients at average risk for developing breast and ovarian cancer:

- The U.S. Preventive Services Task Force Recommendations for Breast Cancer Screening (Average Risk)  
<http://www.uspreventiveservicestaskforce.org/uspstf09/breastcancer/brcanrs.htm>  
<http://www.uspreventiveservicestaskforce.org/uspstf09/breastcancer/brcanrs.htm>
- National Comprehensive Cancer Network Breast Cancer Screening and Diagnosis Guidelines  
[http://www.nccn.org/professionals/physician\\_gls/pdf/breast-screening.pdf](http://www.nccn.org/professionals/physician_gls/pdf/breast-screening.pdf) ([http://www.nccn.org/professionals/physician\\_gls/pdf/breast-screening.pdf](http://www.nccn.org/professionals/physician_gls/pdf/breast-screening.pdf))
- American Cancer Society Guidelines for the Early Detection of Cancer  
<http://www.cancer.org/healthy/findcancerearly/cancerscreeningguidelines/american-cancer-society-guidelines-for-the-early-detection-of-cancer> (<http://www.cancer.org/healthy/findcancerearly/cancerscreeningguidelines/american-cancer-society-guidelines-for-the-early-detection-of-cancer>)

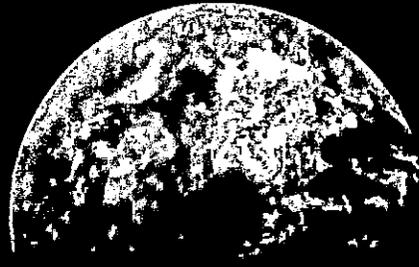
## WHAT CLINICAL OPTIONS ARE AVAILABLE TO REDUCE CANCER RISK IN *BRCA* MUTATION CARRIERS (CONFIRMED THROUGH GENETIC COUNSELING AND TESTING)?

The U.S. Preventive Services Task Force (USPSTF) (<http://www.uspreventiveservicestaskforce.org/uspstf/uspstf09/breastcancer/brcanrs.htm>) recommends that women with positive *BRCA* risk assessment results should be referred for genetic counseling and, if indicated after counseling, *BRCA* testing. (B recommendation). Genetic services should be provided by a trained genetics expert (e.g., genetic counselor, advanced practice nurse in genetics).

There are many available interventions to manage and reduce risk in people with a confirmed genetic susceptibility to breast and ovarian cancer. These may include modified screening plans and additional screening modalities (such as breast MRI), risk-reducing surgeries, and chemoprevention. Increasing data are available on the outcomes of these interventions; uncertainty is often considerable regarding the level of cancer risk associated with a positive family history (<http://www.cancer.gov/Common/PopUps/popDefinition.aspx?id=302456&version=HealthProfessional&language=English>) or genetic test. In this setting, personal preferences are likely to be an important factor in patients' decisions about risk reduction strategies.

The following resources provide detailed information on clinical interventions to reduce cancer risk in *BRCA* mutation carriers:

- National Cancer Institute Genetics of Breast and Ovarian Cancer PDQ  
<http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq> (<http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq>)
- Hereditary Breast and Ovarian Cancer Program's Genetic Testing Results Interpretation Table  
<http://www.nchpeg.org/hboc/result-interpretation-table> (<http://www.nchpeg.org/hboc/result-interpretation-table>)
- Hereditary Breast and Ovarian Cancer Program's Cancer Risks and Screening Guidelines  
<http://www.nchpeg.org/hboc/cancer-risks-screening-guidelines> (<http://www.nchpeg.org/hboc/cancer-risks-screening-guidelines>)
- Mayo Clinic Proceedings: Identification and Management of Women with *BRCA* Mutations or Hereditary Predisposition for Breast and Ovarian Cancer (<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2996153/> (<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2996153/>))
- UpToDate: Management of Hereditary Breast and Ovarian Cancer Syndrome and Patients with *BRCA* mutations  
<http://www.uptodate.com/contents/management-of-hereditary-breast-and-ovarian-cancer-syndrome-and-patients-with-brca-mutations>  
<http://www.uptodate.com/contents/management-of-hereditary-breast-and-ovarian-cancer-syndrome-and-patients-with-brca-mutations>)



# CANCER AND THE ENVIRONMENT

What You Need to Know  
What You Can Do

U.S. DEPARTMENT OF HEALTH  
AND HUMAN SERVICES

National Institutes  
of Health

National Cancer Institute

National Institute of Environmental  
Health Sciences

## CANCER AND THE ENVIRONMENT

This booklet was created by scientists at the National Cancer Institute (NCI) and the National Institute of Environmental Health Sciences (NIEHS) in response to many public requests for information. The content has been guided by responses from a series of *focus groups*\* that were conducted prior to producing the booklet. People from local communities throughout the country participated in these groups.

NCI and NIEHS are 2 of the 27 institutes/centers that make up the National Institutes of Health (NIH), an agency of the Federal Government's Department of Health and Human Services supported by your tax dollars. NIH is the major supporter of medical research in universities and academic centers throughout the country. To date, 102 Nobel Prize winners have been supported by funds from NIH, more than any other scientific institution in the world. For details, go to the NIH Web site at <http://www.nih.gov>.

NCI was established by Congress in 1937 as the Federal Government's principal agency for *cancer* research and training. Research projects include a broad range of topics: the cellular events in the development of cancer; the role of infectious agents or other agents in the environment or workplace; the role of genetic and hormonal factors; the interactions between environmental agents and genetic factors in the development of cancer; improved imaging techniques and biomarkers in the blood or urine for the early detection of cancer; and the role of diet and other chemicals in preventing cancer. Additional activities include tracking cancer trends, coordinating studies to test new drugs, and supporting new drug and vaccine development. Since the passage of the National Cancer Act in 1971, which broadened NCI's responsibilities, the institute has built an extensive network that includes regional and community cancer centers, specialized cancer physicians, and cooperative groups of researchers throughout the country and abroad to test new prevention and treatment agents. NCI's mission also includes the collection and dissemination of health information, programs to promote the incorporation of state-of-the-art cancer treatments into care of cancer patients, and the continuing care of cancer patients and their families. For more information, go to NCI's Web site at <http://www.cancer.gov>.

NIEHS was established by Congress in 1966 for the purpose of reducing human illness caused by hazardous substances in the environment. The National Toxicology Program, which is headquartered at NIEHS, helps coordinate toxicology studies among Federal agencies and identifies substances that might cause cancer. NIEHS conducts and supports extensive biomedical research, disease prevention, and intervention programs, as well as training, education, and community outreach efforts. NIEHS is a leader in understanding the effect of environmental pollution on birth and developmental defects, sterility, Alzheimer's and other brain and nerve disorders, pulmonary diseases, poverty and health, and cancer. For more information, go to the NIEHS Web site at <http://www.niehs.nih.gov>.

\*All terms in *bold italics* are defined in the glossary (see page 35).

The authors dedicate this publication to Dr. Susan Steber Fabro (1942-2002), a scientist at NCI, who provided the leadership to make the booklet a reality.

The importance of the environment can be seen in the differences in cancer rates throughout the world and the change in cancer rates when groups of people move from one country to another. For example, when Asians, who have low rates of prostate and breast cancer and high rates of stomach cancer in their native countries, immigrate to the United States, their prostate and breast cancer rates rise over time until they are nearly equal to or greater than the higher levels of these cancers in the United States. Likewise, their rates of stomach cancer fall, becoming nearly equal to the lower U.S. rates. Lifestyle factors such as diet, exercise, and being overweight are thought to play a major role in the trends for breast and prostate cancers, and infection with the *Helicobacter pylori* bacterium is an important risk factor for stomach cancer. Recently, the rapid rise in the rates of **colorectal** cancer in Japan and China suggests an environmental cause such as lifestyle factors.

Different environmental exposures are linked to specific kinds of cancer. For example, exposure to asbestos is linked primarily to lung cancer, whereas exposure to benzidine, a chemical found in certain dyes (see page 17), is associated with bladder cancer. In contrast, smoking is linked to cancers of the lung, bladder, mouth, colon, kidney, throat, voice box, esophagus, lip, stomach, cervix, liver, and pancreas.

### Factors Inside the Body

Certain factors inside the body make some people more likely to develop cancer than others. For instance, some people either inherit or acquire the following conditions: altered **genes** in the body's **cells**, abnormal hormone levels in the bloodstream, or a weakened immune system. Each of these factors may make an individual more **susceptible** to cancer.



One of the ways scientists know that genes play an important role in the development of cancer is from studying certain rare families where family members over several generations develop similar cancers. It appears that these families are passing on an altered gene that carries with it a high chance of getting cancer. Several genes that greatly increase a person's chance of developing certain cancers (e.g., colon, breast, and ovary) have been identified. Only a very small percentage of people in the general population have abnormal copies of these genes. Cancers caused by these genes, known as **familial cancers**, account for only two to five percent of all cancers.

Gene alterations may also contribute to individual differences in susceptibility to environmental **carcinogens** (cancer-causing substances). For instance, people differ in their ability to eliminate cancer-causing agents from their body to which they have been exposed, or to repair **DNA** damage that was caused by such agents. These gene alterations may also be passed on in families and account for higher rates of cancer in these families. Higher rates of cancer in families may also be related to shared environmental exposures like diet or exposure to carcinogens at work.

smoking) help to protect us from harmful exposures. However, over time, substances in the environment may cause gene alterations, which accumulate inside our cells. While many alterations have no effect on a person's health, permanent changes in certain genes can lead to cancer.

The chance that an individual will develop cancer in response to a particular environmental agent depends on several interacting factors—how long and how often a person is exposed to a particular substance, his/her exposure to other agents, genetic factors, diet, lifestyle, health, age, and gender. For example, diet, alcohol consumption, and certain medications can affect the levels of chemicals in the body that break down cancer-causing substances.

Because of the complex interplay of many factors, it is not possible to predict whether a specific environmental exposure will cause a particular person to develop cancer. We know that certain genetic and environmental factors increase the risk of developing cancer, but we rarely know exactly which combination of factors is responsible for a person's specific cancer. This also means that we usually don't know why one person gets cancer and another does not.

#### INTERPLAY OF FACTORS

There are particular patterns of gene alterations and environmental exposures that make people both more susceptible or more resistant to cancer. One of the challenging areas of research today is trying to identify the unique combinations of these factors that explain why one person will develop cancer and another will not.

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### THE NATURE OF CANCER

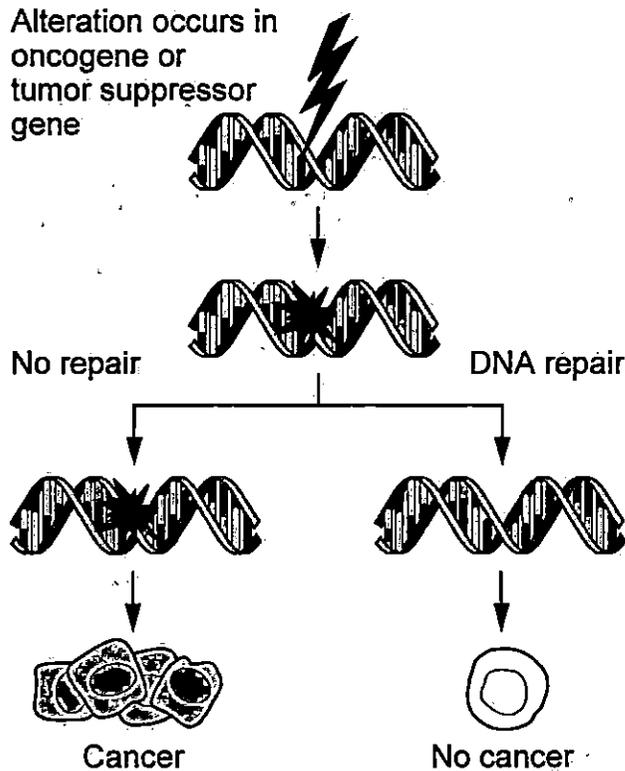
There are more than 100 types of cancer. Cancer begins inside a cell, the basic building block of all living things. Normally, when the body needs more cells, older ones die off and younger cells divide to form new cells that take their place. When cancer develops, however, the orderly process of producing new cells breaks down. Cells continue to divide when new cells are not needed, and a growth or extra mass of cells called a **tumor** is formed. Over time, changes may take place in tumor cells that cause them to invade and interfere with the function of normal **tissues**.

It takes many years for the development of a tumor and even more years until detection of a tumor and its spread to other parts of the body. People exposed to carcinogens from smoking cigarettes, for example, generally do not develop detectable cancer for 20 to 30 years.

There is much evidence to suggest that permanent changes in our genes are responsible for tumor development. These can be inherited or acquired throughout one's lifetime. Scientists have identified more than 300 altered genes

BRCA1  
is a tumor  
suppressor  
gene

that can play a role in tumor development. An alteration in growth-promoting genes, known as **oncogenes**, for example, can signal the cell to divide out of control, similar to having a gas pedal stuck to the floorboard. On the other hand, an alteration in **tumor suppressor genes**, which normally serve as brakes for dividing cells, will allow cells with damaged DNA to continue dividing, rather than repairing the DNA or eliminating the injured cells.



*An alteration in growth-promoting genes, known as oncogenes, can signal the cell to divide out of control. An alteration in tumor suppressor genes will allow cells with damaged DNA to continue dividing, rather than repairing the DNA or eliminating the injured cells.*

One explanation for the fact that cancer occurs more frequently in older people may be that, for a tumor to develop, a cell must acquire several gene alterations that accumulate as we age. As the graph on page 6 illustrates, less than 0.1 percent of the total number of cancer cases in the United States occur in people under the age of 15, whereas nearly 80 percent occur in people age 55 or older.

### Types of Tumors

Tumors are classified as either benign or malignant. **Benign tumors** are not cancer and do not spread to other parts of the body.

1. JAMA. 2017 Jun 20;317(23):2402-2416. doi: 10.1001/jama.2017.7112.

Risks of Breast, Ovarian, and Contralateral Breast Cancer for BRCA1 and BRCA2 Mutation Carriers.

Kuchenbaecker KB(1), Hopper JL(2), Barnes DR(3), Phillips KA(4), Mooij TM(5), Roos-Blom MJ(6), Jervis S(7), van Leeuwen FE(5), Milne RL(8), Andrieu N(9), Goldgar DE(10), Terry MB(11), Rookus MA(5), Easton DF(3), Antoniou AC(3), BRCA1 and BRCA2 Cohort Consortium, McGuffog L(3), Evans DG(12), Barrowdale D(3), Frost D(3), Adlard J(13), Ong KR(14), Izatt L(15), Tischkowitz M(16), Eeles R(17), Davidson R(18), Hodgson S(19), Ellis S(3), Nogues C(20), Lasset C(21), Stoppa-Lyonnet D(22), Fricker JP(23), Faivre L(24), Berthet P(25), Hooning MJ(26), van der Kolk LE(27), Kets CM(28), Adank MA(29), John EM(30), Chung WK(31), Andrulis IL(32), Southey M(33), Daly MB(34), Buys SS(35), Osorio A(36), Engel C(37), Kast K(38), Schmutzler RK(39), Caldes T(40), Jakubowska A(41), Simard J(42), Friedlander ML(43), McLachlan SA(44), Machackova E(45), Foretova L(45), Tan YY(46), Singer CF(47), Olah E(48), Gerdes AM(49), Arver B(50), Olsson H(51).

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Importance: The clinical management of BRCA1 and BRCA2 mutation carriers requires accurate, prospective cancer risk estimates.

Objectives: To estimate age-specific risks of breast, ovarian, and contralateral breast cancer for mutation carriers and to evaluate risk modification by family cancer history and mutation location.

Design, Setting, and Participants: Prospective cohort study of 6036 BRCA1 and

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3820 BRCA2 female carriers (5046 unaffected and 4810 with breast or ovarian cancer or both at baseline) recruited in 1997-2011 through the International BRCA1/2 Carrier Cohort Study, the Breast Cancer Family Registry and the Kathleen Cunningham Foundation Consortium for Research into Familial Breast Cancer, with ascertainment through family clinics (94%) and population-based studies (6%). The majority were from large national studies in the United Kingdom (EMBRACE), the Netherlands (HEBON), and France (GENEPSO). Follow-up ended December 2013; median follow-up was 5 years.

Exposures: BRCA1/2 mutations, family cancer history, and mutation location.

Main Outcomes and Measures: Annual incidences, standardized incidence ratios, and cumulative risks of breast, ovarian, and contralateral breast cancer.

Results: Among 3886 women (median age, 38 years; interquartile range [IQR], 30-46 years) eligible for the breast cancer analysis, 5066 women (median age, 38 years; IQR, 31-47 years) eligible for the ovarian cancer analysis, and 2213 women (median age, 47 years; IQR, 40-55 years) eligible for the contralateral breast cancer analysis, 426 were diagnosed with breast cancer, 109 with ovarian cancer, and 245 with contralateral breast cancer during follow-up. The cumulative breast cancer risk to age 80 years was 72% (95% CI, 65%-79%) for BRCA1 and 69% (95% CI, 61%-77%) for BRCA2 carriers. Breast cancer incidences increased rapidly in early adulthood until ages 30 to 40 years for BRCA1 and until ages 40 to 50 years for BRCA2 carriers, then remained at a similar, constant incidence (20-30 per 1000 person-years) until age 80 years. The cumulative ovarian cancer risk to age 80 years was 44% (95% CI, 36%-53%) for BRCA1 and 17% (95% CI, 11%-25%) for BRCA2 carriers. For contralateral breast cancer, the cumulative risk 20 years after breast cancer diagnosis was 40% (95% CI, 35%-45%) for BRCA1 and 26% (95% CI, 20%-33%) for BRCA2 carriers (hazard ratio [HR] for comparing BRCA2 vs BRCA1, 0.62; 95% CI, 0.47-0.82; P=.001 for difference). Breast cancer risk increased with increasing number of first- and second-degree relatives diagnosed as having breast cancer for both BRCA1 (HR for  $\geq 2$  vs 0 affected relatives, 1.99; 95% CI, 1.41-2.82; P<.001 for trend) and BRCA2 carriers (HR, 1.91; 95% CI, 1.08-3.37; P=.02 for trend). Breast cancer risk was higher if mutations were located outside vs within the regions bounded by positions c.2282-c.4071 in BRCA1 (HR, 1.46; 95% CI, 1.11-1.93; P=.007) and c.2831-c.6401 in BRCA2 (HR, 1.93; 95% CI, 1.36-2.74; P<.001).

Conclusions and Relevance: These findings provide estimates of cancer risk based on BRCA1 and BRCA2 mutation carrier status using prospective data collection and demonstrate the potential importance of family history and mutation location in risk assessment.

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## Comparison of the genotoxic effects induced by 50 Hz extremely low-frequency electromagnetic fields and 1800 MHz radiofrequency electromagnetic fields in GC-2 cells.

Duan W<sup>1</sup>, Liu C, Zhang L, He M, Xu S, Chen C, Pi H, Gao P, Zhang Y, Zhong M, Yu Z, Zhou Z.

### Author information

#### Abstract

Extremely low-frequency electromagnetic fields (ELF-EMF) and radiofrequency electromagnetic fields (RF-EMF) have been considered to be possibly carcinogenic to humans. However, their genotoxic effects remain controversial. To make experiments controllable and results comparable, we standardized exposure conditions and explored the potential genotoxicity of 50 Hz ELF-EMF and 1800 MHz RF-EMF. A mouse spermatocyte-derived GC-2 cell line was intermittently (5 min on and 10 min off) exposed to 50 Hz ELF-EMF at an intensity of 1, 2 or 3 mT or to RF-EMF in GSM-Talk mode at the specific absorption rates (SAR) of 1, 2 or 4 W/kg. After exposure for 24 h, we found that neither ELF-EMF nor RF-EMF affected cell viability using Cell Counting Kit-8. Through the use of an alkaline comet assay and immunofluorescence against  $\gamma$ -H2AX foci, we found that ELF-EMF exposure resulted in a significant increase of DNA strand breaks at 3 mT, whereas RF-EMF exposure had insufficient energy to induce such effects. Using a formamidopyrimidine DNA glycosylase (FPG)-modified alkaline comet assay, we observed that RF-EMF exposure significantly induced oxidative DNA base damage at a SAR value of 4 W/kg, whereas ELF-EMF exposure did not. Our results suggest that both ELF-EMF and RF-EMF under the same experimental conditions may produce genotoxicity at relative high intensities, but they create different patterns of DNA damage. Therefore, the potential mechanisms underlying the genotoxicity of different frequency electromagnetic fields may be different.

PMID: 25688995 DOI: [10.1667/RR13851.1](https://doi.org/10.1667/RR13851.1)

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[Eur J Gynaecol Oncol](#). 2014;35(3):264-9.

## Relationship between exposure to extremely low-frequency electromagnetic fields and breast cancer risk: a meta-analysis.

[Zhao G](#), [Lin X](#), [Zhou M](#), [Zhao J](#).

### Abstract

**OBJECTIVE:** To comprehensively analyze the relationship between human exposure to extremely low frequency electromagnetic fields (ELF-EMFs) and breast cancer and to discuss the potential risk of ELF-EMFs to human breast cancer.

**MATERIALS AND METHODS:** Sixteen research reports of case-control studies which were published from 2000 to 2007 were collected. The fixed effect model (FEM) or the random effect model (REM) was chosen to calculate total ORs depending on the outcomes of the test of homogeneity (Q test); the subgroup was analyzed with the menopause and the non-menopause.

**OUTCOME:** Sixteen research outcome was  $OR_{DL} = 1.10$ , 95% CI = (1.01, 1.20), the  $OR(MH)$  of the non-menopause status group was 1.25, 95% CI = (1.05, 1.49), the  $OR(MH)$  of the menopause status group was  $OR(MH) = 1.04$ , 95% CI = (0.93, 1.18).

**CONCLUSION:** The authors found that ELF-EMFs may be increase the risk of human breast cancer. The women's exposure to ELF-EMFs may be the risk factor of breast cancer when they are non-menopausal.

PMID: 24984538

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**Format:** Abstract[Med Lav](#). 1994 Nov-Dec;85(6):447-62.

## [Carcinogenic risk of extremely-low-frequency electromagnetic fields: state of the art].

[Article in Italian]

[Pira E<sup>1</sup>](#), [Zanetti C](#), [Saia B](#).

### Author information

### Abstract

This paper summarizes the published literature and current problems relating to possible cancerogenic effects of occupational and residential exposure to ELF electromagnetic fields at levels slightly above ambient background. There are several suggestions that such an exposure may increase the risk of cancer, but these studies failed to provide conclusive indications. The present state of uncertainty led to a variety of recommendations and statements being made concerning restrictions to the exposure of people to ELF electromagnetic fields. Attempts to detect direct chromosomal damage from ELF electromagnetic fields have proven negative, while results on cancer promotion have been controversial. On the basis of several epidemiological studies on occupational exposure, an increased risk of leukemia, brain cancer and male breast cancer is apparent; the literature on residential exposure provides some evidence of an effect on childhood cancer, especially leukemia; however, when interpreting these results some major methodological concerns should be kept in mind. In conclusion, the public concern and potential public health impact of this environmental agent argue strongly for addressing further research in order to identify mechanisms of action on biological systems, to define the proper assessment of exposure and to obtain good epidemiological evidence.

PMID: 7731404

[Indexed for MEDLINE]

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8196082[uid]

**Format:** AbstractJ Natl Cancer Inst. 1994 Jun 15;86(12):921-5.

## Breast cancer mortality among female electrical workers in the United States.

Loomis DP<sup>1</sup>, Savitz DA, Ananth CV.

### Author information

### Abstract

**BACKGROUND:** Previous epidemiologic studies have suggested that exposure to electric or magnetic fields in occupational and residential environments may cause cancer. Recent experimental findings provide some support for the hypothesis that exposure to extremely low-frequency electromagnetic fields reduces the pineal gland's nocturnal production of the hormone melatonin, thereby increasing susceptibility to sex hormone-related cancers such as breast cancer.

**PURPOSE:** Our purpose was to assess the evidence that cancer of the female breast might be associated with exposure to extremely low-frequency electromagnetic fields.

**METHODS:** Records of women who had breast cancer as the underlying cause of their death (ICD-9 174) and control subjects (four per case) were selected from computer files of U.S. mortality data for the years 1985-1989. Women 20 years and older at the time of their death were eligible for inclusion if they were residents of and died in one of the 24 states that provided death certification records with occupation and industry codes to the National Center for Health Statistics for at least 1 year during the study interval. Data from death certificates were used to classify the case and control subjects with regard to potential occupational exposure to electric and magnetic fields. Control subjects were a random sample of women who died of any other underlying cause, excluding leukemia and brain cancer.

**RESULTS:** The data analysis contrasted 68 women with breast cancer and 199 controls, all with electrical occupations, with 27,814 women with breast cancer and 110,750 controls, all of whom had other occupations. Electrical workers had excess mortality from breast cancer relative to other employed women [odds ratio (OR) = 1.38; 95% confidence interval (CI) = 1.04-1.82]. Adjusted ORs for specific electrical occupations were 1.73 (95% CI = 0.92-3.25) for electrical engineers, 1.28 (95% CI = 0.79-2.07) for electrical technicians, and 2.17 (95% CI = 1.17-4.02) for telephone installers, repairers, and line workers. There was no excess of breast cancer, however, in seven other occupations held more frequently by women and also involving potentially elevated electrical exposures, including telephone operators, data keyers, and computer operators and programmers.

**CONCLUSIONS:** In light of the limitations inherent in death certification data and the design of this study, any conclusions regarding the hypothesis that exposure to extremely low-frequency electromagnetic fields causes breast cancer among women must be limited. Nevertheless, our

findings are broadly consistent with that hypothesis and encourage further investigation with improvements in study design and data quality.

### Comment in

Re: Breast cancer mortality among female electrical workers in the United States. [J Natl Cancer Inst. 1995]

Re: Breast cancer mortality among female electrical workers in the United States. [J Natl Cancer Inst. 1994]

Re: Are electric or magnetic fields affecting mortality from breast cancer in women? [J Natl Cancer Inst. 1994]

Are electric or magnetic fields affecting mortality from breast cancer in women? [J Natl Cancer Inst. 1994]

PMID: 8196082

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## Life insurance companies deny coverage to those with cancer genes like BRCA

Meredith Knight | May 8, 2016 | Genetic Literacy Project

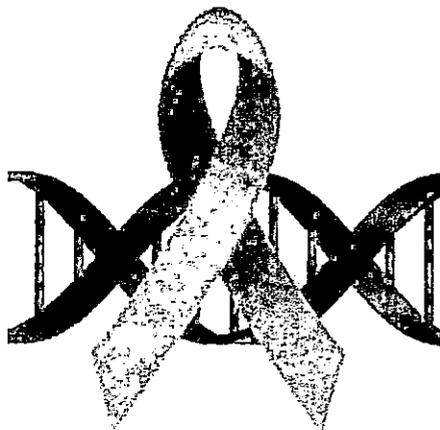
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Many women who've been diagnosed with breast cancer get tested for cancer genes like BRCA 1 and 2. Even after those tests, women can still get healthcare because of Federal legislation barring health insurers from discriminating on a genetic basis. However, the same can not be said of life insurance and men and women who have taken genetic tests for a variety of reasons are being denied coverage.



Fast Company tells the story of a healthy young woman who now can't get life insurance because of a BRCA test:

*Jennifer Marie [not her real name] should be an ideal candidate for life insurance: She's 36, gainfully employed, and has no current medical issues. But on September 15 last year, Jennifer Marie's application for life insurance was denied.*

*"Unfortunately after carefully reviewing your application, we regret that we are unable to provide you with coverage because of your positive BRCA 1 gene," the letter reads. In the U.S., about one in 400 women have a BRCA 1 or 2 gene, which is associated with increased risk of breast and ovarian cancer.*

The Genetic Information Nondiscrimination Act (GINA) protects citizens from genetic discrimination. In 2008 when it passed, the law was already woefully behind the times and under powered. GINA's protections are limited to just two instances: employment and health insurance. Jennifer Marie's story illustrates the emerging dilemma of people who want to know about their genetic risk for disease, but who also want to purchase life insurance to financially assist their families after death. And it shows how unprepared we are for all the potential commercial and administrative uses of our genetic information.

Life insurance companies don't require people to get genetic tests when they're applying for coverage. But companies have started to ask if potential clients have used genetic testing and what those results were. Failing to disclose those tests can result in a rejected application. But as Jennifer found out, the results of the tests can also be cause for rejection. Insurance companies have always asked about family health histories to help evaluate who to cover.

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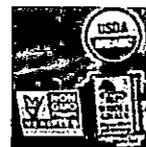
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## [Effects of GSM 1800 MHz radiofrequency electromagnetic fields on DNA damage in Chinese hamster lung cells].

[Article in Chinese]

Zhang DY<sup>1</sup>, Xu ZP, Chiang H, Lu DQ, Zeng QL.

### Author information

### Abstract

**OBJECTIVE:** To study the effects of GSM 1800 MHz radiofrequency electromagnetic fields (RF EMF) on DNA damage in Chinese hamster lung (CHL) cells.

**METHODS:** The cells were intermittently exposed or sham-exposed to GSM 1800 MHz RF EMF (5 minutes on/10 minutes off) at a special absorption rate (SAR) of 3.0 W/kg for 1 hour or 24 hours. Meanwhile, cells exposed to 2-acetylaminofluorene, a DNA damage agent, at a final concentration of 20 mg/L for 2 hours were used as positive control. After exposure, cells were fixed by using 4% paraformaldehyde and processed for phosphorylated form of H2AX (gammaH2AX) immunofluorescence measurement. The primary antibody used for immunofluorescence was mouse monoclonal antibody against gammaH2AX and the secondary antibody was fluorescein isothiocyanate (FITC)-conjugated goat anti-mouse IgG. Nuclei were counterstained with 4, 6-diamidino-2-phenylindole (DAPI). The gammaH2AX foci and nuclei were visualized with an Olympus AX70 fluorescent microscope. Image Pro-Plus software was used to count the gammaH2AX foci in each cell. For each exposure condition, at least 50 cells were selected to detect gammaH2AX foci. Cells were classified as positive when more than five foci were detected. The percentage of gammaH2AX foci positive cells was adopted as the index of DNA damage.

**RESULTS:** The percentage of gammaH2AX foci positive cell of 1800 MHz RF EMF exposure for 24 hours (37.9 +/- 8.6)% or 2-acetylaminofluorene exposure (50.9 +/- 9.4)% was significantly higher compared with the sham-exposure (28.0 +/- 8.4)%. However, there was no significant difference between the sham-exposure and RF EMF exposure for 1 hour (31.8 +/- 8.7)%.

**CONCLUSION:** 1800 MHz RF EMF (SAR, 3.0 W/kg) for 24 hours might induce DNA damage in CHL cells.

PMID: 16836873

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## **SECTION 7**

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# **The Cellular Stress Response: EMF-DNA Interaction**

**2012 Supplement**

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Prepared for the BioInitiative Working Group  
September 2012

## ABSTRACT

The research on stress proteins stimulated by EMF was reviewed by the author in the BioInitiative Report (2007) as well as in the special issue of Pathophysiology (2009) devoted to EMF. This review emphasizes the more recent research on the mechanism of interaction of EMF with DNA. It appears that the DNA molecule is particularly vulnerable to damage by EMF because of the coiled-coil configuration of the compacted molecule in the nucleus. The unusual structure endows it with the self similarity of a fractal antenna and the resulting sensitivity to a wide range of frequencies. The greater reactivity of DNA with EMF, along with a vulnerability to damage, underscores the urgent need to revise EMF exposure standards in order to protect the public. Recent studies have also exploited the properties of stress proteins to devise therapies for limiting oxidative damage and reducing loss of muscle strength associated with aging.

## I. INTRODUCTION

The cellular stress response is a protective reaction of individual cells to potentially harmful stimuli in the environment. It is characterized by the synthesis of a class of proteins referred to as stress proteins. The cellular stress response differs from the more familiar responses of entire organisms to stresses that lead to secretion of cortisol and adrenalin and that result in the activation of various systems throughout the body. The cellular stress response, as the name indicates, is a specific response of individual cells, and stress proteins are the chemical agents that also serve as markers.

The cellular stress response was first described as a reaction to elevated temperature (Ritossa, 1962), which accounts for the proteins initially being called heat shock proteins. Several physical and chemical environmental influences have since been found to evoke the response, and in 1994, Goodman and Blank (1994) were the first to show that the response was stimulated by EMF. In fact, the cells were far more sensitive to EMF than to thermal stimuli, the threshold energy of the EMF stimulus being more than one billion times weaker than an effective thermal stimulus (Blank , Goodman, 1994).

The 'heat shock' response, i.e., hsp synthesis, is activated by a variety of potentially harmful stresses, including physical stimuli like pH and osmotic pressure changes, as well as chemicals such as ethanol and toxic metal ions like Cd<sup>2+</sup>. The ability of EMF in the power frequency (extremely low frequency, ELF) range (Goodman, Blank, 1998) to evoke this response was followed by reports of similar effects due to radio frequency (RF) fields (de Pomerai et al. 2003) and amplitude modulated RF fields (Czyz et al, 2004).

The finding that EMF evoked the cellular stress response had obvious and important biological implications:

- Because the cellular stress response is a reaction to potentially harmful stimuli in the environment, the cells were asserting that *EMF is potentially harmful* to cells.
- Because EMF stimulated protein synthesis, it meant that *EMF causes the two strands of DNA to come apart* for the protein code to be read and for synthesis to proceed.
- Since *EMF can interact with DNA*, it can cause *errors during replication*, as well as during protein synthesis, and higher energy EMF could be expected to cause *DNA strand breaks*, as has been observed (Lai and Singh, 1995).
- The incremental increase of DNA strand breaks with increases in field strength indicates a *dose-response*, evidence in support of EMF as the responsible agent.

## II. CELLULAR STRESS PROTEINS ARE A NEW CLASS OF PROTEINS

Proteins are important components of cells and make up about 50% of the dry weight of most cells. The many different proteins are classified according to their functions, and stress proteins are now recognized as a new class of proteins with functions related to cell protection. Stress proteins join such well-known categories as contractile proteins ( e.g. actin, myosin), catalytic proteins or enzymes ( e.g. pepsin, amylase), transport proteins

(e.g. ATPases for ions across membranes, hemoglobins for blood gases, cytochromes for electrons), etc. Stress proteins were originally described as being synthesized in response to external stimuli and that is currently the area of greatest interest. However, they are also present constitutively.

Cellular stress proteins are synthesized when cells come in contact with stimuli that cause damage to macromolecules (Kultz, 2005), and the stress proteins aid in the repair and transport of these molecules. Because the first stimulus identified was an increase in temperature, the proteins were called 'heat shock' proteins and designated using the original terminology that starts with 'hsp' (for 'heat shock' protein) and a number equal to the molecular weight in kilodaltons.

The transition from heat shock protein to stress protein should alert (perhaps even alarm) the government agencies responsible for setting EMF safety standards. The thermal stimuli that evoked synthesis of protective proteins were believed to be dangerous for cells, but now we see that non-thermal EMF stimuli cause the same protective reactions in cells. The heat shock response and the EMF stress response both relate to the threshold for biological damage, and we should realize that EMF damage is caused by non-thermal stimuli. Compared to the energy needed to stimulate heat shock, EMF requires but a small fraction of the thermal energy needed to produce the same response (Blank et al., 1992).

The government agencies that assess safety of EMF exposure assume that danger is associated with an increase in temperature, i.e., a thermal criterion. It is clear from the responses of cells that the safety of EMF exposure, as indicated by the synthesis of protective stress proteins, is unrelated to the temperature increase. The cells are very sensitive to EMF, and the protective biological response to EMF occurs long before there is a significant change in temperature. It should be obvious that EMF safety standards are based on false assumptions and must be revised to reflect the scientific evidence. Non-thermal EMF stimuli are potentially harmful.

### III. PROTEIN SYNTHESIS

The stress response, like all protein synthesis, indicates that all of the different physical and chemical stimuli that can initiate this response cause the two strands of DNA to come apart for the amino acid code for protein synthesis code to be read. Therefore, the observed stress protein synthesis is evidence that EMF has interacted with the DNA to start this process. The research showing that EMF in both the ELF and RF frequency ranges can also cause DNA strand breaks (Lai, Singh, 1995; 1996; Reflex Report 1994), suggests that the two phenomena are due to the same interaction mechanism, and that there is greater molecular damage with greater EMF energy.

Many research papers and some reviews have been published since the cellular stress response was reported to be stimulated by EMF. In addition to earlier reviews on EMF stimulation of the cellular stress response in the ELF (Goodman, Blank, 1998) and RF (Cotgreave, 2005) ranges, the subject was reviewed in Pathophysiology (Blank, 2009). Also, Calderwood (2007) has edited the volume on cell stress proteins in volume 7 of the series Protein Reviews. A recent (ICEMS, 2010) review on EMF and Bio-Effects includes many papers focused on a variety of possible EMF interaction mechanisms, but does not review the stress response, the stimulation of DNA or biosynthesis.

Section 7 of the Bioinitiative Report summarized both ELF and RF studies, mainly at frequencies 50 Hz, 60 Hz, 900MHz and 1.8 GHz. The citations in that review were not exhaustive, but the different frequencies and many different cells indicated the diversity of results on stimulation of DNA and stress protein synthesis. The many different types of cells that respond to EMF, both *in vivo* and *in vitro*, include epithelial, endothelial and epidermal cells, cardiac muscle cells, fibroblasts, yeast, *E. coli*, developing chick eggs, and dipteran cells.

It is clear that the stress response does not occur in reaction to EMF in all types of cells, and that tissue cultured cells (as opposed to natural cells) are less likely to show an effect of EMF, probably because immortalized cells have been changed significantly to enable them to live indefinitely in unnatural laboratory conditions. Even the same cell line from

two different suppliers can respond differently. Jin et al. (1997) showed that HL60 cells from one supplier reacted to EMF while identically labeled cells from another supplier did not respond. Some cancer cells (e.g., MCF7 breast cancer cells) have responded to EMF (Liburdy et al., 1993; Lin et al., 1998), and Czyz et al. (2004) found that p53-deficient embryonic stem cells showed an increased EMF response, but the wild type did not. Ivancits et al., (2005) found no genotoxic effects (i.e., DNA damage) in lymphocytes, monocytes and skeletal muscle cells, but did find effects with fibroblasts, melanocytes and rat granulosa cells. Lantow et al. (2006) and Simko et al. (2006) found that blood elements, such as lymphocytes and monocytes did not respond. Obviously, the cellular stress response is widespread but not universal.

#### IV. MECHANISM OF PROTEIN SYNTHESIS BY EMF

The stress response has provided an opportunity to investigate EMF interaction with DNA, and in particular, how this results in stimulating DNA to start the synthesis of proteins. Because the DNA sequence is known for hsp70, it was possible to study the effects of changes in the DNA sequence on protein synthesis. As a result of these experiments, it was possible to identify two distinct regions in the promoter region of the HSP 70 gene - an EMF sensitive region that was not sensitive to increased temperature, as well as a region sensitive only to temperature. The EMF sensitive domain contains number of nCTCTn myc-binding sites relative to the transcription initiation site and upstream of the temperature sensitive binding sites (Lin et al. 1999; 2001). These electromagnetic response elements (EMREs) are also found on the *c-myc* promoter which also reacts to EMF.

The EMF sensitivity of the DNA sequences, nCTCTn, was demonstrated by transfecting these sequences into CAT and Luciferase reporter genes and stimulating those genes (with EMF) to synthesize CAT and luciferase, respectively (Lin et al., 1999; 2001). Thus, the HSP70 promoter contains different DNA regions that are specifically sensitive to thermal and non-thermal stressors. This biological mechanism is obviously based on direct interaction with specific segments of DNA, and there is reason to believe that EMF can interact similarly with other segments of DNA. In our experiments, induction of

increased levels of hsp70 by EMF is rapid and occurs at extremely low levels of energy input, 14 orders of magnitude lower than with a thermal stimulus (Blank et al. 1994).

#### V. EMF INTERACTION WITH SIGNALING PATHWAYS

EMF penetrate cells unattenuated and so can interact directly with the DNA in the cell nucleus, as well as with other cell constituents. The above-cited experiments demonstrating the ability of electromagnetic response elements (EMREs) to interact with EMF, after being transferred to another DNA chain, is further support for direct EMF-DNA interaction as the most likely mechanism for EMF initiation of the cellular stress response.

In contrast to EMF, most biological agents are impeded by membranes and require special mechanisms to gain access to the cell interior. Friedman et al, (2007) have demonstrated that, in those situations, the initial step in transmitting extracellular information from the plasma membrane to the nucleus of the cell occurs when NADH oxidase rapidly generates reactive oxygen species (ROS). These ROS stimulate matrix metalloproteinases that allow them to cleave and release heparin binding epidermal growth factor. This secreted factor activates the epidermal growth receptor, which in turn activates the extracellular signal regulated kinase 1/2 (ERK) cascade. The ERK cascade is one of the four mitogen-activated protein kinase (MAPK) signaling cascades that regulate transcriptional activity in response to extracellular stimuli.

Stress protein synthesis can occur by direct interaction of EMF with DNA, as well as by membrane mediated stimulation via chemical signaling. While both mechanisms are possible, it is of interest to note that the body responds directly to physical inputs when there is a need for a rapid response. The body cannot rely upon slowly responding pathways for the synthesis of a relatively large amount of urgently needed protein molecules. The signal pathways function primarily as a mechanism for maintaining homeostasis by minimizing change and responding slowly to stimuli.

## VI. INSIGHTS FROM MUSCLE PROTEIN SYNTHESIS

EMF stimulated protein synthesis may appear to be an unnatural mechanism, but it is essentially the same as the natural process in striated muscle. The only difference is that the electrons in DNA are driven by EMF, while in striated muscle, they are driven by the changes in electric (membrane) potential that cause contraction. Striated muscle is a tissue that requires steady protein synthesis to ensure proper function. Protein synthesis is initiated by the same electric currents that stimulate the muscle contractions. Body builders know that one must stimulate muscle contraction in order to increase muscle mass, and biologists have shown that the electric currents that flow across the muscle membranes during contraction pass through the DNA in the muscle nuclei and stimulate protein synthesis.

Muscle nuclei are not spread evenly throughout a muscle fiber, but are located near the muscle membranes that carry the currents. This means that the DNA in the nuclei can be stimulated every time the muscle is stimulated. The estimated magnitude of electric field along the muscle nuclei,  $\sim 10\text{V/m}$ , provides a large safety margin in muscle, since fields as low as  $3\text{mV/m}$  were found to stimulate biosynthesis in HL60 cells (Blank et al, 1992).

Studies showing effects of EMF on electron transfer reactions in solution suggest that ionic (electric) currents affect electron movements within DNA in much the same way (Blank, 1995). Both electric and EMF (AC magnetic fields) stimulate protein synthesis in HL60 cells and have similar effects on electron transfer in the Na,K-ATPase (Blank and Soo, 2001a; 2001b). This suggests that interaction with DNA, of both electric fields and EMF, initiate stress protein synthesis by a similar mechanism.

Studies on muscle protein synthesis also suggest the possibility of a

frequency code that controls the particular segment of DNA that is activated. Studies have shown that different proteins can be synthesized by changing the frequency of the action potentials that stimulate the process. These experiments were possible because 'fast' and 'slow' muscles contract at different rates because they are composed of different proteins. For this reason it was possible to stimulate muscles at different rates and to study changes in the proteins as a result of changing the frequency of the action potentials (Pette, Vrbova, 1992). The review by Blank (1995) includes many additional experiments that show the importance of the frequency in controlling the segment of the muscle DNA that is affected by the current and translated into protein.

Studies of effects of EMF on well characterized electron transfer reactions, involving cytochrome oxidase, ATP hydrolysis by Na,K-ATPase, and the Belousov-Zhabotinski (BZ) redox reaction, have shown that:

- EMF can accelerate electron transfer rates
- EMF acts as a force that competes with the chemical forces driving a reaction. This means that the effect of EMF varies inversely with the intrinsic reaction rate, and that EMF effects are only seen when intrinsic rates are low. (*N.B. EMF has a greater effect when the system is in a rundown state.*)
- Experimentally determined thresholds are low ( $\sim 0.5 \mu\text{T}$ ).
- Effects vary with frequency, with different optima for the reactions studied: The two enzymes showed broad frequency optima close to the reaction turnover numbers for Na,K-ATPase (60 Hz) and cytochrome oxidase (800 Hz), suggesting that EMF interacted optimally when in synchrony with the molecular kinetics. EMF interactions with DNA in both ELF and RF ranges and do not appear to involve electron transfer reactions with well-defined kinetics.

The effects of EMF on electron transfer reactions were studied in the ELF frequency range, and one would expect differences in the RF range. However, the situation is more

complicated. The effects of EMF on electrons in chemical reactions were detected in the Na,K-ATPase when electric or magnetic fields, each accelerated the reaction only when the enzyme was relatively inactive, i.e., the chemical driving forces were weak. These experiments enabled an estimate of the electron velocity as approximately  $10^3$  m/s (Blank and Soo, 2001a; 2001b), a velocity similar to that of electrons in DNA. An electron moving at a velocity of  $10^3$  m/s crosses the enzyme ( $\sim 10^{-8}$  m) before the ELF field has had a chance to change. This means that a low frequency effect on fast moving electrons in DNA or in enzymes should be viewed as effectively due to a repeated DC pulse. In the RF range, the pulse train is longer.

## VII. DNA IS A FRACTAL ANTENNA

Human DNA is about 2 m long, and the molecule is greatly compacted so that it fits into the nuclei of cells that are microns in diameter.

DNA has a unique double helical structure where two strands of DNA are bound together by hydrogen bonds between pairs of nucleotide bases (one on each strand) and they form a long twisted ribbon with delocalized  $\pi$  electrons that form continuous planar clouds on both surfaces of the ribbon. The result is a structure with two continuous paths that can conduct an electron current along the DNA.

Many studies, initially from the laboratory of Barton at Cal Tech (Hall et al, 1996), have shown that DNA does indeed conduct electrons. As would be expected, the rate of conduction can be influenced by the detailed structure of DNA. Changes, such as hairpin turns and mismatched bases, can lead to the disruption of the ordered double helical structure and anomalies in the rate of electron flow (Arkin et al, 1996; Hall et al, 1997; Lewis et al, 1997; Kelley et al, 1999; Giese, 2002). Electron flow can lead to local charging as well as oxidative damage.

Variations in the rate of electron flow can lead to the accumulation of charge at bottlenecks. The temporary buildup of charge at a site results in strong repulsive forces that can cause a disruption of H-bonds. A net charge can even disrupt the structure of a complex molecule, such as occurs when the four protein chains of hemoglobin

disaggregate in response to a gradual buildup of charge in the hemoglobin tetramer (Blank, 1984; Blank and Soo, 1998). For similar reasons, one would expect disaggregating forces at the DNA site where charge builds up. This would be expected to occur more easily in a compact structure such as DNA in the nucleus.

The tightly coiled DNA in the nucleus uses fractal patterns in order to occupy space efficiently. A fractal is a shape that displays *self-similarity*, where each part of the shape resembles the entire shape. Thus, the double helix is wound into a coil and that coil is wound into a larger coil, and so on. DNA in a cell nucleus is a coiled-coil many times over.

Since the DNA molecule in the nucleus conducts electricity and is organized in a self-similar pattern, it has the two key characteristics of *fractal antennas* when interacting with EMF (Blank, Goodman 2011). Fractal design is desirable for an antenna because it minimizes the overall size, while reacting to a wide range of electromagnetic frequencies. However, these characteristics are not desirable in DNA, because of the many frequencies in the environment that can and do react with DNA. The almost continuous cloud of delocalized electrons along both faces of the 'ribbon' formed by the base pairs provides a conducting path for responding to EMF and makes it more vulnerable to damage. The chemical changes that result from electron transfer reactions, are associated with molecular damage in DNA.

#### VIII. DNA DAMAGE AND CANCER

Stress proteins are essential for cell protection. They help defend cells against damaging forces like increases in temperature and reductions in oxygen supply that could be life-threatening. Similarly, the body generates stress proteins to strengthen cellular resistance to the effects of EM radiation. However, stress protein synthesis is really only an emergency measure that is designed to be effective in the short term. The response to repeated stimuli diminishes with repeated exposure and this could be dangerous.

Thermotolerance, the ability to tolerate higher temperatures as a result of repeated exposures to high temperature, was originally demonstrated at the molecular level in connection with heat shock. Repeated exposure to increased temperature resulted in a decreased heat shock response. A similar mechanism applies when the cellular stress response is stimulated by EMF, since repeated EMF stimuli result in lower production of stress proteins. This could very well be a mechanism by which repeated exposure to EMF can result in less protection and more damage to molecules like DNA. The lower protection predisposes exposed individuals to an increased risk of mutation and initiation of cancer.

DiCarlo and Litovitz (2008) at Catholic University in Washington, D.C. demonstrated the development of EMF tolerance in an experiment performed on chicken embryos. In those eggs exposed to ELF-radiation of 8  $\mu$ T for 30 or 60 minutes at a time, twice a day for four days, production of hsp70 in response to oxygen deprivation declined. The same response was noted in those eggs exposed to RF radiation of 3.5  $\mu$ W/cm<sup>2</sup> for 30 or 60 minutes, once a day, for four days. The researchers noted that these eggs produced 27% less hsp70 following these exposures, and had correspondingly reduced ability to fend off cell damage (reduced *cytoprotection*). Similar experiments have been carried out with short, repeated exposures (in contrast to extended exposures). There too, the rate of stress protein synthesis is reduced with each repetition. The reduction in stress protein synthesis as a result of continuous exposure to EMF would predispose an individual to the accumulation of DNA damage and the development of cancer.

Cancers are believed to be the long term result of the errors in DNA that occur during the normal functioning of cells. Living cells are continuously growing (making protein) and dividing (making DNA), and errors in synthesis occur. The error rate is a very small but finite, so the vast majority of errors is repaired, but not all. When the error rate is too high, the cell activates apoptosis and destroys itself. However, the small number of errors that is retained accumulates over time as mutations, some of which can affect function. It is particularly bad when mutation inactivates a tumor suppressor gene or a

DNA repair gene and enables creation of an oncogene, since this accelerates the development of a cancer.

Although damage can occur during protein synthesis and cell division, as well as upon exposure to oxidizing chemicals, the probability of developing cancer is increased as a result of damage to DNA structure caused by exposure to EMF (Verschaeve, 2008). EMF induced oxidative damage to DNA has even been reported on exposure to high ELF fields (Yokus et al, 2008).

#### IX. STRESS RESPONSE: BIOLOGICAL GUIDE TO SAFETY

The cellular stress response is the way the body tells us that it has come in contact with a potentially harmful stimulus. Since cells react to relatively low levels of EMF, both ELF and RF, one would think that the low biological thresholds for a protective reaction to harmful stimuli would provide critical guidance for the authorities seeking to establish meaningful safety standards. By ignoring the information from the cellular stress response, the authorities appear to be saying that they are better judges of what is harmful to cells than the cells themselves.

Research on the cellular stress response has drawn attention to the inadequacy of EMF safety standards. The synthesis of stress proteins at EMF levels that are currently considered safe indicates that ambient exposure levels can influence the molecular processes involved in protein synthesis needed to provide new molecules and replace damaged molecules. The ability of EMF to interfere with normal function and damage the protein and DNA molecules that are being synthesized is definitely a reason to consider this effect for guidance regarding its health implications. The system of safety standards is not at all protective because processes stimulated at non-thermal levels have been overlooked. The standards must be revised.

The authorities have been misguided in assuming that only thermal stimuli could affect chemical bonds and that non-thermal stimuli cannot cause chemical changes. Non-thermal biological mechanisms activated by EMF have been known for some time, and

some experiments have even been aimed specifically at demonstrating unusual changes in biological systems due to non-thermal EMF stimuli. Bohr and Bohr (2000) showed that both a reaction and its reverse, the denaturation and renaturation of  $\beta$ -lactoglobulin, are accelerated by microwave EMF, and de Pomerai et al (2003) showed that microwave radiation causes protein aggregation in the absence of bulk heating. A clear separation of thermal and non-thermal mechanisms in biology was shown by Mashevich et al (2002) in experiments where chromosomal damage in lymphocytes that had been observed under RF was not seen when the cells were exposed to elevated temperatures. The neglect of non-thermal mechanisms by regulators is based on their ignorance of reactions in biological systems. By greatly underestimating the risk of EMF exposure, they continue to endanger the public.

The cellular stress response is activated by a mechanism that involves interaction of EMF with the DNA molecule. This reaction of DNA, and/or the stress proteins that are synthesized, could be used to develop new EMF safety standards (Blank and Goodman, 2012). A biologically-based measure of EMF radiation could replace the misguided energy-based "specific absorption rate" (SAR). (It should be noted that SAR is the safety standard in the radiofrequency (RF) range, but it fails as a standard for predicting cancer risk in the ELF range.) A standard based on stress proteins would have several advantages compared to SAR:

- it is based on a protective cellular mechanism that is stimulated by a variety of potentially harmful environmental agents
- it is stimulated by a wide range of frequencies in the EM spectrum so there would be no need for different standards in different frequency ranges.

Cancers are believed to arise from mutations in DNA, and changes in DNA induced by interaction with EMF could be a better measure of the biologically effective dose. It may be possible to measure the changes by transcriptional alterations and/or translational changes in specific proteins. A biologically-based standard related to stimulation of DNA

could apply over a much wider range of the electromagnetic spectrum and include ionizing radiation.

#### X. STRESS RESPONSE: GUIDE TO NEW THERAPIES

Since activation of the cellular stress response by EMF was shown to be a protective mechanism, it was only a matter of time before the response would be studied as a potential therapeutic agent. Thermal activation of the stress response has already been shown to be effective in cardiac bypass surgery (Currie et al., 1993; Udelsman et al., 1993; Nitta et al., 1994). Stress protein activation can apparently minimize the oxidative damage of ischemia (low oxygen level in a tissue) reperfusion that occurs when the blood supply is reconnected to the heart after surgery. However, the temperature control required for thermal activation is cumbersome and the technique is not easily applied compared to EMF. A study of non-invasive EMF induction of hsp70, prior to cardiac bypass surgery, has shown that myocardial function can be preserved, and at the same time decrease ischemic injury (George et al, 2008).

EMF activation of stress protein synthesis has a clear advantage over thermal activation. The biological response is not related to the EMF energy, so protective biological responses should occur far below thermal levels. 60 Hz fields were shown to induce elevated levels of hsp70 protein in the absence of elevated temperature (Goodman et al., 1994; Goodman and Blank, 1998; Han et al., 1998; Lin et al., 1998, 1999, 2001; Carmody et al., 2000) in cells including cultured rodent cardiomyocytes (Goodman and Blank, 2002). Also, Di Carlo et al. (1999) and Shallom et al. (2002) confirmed that cardiomyocytes were protected from anoxic damage in EMF exposed chick embryos.

Another potential therapeutic application has come from a study of the stress protein hsp10 in relation to striated muscle function. Kayani et al (2010) at the University of Liverpool found that this stress protein can prevent the age-related deterioration of muscle strength in skeletal muscle of transgenic mice. Hsp10 is often linked with hsp60 in supporting mitochondrial function. In cardiac myocytes this combination protects mitochondrial function as well as preventing cell deaths induced by ischemia-reperfusion.

These results suggest that mitochondrial hsp10 and hsp60 in combination or individually play an important role in maintaining mitochondrial integrity and ability to generate ATP, which are crucial for survival of cardiac myocytes during ischemia/reperfusion.

Research on therapeutic effects using stress proteins is obviously just beginning and we can expect other applications where EMF is used to generate this group of therapeutic agents essentially instantaneously and in situ.

## XI. THE ENVIRONMENTAL EMF ISSUE AND CONCLUSIONS

Research has shown that the EMF-activated cellular stress response:

- is an effective protective mechanism for cells exposed to a wide range of EMF frequencies
- thresholds are very low (safety standards must be reduced to limit biological responses)
- mechanism involves direct interaction of EMF with the DNA molecule (claims that there are no known mechanisms of interaction are patently false)
- the coiled-coil structure of DNA in the nucleus makes the molecule react like a fractal antenna to a wide range of frequencies (there is a need for stricter EMF safety standards)
- biologically-based EMF safety standards could be developed from the research on the stress response.

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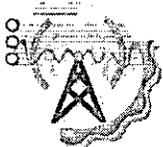
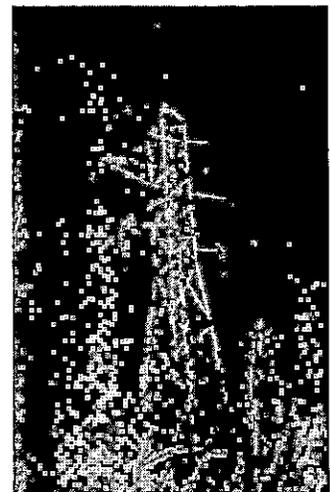
## Electric and Magnetic Fields (EMF): Health Concerns

EMF exposure is very common, and so are questions about what this exposure may mean. The following sections provide answers to some common questions about EMF and concerns about health.



### What is EMF?

**Electric and magnetic fields (EMF)** are areas of energy that surround any electrical device. Power lines, electrical wiring, computers, televisions, hair dryers, household appliances and everything else that uses electricity are sources of EMF. The magnetic field is not blocked by buildings so outdoor sources like power lines can add to the EMF inside your home. However, **the field decreases rapidly with distance so that most homes are too far from high voltage lines to matter.**



### How Are Electromagnetic Fields Measured?

EMF are commonly measured in units of **gauss (G)** by an instrument known as a gaussmeter. A **milligauss (mG)** is 1000 times smaller than a gauss.



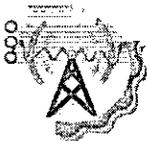
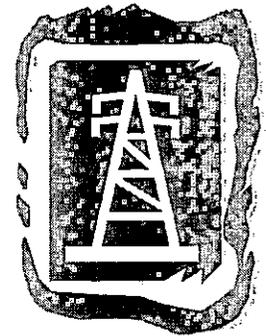
## What Are Typical EMF Levels Within A Home?

In a study that measured EMF in almost 1000 homes in the United States, 50% had average EMF levels of 0.6 mG or less, and 95% had average EMF levels below 3 mG. Keep in mind that these are *average* EMF levels within a home. EMF levels can be higher (5 mG or more) when you are near a household appliance (or anything else that uses electricity). EMF levels rapidly become weaker as you move away from the source.



## How High Are EMF Levels Near Power Lines?

Power lines that send electricity between towns and into neighborhoods generally have the highest voltage. They are bigger and have more wires than the distribution lines that are common on most streets. The high voltage lines can have EMF levels of 30 to 90 mG underneath the wires, depending on the voltage, height, and placement of the lines. EMF levels decrease rapidly with distance from the lines. At 300 feet (a football field), EMF is at background levels. In some cases, even closer distances are at background. The distribution lines that run up and down every street are smaller, contain lower voltage and are of less concern.



## Is EMF Exposure Harmful?

Despite extensive research over the past 20 years, the health risk caused by EMF exposure remains an open question. Two national research organizations (the National Research Council and the National Institute of Health) have looked at the studies and have concluded that there is not strong evidence that EMF exposures pose a health risk. However, some studies have shown an association between household EMF exposure and a small increased risk of childhood leukemia at average exposures above 3 mG. For cancers other than childhood leukemia, there is less evidence for an effect. For example, workers that repair power lines and railway workers can be exposed to much higher EMF

levels than the general public. The results of cancer studies in these workers is mixed. Some studies have suggested a link between EMF exposure in electrical workers and leukemia and brain cancer. Other similar studies have not found such associations. There is also some evidence that utility workers exposed to high levels of EMF may be at increased risk of developing amyotrophic lateral sclerosis (Lou Gehrig's Disease).

Although the current scientific evidence provides no definitive answers as to whether EMF exposure can increase health risks, there is enough uncertainty that some people may want to reduce their exposure to EMF.



## How Can I Reduce My EMF Exposure?

EMF exposure depends on what EMF sources are nearby and how much time you spend near them.

If you would like to reduce your exposure to EMF, you can take simple steps such as:

- Increase distance: for example, sit at arm's length from your computer or re-position electric alarm clocks farther away from your body while in bed.
- Repair faulty wiring which may be generating higher than usual EMF.
- Turn off electrical devices such as televisions and computers when not in use.
- Use electric blankets to warm the bed, turning them off before getting into bed.



## What Should I Do if a Home I Want To Buy is Near High Voltage Lines?

If the power lines are more than 300 feet away, there should be no cause for concern. At this distance EMF from the lines is no different from typical levels around the home.

If the power lines are less than 300 feet away from the home, you may want to obtain EMF measurements in the yard. Most electric utilities in Connecticut will take measurements for free. There are also private firms that will charge a fee for measurements. To understand your measurement, consider that typical EMF levels found inside homes

range from 0.1 to 4 mG. EMF levels above this range are not necessarily hazardous, but indicate EMF levels above what's typical background inside a home.

Deciding where to live rests upon different considerations for each individual. EMF exposure is just one of many factors in this decision. Other environmental health issues around a home can include: radon, lead paint, asbestos, soil or groundwater contamination, local traffic and noise. All of these factors should be considered when evaluating the home environment.

## What are Best Management Practices (BMPs)?

When new power lines are constructed, they have the potential to increase EMF levels in an area. The Connecticut Siting Council (CSC) reviews these plans. To ensure that the public's exposure to EMF is kept to a minimum, the CSC released a set of BMPs to be followed when constructing new lines. The plans for new lines and their adherence to the BMPs will be on file in town offices and are typically discussed at open forums prior to construction.



## Where Can I Find More Information?

National Institute of Environmental Health Sciences report on health effects from EMF  
<http://www.niehs.nih.gov/health/topics/agents/emf/>

California Dept of Health Services: Electric and Magnetic Fields  
[http://www.ehib.org/cma/topic.jsp?topic\\_key=7](http://www.ehib.org/cma/topic.jsp?topic_key=7)

Connecticut Siting Council Best Management Practices  
[http://www.ct.gov/csc/lib/csc/emf\\_bmp/emf\\_bmp\\_12-14-07.doc](http://www.ct.gov/csc/lib/csc/emf_bmp/emf_bmp_12-14-07.doc)

World Health Organization: International EMF Project  
<http://www.who.int/peh-emf/en/>



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Revised 4/2008

We've made some changes to EPA.gov. If the information you are looking for is not here, you may be able to find it on the EPA Web Archive or the January 19, 2017 Web Snapshot.



## **News Releases from Headquarters › Chemical Safety and Pollution Prevention (OCSPP)**

# **EPA Takes Action to Prevent Poisonings from Herbicide**

12/15/2016

Contact Information:  
[press@epa.gov](mailto:press@epa.gov)

**WASHINGTON** – The U.S. Environmental Protection Agency (EPA) is finalizing safety measures to stop poisonings caused by ingestion of the herbicide paraquat, which can also cause severe injuries or death from skin or eye exposure.

Since 2000, there have been 17 deaths – three involving children – caused by accidental ingestion of paraquat. These cases have resulted from the pesticide being illegally transferred to beverage containers and later mistaken for a drink and consumed. A single sip can be fatal. To prevent these tragedies, EPA is requiring:

- new closed-system packaging designed to make it impossible to transfer or remove the pesticide except directly into the proper application equipment;
- special training for certified applicators who use paraquat to emphasize that the chemical must not be transferred to or stored in improper containers; and
- changes to the pesticide label and warning materials to highlight the toxicity and risks associated with paraquat.

In addition to the deaths by accidental ingestion, since 2000 there have been three deaths and many severe injuries caused by the pesticide getting onto the skin or into the eyes of those working with the herbicide. To reduce exposure to workers who mix, load and apply paraquat, EPA is restricting the use of paraquat to certified pesticide applicators only. Uncertified individuals working under the supervision of a certified applicator will be prohibited from using paraquat.

Paraquat is one of the most widely-used herbicides in the U.S. for the control of weeds in many agricultural and non-agricultural settings and is also used as a defoliant on crops such as cotton prior to harvest.

EPA proposed similar measures last March and took public comment.

Actions on specific pesticides are one way that EPA is protecting workers from pesticide exposure. EPA's Final Certification and Training and Worker Protection Standard rules will also protect pesticide applicators and farmworkers.

Learn more about paraquat and the new measures to reduce risk: <https://www.epa.gov/ingredients-used-pesticide-products/paraquat-dichloride>

Learn about EPA's Certification and Training Rule: <https://www.epa.gov/pesticide-worker-safety/revised-certification-standards-pesticide-applicators>

Learn about EPA's Worker Protection Standard: <https://www.epa.gov/pesticide-worker-safety/revisions-worker-protection-standard>

To View the docket on [www.regulations.gov](http://www.regulations.gov): EPA-HQ-OPP-2011-0855-0112

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Letter

## The correlations between BRCA1 defect and environmental factors in the risk of breast cancer

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**ABSTRACT** — The risk factors for breast cancer, the most common female malignant cancer, include environmental factors such as radiation, tobacco, a high-fat diet, and xenoestrogens as well as hormones. In addition, BRCA1 and BRCA2 are the most well-known genetic factors that increase risk for breast cancer. Coincidence of those environmental and genetic factors might augment the risk of tumorigenesis of breast. To verify this hypothesis, we briefly evaluated the carcinogenic potency of various environmental factors in the absence or presence of BRCA1 as a genetic factor in a normal mammary epithelial cell line, MCF10A. Many environmental factors tested increased cellular ROS level in the absence of other insult. In addition, TCDD, DMBA, 3MC, and BPA enhanced the BaP-induced ROS production. BRCA1 knockdown (BRCA1-KD) cells by siRNA significantly induced cellular accumulation of ROS compared to control cells. In this setting, the addition of paraquat, TCDD, DMBA, 2OHE2 or 4OHE2 significantly augmented ROS generation in BRCA1-KD MCF10A cells. Measurements of BaP-DNA adduct formation as a marker of DNA damage also revealed that BRCA1 deficiency leads increased DNA damage. In addition, TCDD and DMBA significantly increased BaP-DNA adduct formation in the absence of BRCA1. These results imply that elevated level of ROS is correlated with increase of DNA damage in BRCA1 defective cells. Taken together, our study suggests that several environmental factors might increase the risk of tumorigenesis in BRCA1 defective breast epithelial cells.

**Key words:** BRCA1, Genetic factor, Environmental factors, Tumorigenesis, Breast cancer

### INTRODUCTION

Primary risk factors of breast cancer include exposure to environmental factors such as radiation, tobacco and xenoestrogen (Ibarluzea *et al.*, 2004; Wolff *et al.*, 1996; Lichtenstein *et al.*, 2000; Nathanson *et al.*, 2001). Main molecular pathogenesis of these environmental factors is attributed to oxidative stresses. There are accumulating data that residual oxidative stresses from these xenobiotics promote tumorigenesis (Dunnick *et al.*, 1995). For

complete detoxification and excretion of xenobiotics, the cooperative processes of phase I and phase II enzymes are required (Xu *et al.*, 2005). In phase I, enzymes such as cytochrome P450 oxidases (CYPs) introduce reactive or polar groups into xenobiotics. These modified compounds are then conjugated to polar compounds in phase II, and excreted out by phase III enzymes (Denison and Nagy, 2003).

Mutations of BRCA1 have been identified as to be responsible for about half of inherited cases of breast

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cancer (Easton *et al.*, 1993). Although major function of BRCA1 is known as a classical tumor suppressor gene, we have demonstrated that BRCA1 regulates transcription of phase I and II enzymes upon exposure to various exogenous stresses (Kang *et al.*, 2006; 2008a and 2008b). BRCA1 can stimulate antioxidant gene expression and modulate intracellular reactive oxygen species (ROS) levels through enhancing the activity of the antioxidant response transcription factor, NRF2 (Bae *et al.*, 2004; Kang *et al.*, 2012). Furthermore, BRCA1 is also engaged in the cells' responses to xenobiotic stresses by up-regulating AhR/ARNT (aryl hydrocarbon receptor/aryl hydrocarbon receptor nuclear translocator)-driven transcription (Kang *et al.*, 2006, 2008a). BRCA1 stabilizes ARNT and modulates transcriptional regulation of CYP1A1 and CYP1B1 following xenobiotic stress exposure (Kang *et al.*, 2006). Therefore, BRCA1 can preserve the integrity of cellular macromolecules, especially genomic DNA by reducing protein nitration and hydrogen peroxide levels (Saha *et al.*, 2009).

In this context, defects in both phase I and II systems resulting from a BRCA1 deficiency may hamper sufficient cytoprotection against environmental insults, which could result in increased ROS production, DNA damage and tumorigenesis in the mammary gland. Here, we evaluated the role of environmental risk factors in the absence of BRCA1 on oxidative stress and DNA damage.

## MATERIALS AND METHODS

### Cell culture and reagents

MCF10A and 293 cells from American Type Culture Collection (Manassas, VA, USA) were cultured as described previously (Kang *et al.*, 2008a, 2012). Benzo[a]pyrene (BaP), 7,12-Dimethylbenz[a]anthracene (DMBA), 3-Methylcholanthrene (3MC), 2-hydroxyestradiol (2OHE2), 4-hydroxyestradiol (4OHE2), sodium selenite, styrene oxide, cadmium chloride, and bisphenol A (BPA) were purchased from Sigma (St. Louis, MO, USA). PCB (3,3',4,4',5-Pentachlorobiphenyl) was obtained from AccuStandard, Inc. (New Haven, CT, USA) and TCDD (2,3,7,8-Tetrachlorodibenzodioxin) was purchased from Ultra Scientific, Inc. (North Kingstown, RI, USA). Radio-labeled [<sup>3</sup>H]BaP was purchased from American Radiolabeled Chemicals, Inc. (St. Louis, MO, USA). Irradiations of UVA and UVC were performed using CL-1000L UV crosslinker (UVP, Inc., Upland, CA, USA) and Spectrolinker XL-1000 UV crosslinker (Spectronics, Westbury, NY, USA), respectively.

### Transfection of siRNA

Control (non-targeting scrambled) and BRCA1-siRNAs were purchased from Dharmacon, Inc. (Lafayette, CO, USA). Their sequences and transfection method were described previously (Kang *et al.*, 2011a, 2012).

### Measurement of ROS production

Measurements of ROS were performed by using CM-H<sub>2</sub>DCFDA (2',7'-dichlorofluorescein diacetate) as described previously (Kang *et al.*, 2011b). After incubation of environmental factors with or without 5 μM BaP for 24 hr, cells were treated with 5 μM of CM-H<sub>2</sub>DCFDA. Fluorescence was measured using Ultra 384 Fluorometer (Tecan, Männedorf, Switzerland) at 495/535 nm at the Genomics and Epigenomics Shared Resource at Georgetown University Medical Center.

### Measurement of [<sup>3</sup>H]BaP-induced DNA adducts

To determine BaP-induced DNA adducts, we used [<sup>3</sup>H]BaP-DNA binding assay (Kang and Lee, 2005; Kang *et al.*, 2011a, 2011b). After 24 hr incubation of cells with environmental factors in the absence or presence of 5 nM of [<sup>3</sup>H]BaP, genomic DNAs were isolated using Wizard SV Genomic DNA purification system (Promega, Madison, WI, USA). The radioactivity of [<sup>3</sup>H]BaP-DNA adducts in equal amount of DNA was counted using Beckman Coulter liquid scintillation counter LS6500 (Fullerton, CA, USA).

### Reporter gene assay

Cells seeded in 24-well-plates and transfected with a reporter gene (GAL4-DBD-Luc) and expression vectors for GAL4-BRCA1 AD1 and AhR (Kang *et al.*, 2008a) using Lipofectamine Plus (Invitrogen). Then cells were treated with various agents for 24 hr when they were harvested, lysed and used for luciferase assays were performed as previously (Kang *et al.*, 2008a). The luminescence signal was measured by the Wallac Victor<sup>2</sup> microplate reader (Perkin-Elmer Life Sciences, Boston, MA, USA) at the Genomics and Epigenomics Shared Resource at Georgetown University Medical Center.

### Statistical analysis

All experiments were performed more than three times. We used ANOVA analysis and Tukey's multiple comparison procedure to adjust for p values. The test is performed at 5% significance level. \* or \* means the difference is significant after adjusting for multiple comparison.

## RESULTS AND DISCUSSION

For evaluation of carcinogenesis in cellular model system, we have employed an *in vitro* assay system using benzo[a]pyrene (BaP) as a carcinogenic insult in BRCA1-knockdown (BRCA1-KD) cells to demonstrate oxidative stress induction and genomic DNA damage in BRCA1 defective cells (Kang *et al.*, 2011b). To address the tumorigenic potency of the various environmental factors when genetic factors are involved, we assessed the risk changes using our monitoring system.

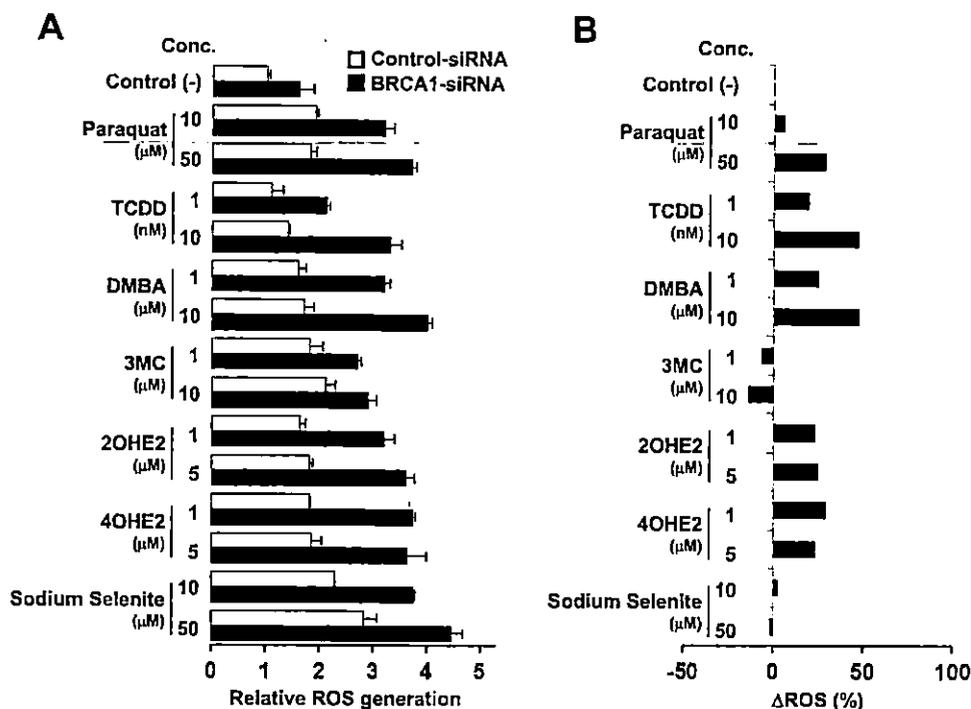
First, we profiled the potency of ROS production by various environmental factors in MCF10A normal mam-

mary epithelial cell (Soule *et al.*, 1990). The concentrations of various environmental factors were comparable to US Drinking Water Standards and Health Advisories (Supplementary Table 1). As expected, exposure to several xenobiotics increased cellular ROS level (Table 1). Most of the environmental factors tested (except for PCB, cadmium, BPA, UVA, and UVC) significantly induced cellular ROS level. We also measured the changes of ROS level by these environmental factors in the presence of BaP as a carcinogenic insult. Incubation of 5  $\mu$ M BaP alone significantly increased ROS level (2.25 fold higher than control). In this setting, co-incubation with TCDD, DMBA and 3MC significantly augmented BaP-induced

**Table 1.** Effects of various environmental factors on the level of cellular ROS production in the absence or presence of BaP.

Environmental Factor	Conc.	ROS	
		(-)	w/ BaP
Control	-	1.00 $\pm$ 0.04	2.25 $\pm$ 0.24
Paraquat	10 $\mu$ M	1.47 $\pm$ 0.05*	3.62 $\pm$ 0.34
	25 $\mu$ M	2.02 $\pm$ 0.20*	4.12 $\pm$ 0.68
TCDD	1 nM	1.16 $\pm$ 0.13	2.83 $\pm$ 0.15
	10 nM	1.31 $\pm$ 0.14*	3.38 $\pm$ 0.21#
DMBA	1 $\mu$ M	1.74 $\pm$ 0.24*	7.24 $\pm$ 0.87#
	10 $\mu$ M	1.93 $\pm$ 0.20*	8.18 $\pm$ 0.32#
3MC	1 $\mu$ M	1.29 $\pm$ 0.23*	4.49 $\pm$ 0.05#
	5 $\mu$ M	1.39 $\pm$ 0.04*	5.02 $\pm$ 0.23#
PCB	1 $\mu$ M	0.97 $\pm$ 0.07	2.42 $\pm$ 0.12
	10 $\mu$ M	0.82 $\pm$ 0.08	1.94 $\pm$ 0.06
2OHE2	1 $\mu$ M	1.43 $\pm$ 0.04*	2.85 $\pm$ 0.00
	5 $\mu$ M	2.54 $\pm$ 0.35*	4.20 $\pm$ 0.22
4OHE2	1 $\mu$ M	1.50 $\pm$ 0.18*	3.00 $\pm$ 0.05
	5 $\mu$ M	1.99 $\pm$ 0.21*	4.31 $\pm$ 0.13
Sodium Selenite	1 $\mu$ M	1.40 $\pm$ 0.04*	2.93 $\pm$ 0.26
	5 $\mu$ M	1.63 $\pm$ 0.23*	3.66 $\pm$ 0.10
Styrene Oxide	50 $\mu$ M	1.77 $\pm$ 0.32*	2.55 $\pm$ 0.29
	100 $\mu$ M	1.82 $\pm$ 0.00*	2.75 $\pm$ 0.11
Cadmium Chloride	100 $\mu$ M	0.93 $\pm$ 0.26	2.03 $\pm$ 0.03
	200 $\mu$ M	0.58 $\pm$ 0.03	1.43 $\pm$ 0.09
BPA	1 nM	0.94 $\pm$ 0.16	2.44 $\pm$ 0.10
	10 nM	1.05 $\pm$ 0.04	2.76 $\pm$ 0.38#
Ethanol	1 %	1.19 $\pm$ 0.11	2.84 $\pm$ 0.10
	5 %	1.31 $\pm$ 0.16*	2.26 $\pm$ 0.15
UVA	0.2 J/cm <sup>2</sup>	1.21 $\pm$ 0.12	2.56 $\pm$ 0.30
UVC	0.2 J/cm <sup>2</sup>	1.22 $\pm$ 0.12	2.93 $\pm$ 0.48

\*and #; significant increase compared to without or with BaP treated control, respectively.  $P < .05$ .



**Fig. 1.** Effect of various environmental factors on the ROS production in BRCA1 deficient MCF10A cells. (A) The ROS levels were measured after incubation with indicated environmental factors in the presence of BaP (5 μM) for 24 hr in control- or BRCA1-siRNA treated MCF10A cells. (B) The change of ROS ( $\Delta$ ROS) calculated by  $[(R_{BF}/R_{CF}) - (R_B/R_C)] / (R_B/R_C) * 100$  from values in (A). Where  $R_C$  is ROS level in control-siRNA-transfected cells,  $R_B$  is ROS level in BRCA1-siRNA-transfected cells,  $R_{CF}$  is ROS level in control-siRNA-transfected cells treated with environmental factor, and  $R_{BF}$  is ROS level in BRCA1-siRNA-transfected cells treated with environmental factor.

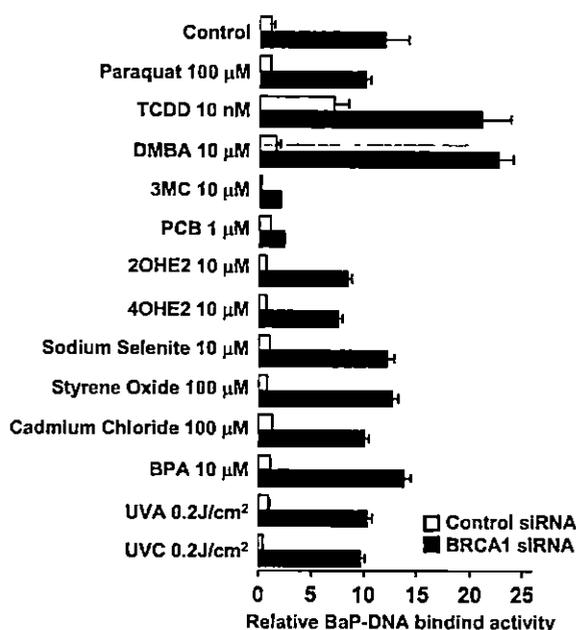
ROS level in MCA10A cells (Table 1). Intriguingly, BPA (10 nM) incubation synergistically increased BaP induced ROS level, while BPA itself did not induce ROS generation.

Next, we investigated whether a genetic factor modulates ROS production induced by environmental factors. After knockdown of BRCA1 in MCF10A cells, the changes of ROS generation was measured. As reported earlier (Saba *et al.*, 2009; Kang *et al.*, 2011a; Martinez-Outschoorn *et al.*, 2012a, 2012b; Kang *et al.*, 2012), abrogation of BRCA1 significantly increase ROS level in the control-siRNA treated cells (Fig. 1A). Although ROS levels were changed by all environmental factors in some degrees in control-siRNA transfected MCA10A cells, significant enhancement of ROS production was only observed in paraquat, TCDD, DMBA, 2OHE2, 4OHE2 treated BRCA1-KD MCF10A cells (Fig. 1B). Interestingly, paraquat, TCDD, and DMBA commonly enhanced ROS production both in the pres-

ence of BaP and in the absence of BRCA1. These results implicate that several factors might potentiate the risk factors (i.e., ROS production) in BRCA1 deficiency related breast cancer.

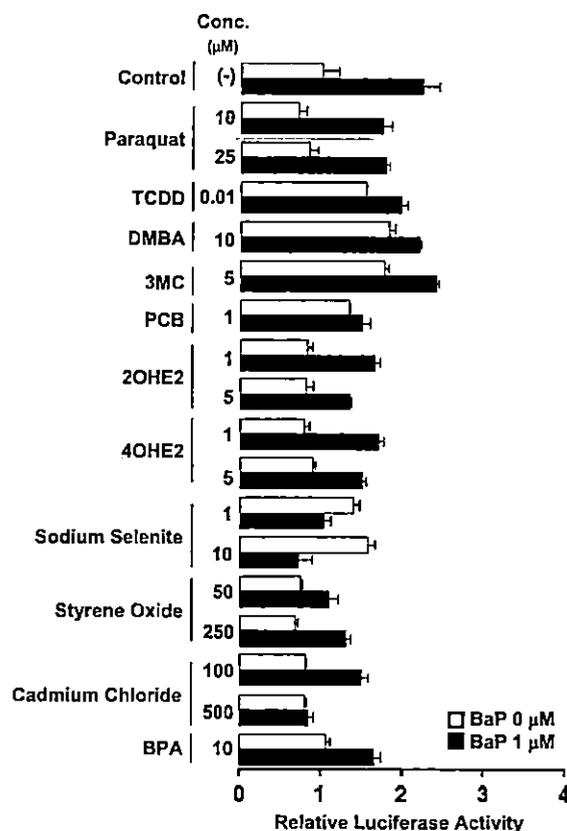
Since oxidative stresses lead to genotoxicity, we examined whether there are positive correlations between ROS levels and DNA damage. We determined the [ $^3$ H] BaP-DNA binding as a measure of DNA damage (Kang and Lee, 2005; Kang *et al.*, 2011a, 2011b). Previously we reported that the results from [ $^3$ H]BaP-DNA binding assay are well correlated to the results from *in vitro* [ $^{32}$ P] postlabeling assay using TLC plates (Kang *et al.*, 2011a, 2011b). In control MCF10A cells, the level of [ $^3$ H]BaP-DNA adduct was significantly elevated in BRCA1-KD cells. Interestingly, TCDD itself markedly increased the [ $^3$ H]BaP-DNA adduct formation in control-siRNA transfected MCF10A cells. Under this condition, only TCDD and DMBA significantly raised DNA damage in BRCA1 deficient cells (Fig. 2). Unexpectedly,

## Environmental factors in carcinogenesis of BRCA1-defective cells



**Fig. 2.** Effect of various environmental factors on BaP-DNA adduct formation. To facilitate DNA damage, MCF10A cells, transfected with either control- or BRCA1-siRNA, were further incubated 5 nM of [<sup>3</sup>H]BaP for 24 hr then the amounts of BaP-DNA adduct were measured as described in materials and methods.

[<sup>3</sup>H]BaP-DNA levels in 3MC and PCB treated BRCA1-KD MCF10A cells were significantly reduced compared to control. It is still unclear how 3MC and PCB inhibits the formation of BaP-DNA adduct formation. Previously it has been reported that BaP-DNA adduct formation was reduced when [<sup>3</sup>H]BaP and calf thymus DNA were incubated with microsomal protein from 3MC-induced rats in the presence of unoxidized beta carotene (Salgo *et al.*, 1999). It was also reported that pretreatment of CB 126 (3,3',4,4',5-pentachlorobiphenyl; a dioxin-like PCB) reduced the hepatic BaP-DNA adduct formation in marine flatfish dab (van Schanke *et al.*, 2000). We could postulate that these results might come from the activation of AhR by 3MC and PCB. As 3MC and PCB are well-known to activate AhR (Abdelrahim *et al.*, 2006), activated AhR system might clear metabolites of [<sup>3</sup>H]BaP before incorporation into DNA. Alternatively, these environmental factors might differentially affect enzyme activities of phase I system. As an example, CB 126 was reported as a strong inhibitor of CYP1B1 (Pang *et al.*, 1999). Although TCDD is also known as an AhR ligand, mutagenic analyses of AhR suggested that TCDD has a different AhR



**Fig. 3.** Effect of various environmental factors on the transcriptional activation by BRCA1 AD1-AhR. 293 cells were transfected with a reporter plasmid (GAL4-DBD-Luc) and expression vectors for GAL4-BRCA1 AD1 and AhR. The environmental factors were treated as indicated for 24 hr and luciferase activity was measured as described in materials and methods.

binding mode (Denison *et al.*, 2011). Indeed, TCDD has been reported to increase the BaP-DNA adducts in several experimental settings (Carvan *et al.*, 1995; Harrigan *et al.*, 2006; de Waard *et al.*, 2008). Similar to BaP, DMBA itself can form high level of DNA adduct even in the absence of enzymatic or chemical activation (Bryla and Weyand, 1992). Under our experimental setting, DMBA enhanced the BaP-DNA adduct formation in a BRCA1-dependent manner. These data implicate that there might be positive correlation between elevated level of ROS and DNA damage or tumorigenesis by these environmental factors.

Since BRCA1 plays crucial role in detoxification of xenobiotics through interaction with AhR (Kang *et*

*al.*, 2008b), we measured effects various environmental factors on the transcriptional activity of BRCA1. 293 cells were transfected with a reporter plasmid containing GAL4-DNA binding domain (DBD) in the upstream of luciferase reporter gene and expression plasmids for BRCA1 AD1 fused to GAL4 DBD (GAL4-BRCA1 AD1) and AhR (Kang *et al.*, 2008a). Then, the transfected cells were further treated with various environmental factors and reporter gene activity was monitored to determine the effect of these factors on BRCA1 AD1-AhR-mediated transcriptional activity. Under these conditions, BaP enhanced BRCA1 AD1-AhR-mediated transcriptional activation. Most of environmental factors themselves showed little or no effects on the transcriptional activation by BRCA1 AD1-AhR except for TCDD, DMBA, 3MC, PCB, and sodium selenite (Fig. 3). Interestingly, sodium selenite markedly induced the BRCA1 AD1-AhR-mediated transcription in the absence of BaP. Co-treatment of BaP with these environmental factors exhibited no significant effects (paraquat, TCDD, DMBA, and 3MC) or rather antagonistic effects (PCB, 2OHE2, 4OHE2, sodium selenite, sodium oxide, cadmium, and BPA) on the BaP-induced transcriptional activation of BRCA1 AD1-AhR. Thus combination of some environmental factors might augment the impairment of defense mechanism of BRCA1 against xenobiotic stress.

In this study, we assessed the risks of various environmental factors for increase of ROS production or ROS-induced DNA damage. We included the environmental factors such as 1) the polycyclic aromatic hydrocarbon (PAH) family (BaP and DMBA), 2) pesticides (PCB and paraquat), 3) chemicals causing mammary gland tumors in mice (styrene oxide), 4) heavy metal (cadmium), 5) radiation (UVA and UVC), 6) catechol estrogen (2OHE2 and 4OHE2), 7) a herbicide (TCDD), and 8) xenoestrogen BPA. Interestingly, TCDD and DMBA commonly increased the BaP-induced ROS production in both control and BRCA1 defective normal breast epithelial MCF10A cells. In addition, TCDD and DMBA drastically enhanced the BaP-DNA adduct formation in BRCA1 deficient cells. These results suggest that concurrent exposure to environmental factors increases the risk of breast cancer carrying genetic factors such as BRCA1 defect.

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## CHAPTER 2

### RECOMMENDATIONS I: PERENNIAL GRASSES AND GRASSES MIXED WITH HERBACEOUS BROADLEAVED WEEDS

#### GENERAL CONSIDERATIONS

2.1. Perennial grass weeds, or mixtures of grass and herbaceous broadleaved weeds, frequently compete with newly planted tree crops during the first five years after planting. Only occasionally has the competition for moisture and nutrients by these weeds been shown seriously to retard crop growth, the main risk to the crop arising from excessive shading and "smothering" from weeds that are taller than the crop and often fall over it at the end of the growing season as the aerial parts die away. "Smothering" appears to damage crops mechanically and by creating conditions favourable for pathogenic fungi, as well as by creating excessive shade.

2.2. As improvements in crop growth due to removal of weed competition for moisture and nutrients are unpredictable and often small, the main object of weed control in these situations is to prevent smothering. Unfortunately, the variation in weed floras and their vigour from site to site, and the variation in tolerance of smothering between crop species, makes it impossible to quantify the extent of weed control required to avoid smothering. Foresters must use their own experience of their area to assess the degree of weed control required.

2.3. Five herbicides, atrazine, chlorthiamid, dalapon, dichlobenil and paraquat, are fully recommended for grass and grass/herbaceous broadleaved weed mixtures. Each one differs in the spectrum of weeds it controls well. The choice of herbicide depends initially on its ability to leave the crop undamaged, but thereafter the choice depends largely on the differences in weed species controlled and the cost. If herbaceous broadleaved weeds are really a problem, then herbicides which mainly control grasses (dalapon and atrazine) should not be used. However, in most situations, grasses form the most important and potentially dangerous fraction of the weed flora, and the particular species of grass present will largely dictate which herbicide should be used. Table 2 shows the susceptibility of the major grass species found in British forestry to these five fully recommended herbicides. It should be noted that the performance of each herbicide will vary with soil type, weather conditions and time of application.

2.4. Figure 1 is a decision tree which should help foresters decide which herbicide to use for their particular weed situations. The final decision may often be a compromise, and will often depend on the major weeding type in a forest.

#### CONTROL OF GRASS AND GRASS/HERBACEOUS BROADLEAVED WEEDS BEFORE PLANTING

2.5. Control of grass and herbaceous broadleaved weeds before planting is not commonly practised in Britain. New ground is often ploughed before planting and this gives adequate initial suppression of the weeds, whilst on ground to be replanted, the previous crop has frequently prevented the development of weeds. Even when weeds are present, these can be more conveniently controlled after planting rather than before.

2.6. However, in some situations the control of existing weeds before planting can make the planting and establishment of the crop easier. In these situations it is rarely necessary to apply the herbicide to the

## Electromagnetic fields and DNA damage

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### Abstract

A major concern of the adverse effects of exposure to non-ionizing electromagnetic field (EMF) is cancer induction. Since the majority of cancers are initiated by damage to a cell's genome, studies have been carried out to investigate the effects of electromagnetic fields on DNA and chromosomal structure. Additionally, DNA damage can lead to changes in cellular functions and cell death. Single cell gel electrophoresis, also known as the 'comet assay', has been widely used in EMF research to determine DNA damage, reflected as single-strand breaks, double-strand breaks, and crosslinks. Studies have also been carried out to investigate chromosomal conformational changes and micronucleus formation in cells after exposure to EMF. This review describes the comet assay and its utility to qualitatively and quantitatively assess DNA damage, reviews studies that have investigated DNA strand breaks and other changes in DNA structure, and then discusses important lessons learned from our work in this area.

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**Keywords:** Electromagnetic field; DNA damage; Comet assay; Radiofrequency radiation; Cellular telephone

### 1. The comet assay for measurement of DNA strand breaks

DNA is continuously damaged by endogenous and exogenous factors and then repaired by DNA repair enzymes. Any imbalance in damage and repair and mistakes in repair result in accumulation of DNA damage. Eventually, this will lead to cell death, aging, or cancer. There are several types of DNA lesions. The common ones that can be detected easily are DNA strand breaks and DNA crosslinks. Strand breaks in DNA are produced by endogenous factors, such as free radicals generated by mitochondrial respiration and metabolism, and by exogenous agents, including UV, ionizing and non-ionizing radiation, and chemicals.

There are two types of DNA strand breaks: single- and double-strand breaks. DNA single-strand breaks include frank breaks and alkali labile sites, such as base modification, deamination, depurination, and alkylation. These are the most commonly assessed lesions of DNA. DNA double-strand breaks are very critical for cells and usually they are

lethal. DNA strand breaks have been correlated with cell death [1–5], aging [6–8] and cancer [9–13].

Several techniques have been developed to analyze single- and double-strand breaks. Most commonly used is microgel electrophoresis, also called the 'comet assay' or 'single cell gel electrophoresis'. This technique involves mixing cells with agarose, making microgels on a microscope slide, lysing cells in the microgels with salts and detergents, removing proteins from DNA by using proteinase K, unwinding/equilibrating and electrophoresing DNA (under highly alkaline condition for assessment of single-strand breaks or under neutral condition for assessment of DNA double-strand breaks), fixing the DNA, visualizing the DNA with a fluorescent dye, and then analyzing migration patterns of DNA from individual cells with an image analysis system.

The comet assay is a very sensitive method of detecting single- and double-strand breaks if specific criteria are met. Critical criteria include the following. Cells from tissue culture or laboratory animals should be handled with care to minimize DNA damage, for instance, by avoiding light and high temperature. When working with animals exposed to EMF *in vivo*, it is better to anesthetize the animals with CO<sub>2</sub> before harvesting tissues for assay. Antioxidants

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such as albumin and sucrose, or spin-trap molecules such as  $\alpha$ -phenyl-*tert*-butyl nitron (PBN), should be added during dispersion of tissues into single cells. Cells should be lysed at 0–4 °C to minimize DNA damage by endonucleases. Additionally, antioxidants such as tris and glutathione, and chelators such as EDTA, should be used in the lysing solution. High concentrations of dimethylsulfoxide (DMSO) should be avoided due to its chromatin condensing effect. Treatment with proteinase K (PK; lyophilized DNase-free proteinase-K from Amresco is ideal) at a concentration of 0.5–1 mg/ml (depending upon cell type and number of cells in the microgel) should be used for 1–2 h at 37 °C to reveal all possible strand breaks which otherwise may go undetected due to DNA–protein crosslinks. Longer times in PK will lead to loss of smaller pieces of DNA by diffusion. Glass slides should be chosen based on which high resolution agarose (3:1 high resolution agarose from Amresco is ideal) will stick well to the slide and on the ability of the specimen to be visualized without excessive fluorescence background. Choice of an electrophoresis unit is important to minimize slide-to-slide variation in DNA migration pattern. A unit with uniform electric field and buffer recirculation should be used. Electrophoresis buffers should have antioxidants and chelators such as DMSO and EDTA. DNA diffusion should be minimized during the neutralization step by rapidly precipitating the DNA. Staining should employ a sensitive fluorescent dye, such as the intercalating fluorescent labeling dye YOYO-1. A cell-selection criteria for analysis should be set before the experiment, such as not analyzing cells with too much damage, although, the number of such cells should be recorded.

There are different versions of the comet assay that have been modified to meet the needs of specific applications and to improve sensitivity. Using the most basic form of the assay, one should be able to detect DNA strand breaks in human lymphocytes that were induced by 5 rad of gamma-ray [14,15].

## 2. Radiofrequency radiation (RFR) and DNA damage

In a series of publications, Lai and Singh [16–19] reported increases in single- and double-strand DNA breaks, as measured by the comet assay, in brain cells of rats exposed for 2 h to a 2450-MHz RFR at whole body specific absorption rate (SAR) between 0.6 and 1.2 W/kg. The effects were blocked by antioxidants, which suggested involvement of free radicals. At the same time, Sarkar et al. [20] exposed mice to 2450-MHz microwaves at a power density of 1 mW/cm<sup>2</sup> for 2 h/day over a period of 120, 150, and 200 days. Rearrangement of DNA segments were observed in testis and brain of exposed animals. Their data also suggested breakage of DNA strands after RFR exposure. Phillips et al. [21] were the first to study the effects of two forms of cell cellular phone signals, known as TDMA and iDEN, on DNA damage in Molt-4 human lymphoblastoid cells using the comet

assay. These cells were exposed to relatively low intensities of the fields (2.4–26  $\mu$ W/g) for 2–21 h. They reported both increased and decreased DNA damage, depending on the type of signal studied, as well as the intensity and duration of exposure. They speculated that the fields may affect DNA repair in cells. Subsequently, different groups of researchers have also reported DNA damage in various types of cells after exposure to cell phone frequency fields. Diem et al. [22] exposed human fibroblasts and rat granulosa cells to cell phone signal (1800 MHz; SAR 1.2 or 2 W/kg; different modulations; for 4, 16 and 24 h; intermittent 5 min on/10 min off or continuous). RFR exposure induced DNA single- and double-strand breaks as measured by the comet assay. Effects occurred after 16 h of exposure to different cell phone modulations in both cell types. The intermittent exposure schedule caused a significantly stronger effect than continuous exposure. Gandhi and Anita [23] reported increases in DNA strand breaks and micronucleation in lymphocytes obtained from cell phone users. Markova et al. [24] reported that GSM signals affected chromatin conformation and  $\gamma$ -H2AX foci that co-localized in distinct foci with DNA double-strand breaks in human lymphocytes. The effect was found to be dependent on carrier frequency. Nikolova et al. [25] reported a low and transient increase in DNA double-strand breaks in mouse embryonic stem cells after acute exposure to a 1.7-GHz field. Lixia et al. [26] reported an increase in DNA damage in human lens epithelial cells at 0 and 30 min after 2 h of exposure to a 1.8-GHz field at 3 W/kg. Sun et al. [27] reported an increase in DNA single-strand breaks in human lens epithelial cells after 2 h of exposure to a 1.8-GHz field at SARs of 3 and 4 W/kg. DNA damage caused by the field at 4 W/kg was irreversible. Zhang et al. [28] reported that an 1800-MHz field at 3.0 W/kg induced DNA damage in Chinese hamster lung cells after 24 h of exposure. Aitken et al. [29] exposed mice to a 900-MHz RFR at a SAR of 0.09 W/kg for 7 days at 12 h per day. DNA damage in caudal epididymal spermatozoa was assessed by quantitative PCR (QPCR) as well as by alkaline and pulsed-field gel electrophoresis. Gel electrophoresis revealed no significant change in single- or double-strand breaks in spermatozoa. However, QPCR revealed statistically significant damage to both the mitochondrial genome and the nuclear  $\beta$ -globin locus. Changes in sperm cell genome after exposure to 2450-MHz microwaves have also been reported previously by Sarkar et al. [20]. Related to this are several publications that have reported decreased motility and changes in morphology in isolated sperm cells exposed to cell phone radiation [30], sperm cells from animals exposed to cell phone radiation [31], and cell phone users [32–34]. Some of these *in vivo* effects could be caused by hormonal changes [35,36].

There also are studies reporting no significant effect of cell phone RFR exposure on DNA damage. After RFR-induced DNA damage was reported by Lai and Singh [16] using 2450-MHz microwaves and after the report of Phillips et al. [21] on cell phone radiation was published, Motorola funded a series of studies by Roti Roti and colleagues [37] at

Washington University to investigate DNA strand breaks in cells and animals exposed to RFR. None of the studies reported by this group found significant effects of RFR exposure on DNA damage [38–40]. However, a different version of the comet assay was used in these studies. More recently, four additional studies from the Roti-Roti laboratories also reported no significant effects on DNA damage in cells exposed to RFR. Li et al. [41] reported no significant change in DNA strand breaks in murine C3H10T1/2 fibroblasts after 2 h of exposure to 847.74- and 835.02-MHz fields at 3–5 W/kg. Hook et al. [42] showed that a 24-h exposure of Molt-4 cells to CDMA, FDMA, iDEN or TDMA-modulated RFR did not significantly alter the level of DNA damage. Lagroye et al. [43,44] also reported no significant change in DNA strand breaks, protein–DNA crosslinks, and DNA–DNA crosslinks in cells exposed to 2450-MHz RFR.

From other laboratories, Vijayalaxmi et al. [45] reported no increase in DNA strand breaks in human lymphocytes exposed *in vitro* to 2450-MHz RFR at 2.135 W/kg for 2 h. Tice et al. [46] measured DNA single-strand breaks in human leukocytes using the comet assay after exposure to various forms of cell phone signals. Cells were exposed for 3 or 24 h at average SARs of 1.0–10.0 W/kg. Exposure for either 3 or 24 h did not induce a significant increase in DNA damage in leukocytes. McNamee et al. [47–49] found no significant increase in DNA breaks and micronucleus formation in human leukocytes exposed for 2 h to a 1.9-GHz field at SAR up to 10 W/kg. Zeni et al. [50] reported that a 2-h exposure to 900-MHz GSM signal at 0.3 and 1 W/kg did not significantly affect levels of DNA strand breaks in human leukocytes. Sakuma et al. [51] exposed human glioblastoma A172 cells and normal human IMR-90 fibroblasts from fetal lungs to cell phone radiation for 2 and 24 h. No significant changes in DNA strand breaks were observed up to a SAR of 800 mW/kg. Stronati et al. [52] showed that 24 h of exposure to 935-MHz GSM basic signal at 1 or 2 W/kg did not cause DNA strand breaks in human blood cells. Verschaeve et al. [53] reported that long-term exposure (2 h/day, 5 days/week for 2 years) of rats to 900-MHz GSM signal at 0.3 and 0.9 W/kg did not significantly affect levels of DNA strand breaks in cells.

### 3. Extremely low frequency electromagnetic fields (ELF EMF) and DNA damage

To complete the picture, a few words on the effects of ELF EMF are required, since cell phones also emit these fields and they are another common form of non-ionizing EMF in our environment. Quite a number of studies have indicated that exposure to ELF EMF could lead to DNA damage [54–69]. In addition, two studies [70,71] have reported effects of ELF fields on DNA repair mechanisms. Free radicals and interaction with transitional metals (e.g., iron) [60,62,63,69] have also been implicated to play a role in the genotoxic effects observed after exposure to these fields.

### 4. Some considerations on the effects of EMF on DNA

From this brief literature survey, no consistent pattern of RFR exposure inducing changes in or damage to DNA in cells and organisms emerges. However, one can conclude that under certain conditions of exposure, RFR is genotoxic. Data available are mainly applicable only to radiation exposure that would be typical during cell phone use. Other than the study of Phillips et al. [21], there is no indication that RFR at levels that one can experience in the vicinity of base stations and RF-transmission towers could cause DNA damage.

Differences in experimental outcomes are expected since many factors could influence the outcome of experiments in EMF research. Any effect of EMF has to depend on the energy absorbed by a biological organism and on how the energy is delivered in space and time. Frequency, intensity, exposure duration, and the number of exposure episodes can affect the response, and these factors can interact with each other to produce different effects. In addition, in order to understand the biological consequence of EMF exposure, one must know whether the effect is cumulative, whether compensatory responses result, and when homeostasis will break down. The contributions of these factors have been discussed in a talk given by one of us (HL) in Vienna, Austria in 1998 [72].

Radiation from cell phone transmission has very complex patterns, and signals vary with the type of transmission. Moreover, the technology is constantly changing. Research results from one type of transmission pattern may not be applicable to other types. Thus, differences in outcomes of the research on genotoxic effects of RFR could be explained by the many different exposure conditions used in the studies. An example is the study of Phillips et al. [21], which demonstrated that different cell phone signals could cause different effects on DNA (i.e., an increase in strand breaks after exposure to one type of signal and a decrease with another). This is further complicated by the fact that some of the studies listed above used poor exposure procedures with very limited documentation of exposure parameters, e.g., using an actual cell phone to expose cells and animals, thus rendering the data from these experiments as questionable.

Another source of influence on experimental outcome is the cell or organism studied. Many different biological systems were used in the genotoxicity studies. Different cell types [73] and organisms [74,75] may not all respond similarly to EMF.

Comment about the comet assay also is required, since it was used in many of the EMF studies to determine DNA damage. Different versions of the assay have been developed. These versions have different detection sensitivities and can be used to measure different aspects of DNA strand breaks. A comparison of data from experiments using different versions of the assay could be misleading. Another concern is that most of the comet assay studies were carried out by experimenters who had no prior experience with this technique and mistakes

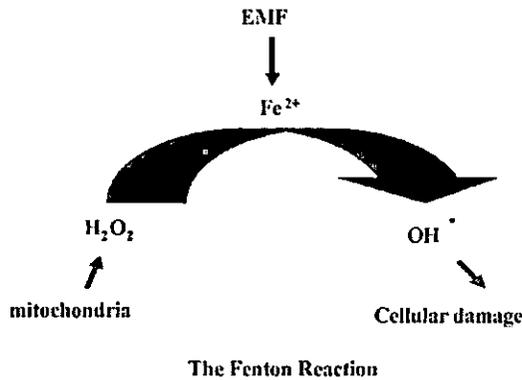


Fig. 1. A representation of the Fenton reaction and its role as a mediator in EMF-induced bioeffects.

were made. For example, in the study by Lagroye et al. [43] to investigate the effect of PK digestion on DNA migration after RFR exposure, PK was added to a lysing solution containing the detergent Triton X-100, which would inactivate the enzyme. Our experience indicates that the comet assay is a very sensitive and requires great care to perform. Thus, different detection sensitivities could result in different laboratories, even if the same procedures are followed. One way to solve this problem of experimental variation is for each research team to report the sensitivity of their comet assay, e.g., the threshold of detecting strand breaks in human lymphocytes exposed to X-rays. This information has generally not been provided for EMF-genotoxicity studies. Interestingly, when such information was provided, a large range of sensitivities have been reported. Malyapa et al. [40] reported a detection level of 0.6 cGy of gamma radiation in human lymphocytes, whereas McNamee et al. [76] reported 10–50 cGy of X-irradiation in lymphocytes, which is much higher than the generally acceptable detection level of the comet assay [15].

A drawback in the interpretation and understanding of experimental data from bioelectromagnetics research is that there is no general acceptable mechanism on how EMF affects biological systems. The mechanism by which EMF produces changes in DNA is unknown. Since the energy level associated with EMF exposure is not sufficient to cause direct breakage of chemical bonds within molecules, the effects are probably indirect and secondary to other induced biochemical changes in cells.

One possibility is that DNA is damaged by free radicals that are formed inside cells. Free radicals affect cells by damaging macromolecules, such as DNA, protein, and membrane lipids. Several reports have indicated that EMF enhances free radical activity in cells [18,19,61,62,77,78], particularly via the Fenton reaction [62]. The Fenton reaction is a process catalyzed by iron in which hydrogen peroxide, a product of oxidative respiration in the mitochondria, is converted into hydroxyl free radicals, which are very potent and cytotoxic molecules (Fig. 1).

It is interesting that ELF EMF has also been shown to cause DNA damage. Furthermore, free radicals have been implicated in this effect of ELF EMF. This further supports the view that EMF affects DNA via an indirect secondary process, since the energy content of ELF EMF is much lower than that of RFR. Effects via the Fenton reaction predict how a cell would respond to EMF. For instance:

- (1) Cells that are metabolically active would be more susceptible to EMF, because more hydrogen peroxide is generated by mitochondria to fuel the reaction.
- (2) Cells that have high level of intracellular free iron would be more vulnerable to EMF. Cancer cells and cells undergoing abnormal proliferation have higher concentrations of free iron because they uptake more iron and have less efficient iron storage regulation. Thus, these cells could be selectively damaged by EMF. Consequently, this suggests that EMF could potentially be used for the treatment of cancer and hyperplastic diseases. The effect could be further enhanced if one could shift anaerobic glycolysis of cancer cells to oxidative glycolysis. There is quite a large database of information on the effects of EMF (mostly in the ELF range) on cancer cells and tumors. The data tend to indicate that EMF could retard tumor growth and kill cancer cells. One consequence of this consideration is that epidemiological studies of cancer incidence in cell phone users may not show a risk at all or even a protection effect.
- (3) Since the brain is exposed to rather high levels of EMF during cell phone use, the consequences of EMF-induced genetic damage in brain cells are of particular importance. Brain cells have high levels of iron. Special molecular pumps are present on nerve cell nuclear membranes to pump iron into the nucleus. Iron atoms have been found to intercalate within DNA molecules. In addition, nerve cells have a low capacity for DNA repair, and DNA breaks could easily accumulate. Another concern is the presence of superparamagnetic iron-particles (magnetites) in body tissues, particularly in the brain. These particles could enhance free radical activity in cells and thus increase the cellular-damaging effects of EMF. These factors make nerve cells more vulnerable to EMF. Thus, the effect of EMF on DNA could conceivably be more significant on nerve cells than on other cell types of the body. Since nerve cells do not divide and are not likely to become cancerous, the more likely consequences of DNA damage in nerve cells include changes in cellular functions and in cell death, which could either lead to or accelerate the development of neurodegenerative diseases. Double-strand breaks, if not properly repaired, are known to lead to cell death. Cumulative DNA damage in nerve cells of the brain has been associated with neurodegenerative diseases, such as Alzheimer's, Huntington's, and Parkinson's diseases. However, another type of brain cell, the glial cell, can become cancerous as a result of DNA damage. The question is whether the damaged cells

would develop into tumors before they are killed by EMF due to over accumulation of genetic damages. The outcome depends on the interplay of these different physical and biological factors—an increase, decrease, or no significant change in cancer risk could result from EMF exposure.

- (4) On the other hand, cells with high amounts of antioxidants and antioxidative enzymes would be less susceptible to EMF. Furthermore, the effect of free radicals could depend on the nutritional status of an individual, e.g., availability of dietary antioxidants, consumption of alcohol, and amount of food consumption. Various life conditions, such as psychological stress and strenuous physical exercise, have been shown to increase oxidative stress and enhance the effect of free radicals in the body. Thus, one can also speculate that some individuals may be more susceptible to the effects of EMF exposure.

Additionally, the work of Blank and Soo [79] and Blank and Goodman [80] support the possibility that EMF exposure at low levels has a direct effect on electron transfer processes. Although the authors do not discuss their work in the context of EMF-induced DNA damage, the possibility exists that EMF exposure could produce oxidative damage to DNA.

## 5. Lessons learned

Whether or not EMF causes biological effects, let alone effects that are detrimental to human health and development, is a contentious issue. The literature in this area abounds with apparently contradictory studies, and as presented in this review, the literature specific to the effects of RFR exposure on DNA damage and repair in various biological systems is no exception. As a consequence of this controversy, there are several key issues that must be addressed—contrary data, weight of evidence, and data interpretation consistent with known science.

Consider that EMF does not share the familiar and comforting physical properties of chemical agents. EMF cannot be seen, tasted, smelled, or felt (except at high intensities). It is relevant, therefore, to ask, in what ways do scientists respond to data, especially if that data are contrary to their scientific beliefs or inconsistent with long-held hypotheses? Often such data are ignored, simply because it contradict what is accepted as conventional wisdom. Careful evaluation and interpretation of data may be difficult, because technologies used to expose biological systems to EMF and methodologies used to assess dosimetry generally are outside the experience of most biomedical scientists. Additionally, it is often difficult to assess differences in methodologies between studies, one or more of which were intended to replicate an original investigation. For instance, Malyapa et al. [40] reported what they claimed to be a replication of the work of Lai and Singh [16]. There were, however, significant differences

in the comet analyses used by each group. Lai and Singh precipitated DNA in agarose so that low levels of DNA damage could be detected. Malyapa et al. did not. Lai and Singh treated their samples with PK to digest proteins bound to DNA, thus allowing DNA to move toward the positive pole during electrophoresis (unlike DNA, most proteins are negatively charged, and if they are not removed they will drag the DNA toward the negative pole). The Malyapa et al. study did not use PK. There were other methodological differences as well. Such is also the case in the study of Hook et al. [42], which attempted to replicate the work of Phillips et al. [21]. The latter group used a PK treatment in their comet assay, while the former group did not.

While credibility is enhanced when one can relate data to personal knowledge and scientific beliefs, it has not yet been determined how RFR couples with biological systems or by what mechanisms effects are produced. Even carefully designed and well executed RFR exposure studies may be summarily dismissed as methodologically unsound, or the data may be interpreted as invalid because of inconsistencies with what one believes to be correct. The quintessential example is the belief that exposure to RFR can produce no effects that are not related to the ability of RFR to produce heat, that is, to raise the temperature of biological systems [81,82]. Nonetheless, there are many examples of biological effects resulting from low-level (athermal) RFR exposure [83,84]. Consider here the work of Mashevich et al. [85]. This group exposed human peripheral blood lymphocytes to an 830-MHz signal for 72 h and at different average SARs (SAR, 1.6–8.8 W/kg). Temperatures ranged from 34.5 to 38.5 °C. This group observed an increase in chromosome 17 aneuploidy that varied linearly with SAR. Temperature elevation alone in the range of 34.5–38.5 °C did not produce this genotoxic effect, although significant aneuploidy was observed at higher temperatures of 40–41 °C. The authors conclude that the genotoxic effect of the radiofrequency signal used is elicited through a non-thermal pathway.

Also consider one aspect of the work of Phillips et al. [21]. In that study, DNA damage was found to vary in direction; that is, under some conditions of signal characteristics, signal intensity, and time of exposure, DNA damage increased as compared with concurrent unexposed controls, while under other conditions DNA damage decreased as compared with controls. The dual nature of Phillips et al.'s [21] results will be discussed later. For now consider the relationship of these results to other investigations. Adey et al. [86] performed an *in vivo* study to determine if rats treated *in utero* with the carcinogen ethylnitrosourea (ENU) and exposed to an 836.55-MHz field with North American Digital Cellular modulation (referred to as a TDMA field) would develop increased numbers of central system tumors. This group reported that rather than seeing an increase in tumor incidence in RFR-exposed rats, there was instead a decrease in tumor incidence. Moreover, rats that received no ENU but which were exposed to the TDMA signal also showed a decrease in the number of spontaneous tumors as compared

with animals exposed to neither ENU nor the TDMA signal. This group postulated that their results may be mechanistically similar to the work of another group. Stammberger et al. [87] had previously reported that rats treated *in utero* with ENU and then exposed to low doses of X-irradiation exhibited significantly reduced incidences of brain tumors in adult life. Stammberger and colleagues [87] hypothesized that low-level X-irradiation produced DNA damage that then induced the repair enzyme O<sup>6</sup>-alkylguanine-DNA alkyltransferase (AT). Numerous groups have since reported that X-irradiation does indeed induce AT activity (e.g., [88,89]). In this context, it is significant that Phillips et al. [21] found that cells exposed *in vitro* to a TDMA signal identical to that used in the study of Adey et al. [86] produced a decrease in DNA damage under specific conditions of intensity and time of exposure (lower intensity, longer time; higher intensity, shorter time). These results raise the intriguing possibility that the decrease in tumor incidence in the study of Adey et al. [86] and the decrease in DNA damage in the study of Phillips et al. [21] both may have been the result of induction of AT activity resulting from DNA damage produced by exposure to the TDMA signal. This remains to be investigated.

Because the issue of RFR-induced bioeffects is contentious, and because the issue is tried in courtrooms and various public forums, a term heard frequently is weight of evidence. This term generally is used to describe a method by which all scientific evidence related to a causal hypothesis is considered and evaluated. This process is used extensively in matters of regulation, policy, and the law, and it provides a means of weighing results across different modalities of evidence. When considering the effects of RFR exposure on DNA damage and repair, modalities of evidence include studies of cells and tissues from laboratory animals exposed *in vivo* to RFR, studies of cells from humans exposed to RFR *in vivo*, and studies of cells exposed *in vitro* to RFR. While weight of evidence is gaining favor with regulators [90], its application by scientists to decide matters of science is often of questionable value. One of the reasons for this is that there generally is no discussion or characterization of what weight of evidence actually means in the context in which it is used. Additionally, the distinction between weight of evidence and strength of evidence often is lacking or not defined, and differences in methodologies between investigators are not considered. Consequently, weight of evidence generally amounts to what Krinsky [90] refers to as a "seat-of-the-pants qualitative assessment." Krinsky points out that according to this view, weight of evidence is "a vague term that scientists use when they apply implicit, qualitative, and/or subjective criteria to evaluate a body of evidence." Such is the case in the reviews by Juutilainen and Lang [91] and Verschaeve and Maes [92]. There is little emphasis on a critical analysis of similarities and differences in biological systems used, exposure regimens, data produced, and investigator's interpretations and conclusions. Rather, there is greater emphasis on the number of publications either finding or not finding an effect of RFR exposure on some endpoint.

To some investigators, weight of evidence does indeed refer to the balance (or imbalance) between the number of studies producing apparently opposing results, without regard to critical experimental variables. While understanding the role these variables play in determining experimental outcome could provide remarkable insights into defining mechanisms by which RFR produced biological effects, few seem interested in or willing to delve deeply into the science.

A final lesson can be derived from a statement made by Gos et al. [93] referring to the work of Phillips et al. [21]. Gos and colleagues state, "The results in the latter study (Phillips et al., 1998) are puzzling and difficult to interpret, as no consistent increase or decrease in signal in the comet assay at various SARs or times of exposure was identified." This statement is pointed out because studies of the biological effects of exposure to electromagnetic fields at any frequency are often viewed as outside of or distinct from what many refer to as *mainstream science*. However, what has been perceived as an inconsistent effect is indeed consistent with the observations of bimodal effects reported in hundreds of peer-reviewed publications. These bimodal effects may be dependent on concentration of an agent, time of incubation with an agent, or some other parameter relating to the state of the system under investigation. For instance, treatment of B cells for a short time (30 min) with the protein kinase C activator phorbol 12,13-dibutyrate increased proliferative responses to anti-immunoglobulin antibody, whereas treatment for a longer period of time ( $\geq 3$  h) suppressed proliferation [94]. In a study of  $\kappa$ -opioid agonists on locomotor activity in mice, Kuzmin et al. [95] reported that higher, analgesic doses of  $\kappa$ -agonists reduced rearing, motility, and locomotion in non-habituated mice. In contrast, lower, subanalgesic doses increased motor activity in a time-dependent manner. Dierov et al. [96] observed a bimodal effect of all-trans-retinoic acid (RA) on cell cycle progression in lymphoid cells that was temporally related to the length of exposure to RA. A final example is found in the work of Rosenstein et al. [97]. This group found that the activity of melatonin on depolarization-induced calcium influx by hypothalamic synaptosomes from rats sacrificed late evening (2000 h) depended on melatonin preincubation time. A short preincubation time (10 min) stimulated uptake, while a longer preincubation (30 min) inhibited calcium uptake. These effects were also dependent on the time of day when the rats were sacrificed. Effects were maximal at 2000 h, minimal at 2400 h, and intermediate at 400 h. At 1000 h, only inhibitory effects of melatonin on calcium uptake were observed. These examples point out that what appears to be inconsistency may instead be real events related to and determined by the agents involved and the state of the biological system under investigation. The results of Phillips et al. [21] may be the result of signal modulation, signal intensity, time of exposure, or state of the cells. The results may indicate a bimodal effect, or they may, as the investigators suggest, represent time- and signal-dependant changes in the balance between damage and repair because of direct or indirect effects of RFR exposure on repair mechanisms.

## 6. Summary

Exposure of laboratory animals *in vivo* and of cultured cells *in vitro* to various radiofrequency signals has produced changes in DNA damage in some investigations and not in others. That many of the studies on both sides of this issue have been done well is encouraging from a scientific perspective. RFR exposure does indeed appear to affect DNA damage and repair, and the total body of available data contains clues as to conditions producing effects and methodologies to detect them. This view is in contrast to that of those who believe that studies unable to replicate the work of others are more credible than the original studies, that studies showing no effects cancel studies showing an effect, or that studies showing effects are not credible simply because we do not understand how those effects might occur. Some may be tempted to apply incorrectly the teachings of Sir Karl Popper, one of the great science philosophers of the 20th century. Popper proposed that many examples may lend support to an hypothesis, while only one negative instance is required to refute it [98]. While this holds most strongly for logical subjects, such as mathematics, it does not hold well for more complex biological phenomena that are influenced by stochastic factors. Each study to investigate RFR-induced DNA damage must be evaluated on its own merits, and then studies that both show effects and do not show effects must be carefully evaluated to define the relationship of experimental variables to experimental outcomes and to assess the value of experimental methodologies to detect and measure these outcomes (see Section 2).

The lack of a causal or proven mechanism(s) to explain RFR-induced effects on DNA damage and repair does not decrease the credibility of studies in the scientific literature that report effects of RFR exposure, because there are several plausible mechanisms of action that can account for the observed effects. The relationship between cigarette smoking and lung cancer was accepted long before a mechanism was established. This, however, occurred on the strength of epidemiologic data [99]. Fortunately, relevant epidemiologic data relating long-term cell phone use (>10 years) to central nervous system tumors are beginning to appear [84,100–102], and these data point to an increased risk of acoustic neuroma, glioma and parotid gland tumors.

One plausible mechanism for RFR-induced DNA damage is free radical damage. After finding that two free radical scavengers (melatonin and N-tert-butyl- $\alpha$ -phenylnitron) prevent RFR-induced DNA damage in rat brain cells, Lai and Singh [62] hypothesized that this damage resulted from free radical generation. Subsequently, other reports appeared that also suggested free radical formation as a result of RFR exposure [103–105]. Additionally, some investigators have reported that non-thermal exposure to RFR alters protein structure and function [106–109]. Scientists are familiar with molecules interacting with proteins through lock-and-key or induced-fit mechanisms. It is accepted that such interactions provide energy to change protein conformation and protein

function. Indeed, discussions of these principles are presented in introductory biology and biochemistry courses. Perhaps then it is possible that RFR exposure, in a manner similar to that of chemical agents, provides sufficient energy to alter the structure of proteins involved in DNA repair mechanisms to the extent that their function also is changed. This has not yet been investigated.

When scientists maintain their beliefs in the face of contrary data, two diametrically opposed situations may result. On the one hand, data are seen as either right or wrong and there is no discussion to resolve disparities. On the other hand, and as Francis Crick [110] has pointed out, scientists who hold theoretically opposed positions may engage in fruitful debate to enhance understanding of underlying principles and advance science in general. While the latter certainly is preferable, there are external factors involving economics and politics that keep this from happening. It is time to acknowledge this and embark on the path of fruitful discussion. Great scientific discoveries await.

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SINCE 1828 MENU

# endangered species

*noun*

Popularity: Bottom 30% of words

## Definition of ENDANGERED SPECIES

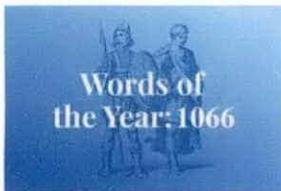
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**BUSINESS**

## Duke Energy earnings for 2016 down from previous year

BY BRUCE HENDERSON  
*bhenderson@charlotteobserver.com*

FEBRUARY 16, 2017 7:51 AM

Duke Energy reported \$2.1 billion in 2016 earnings Thursday, a 23 percent drop from the \$2.8 billion of the previous year, but reached the high end of its earnings per share target.

The company said it would expand its capital spending over the next five years to invest heavily in updating its grid, measures that can improve reliability for customers.

Duke reported earnings per share of \$3.11, down from \$4.05 for the full year of 2015. Adjusted for one-time costs, including a currency-related loss on the sale of its Latin America businesses that had been expected, earnings were \$4.69 a share compared to \$4.54 in 2015.

Duke had expected a strong third quarter and the early close of Duke's acquisition of Piedmont to push 2016 earnings to the high end of its guidance costs of \$4.50 to \$4.70 a share, not including repair costs from Hurricane Matthew in October.

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Favorable weather, including a warm summer that had customers cranking up their air conditioning, and an early close, in October, of the Duke's acquisition of Piedmont Natural Gas boosted yearly earnings, the company said.

Chairman and CEO Lynn Good, in a statement, called 2016 "a transformational year for Duke Energy as we acquired Piedmont Natural Gas and exited our international business, positioning the company for more consistent earnings and cash flow growth. We continue to advance our long-term growth strategy to modernize the energy grid, generate cleaner energy and expand natural gas infrastructure."

Duke set its 2017 target for adjusted earnings in a range between \$4.50 to \$4.70 a share.

Duke reported a fourth-quarter loss of \$227 million, or 33 cents a share, compared to the \$477 million in profits and 69 cents a share of one year earlier. Duke attributed the loss largely to the sale of its international business.

Adjusted earnings for the quarter were 81 cents a share, meeting the consensus of analysts surveyed by Zacks Investment Research, compared to 87 cents in adjusted earnings a year earlier.

## **Grid reliability**

Duke also said it would boost spending on capital projects, largely tied to grid improvements, by 25 percent over the next five years to \$37 billion.

Longer term, the company plans to spend \$25 billion over the next decade in modernizing the grid and \$11 billion on cleaner energy such as natural gas, solar and wind. Duke also wants to nearly double its earnings from natural gas.

Grid improvements will be aimed at improving reliability, such as by placing power lines underground and limiting outages due to storms, and in “smart grid” improvements that use digital technology to give customers more information and options about their energy use.

Duke, the second-largest U.S. electric utility by market capitalization, returned to core businesses in 2016 by dumping its volatile businesses in Latin America and closing its \$4.9 billion merger with Piedmont Natural Gas.

Duke owns a 47 percent stake in a company that will build and own a natural gas pipeline from West Virginia to eastern North Carolina. Duke expects an environmental study of the Atlantic Coast Pipeline to be completed by late June, with federal approval 90 days later.

Chief financial officer Steve Young, in a Bloomberg broadcast interview, said Duke likes President Donald Trump’s emphasis on building infrastructure such as pipelines. Duke hopes regulatory approvals for those projects would be approved quicker under the Trump administration, he said.

Young said Duke, after the Piedmont acquisition and shedding the international and merchant plant businesses in the Midwest, will continue to look for expansion opportunities.

“We’ve got the businesses that we want and we’re going to develop them organically; great opportunities there,” he told Bloomberg. “We will keep our eye on the markets for assets as we have done in the past. We’re opportunistic and we’ll be frugal with our cost of capital, but great opportunities out there as we build the grid of the United States and decarbonize.”

Duke received a construction and operating license for the Lee nuclear plant in South Carolina but hasn’t decided whether to build it. Cost overruns for two nuclear stations under construction in South Carolina and Georgia threaten to topple Japan’s Toshiba, which owns Westinghouse, designer of the reactors the plants will use.

*Bruce Henderson: 704-358-5051, @bhender*

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[Breast cancer genetics. BRCA1 and BRCA2: the main genes for disease predisposition].

[Article in Spanish]

Ruiz-Flores P(1), Calderón-Garcidueñas AL, Barrera-Saldaña HA.

Author information:

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Breast cancer is among the most common world cancers. In Mexico this neoplasm has been progressively increasing since 1990 and is expected to continue. The risk factors for this disease are age, some reproductive factors, ionizing radiation, contraceptives, obesity and high fat diets, among other factors. The main risk factor for BC is a positive family history. Several families, in which clustering but no mendelian inheritance exists, the BC is due probably to mutations in low penetrance genes and/or environmental factors. In families with autosomal dominant trait, the BRCA1 and BRCA2 genes are frequently mutated. These genes are the two main BC susceptibility genes. BRCA1 predispose to BC and ovarian cancer, while BRCA2 mutations predispose to BC in men and women. Both are long genes, tumor suppressors, functioning in a cell cycle dependent manner, and it is believed that both switch on the transcription of several genes, and participate in DNA repair. The mutations profile of these genes is known in developed countries, while in Latin America their search has just began. A multidisciplinary group must be responsible of the clinical management of patients with mutations in BRCA1 and BRCA2, and the risk assignment and Genetic counseling must be done carefully.

PMID: 11332051 [Indexed for MEDLINE]



# Power Lines, Electrical Devices and Extremely Low Frequency Radiation

## What is extremely low frequency (ELF) radiation?

Radiation is the emission or sending out of energy from any source. X-rays are an example of radiation, but so is the light that comes from the sun and the heat that is constantly coming off our bodies.

When talking about radiation and cancer, many people think of specific kinds of radiation such as x-rays or the radiation in nuclear reactors. But these are not the only types of radiation that concern us when we think about radiation risks to human health.

Radiation exists across a spectrum from very high-energy (also referred to as high-frequency) radiation to very low-energy (or low-frequency) radiation. This is sometimes referred to as the **electromagnetic spectrum**.

Examples of high-energy radiation include x-rays and gamma rays. They, as well as some higher energy ultraviolet (UV) rays, are classified as **ionizing radiation**, which means that they have enough energy to remove an electron from (ionize) an atom. Ionizing radiation can damage the DNA inside cells, which can lead to mutations and the uncontrolled cell growth we know as cancer.

to many substances other than the one being studied, and these other exposures could affect the results.

In most cases neither type of study provides conclusive evidence on its own, so researchers usually look at both lab-based and human studies when trying to figure out if something can cause cancer.

## Studies in the lab

Several large studies have looked at the possible effects of ELF magnetic fields on cancer in rats and mice. These studies expose the animals to magnetic fields much stronger than what people are normally exposed to at home, with fields ranging from 2 to 5000 microtesla ( $\mu\text{T}$ ). Most of these studies have found no increase in the risk of any type of cancer. In fact, the risk of some types of cancer was actually lower in the animals exposed to the ELF radiation. One study did show an increased risk of tumors that start in thyroid cells, called C-cells, in male rats at some exposures. This increased risk was not seen in female rats or in mice, and was not seen at the highest field strength. These inconsistencies, and the fact that these findings were not consistently seen in the other studies, make it hard for scientists to conclude that the observed increased risk of tumors is from the ELF radiation.

Other studies in mice and rats have looked specifically for increases in leukemia and lymphoma as a result of exposure to ELF radiation, but these studies have also not found a link.

## Studies in people

Studying the effects of ELF radiation in people can be hard, for many reasons:

Exposure to ELF radiation is very common, so it's not possible to compare people who are exposed with people who aren't exposed. Instead, studies try to compare people exposed at higher levels with people exposed at lower levels.

It is very hard to determine how much ELF radiation a person has been exposed to, especially over a long period. As far as we know, the effects of ELF radiation do not add up over time, and there is no test that can measure how much exposure a person has had.

Researchers can get a snapshot of ELF exposures by having a person wear a device that records their exposure levels over hours or days. Or, researchers can measure the magnetic or electrical field strength in a person's home or workplace settings.

Other options include estimating exposure based on the wiring configuration of someone's workplace/home or on its distance from power lines. But these methods result in exposure estimates that have a lot of uncertainty and that can produce biased estimates of total exposure. They typically do not account for a person's ELF exposures while in other places, they don't measure ELF exposures in every location that person has ever lived or worked over their lifetime. As a result, there are no good ways to accurately estimate someone's long-term exposure, which is what matters most when looking for possible effects on cancer risk.

## In children

- A number of studies have looked at a possible link between ELF radiation from **magnetic fields** in the home and childhood leukemia (</cancer/leukemia-in-children.html>), with mixed results. Still, when the findings from these studies are combined, a small increase in risk is seen for children at the highest exposure levels compared to those with the lowest exposure levels. Studies looking at the effect of ELF *electric fields* on childhood leukemia have not found a link.

Studies have generally not found any strong links between ELF electric or magnetic fields and other types of childhood cancers.

## In adults

Although several studies have looked at possible links between ELF exposures in adults and cancer, most have not found a link.

Extremely low frequency (ELF) radiation is at the low-energy end of the electromagnetic spectrum and is a type of **non-ionizing radiation**. Non-ionizing radiation has enough energy to move atoms around or make them vibrate, but not enough to directly damage DNA. ELF radiation has even lower energy than other types of non-ionizing radiation like radiofrequency radiation, visible light, and infrared.

With most types of radiation, the electric and magnetic fields are coupled. Because they act as one, they are considered together as an electromagnetic field (EMF). But with ELF radiation, the magnetic field and the electrical field can exist and act independently, so they are often studied separately. Typically, we use the term “magnetic field” to indicate ELF radiation from a magnetic field, while we use “electric field” to mean ELF radiation from an electric field.

The possible link between electromagnetic fields and cancer has been a subject of controversy for several decades. It's not clear exactly how electromagnetic fields, a form of low-energy, non-ionizing radiation, can increase cancer risk. Plus, because we are all exposed to different amounts of these fields at different times, the issue has been hard to study.

## Electric and magnetic fields

All radiation on the electromagnetic spectrum is produced by the interactions of 2 forces, referred to as **fields**. Radiation has both an electric field and a magnetic field.

**Electric fields** are the forces acting on charged particles (parts of atoms), like electrons or protons, which cause them to move. Electric current is simply the flow of electrons produced by an electric field. The strength of an electric field is often expressed as volts per meter (V/m) or, for stronger fields, as kilovolts per meter (kV/m), where a kilovolt is 1000 volts.

A **magnetic field** is created when charged particles are in motion. The strength of a magnetic field can be expressed in many different units, including tesla (T), microtesla ( $\mu\text{T}$  or one millionth of a tesla), and gauss (G), where one G equals 100  $\mu\text{T}$ .

# How are people exposed to ELF radiation?

Generating, transmitting, distributing, and using electricity all expose people to ELF radiation. Power lines, household wiring, and any device that uses electricity can generate ELF radiation. Thus any electric device, from refrigerators and vacuum cleaners to televisions and computer monitors (when they are on) are sources of ELF radiation. Even electric blankets expose people to ELF radiation.

How much electromagnetic radiation you are exposed to depends on the strength of the electromagnetic field, your distance from the source of the field, and the length of time you are exposed. The highest exposure occurs when the person is very close to a source putting out a strong field and stays there for a long period.

# Does ELF radiation cause cancer?

Researchers use 2 main types of studies to try to figure out if something causes cancer.

- **Lab studies:** In lab studies, animals are exposed to different levels of the substance (sometimes at extremely high levels) to see if this exposure causes tumors or other health problems. Researchers might also expose normal human cells in a lab dish to see if this causes the types of changes that are seen in cancer cells. It's not always clear that the results from these types of studies directly apply to humans, but lab studies are a good way to find out if an exposure might possibly cause cancer.
- **Studies in people:** Other types of studies look at cancer rates in different groups of people. Such a study might compare the cancer rate in an exposed group to the rate in a group with lower exposures, or to a group not exposed at all. Sometimes the exposed group's cancer rate is compared to the cancer rate in the general population. But it can be hard to know what the results of these studies mean, because many other factors might affect the results. For example, people are typically exposed

# What expert agencies say

Several national and international agencies study different exposures in the environment to determine if they can cause cancer. (Something that causes cancer or helps cancer grow is called a *carcinogen*.) The American Cancer Society looks to these organizations to evaluate the risks based on evidence from laboratory, animal, and human research studies.

Based on animal and human evidence like the examples above, some expert agencies have evaluated the cancer-causing nature of ELF radiation.

The **International Agency for Research on Cancer (IARC)** is part of the World Health Organization (WHO). One of its major goals is to identify causes of cancer. In 2002, IARC considered the evidence for ELF magnetic and electric fields separately:

- It found “limited evidence” in humans for the carcinogenicity of ELF *magnetic* fields in relation to childhood leukemia, with “inadequate evidence” in relation to all other cancers. It found “inadequate evidence” for the carcinogenicity of ELF magnetic fields based on studies in lab animals.
- It found “inadequate evidence” for the carcinogenicity of ELF *electric* fields in humans.

Based on this assessment, IARC has classified ELF *magnetic* fields as “possibly carcinogenic to humans.” It has classified ELF *electric* fields as “not classifiable as to their carcinogenicity to humans.”

In 1999, the US **National Institute of Environmental Health Sciences (NIEHS)** described the scientific evidence suggesting that ELF exposure poses a health risk as “weak,” but noted that it cannot be recognized as entirely safe, and considered it to be a “possible” human carcinogen.

# How can I avoid exposure to ELF radiation?

It's not clear that exposure to ELF radiation is harmful, but there are things you can do to lower your exposure if you are concerned. Your exposure is based on the strength of the ELF radiation coming from each source, how close you are to each, and how long you spend in the field.

The NIEHS recommends that people concerned about their exposure to EMF (and ELF radiation) find out where their major EMF sources are and move away from them or limit the time spent near them. For example, moving even an arm's length away from a source can dramatically lower exposure to its field.

## Power lines

People who are concerned about ELF radiation exposure from high-power electrical lines should keep in mind that the intensity of any exposure goes down significantly as you get farther away from the source. On the ground, the strength of the electromagnetic field is highest directly under the power line. As you get farther away, you are exposed to less and less, with the level eventually matching normal home background levels. The electromagnetic field directly under a power line is typically in the range of what you could be exposed to when using certain household appliances.

If you are concerned about your exposure to electromagnetic sources around you (including power lines), you can measure the field strength with a device called a *gaussmeter*.

Written by    Additional resources    References

The American Cancer Society medical and editorial content team



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Mr. Christopher J. Ayers  
Executive Director Public Staff  
4326 Mail Service Center  
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State of North Carolina Utilities Commission Docket No. E-2, Sub 1150 (Cleveland Matthews Project)

I am enclosing a copy of an email to Duke Energy Progress to move the proposed line and to not involve my property (parcels on Elevation Road and Gum Swamp Road).

Thank you,

Kimberly Canady

A handwritten signature in cursive script that reads "Kimberly Canady".

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Clerk's Office  
N.C. Utilities Commission

CenturyLink Webmail

kimberlycanady@centurylink.net

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**Duke Energy Cleveland Matthews Project-Request to move project**

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**From :** KIMBERLY CANADY <kimberlycanady@centurylink.net> Tue, Oct 10, 2017 10:43 PM  
**Subject :** Duke Energy Cleveland Matthews Project-Request to move project  
**To :** Miranda Gregory <Miranda.Gregory@duke-energy.com>

Ms. Gregory, you are the only Duke Energy employee that has reached out to me by e-mail, therefore, I am sending my request to you.

Please move the proposed center line no closer than 500 feet beyond the boundary of my property (parcels on Elevation Road and Gum Swamp Road). I have been diagnosed with a rare hereditary gene mutation that predisposes me to cancer. My children *may* have also inherited the gene mutation. Living close to a high voltage power line, with EMF exposure and possibly exposure to herbicides used in maintaining the line is too great of a risk. In Appendix C, page 38 of 51 in the docket, in the section on Electric and Magnetic Fields, states in the absence of widespread government standards, it becomes a matter of personal responsibility to weigh the potential risks associated with EMF's and determine your response. I am responding by making a written request to not involve my property in the proposed Duke Energy Cleveland Matthews project.

As docket E-2, Sub 1150 states, Mr. Chris Ayers with the NC Utilities Commission and the Honorable Josh Stein represent the using and consuming public. I will forward a printed copy of this request to their respective addresses listed in the docket.

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Raleigh, NC 27699-4300

Hearing request in regards to the State of North Carolina Utilities Commission Docket No. E-2, Sub 1150 (Cleveland Matthews Project)

I am writing to request a hearing on this project, so that I may voice my opposition to the construction of the transmission line across my property. I am a property owner in segment 33 of the proposed route for this project, in the Four Oaks area of Johnston County. I had no personal contact with Duke Energy employees regarding this project, prior to the determination of the proposed route, and therefore my comments and concerns were not addressed prior to the public deadline for comments. I specifically have concerns related to public notification of the project, the proposed route across my property, health concerns and environmental impact.

Docket No., E-2, Sub 1150 is an application of Duke Energy progress, LLC to construct approximately 11.5 miles of new 230kV transmission line in the Cleveland area of Johnston County. This statement to the Utilities Commission should be a mirror image of what was presented to the public prior to the close of public comment.

The preferred route is 11.5 miles and is primarily not located in the Cleveland area of Johnston County. There are other routes that are shorter and are located in the geographical area that Duke Energy has described. In section 2.1.1 of the Routing Necessity and Environmental Report by Burns McDonnell for Duke Energy it states, "A new substation site in the vicinity of Cleveland Road and Matthews Road would be advantageous to continue providing this area of developing Johnston County with reliable electric service. The new substation and associated transmission line would provide greater capacity and enhanced service reliability to the area to support residential and commercial growth." I would like to ask that the transmission line stay in the general area where it is needed.

I would like to ask that representatives from the Utilities Commission perform a site visit to my property. A site visit and consideration of this hearing request would be greatly appreciated.

Kimberly L. Canady

*Kimberly L. Canady*  
950 Gum Swamp Rd.  
Four Oaks, NC 27524

October 24, 2017

Mr. Christopher J. Ayers  
Executive Director Public Staff  
4326 Mail Service Center  
Raleigh, NC 27699-4300

The State of North Carolina Utilities Commission Docket No. E-2, Sub 1150 (Cleveland Matthews Project)

I am writing to express my concerns with the Application of Duke Energy Progress, LLC for a Certificate of Environmental Compatibility and Public Convenience and Necessity to Construct Transmission Line in the Cleveland Area of Johnston County, North Carolina, Docket E-2, Sub 1150.

I live in Segment 33 of the proposed route. I inherited the land that Duke Energy Progress (DEP) would like to take for its use from my grandfather. My brother and I own the farm jointly. I live on approximately 2 acres, adjacent the farm with my husband of 23 years, 17-year-old daughter and 10-year-old son. I have lived adjacent to this property my entire life, 43 years, with the exception of 9 months that I was away at school. My brother lives in Benson, NC. My father died of kidney cancer at 44 years old in 1992, prior to my grandfather passing away in 1998.

I have included documentation related to my family heritage. Elijah Lassiter who lived from 1782-1848 was a soldier of the Revolution, founded the Lassiter family (in this area) and for almost 200 years, held land on the north and south side of Black Creek in what is now Elevation Township. That is where I live. There are 5 of Elijah Lassiter's descendants between the ages of 10-17, in our immediate families, that would potentially inherit land from either myself and my brother, Marty R. Lassiter, or my aunt, Linda L. Keen.

Certainly, I would have raised concerns with Duke Energy Progress, if the November 4, 2016 letter would have been written more clearly. There are a number of landowners along the chosen route, in addition to myself that feel that public notification was inadequate. Not all landowners received the SINGLE notification that was mailed November 4, 2016, prior to the close of public comment (per landowners Linda and Russell Keen). Other landowners have stated to me that they did not get the certified letter that was dated April 20 (per landowners Roy and Sue Massengill and Oliver L. Canaday). I received the Nov 4 letter (**that should have been required to be sent out certified mail**), but did not understand the potential impact of the project from Duke Energy Progress' correspondence. **The Nov 4 letter proved to be a key in the determination of the route because public comment was used in route selection.** My brother, Marty R. Lassiter has stated that he would have had no clue about project if he were not on good terms with me, his sister. We have been told by DEP representatives that correspondence goes to 1<sup>st</sup> on the deed. My opinion is that this process of notification is unacceptable and any correspondence needs to go to both parties, since both parties are legally responsible for the land. My brother has stated that he felt like something was trying to be slid in on us. It is important to note too that he owns a parcel within 500 feet of the proposed line that bears only his name on the deed.

Specifically, I'd like to address the statement of not understanding the impact of the project from the letter. Per the article [www.ncbi.nlm.nih.gov](http://www.ncbi.nlm.nih.gov) (National Institutes of Health) the average US resident, reads at or below an 8<sup>th</sup> grade level. I feel that correspondence from any entity that could exercise the use of eminent domain should be required to write at or below an eighth-grade reading level.

The attached November 4, 2016 letter from Duke Energy Progress' subject line states "Important information about a Duke Energy investment in your community." What is important to note is **I DO NOT live in the Cleveland area of Johnston County.** This subject line is misleading.

Also, the November 4, 2016 letter states that Duke Energy Progress was "writing to inform you that Duke Energy identified the need for a transmission-to-distribution substation in the **Cleveland area of Johnston County** to meet the projected growth of the area." It further states that DEP needs to construct a new transmission tap line. According to [www.merriam-webster.com](http://www.merriam-webster.com) the definition that most closely applies to electricity is "an intermediate point in an electric circuit where a connection may be made." I do not have a background in electricity or utilities. My thought process was that Duke Energy Progress wanted to connect to an electrical line that currently exists on my land. Duke Energy Progress failed to notify me in a clear concise way that they needed or would potentially need a **125-150 ft. easement through my property** to build 65-85 ft. H frame structures with high voltage power lines to carry electricity to the new substation at the intersection of Matthews Road and Polenta Road **prior to the close of comment.** The previous sentence is easy for anyone to understand and is an example of being transparent. My concern is that Duke Energy Progress' intent was to be ambiguous enough in their notification as to not solicit a response from all landowners. Lack of transparency on the part of Duke Energy Progress is not, "doing the right thing."

I am enclosing information about the Lassiter's settling in the area our homeplace is located, and factors that I think should have been considered when siting on our family homeplace.

I am asking that you please consider this information when determining the final route selection.

Thank you,



Kimberly L. Canady



Nov. 4, 2016

**LASSITER, MARTY R CANADY, KIMBERLY L  
950 GUM SWAMP RD  
FOUR OAKS, NC 27524-0000**

**Important information about a Duke Energy investment in your community**

Dear Property Owner:

Duke Energy's electrical system is essential to meeting customers' needs, and system reliability is a responsibility that we take seriously. We work to ensure a resilient and secure smart grid to provide reliable service today and in the future. We are committed to being responsive to customers' needs, providing accurate information as well as communicating frequently and transparently with the community.

We are writing to inform you that Duke Energy identified the need for a transmission-to-distribution substation in the Cleveland area of Johnston County to meet the projected growth of the area. The substation will convert the 230-kilovolt (230-kV) transmission line voltage down to 23 kV to serve homes and businesses through local distribution lines. To serve the new substation, we need to construct a new 230-kV transmission tap line to run from one of three existing transmission lines to the new substation.

The new substation will be constructed on land currently owned by Duke Energy on the southeast corner of the intersection of Matthews Road and Polenta Road. The new transmission tap line to feed the substation will connect to one of three existing 230-kV lines: the Lee Sub-Milburnie 230-kV line to the northeast; the Erwin-Selma 230-kV line to the southeast; or the Erwin-Milburnie 230-kV line to the west. The total new investment in this Johnston County system upgrade for the Cleveland area is estimated to be approximately \$28.4 million.

The general locations and proposed alignments of the various alternative routes currently under consideration for the new transmission tap line are depicted on the enclosed map. You are receiving this letter and invitation to a public information open house because your property (or more than one property) falls within 500 feet of the centerline of one of the potential routes being considered for the new transmission tap line. Our goal is to minimize impacts to personal property, homes, businesses, the environment and cultural resources.

**We invite you to attend one of two informational open houses  
to learn more about this important project:**

**Wednesday, Nov. 16, 2016 | 4-7 p.m.  
C3 Church  
8246 Cleveland Road | Clayton, NC 27520**

**Thursday, Nov. 17, 2016 | 4-7 p.m.  
Johnston County Community College – Tart Building  
245 College Road | Smithfield, NC 27577**

Public participation is a vital part of this process, and that's why we hope you'll attend one of these meetings and provide your input. The meetings will be set up in an open house format, allowing you to attend as your schedule permits. Instead of a presentation, various information stations will be set up with Duke Energy subject matter experts there to address your questions and provide information including visual displays of the project, an estimated timeline and other pertinent information about the project.

There will be opportunities at each of the open house events to ask questions and formally submit your comments and concerns. All public input becomes part of the official data collection record that we carefully consider during the siting evaluation process and before selecting a preferred route. There will be additional opportunities to formally submit comments and concerns to be considered as part of the siting process for an additional 30 days through Friday, Dec. 16, 2016.

### **Next Steps**

We anticipate announcing the preferred route in early to mid-spring of 2017 following the careful review of all public input and extensive expert analysis. After determining the preferred route, Duke Energy will begin surveying the preferred route to establish its precise location and identify the easements required on individual property parcels. Company representatives will work with landowners along the selected route to survey the land and discuss the easement process.

By late spring/early summer 2017, we plan to file the formal Certificate of Public Convenience and Necessity (CPCN) application for the 230-kV transmission line to request approval by the North Carolina Utilities Commission as required by North Carolina law. Construction is expected to begin by summer of 2018 and be completed before the end of 2019.

**No construction will begin until the North Carolina Utilities Commission grants final approval.**

If you are unable to attend either of the open house events or if you have additional questions about the project, please contact us at the toll-free number or email address provided below. Information made available at the open houses will also be found at a project-specific website beginning Nov. 16, 2016.

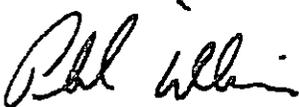
**Website:** [www.duke-energy.com/cleveland-matthews](http://www.duke-energy.com/cleveland-matthews) (available beginning Nov. 16, 2016)

**Email:** [CarolinasEast@duke-energy.com](mailto:CarolinasEast@duke-energy.com)

**Call:** 866.297.5886

We are committed to communicating with you throughout this process. We appreciate your patience and cooperation as we work through this important project to meet the growing demand for power in your community.

Sincerely,



**Phil Williams**  
Project Manager

Enclosures (1)

Property Identification Numbers (PIN): 165200-63-3989

[lassiters1.jpg](#)

Page Four — THE FOUR OAKS NEWS — Wednesday, March 9, 1983

# Looking for the first La

## LOOKING FOR THE FIRST LASSITERS

According to the census records and land records I have found Elijah Lassiter (1762-1848), a native of Northampton County, North Carolina and a soldier of the Revolution, was the founder of the Lassiter family which for almost 200 years has held land on the north side of Black Creek in what is now Elevation Township. Actually the Lassiter land also lies on the south side of Black Creek (the Shade Lassiter estate, the David Lassiter estate, the Charlie Lassiter estate, the Paul Lassiter estate and Tom Lassiter's land) and on the north side of Middle Creek (the Willis A. Lassiter farm).

By 1827 Elijah had purchased more than 1,500 acres reaching from Sassarixa Swamp where it joined the Olive land, up Black Creek and north across the Lassiter road and the Hunter Road to Middle Creek. I am aware that this is a general description, I have not made an effort to add land bought by Elijah's descendants. Today several other families hold land in the area. Several of them are a result of marriages into the Lassiter family.

## FOOT AND FIELD RESEARCH

The figures and facts which can be mined from the courthouse and the library tell the story very well but there is another satisfaction in seeing how the land lies and talking with people who have lived on the family land all their lives and carry some history of their families in the memories of their childhood.

Sunday afternoon, Feb. 27, I went to visit Paul Langdon (son of James and Rebecca Lassiter Langdon) who has lived on the King Mill Pond Road for all but the first five years of his life. The question I put to him was, "Where are the oldest Lassiter cemeteries you know about?" The problem on which we actually worked was finding the homesites of the first Lassiters to settle the land. We knew that their houses were gone, looking for their graves seemed the most reasonable procedure. Generally a homesteader would pick a house site on a well drained hill near a spring and when death first struck his family he would start a cemetery on the highest hill near the house.

## Four Oaks History

By

James Bryan Creech

Paul knew where the home of his grandfather William Henry Lassiter (1845-1918) stood, it was on the site of some tobacco barns near the rear of the James Langdon house, Paul's home until he built a new house nearby a few years ago. William Henry kilned brick from clay on the site to build the four chimneys and pillars of his house.

William Henry's father, Alfred Lassiter, (1823-1905) built his home by a natural spring at the rear of these houses. It was a little nearer Black Creek. Both of these houses are gone, the William Henry Lassiter house burned in the late 1920s. But there were two generations before William Henry and Alfred, William (1791-1874?) and the founder of the Johnston County tribes, Elijah (1762-1848). Where did they live and where are they buried? Nearby, I think. Somewhere among the Lassiters there may be someone who knows where Elijah settled in the late 1780s. If so, I will be glad to hear from you.

## THE OLDEST LASSITER CEMETERY?

The oldest cemetery Paul remembers is on land owned by Terry Parker, land previously owned by Garnet Lassiter, Albert Lassiter (his father), John William Lassiter (his grandfather) and so on back by way of

[lassiters2.jpg](#)

# ssiters near Elevation

Alfred to William to Elijah. The site of the cemetery is on a hill east of Paul Langdon's pond, less than a quarter mile from the rear of Russell Lassiter's home. The path which passes by the site continues to the "ball diamond field" and Black Creek. No trace of the old cemetery can now be found but Paul can remember the wooden markers and the plank houses which marked the graves in the 1920s. The plank houses which were built over the graves stood about four feet high, had gabled roofs and plank walls and were the size of the graves.

Not far south of this site was the Ligah Lassiter bridge which may have been the first bridge across Black Creek in this area. I assume that the bridge was named for Elijah Lassiter and if he built it, it was built before 1848. It seems likely to me that Elijah built his house in this vicinity probably near the cemetery. William, his oldest son, also must have lived nearby because it is known that Alfred and Joseph, sons of William, had their homes nearby. Alfred's home was on the Paul Langdon farm and Joseph's home was in the yard of the present home of Mr. and Mrs. Grover Langdon. Joseph Lassiter, (1837-1904), Russell Lassiter's grandfather, and Joseph's wife, Martha Woodall Lassiter are buried in a small graveyard just southwest of the Russell Lassiter home on Lassiter Road. Graves in the Joseph Lassiter cemetery also had small plank houses built over them. These structures were removed in 1936 when James Lassiter, Joseph's son and Russell's father, died and was buried with his parents. About 1925, James built the large and fine house on Lassiter Road in which Russell now lives. James' first house was on the south side of Sassarixa near the home of the late

Jesse Lassiter (son of James).

## WHERE DID THE FIRST LASSITERS LIVE?

I theorize that Elijah Lassiter built his house, raised his family, died and was buried on the land bounded by Black Creek, Black Creek Road, the northside of Sassarixa, King Pond Road and the branch which runs from the pond to Black Creek. I think William may have lived in the same area because three (perhaps more) of his children received land in the area or immediately adjoining. If there exists evidence which would more accurately locate the homes of Elijah and William Lassiter, his son, I will be glad to publish it here. Grover Langdon remembers that there was an old graveyard in a field near the Joseph Lassiter house which was in his (Grover's) yard, so there may have been Lassiters living north of Sassarixa before Joseph built there before the 1861-1865 War. Mrs. Grover Langdon is the former Iva Lassiter, granddaughter of Joseph Lassiter and daughter of James Lassiter.

## WHO HAS THE PICTURES?

Publishing individual photographs in this column can be a problem because of space but I would like to borrow some group pictures of the first Lassiter families to be photographed. Are there family pictures of the John William Lassiters, the Joseph A. Lassiters, the Henry Lassiters, the Robert I. Lassiters, the Shadrack Lassiters (He was Robert I. Lassiter's father) and others including the Lassiter daughters and the families they produced? Pictures may be left at the Four Oaks News office. Please bring complete written identification of the individuals in the pictures, relevant dates and information as to marriages, careers and places of residence.

tribes1.jpg

# The 12 Johnston County

## The Twelve Tribes of Elijah Lassiter

Considering the fact that he was born 221 years ago and taking into account the fact that he was illiterate, a surprising amount of information is known about Elijah Lassiter, the founder of the Lassiter family in Johnston County. His father was James Lassiter Sr. of Northampton County. I have not found the name of his mother, Elijah, who was born in 1762, served two, three months hitches in the Revolutionary Army and in the course of that service twice marched from Smithfield to Cross Creek (Fayetteville). If he was marched along Green's Path or the Averasboro Road he walked over some of the land along Black Creek to which he returned to settle in the early 1790s. In the census of 1800 Elijah's family was the only Lassiter family in Johnston County.

Three facts I have not learned: the name of his wife (Perhaps he married before leaving Northampton County, I did not find a marriage record in the Court-house), and exact location of his homeplace and the site of his grave. My first guess as to his homesite: the north side of Black Creek less than one mile upstream from the bridge at Pete Lassiter's. My second guess: the southwest side of Sassarixa less than one and a half miles upstream from where it passes under Black Creek Road near the Olive hill. The site of an old bridge (the "Ligah" Lassiter bridge) upstream from the present bridge and the site of a now gone cemetery on Terry Parker's land makes me favor a site on Terry's land or on Paul Langdon's homeplace. If someone knows exactly where Elijah cleared his first land and built his house I will be happy to publish the evidence. I also would like to know where William Lassiter (1791-1874), Elijah's oldest son, built his house and raised his family. I suspect William lived in the same general area, I know some of his children did.

### Six Sons and Six Daughters

The most impressive crop which Elijah Lassiter produced on his 1,500 acres (chiefly pine woods) was 12

## Four Oaks History

By

James Bryan Creech

children who survived to marry and establish families of their own. Whether he accomplished this with one wife or two I do not know. And I do not know the history of all his children but several of them lived long enough to produce large families.

The oldest son, William, who was born in 1791 married Lucy (Luvey or Lewey) Stephenson in 1813. William was the executor of his father's will in 1848. William's own family consisted of six sons and four daughters. They must wait for another installment in order to provide space here for Elijah's 12.

I do not know the exact order of birth of the other five sons but I will name them anyway. Jason married Thena H. Cotton in 1825. Jason and his family moved out of Johnston County, I think. They were gone before the 1840 census. Another possibility is that Jason died.

tribes2.jpg

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# tribes of Elijah Lassiter

Shadrach Lassiter (born 1803) married Lucy Johnson in 1825. They remained on the Lassiter plantation and produced some of the Lassiter families who still live in the Lassiter community in Elevation Township. Shadrach does not appear in the 1870 census, he probably died before that time.

Elisha Lassiter married Obedience (Beedy) Carrell in 1827 and they too stayed on the Lassiter land. James Lassiter married Lucy Lockhart and they established the Lassiter line from which come The Smithfield Herald Lassiters, the Bentonville Lassiters and others.

Elijah Lassiter Jr. married Mary (Polly) Tomlinson of Johnston County in 1826 and they moved to Pike County, Ala. So did Elijah Jr.'s sister Sally. So there are in Alabama some Lassiters who have hundreds of relatives in Johnston County today. Other Johnston County families also moved to Alabama in the early 1800's

## And Six Daughters

Elijah's six daughters married men with Johnston County names and probably settled in southern Johnston County with the exception of Sarah (Sally) who married Solomon Whittenton and moved with her husband and her brother, Elijah Jr. and his wife to Pike County, Ala. Elijah Jr. and Sarah (still in Alabama) appointed D.R. Whittenton of Johnston County as their attorney to collect their part of Elijah Sr.'s estate at his

death in 1848.

Elijah Sr.'s daughter, Lucy, married James Johnson. Penelope married George Stephenson. Tabitha married George Johnson. Patsy married John Carrol and Mary married Gideon Woodall.

Thus were established 12 families from Elijah Lassiter, the first of his name to settle in Johnston County. I do not plan in this series of articles to trace all of these lines. Readers who recognize here some of their folks can track them into this century by visiting the courthouse and the Johnston County Room in the Smithfield library. I do intend to continue with some of these lines until I connect them with some living Lassiters. Those I choose are not to be considered more important than their kin. Knowing it is impossible to do a professional genealogy given my expertise, time and newspaper space, I choose to follow the lines of some of the Lassiters I know including some who came to live in Four Oaks.

If there be among our gentle readers some who are working on a Lassiter family tree, I gladly will show you the unorganized notes which I have collected. I repeat, I do not have any information which cannot be found in the courthouse or the county library. But I will be glad to talk to you, especially if you will bring me some Lassiter family pictures more than 50 years old.

Page Four — THE FOUR OAKS NEWS — Wednesday, March 9, 1983

# Looking for the first La

## LOOKING FOR THE FIRST LASSITERS

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[elijah1.jpg](#)

# Some of the first Lassiter

## THE FIRST JOHNSTON COUNTY LASSITERS

The first Lassiter which I can connect with the Lassiter families of Black Creek is Elijah Lassiter who was born in Northampton County in 1762. Since Northampton County is in northeastern North Carolina on the Virginia border it is probable that the family of James Lassiter Sr. (Elijah's father) came from the eastern shore of Virginia. It is a pattern of migration which was followed by several of the first families of Johnston County just after the Revolution.

In his will made in 1804 James Lassiter Sr. named as heirs his wife, Lydia, seven sons, Shadrack, Elias, Greene, Elijah, James, Kinchen and Jordan and four daughters, Penelope, Henretty, Abigail and Reiniford. It was Elijah who came to Johnston County after the Revolution, settled on Black Creek in what is now Elevation Township and started the Lassiter family which still inhabits much of that part of Johnston County.

### Elijah Tells His Story Firsthand

Although he was illiterate and signed his name with an 'X' Elijah left a firsthand account of his early life. On the 25th of February 1834 Elijah Lassiter made an appearance in the Court of Pleas and Quarter Sessions of Johnston County and swore to the following deposition. Elijah was about 71 at the time and he had forgotten some names and dates and he no longer had the papers to prove his military service but he told what he did for the new country in the Great Revolution and qualified for the benefits of the Pension Act of 1832. Elijah's deposition tells something about the life of a young soldier in the Revolution as well as some Lassiter family history so it is copied below in its entirety.

### State of North Carolina

### Johnston County

### Court of Pleas and Quarter Sessions

February 1834

For this day the 25th of February 1834 Elijah Lassi-

## Four Oaks History

By

James Bryan Creech

ter, a soldier of the Revolution, born 4th of August 1762, aged 71 years, makes his appearance in open Court — the Justice of said Court being present — and deposes to the following facts in order to entitle himself to the benefit of the Pension Act of 1832.

That he is at present a resident of the County of Johnston and has been for nearly fifty years having removed to this County from Northampton County N. Carolina where he was born — that he has a record of his age now in his possession — that in the year 1778 or 1779 when he was in his sixteenth or seventeenth year he was drafted in the Militia of this State — that his company rendezvoused at the Wingfield Courthouse as it is called on the 8th day of — 1778 or 1779 where he joined the Regiment under the command of General Caswell — that a Captain Peterson had the command of his company — that the term for which he was drafted was 3 months — and that he faithfully served through this term — that he with his company and the Regiment commanded by General Caswell, who went along in person, marched from the place of Rendezvous to Smithfield, Johnston County, where they remained for a week or ten days — they then marched across the Cape Fear River above what is now the Town of Fayette-

[elijah2.jpg](#)

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# 's from Johnston County

teville. Crossed the Yadkin at Shallow Ford and then took down the Pee Dee River in the State of South Carolina to — Ferry — near which place an engagement took place between the American and British forces — that here the American army was commanded by Gen. Gates — and defeated.

At the ferry this deponent was taken seriously ill and was confined by sickness to his bed for five weeks and five days — that after his recovery so as to be able to travel he returned home — That he returned home in September and he well recollects having received a paper from some of the officers but who he has forgotten for he is an illiterate man certifying his having served 3 months.

He was not however in the engagement where Gen. Gates was defeated because of his serious illness — but he heard the report of the guns when in his bed.

He remained at home for nearly two years. He was again drafted for another 3 months service — that his Company met at the Town of Halifax on the 1st day of July in either 1780 or 1781 — where Joel Sherwood took Command as Captain of the Company and Elijah Dougherty was Lieutenant — several other companies met at the same place and time — and they as well as his own Company were commanded by Major Hogg — that they marched from Halifax to Smithfield — then to Cross Creek which is now the Town of Fayetteville — then by Duplin Court House down to the Town of Wilmington — that they remained about a fortnight or three weeks at Cross Creek for the purpose of giving check to the forces who were there and had been previously committing great depredations upon the private property of the inhabitants — their object in marching to Wilmington was to meet the British army at that

place — but just before they reached that place the British took shipping — that they remained at Wilmington until they received intelligence of the surrender of Lord Cornwallis — when this deponent and his company with a few others marched back to Tarborough — where he with his Company received a discharge — that this last tour was for three months — his term of service for his country embracing altogether a period of 6 months.

He makes oaths that he has never received a pension for this service either from the State Government or the U. States Government.

Elijah Lassiter came into Court and after having the above Declaration for a pension read over to him in the presence of the Court makes oath that the facts therein set forth are true to the best of his knowledge and belief.

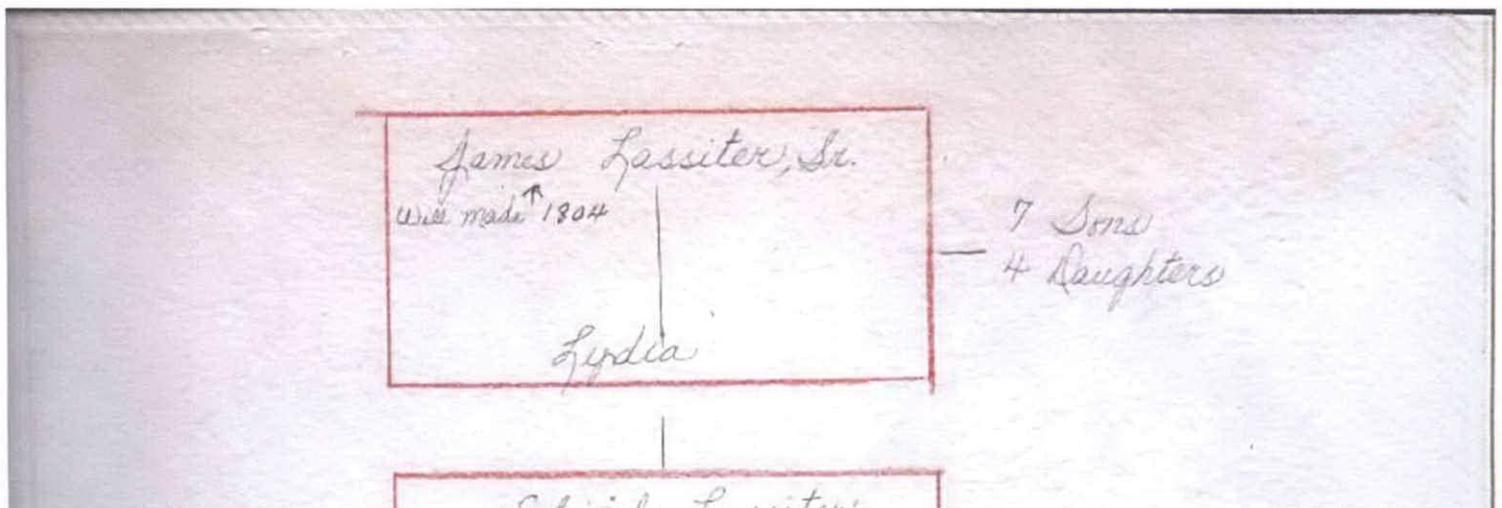
R.M. Sanders, Clerk

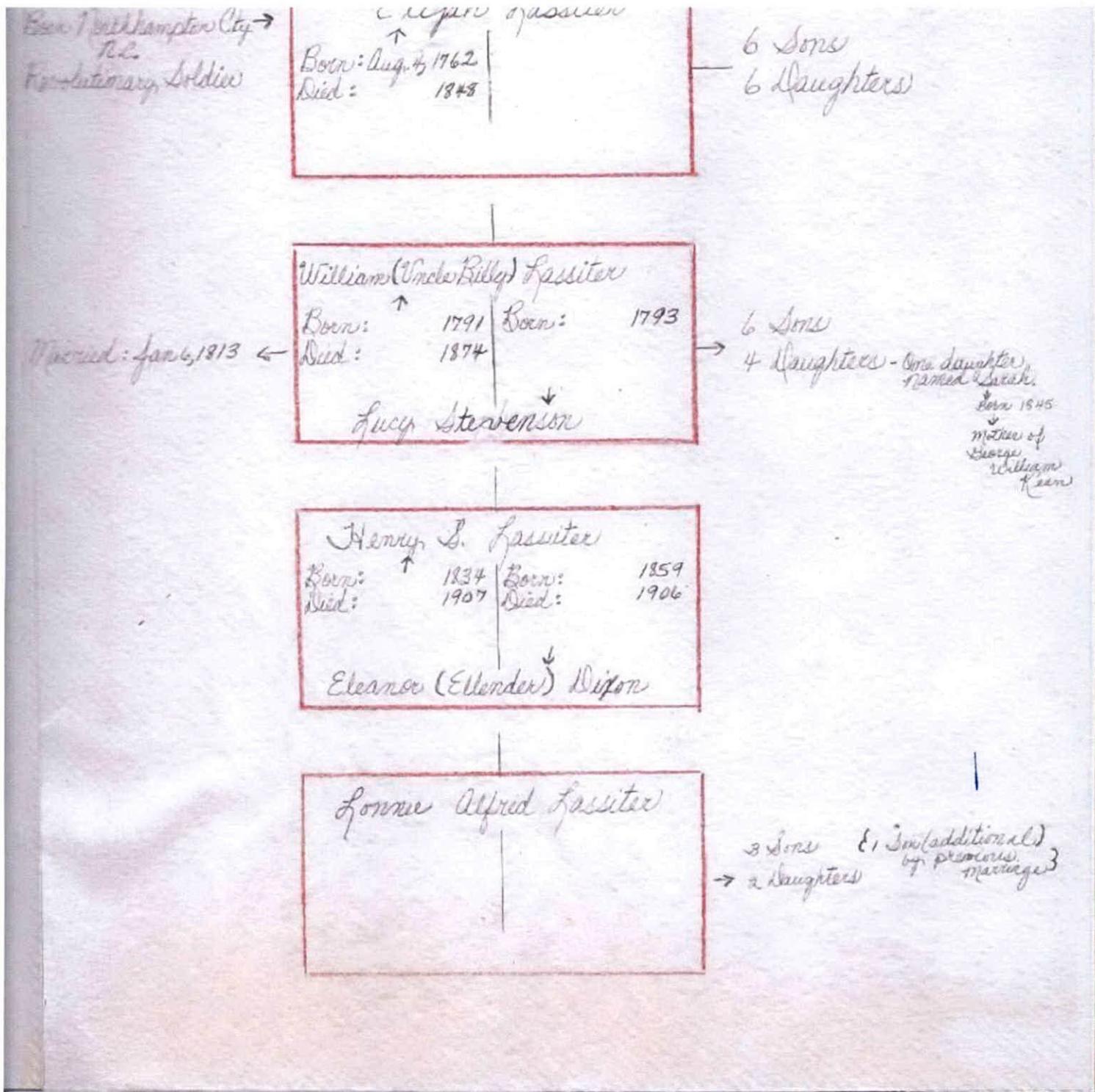
Elijah his mark X Lassiter

In 1800 the family of Elijah Lassiter was the only Lassiter family in the county. At least three other Lassiter men held land in the county before 1800. They were residents of other counties: George Lassiter from Duplin County and George Lassiter Jr. from Sampson County. Robert Lassiter who held a 1759 grant (his records are in the State Archives) must have resided outside the county also, he does not appear in the census.

Why did Elijah Lassiter move from Northampton County to Johnston County after the Revolution? My guess is that he gave the land along Black Creek a good looking over as he marched across it twice in two years and decided he liked the looks of it. By 1827 he owned more than 1,500 acres chiefly lying between Black and Middle Creeks.

familytree1.jpg





familytree2.jpg





Through out books red underlines denotes direct descendent of Linda Lee Hassiter Keen. Blue underlines denotes direct descendent of Russell Lewis Keen.

William (Uncle Billy) Hassiter & Lucy Stoverson had a daughter named Sarah (born 1945). She was the mother of George William Keen. George married Adelia, daughter of James O. Hassiter, Jr., Granddaughter of Virginia Elder James O. Hassiter, Great grand daughter of Elijah Hassiter.

Things that were done right in this project:

— Short route

— parallel a road

— 10 events provided landowners and community members the opportunity to give detailed input to the project team.

— Company presented two tower structure options

why: because it would have disrupted businesses, ruin the mainly rural appearance of the countryside and disrupt watersheds and wildlife habitat.

# Final route selected for controversial power line project through part of northern Harford



By Allan Vought  
The Aegis

OCTOBER 18, 2017, 12:55 PM

**T**ransource Energy has announced the routes that it will file with state regulators in Maryland and Pennsylvania for its Independence Energy Connection high voltage overhead electric transmission line project that has sparked community opposition in Harford County and neighboring York County, Pa.

The company had been looking for several routes to connect a new electric switching substation planned near the Susquehanna River in York County with the existing Conastone switching station near Norrisville in Harford County, a distance of about 16 miles. A similar connection is planned between switching facilities in south central Pennsylvania and Washington County, Md., near Smithsburg, about 29 miles.

The final proposed Harford County route unveiled by Transource Monday parallels Route 23 to the west for about 3 miles from the state line to Conastone Station.

Transource was contracted by PJM Interconnection, the regional power grid operator for the affected the area, to build the \$320 million project.

In a news release, Transource stated that PJM identified the need for the infrastructure upgrade “to alleviate congestion on the high-voltage electric grid and benefit customers in the region, including parts of Pennsylvania and Maryland.”

Steve Herling, vice president of planning for PJM, which operates the power distribution grid for 65 million people in 13 states and the District of Columbia, stated in a recent letter to The Aegis that the grid operator “performed extensive analysis of this highly congested area where limitations to move electricity efficiently have been a chronic problem.”

"This solution is the most reliable and cost effective and will save consumers millions in the long run," Herling wrote of the IEC.

But opponents on both sides of the Mason-Dixon Line that forms the border between northern Harford and southern York counties, have challenged both the need for the project and why Transource is not following existing power line routes.

"There have been no published studies to determine if the energy that is to be sent over the new transmission power line towers can be accommodated by the use of existing lines and towers," Norrisville resident Aimee C. O'Neill wrote in a letter published by The Aegis last month.

O'Neill, who could not be reached for comment Tuesday, is co-chair of a group formed this summer to oppose the project called Stop Transource Power Lines MD.

Harford County opponents who have spoken out publicly about the project, many of them landowners, say the proposed power lines will disrupt businesses and ruin the mainly rural appearance of the countryside and disrupt watersheds and wildlife habitat.

Opponents also say they believe some properties in agricultural preservation programs will be negatively affected by the new power lines.

Stop Transource Power Lines MD plan to hold an informational meeting Wednesday evening at Pond View Farm in White Hall to discuss the final power line route announcement and what steps to take next.

O'Neill and others in the organization have been critical of what they say has been a lack of interest among elected officials concerning the impact of the project, although the northern Harford area's County Council representative, Chad Shrodes, has worked closely with the opponents all summer. The area's state legislators also have attended one or more of the community meetings this summer.

The opposition group also has worked through the Jarrettsville/Norrisville Community Advisory Board to inform Harford County residents about the project and the reasons for its opposition.

Transource hosted two community information meetings about the project in Harford County over the summer, the last in Norrisville in August, which was attended by nearly 200 people.

The company stated in its news release that it presented more than 250 miles of route options in the east and west segments of the IEC project for review.

**"The 10 events provided landowners and community members the opportunity to give detailed input to the project team," the news release states. "All submitted input was incorporated into determining the final proposed routes."**

**"Transource worked to balance the public input with a variety of factors such as existing land use, sensitive species and habitats, soils and topography, historic and cultural resources and the opportunity to parallel existing infrastructure," the release continues.**

**The company also stated that in addition to routing options, it presented two tower structure options — lattice or monopole. The majority of comments received supported the monopole option, according to the company, and that is what it will use, "except in areas where engineering or construction needs dictate another structure type."**

**"By including community members in the siting process, rather than engaging them after decisions were made, we were able to consider and accommodate many landowner requests," said Todd Burns, Transource director, in a statement.**

**"The input gathered over the last few months was a critical component of our decision-making process," Burns continued. "We are confident that the route selection strikes the balance between building the required infrastructure that powers our homes and economy, while respecting land use and the environment in these communities. We look forward to continuing to work with these communities as an engaged partner as we move forward with the regulatory approval phase of the project."**

**Transource said it is directly notifying involved landowners, as well as people who have been part of the community input process.**

**The project and the final routes for the power lines must still be approved by the Maryland Public Service Commission and the Pennsylvania Public Utilities Commission. Transource said it plans to file applications with both by the end of the year.**

**Construction of the IEC is expected to begin in 2019, with a project in-service date of mid-2020, the company said.**

**Additional information can be found on the project website at [www.TransourceEnergy.com/Projects/Independence](http://www.TransourceEnergy.com/Projects/Independence).**

My name is Randy Johnson. My address is 935 Parkertown Road Four Oaks. I am 41 years old, I live there with my wife Casey, daughters Carly Rae 7, and Cara Ryland 3. I am opposed to Duke Progress running their transmission line across my property. Their communication to the public, the bias in their matrix, and fraud in their docket should be enough for the Utilities Commission to make Duke Energy find an alternative route for this line. Preferably in the area that caused the need for it, and that will benefit from it in the future.

## COMMUNICATION

Duke Energy mailed out letters about open house. They used responses and information from the open houses to determine the southern route was selected as the best route.

Page 18. Direct testimony of Timothy J. Same. "minimal input from concerned landowners as opposed to much greater input along the other lowest scoring routes indicating less chance of construction or access issues and a more positive public perception of the project".

Page 4-24 docket e2, sub 1150 states

"The disparity between comments received for the western routes versus the southern routes illustrates the general level of interest and/or concern from landowners and the public along these routes.

Page 10 direct testimony of Timothy J. Same

"an informational letter and small scale

map describing the project and advertising the workshops was mailed to all property owners within 500 feet of alternative routes"

If you did not receive a letter about workshops, how was one to make a comment. You can see that there are several letters sent to Utilities Commission from property owners that did not receive letter. In addition, the map sent with the letter has a map that does not have any road names on them, other than highways (40,70,95,301). A person could not look at this map and tell if their property was affected or not.

In appendix c page 6 of 51, depicts a map with segments (17-24). You can see that every state maintained road is listed and is a more accurate map. A person could actually tell where their property is and respond. To further prove the map the people received along the southern route is vague, Duke Energy admits to it.

Page 6, of Duke Energy response to question 6.

6. The letter dated July 31, 2017, to Christopher Ayers from Randy Johnson (submitted into the Commission's docket system on August 15, 2017) includes an attachment purporting to be a map of part of DEP's selected route. Is the map accurate? If this map is accurate, the route appears to cross some 12-15 parcels rather than following property lines. Please discuss the implications of moving

the route to the west or east to follow property lines or road(s), so as to reduce the number of parcels being bisected by the route.

**Response:**

Yes, the referenced map from the letter dated July 31, 2017, to Christopher Ayers from Randy Johnson is somewhat illegible, but it appears to be accurate.

This is the interactive map that was available online, that I scanned into an email and sent to them. If it somewhat illegible to an engineer, that how does a person without an engineering degree interpret and respond.

This leads to my next point in their communication. The duke energy Cleveland Matthews website was listed on this communication dated nov 4. You could go online and view the interactive map. Problem. There is little to no rural broadband serving the parkertown road area of four oaks. I personally have signed up with centurylink on 2 separate occasions. Once in 2008, and again in july of 2017. I cancelled it twice and have records of it. It would not download my first graders summer time curriculum, much less an interactive map. So, if you do not have internet you could not effectively see where this went across your property and respond accordingly. In addition and most importantly, this eliminated several landowners on our route to have a voice. Reason, many of the landowners on this route are in their 60's, 70's and 80's years of age. Many of these people have no interest in computers or the internet. Most people this age are not going to drive to an open house in the late afternoon when traffic is bad or after dark. The scoring matrix they used gave a lower score with property owners of more than 1 acre and 5 acres.

At least  
11 people can  
not do this

Page 4-10 docket e-2, sub1150

“To determine residential land use score, the acreage of parcels within ROW that were 5 acres or less were multiplied by 2 and parcels that were greater than 5 acres were multiplied by one”

The matrix that was used discriminated against older residents which typically own the larger parcels along their right of way, and in my opinion was intentional in hopes of not getting a response.

Matrix

Page 7 docket e2 sub 1150 testimony timothy j same

“the primary goals regarding routing were to:

\*minimize overall impacts by paralleling existing ROWS, including transmission lines, highways, and roads where possible

\*maximize the distance of the line from existing residences

\*minimize the overall length of route

Duke energy only accomplished 1 of the 3 goals set forth in docket. They did not use any existing ROWS, highways or roads and they chose the longest route in the matrix. One has to wonder how much effort was put into actually achieving these goals.

The matrix that Duke energy used to assign values is biased toward property owners that own larger parcels of land. Parcels of one acre to 5 acres with a residence received a value of 5. In addition, businesses were given a value of 5.

Page 12 direct testimony of timothy j same

Item 14 businesses within 500 feet was a line item

Page 8-2 docket # e-2 sub 1150

No business or public facilities within 500 feet of centerline

Title 26: Internal Revenue  
PART 1—INCOME TAXES (CONTINUED)

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### **§1.175-3 Definition of “the business of farming.”**

The method described in section 175 is available only to a taxpayer engaged in “the business of farming”. A taxpayer is engaged in the business of farming if he cultivates, operates, or manages a farm for gain or profit, either as owner or tenant. For the purpose of section 175, a taxpayer who receives a rental (either in cash or in kind) which is based upon farm production is engaged in the business of farming.

### **§1.175-4 Definition of “land used in farming.”**

(a) *Requirements.* For purposes of section 175, the term *land used in farming* means land which is used in the business of farming and which meets both of the following requirements:

(1) The land must be used for the production of crops, fruits, or other agricultural products, including fish, or for the sustenance of livestock. The term *livestock* includes cattle, hogs, horses, mules, donkeys, sheep, goats, captive fur-bearing animals, chickens, turkeys, pigeons, and other poultry. Land used for the sustenance of livestock includes land used for grazing such livestock.

(2) The land must be or have been so used either by the taxpayer or his tenant at some time before or at the same time as, the taxpayer makes the expenditures for soil or water conservation or for the prevention of the erosion of land. The taxpayer will be considered to have used the land in farming before making such expenditure if he or his tenant has employed the land in a farming use in the past

The above negates the duke energy docket e-2 sub1150. There are businesses along this route, and most importantly these businesses provide food and fiber that sustain our population and is just as important as any other factor quantified in this matrix. According to NCDA, a 74 billion dollar business in 2016!

In addition, Ag Carolina financial, the farm service agency, nash equipment company, east coast equipment, crop production service and well as many other vendors I utilize for my farming business would contend that there are businesses along this route as well. Reason, their businesses depends on farming.

## NEED

Initial communication dated nov 4 2016

Duke energy identified the need for a transmission to distribution substation in the Cleveland area of Johnston county to meet projected growth in the area.

Fact :I DO NOT LIVE IN THE CLEVELAND AREA OF JOHNSTON COUNTY

It is a fact that this area of the county is growing, subdivisions are being constructed a high rate. My contention is that now is the time for developers and duke energy get together and plan for this right of way to supply these developments the necessary power. The developer already has to leave out "green space" which is also quantified in matrix with value of 5) Duke energy is a for profit company, they will be the ones making money off of the substation and power lines the second they hook up new customers. It baffles me that they want to construct a line almost 12 miles across peoples property that will not benefit at all from this line. This area created the need. They need to deal with the transmission line.

Reason 1. A house in a subdivision will gain little to no value once it is built, only market fluctuations will cause it to go up or down in value. The house just gets older, depreciates, and only upgrades to the existing structure could make it go up or down. In most cases, these people are limited to adding structures or improvements or most likely members of an hoa, that determines what they can and cannot do.

My 40+acres has unlimited potential. I do not want it depreciated or restricted to serve the needs of others because of poor planning between the county, developers and duke energy. I did not create this problem. This may sound selfish to some, but I bought this property and have maintained it and built it to what it is today.

Note: Duke energy brochure at Johnston county cooperative extension service building

Restricts what I can plant or do with my property. Also provides me information that states all the safety steps when working around power lines and poles. As of today, I do not have any restrictions or have to worry about this. This restricts profitability of my farm and creates additional safety measures I have to adhere to.

NOTE

Direct testimony of timothy j same page 19 docket e-2, sub 1150

“the preferred route was one of the least overall impacting routes(fifth lowest scoring) in the numerical evaluation performed for the proposed project.

Translation...there were other routes that scored better, but this route will be the easiest to construct and maintain because somebody has already cleared it and already maintain it and their was little response from affected landowners.

Contention

I do not participate in the profit of duke energy. If duke sees a need in this part of the county, and they are going to realize a profit from it, then they need to invest in the engineering, construction and maintaining of the transmission line to the “area of need”. My property is not a willing participant in the profitability of duke energy. I understand it may take a little effort, but if the profit is great enough, then they should proceed with the steps it takes to use the lower scoring routes that keeps the transmission line where it is needed. If the profit is not there in the area it is needed, then do not do it all until duke can figure out how to put the line in the area it is needed. One of 2 things will happen, developers and existing landowners in that area will come together with a plan for the right of way that will serve continued growth, or the developers will stop building until this plan is put into place. THE people in the southern part of the county should not have their properties ruined, lives affected, homes affected for growth in another part of the county that they have nothing to do with.

In closing,

Many people did not receive letters about open houses and that has been documented.

OLDER residents had little to no way to voice their opinions about this project which weighed heavily in duke consideration of proposed route

The matrix duke utilized favored the southern routes intentionally, stating no businesses, and given higher values for parcels less than 5 acres and the highest value for green space.

There were other routes that scored better. Fact. In direct testimony. Timothy j same

The lowest scoring routes were in the area of need. It needs to stay there. Again, I do not participate in their profit, so if it costs more to build in that area and you can make a profit, then build it in that area, if you cannot put in area of need profitably, then do not build it. The marketplace can decide.

My farm is in the Johnston County Voluntary Agriculture District

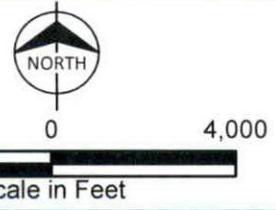
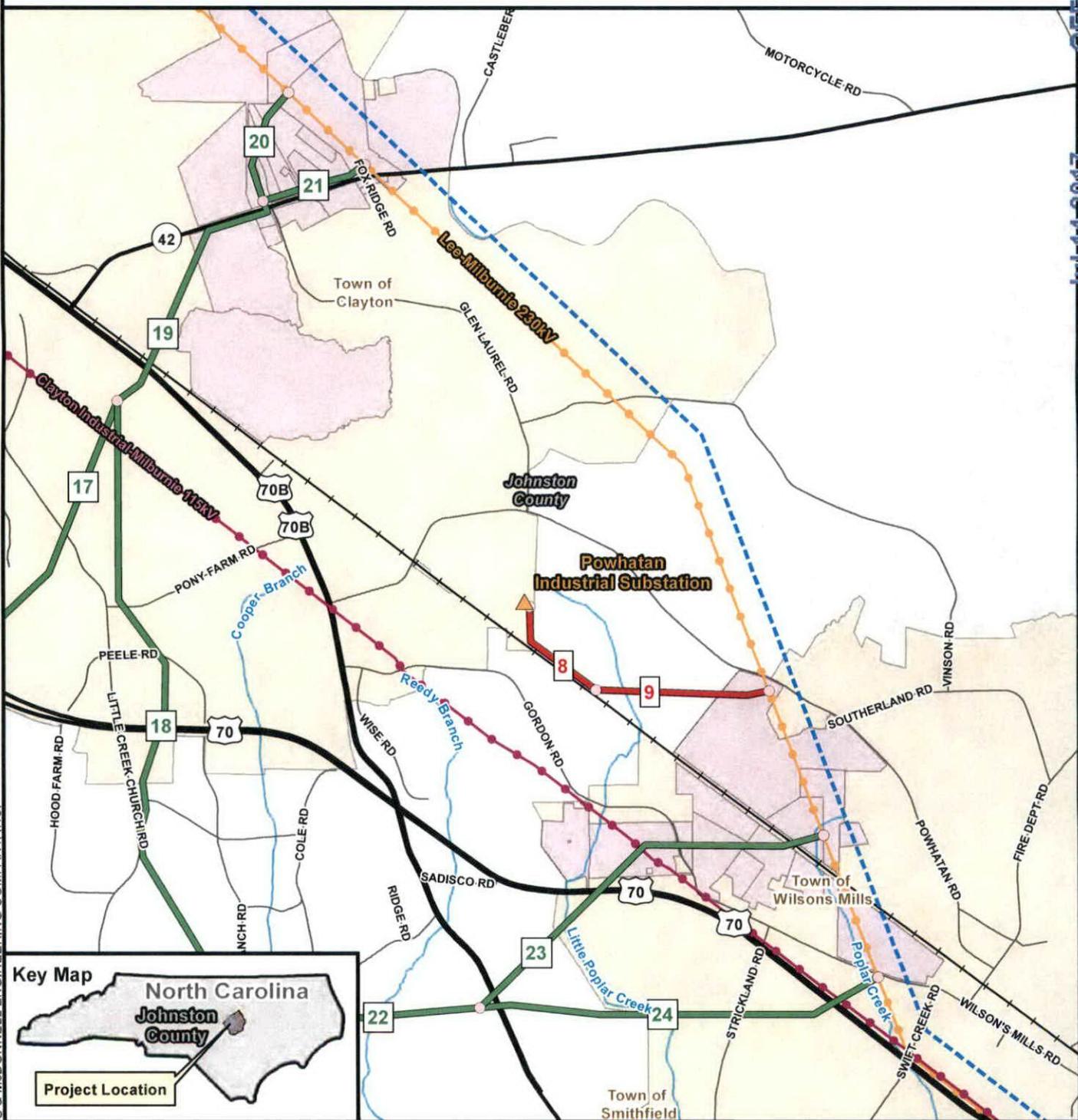
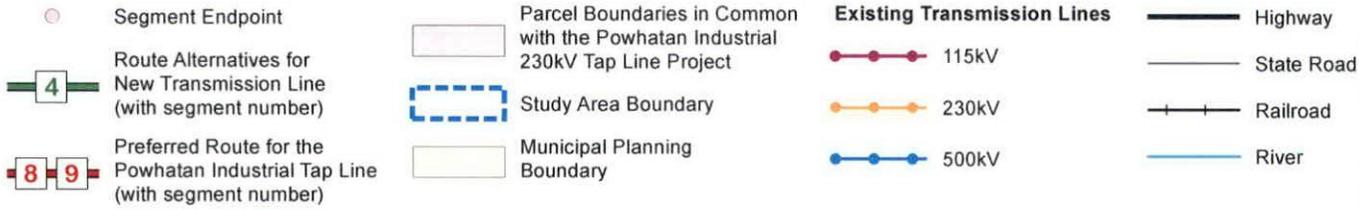
My youngest daughter, Cara is 3 years old. I mention this because her whole entire life has had her parents being consumed with the threat of eminent domain on our property. 2015-2016 CSX wanted our property for a intermodal hub. 2016-2017 duke energy want to cut her inheritance in half and create an unsafe environment for her to ride her 4 wheeler or work along side her sister in our fields and pastures.

She has been robbed of countless hours of spending time with her parents because they have been constantly studying and researching for hearings just like this one in hopes of saving our property. I cannot go back and give her those hours back. I missed them.

Based on all the facts mentioned above, I ask that the utilities commission to "NOT APPROVE" the construction of the Cleveland Matthews line on the proposed preferred route. The route needs to be in the area of need on the lowest scoring route in that area. Thank you.

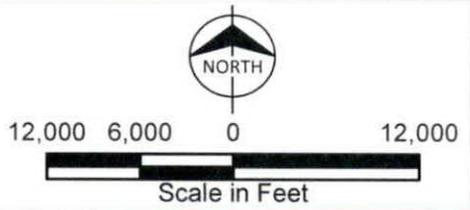
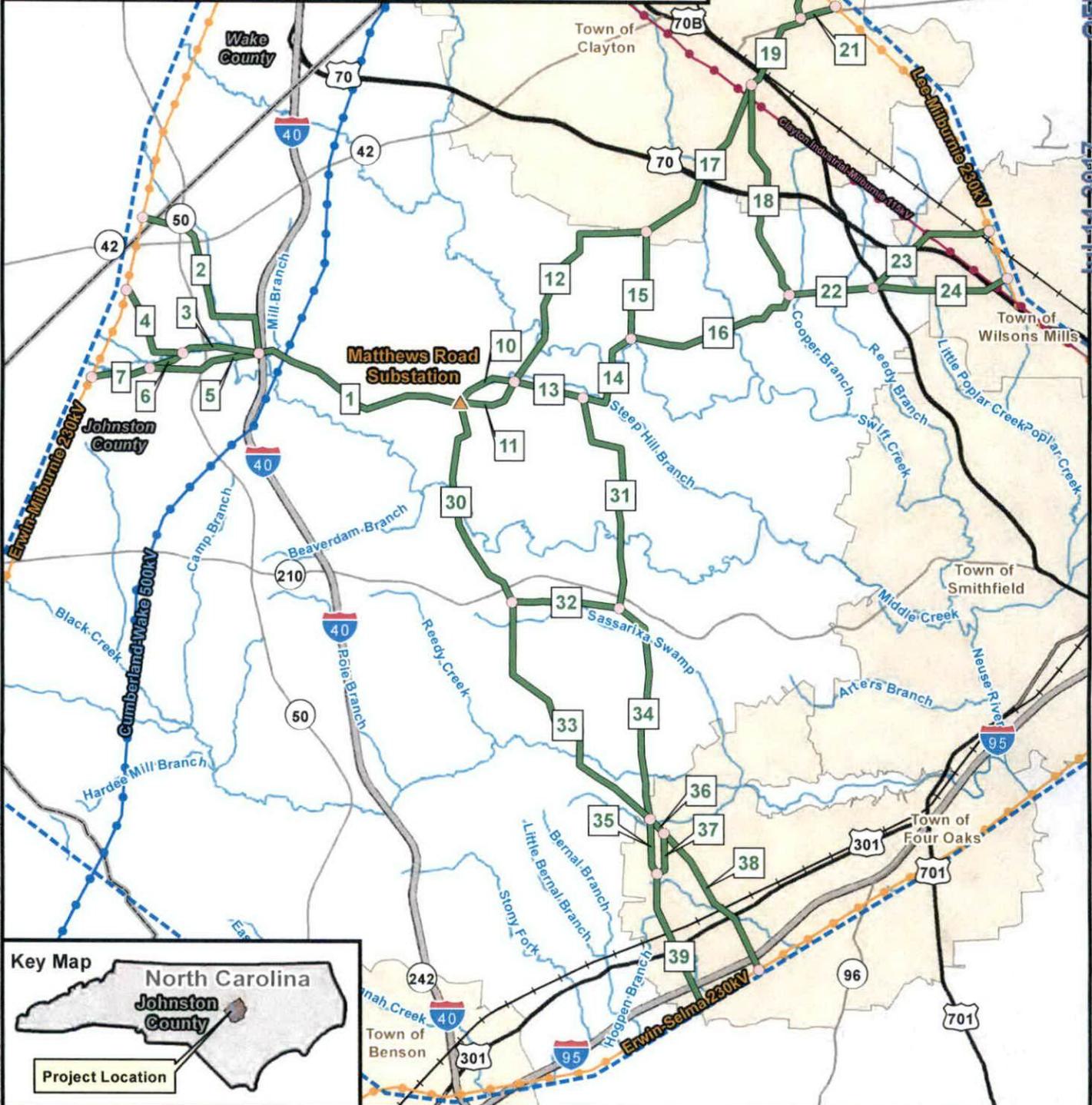
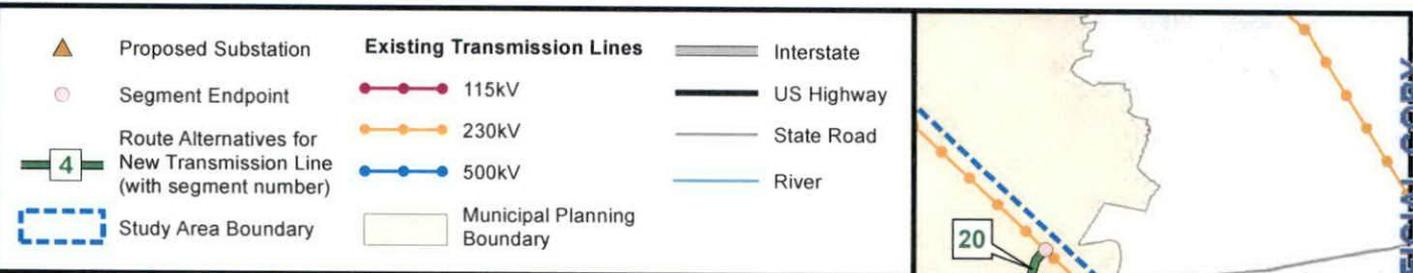


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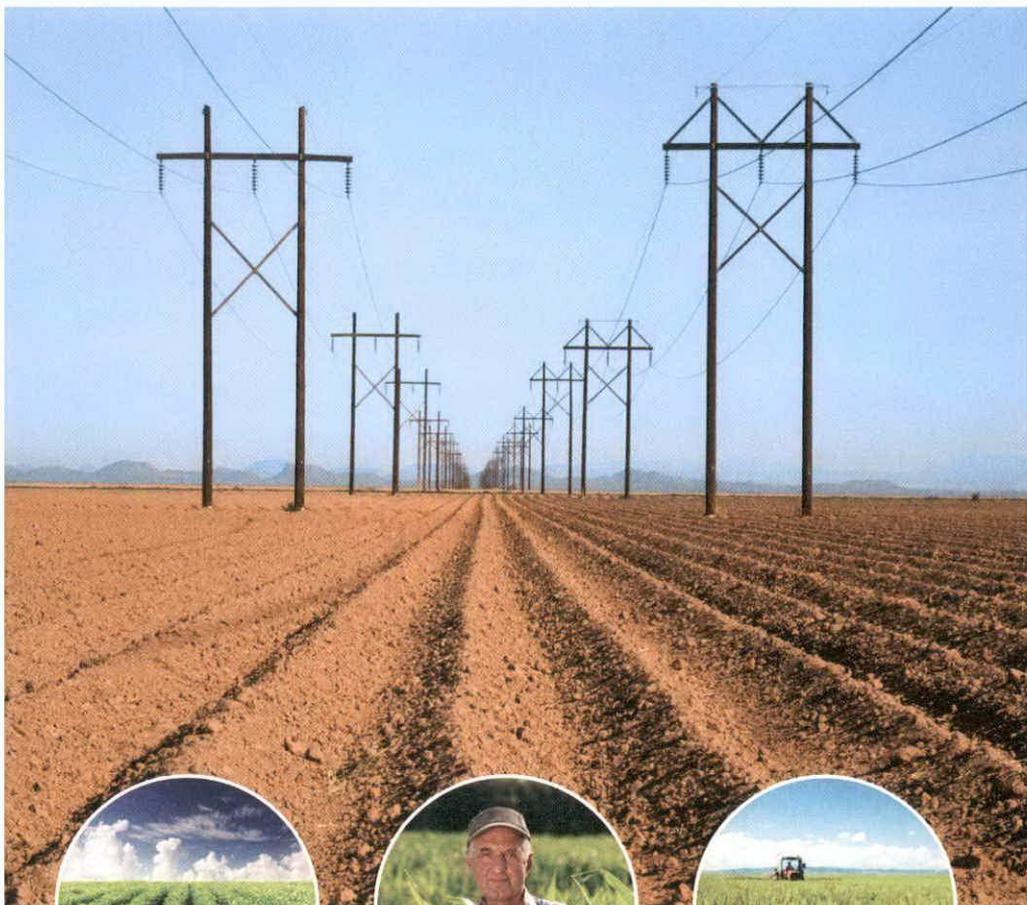
Duke Energy  
 Cleveland - Matthews Road  
 Project  
 New 230kV Transmission Line  
 In Common with the  
 Powhatan Industrial Project

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Duke Energy  
 Cleveland - Mathews Road  
 Project  
 New 230kV Transmission Line  
 Route Alternatives

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# A Guide for Farmers

STAYING SAFE AROUND POWER LINES





### Lakes and ponds

Lakes and ponds may not be installed without **detailed plan review and prior written approval**. Approval is subject to size limitations and other restrictions such as minimum distances to Duke Energy line support structures or guy anchors. Duke Energy Florida right-of-way restrictions do not allow for any retention ponds within transmission line rights of way.



### Trees, shrubs and other vegetation

In order to ensure safe and reliable transmission line operation, the planting of any tree or shrub is subject to area-specific restrictions. Duke Energy may exercise the right to cut “danger trees” outside the right-of-way limits as required to properly maintain and operate transmission lines. Vegetation that is not in compliance is subject to removal without notice.

To learn more, see your area's Duke Energy Electric Transmission Right-of-Way Guidelines/Restrictions document on our website.

## \*Guidelines/Restrictions links

**Carolinas:** [www.duke-energy.com/pdfs/ROW\\_Carolinas\\_Guidelines-Map.pdf](http://www.duke-energy.com/pdfs/ROW_Carolinas_Guidelines-Map.pdf)

**Florida:** [www.duke-energy.com/pdfs/ROW\\_Florida\\_Guidelines-Map.pdf](http://www.duke-energy.com/pdfs/ROW_Florida_Guidelines-Map.pdf)

**Midwest:** [www.duke-energy.com/pdfs/ROW\\_Midwest\\_Guidelines-Map.pdf](http://www.duke-energy.com/pdfs/ROW_Midwest_Guidelines-Map.pdf)

**? Questions?** Call your area's Customer Call Center.

Duke Energy Progress: **800.452.2777**

Duke Energy Carolinas: **800.777.9898**

Duke Energy Florida: **800.700.8744**

Duke Energy Indiana: **800.521.2232**

Duke Energy Kentucky or Ohio: **800.544.6900**

**At Duke Energy, we're committed to providing safe and reliable electricity to our customers.** In order to do so, we must be able to maintain safe, unobstructed transmission lines. Interference – even unintentional – from the public can result in electrical flashes that are dangerous to both people and property, and it can also cause power outages that affect surrounding industries.

## Safety

Because our transmission lines are not insulated, anything that provides a path from wire to wire or from wire to ground – such as smoke, spray or debris near lines – can cause an electrical flash, which can be extremely dangerous to people and property. If a flash occurs, our equipment shuts the line down immediately, much like the circuit breakers in your home.

Learn more in our Look Up and Live brochure at [duke-energy.com/pdfs/lookupandlive.pdf](http://duke-energy.com/pdfs/lookupandlive.pdf)



## Possible hazards and right-of-way guidelines

In the course of doing your job, you may encounter or perform work near transmission lines that could potentially shut off the power to the surrounding area. **For your safety and safety of others, be aware of hazards, some of which are outlined to the right.** Also refer to the Duke Energy Electric Transmission Right-of-Way Guidelines/Restrictions document for your area.\*

### Hazards

- Smoke from field burning
- Spray from hog waste
- Agricultural irrigation and spray from livestock confinement facilities
- Construction or farming equipment near transmission line facilities
- Airplane crop dusting

## Liability

When transmission lines are shut down due to interference, the outages can cause serious monetary loss to industries that rely on uninterrupted power for their processes. Their loss of production can result in legal action against you to recover their losses.



⊘ Activities like spraying near overhead power lines can cause interference. See the previous page for a list of other potential hazards.

## Right-of-way guidelines



### Structures, equipment or storage

It is important that transmission line right-of-way corridors remain **clear of all types of structures and obstructions**. Keeping the right of way clear provides unobstructed access by Duke Energy crews and our contractors. Safety always comes first.



### Grading or earth work

Changes of grade within the right of way are not allowed without prior written approval by a Duke Energy Asset Protection Specialist. Grade elevation changes may cause code violations. Before any grading will be allowed, detailed grading plans must be reviewed by a Duke Energy Asset Protection Specialist and approved in writing. In order to ensure the integrity of transmission line poles, towers and guy anchors, **no grading is allowed within 25 feet of transmission line facilities**.



### Fences

Fences that prevent access and that do not adhere to Duke Energy transmission right-of-way restrictions **are not allowed**. Example: Fence crossings require a 16-foot gate for access.



### Lakes and ponds

Lakes and ponds may not be installed without **detailed plan review and prior written approval**. Approval is subject to size limitations and other restrictions such as minimum distances to Duke Energy line support structures or guy anchors. Duke Energy Florida right-of-way restrictions do not allow for any retention ponds within transmission line rights of way.



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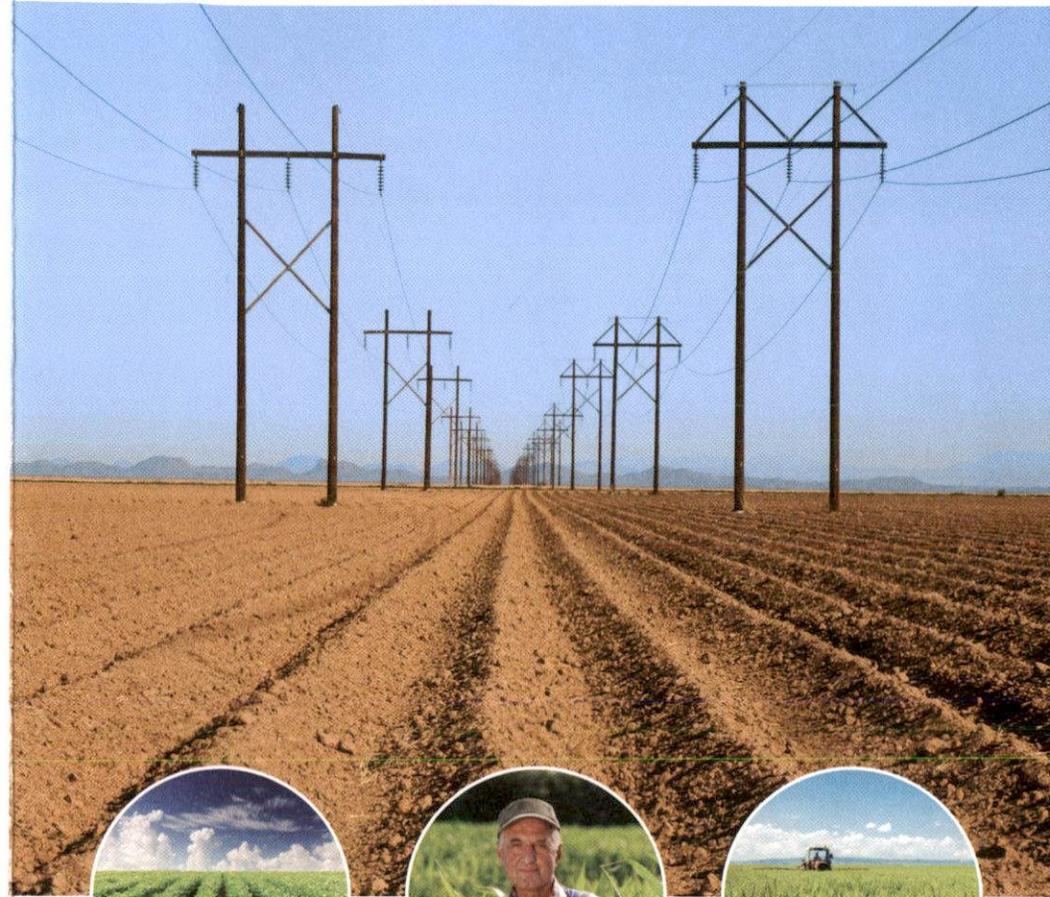
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# A Guide for Farmers

STAYING SAFE AROUND POWER LINES



An ordinance adopted under this Part or Part 3 of this Article shall provide for the establishment of an agricultural advisory board, organized and appointed as the county or city that adopted the ordinance shall deem appropriate. The county or city that adopted the ordinance may confer upon this advisory board authority to:

- (1) Review and make recommendations concerning the establishment and modification of agricultural districts;
- (2) Review and make recommendations concerning any ordinance or amendment adopted or proposed for adoption under this Part or Part 3 of this Article;
- (3) Hold public hearings on public projects likely to have an impact on agricultural operations, particularly if such projects involve condemnation of all or part of any qualifying farm;
- (4) Advise the governing board of the county or city that adopted the ordinance on projects, programs, or issues affecting the agricultural economy or way of life within the county;
- (5) Perform other related tasks or duties assigned by the governing board of the county or city that adopted the ordinance. (1985 (Reg. Sess., 1986), c. 1025, s. 1; 2005-390, ss. 3, 13.)

#### **§ 106-740. Public hearings on condemnation of farmland.**

An ordinance adopted under this Part or Part 3 of this Article may provide that no State or local public agency or governmental unit may formally initiate any action to condemn any interest in qualifying farmland within a voluntary agricultural district under this Part or an enhanced voluntary agricultural district under Part 3 of this Article until such agency has requested the local agricultural advisory board established under G.S. 106-739 to hold a public hearing on the proposed condemnation.

- (1) Following a public hearing held pursuant to this section, the board shall prepare and submit written findings and a recommendation to the decision-making body of the agency proposing acquisition.
- (2) The board designated to hold the hearing shall have 30 days after receiving a request under this section to hold the public hearing and submit its findings and recommendations to the agency.
- (3) The agency may not formally initiate a condemnation action while the proposed condemnation is properly before the advisory board within these time limitations. (1985 (Reg. Sess., 1986), c. 1025, s. 1; 2005-390, ss. 3, 14.)

#### **§ 106-741. Record notice of proximity to farmlands.**

- (a) Any county that has a computerized land records system may require that such records include some form of notice reasonably calculated to alert a person researching the title of a particular tract that such tract is located within one-half mile of a poultry, swine, or dairy qualifying farm or within 600 feet of any other qualifying farm or within one-half mile of a voluntary agricultural district.
- (b) In no event shall the county or any of its officers, employees, or agents be held liable in damages for any misfeasance, malfeasance, or nonfeasance occurring in good faith in connection with the duties or obligations imposed by any ordinance adopted under subsection (a).

# Agricultural REVIEW

Volume: 91 - No. 4

April 2016

Raleigh, N.C.

## Save the date: Got to Be NC Festival runs May 20-22

The Got to Be NC Festival returns to the State Fairgrounds in Raleigh May 20-22, highlighting agriculture and agribusiness, the state's No. 1 industry.

This family-friendly event proudly showcases the best of North Carolina at the Got to Be NC Food, Wine & Beer Homegrown Fare, presented by Lowes Foods. Also, there will be plenty of kids' activities, lumberjack shows, tractor displays, food, carnival rides and a barbecue cooking contest fundraiser.

If you love antique tractors and farm equipment, you won't have any problem finding them in all colors and sizes. Restored examples, along with rare tractors are on display each day, with many participating in the daily tractor parade at 1 p.m.

Festival admission is free, with pay-as-you-go rides and food, and \$3 admission for the food, wine and beer expo. Gates open Friday from noon to 10 p.m., Saturday, 9 a.m. to 10 p.m. and Sunday 9 a.m. to 8 p.m.

To find out more about the festival, go to <http://gottobencc-festival.com/>.

## Caution urged with spring debris burning

The N.C. Forest Service is urging residents across the state to think safety and exercise caution during the spring fire season, which typically lasts from March to May.

During the spring fire season, people do a lot of yard work that often includes burning leaves and yard debris. There are many factors to consider before doing any burning.

"If you're thinking about burning debris, contact your county forest ranger first," said Agriculture Commissioner Steve Troxler. "The forest ranger can offer technical advice and explain the best options to help maximize safety for people, property and the forest."

For people who choose to burn debris, the NCFCS urges them to adhere to the following tips to protect property and prevent wildfires:

- Consider alternatives to burning. Some yard debris, such as leaves and grass, may be more valuable if composted.
- Check with your county fire marshal's office for local laws on burning debris. Some communities allow burning only during specified hours; others forbid it entirely.
- Make sure you have an approved burning permit, which can be obtained at any NCFCS office, county-approved burning permit agent, or online at <http://ncforestservice.gov>.

- Check the weather. Don't burn if conditions are dry or windy.

- Only burn natural vegetation from your property. Burning household trash or any other man-made materials is illegal. Trash should be hauled away to a convenience center.

- Plan burning for the late afternoon when conditions are typically less windy and more humid.

- If you must burn, be prepared. Use a shovel or hoe to clear a perimeter around the area where you plan to burn.

- Keep fire tools ready. To control the fire, you will need a hose, bucket, a steel rake and a shovel for tossing dirt on the

fire.

- Never use flammable liquids such as kerosene, gasoline or diesel fuel to speed burning.

- Stay with your fire until it is completely out. In North Carolina, human carelessness leads to more wildfires than any other cause. In fact, debris burning is the No. 1 cause of wildfires in the state.

- These same tips hold true for campfires and barbecues, too. Douse burning charcoal briquettes or campfire thoroughly with water. When the coals are soaked, stir them and soak them again. Be sure they are out cold and carefully

(See Spring burning, pg. 2)

## PayNow link makes it easier to pay for agronomic services

Customers are pretty accustomed to loading up their online retail shopping carts, hitting the pay button and simply waiting for their merchandise to arrive in a few days. Online shopping is convenient, and it doesn't involve waiting in line or finding a parking space.

To help customers easily pay for services, the Agronomic Services Division recently rolled out a new PayNow feature on its PALS website, which lets customers pay by credit card and get instant access to their test results.

PALS stands for Public Access Laboratory-information-management System, a website where



Agronomic customers can now pay their testing fees online using a new PayNow feature on the Agronomic Services Division website.

farmers, homeowners and crop advisers can check on results of tests of soil, plant tissue, waste,

solution, soil-less media and nematode assays.

Before the PayNow feature

was added, customers would have to mail a check to the division to cover testing fees. Regular customers could create an escrow account for fees to be drawn against, but the process wasn't as quick and easy.

Since the feature's soft launch in mid-January, clients have found their way to the feature, without any real promotion of it.

"I've been pleased with the response so far," said Colleen Hudak-Wise, director of the Agronomic Services Division.

(See PayNow, pg. 8)

## From the tractor

by Agriculture Commissioner Steve Troxler



Commissioner Troxler

In late March, I was pleased to help N.C. A&T State University recognize Don and Alease Williams of Sampson County as the 2016 Small Farmers of the Year. This year marked the 30th anniversary of Small Farms Week, which highlights the important contributions of small farms

across this state.

The Williamses' farm produces free-range pasture pork from farrow to finish using heritage breeds Hampshire and Chester White.

According to the 2012 Census of Agriculture, we have around 50,000 farms in the state. Of that, 82 percent of them are 180 acres or less in size.

The economic impact of these businesses is anything but small, especially in our rural communities where farm dollars support ag supply stores, trucking operations and other community businesses and government.

North Carolina's agriculture and agribusiness industry is \$76 billion strong, leading our economy and creating jobs in areas

where big industry doesn't exist. This industry has been the foundation of our state and has helped build some of our biggest cities. I cannot begin to imagine what North Carolina would be without agriculture and agribusiness.

And we are working hard to support this industry and position it for future growth. I am confident that agriculture and agribusiness will be a \$100 billion industry moving forward.

Some people may look at agriculture as an industry of the past, but it is anything but that. In fact, with a growing global population that will need to eat, we are going to need each and every farm we can get to keep up with demand.

That represents a tremendous opportunity. We are fortunate in

North Carolina to have many farmers such as the Williamses, who understand what it takes to produce food. They are good at it and we can all be thankful for that. Their success means we have a locally grown safe, affordable and abundant supply of food choices.

As we head into the growing season, I encourage you to support your local farmers whenever possible. Look for local products in your grocery stores, at restaurants and at farmers markets and roadside stands. Your support helps boost our economy and keep agriculture strong in this state.

And thank you to those who toil to provide us with safe wholesome foods.

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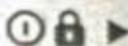
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Cleveland Electrical...  
forums.johnstoncounty.today



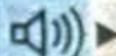
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23, 2017



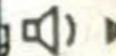
**robbie**

Well-Known Member



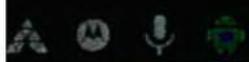
!!! Got MY Official Letter in the mail!!! The  
mission line is NOT going to be running  
nd my house!!! 😊

r picked the route we went to the meeting  
nd said was a no brainer!!! 😊



sday at 5:31 PM

t likes this.



4G LTE 9:26



Well-Known Member

which route did they pick?

Wednesday at 7:03 PM



Yuri  
New Member



The south route to Parkertown. Big surprise.

Wednesday at 7:19 PM

SIM Tray

cynadon likes this.



cynadon



DWK  
Well-Known Member



certdude said: ↑

*Thats a big freaken substation for a minor voltage change, and local distribution. They ought to take half that land and make a park out of it.*



ce it was discussed here before, the lady that owns the prospective park property that offered to the county, also owns the large tract that I be sold to developers soon. The public park developed, would just be there to be a draw the new homes being built, rather than making the ugly mega substation be a deficit to prospective home buyers. It's a "trade-off" deal, and she gets a nice tax benefit to boot. And the station infrastructure gets built to service more new subdivisions. That's just in my humble opinion, mind you.

Agreed, it's much larger than needed. Most substations of similar voltage changes are about 5-6 acres.

[View attachment 2961](#) [View attachment 2960](#) [View attachment 2962](#)



Thank you for those excellent comparative maps! When compared to the substation in Raleigh, the proposed Matthews substation is VERY LARGE - roughly 4 times the size of Raleigh's and far larger than any rural area would ever need. Does it seem to anybody else that this project went "under the radar", while the proposed park was very visibly publicized? And why? Personally, I think that the site location there on Matthews and Polenta for a large substation is not the best location for the Cleveland community, as it immediately industrializes a rural area. My husband, who works at a data center, believes that the large electrical capacity of the Matthews substation could reasonably support some type of very large future facilities - such as "data centers" or another "Grifols type" or "Talecris" facility, or something less desirable and



location there on Matthews and Polenta for a large substation is not the best location for the Cleveland community, as it immediately industrializes a rural area. My husband, who works at a data center, believes that the large electrical capacity of the Matthews substation could reasonably support some type of very large future facilities - such as "data centers" another "Grifols type" or "Talecris" facility, or maybe even something less desirable, and that Duke Energy is building out the infrastructure now to possibly attract future businesses - not necessarily a bad thing - but I believe that might be the real reason why they're building a substation of that size out here right now. I will need to research the topic, but it would be interesting to know the types of businesses that these large substations typically service. And I don't believe for one minute that the relatively sudden infrastructure building of that large substation after many years, has much to do with "remediating power outages" in Cleveland either, since there's no real profit in that for them. Again, just my humble opinion, but maybe worth tracking this.

route. How far out do we go. I  
counted 9 to 15  
As far as footage

Today 3:48 PM

Hi, Casey - we look at a 1,000-  
ft corridor. In other words, 500-  
ft each side of centerline of the  
proposed right of way.

Thanks

I just got back to my desk and



iMessage



Ok



Talk later



Thanks

I just got back to my desk and checked our data tables. We show 9 homes within 500ft of centerline along Segment 39 and 12 homes within 500ft along Segment 38.

I'm also asking our siting team to verify and confirm.

Ok thank you



Message



Ok



Talk later



So far she said

I spoke to the lead over the survey crew and he contacted the vendor tonight about the issues. We stopped all machine cutting and they will only be cutting by hand. We made it clear nothing 6" or larger is to be cut and not a single cigarette butt left behind.

I'll contact Tracy tomorrow to talk to her directly and make sure we set things right.

I think you may have some big problems. The adams are not happy

I don't imagine they are. I'll do what I can to make it right.

They are still out looking randy is heading over too



I called the project manager and surveying lead and left messages for them to call me back.

Ok

She has about 20 trees down so far she said

I spoke to the lead over the survey crew and he contacted the vendor tonight about the

Drew

owners would get notice so they could be on property if chose

That wasn't our land surveyors. I confirmed that this morning. Not sure what those stakes are. Wondering if they are underground utility markers.

I'll hopefully know more tomorrow.

Trees were cut down too

On Amy's land?

No south of me

Cross 301 and go past where cell tower is

Oh, ok. The crews doing the centerline survey cut trees/vegetation down for line of

It was north. Beside cell tower

I am glad y'all talked with her finally I know she had left several messages.

Our land surveyors started in the south and just crossed I-95 today. They definitely aren't working north of you yet. I'm not sure what's happening. I have calls out to see who else might be out in the field.

I really think we would be south

This was south

Ah, ok.

But why did they do Amy in middle

Buzz told randy property

Commissioner Gray  
Commissioner Beatty  
Commissioner Clodfelter

Dr. Casey Johnson Exhibit 1  
1A

My name is Dr. Casey L. Johnson. I am 36 years old and married to Randy Johnson. Together we own a ~~20~~ 40 acre farm on Parkertown Rd. in Four oaks. We have two daughters Carly Rae 6 and Cara 3. We purchased our land in 2007 and built our forever home in 2008. Early in our marriage we worked tirelessly to pay down on our home and property before we had children. my husband as a farmer and full-time employee at Vermeer midatlantic and myself as a full time pharmacist and working relief on my days off for other local pharmacies.

When we envisioned our future for ourselves, and now our girls, we never thought we would face two threats against our property in as much as 12 months. One when our local government offered our property along with 500 surrounding acres to CSX for an intermodal rail hub and now a multi billion dollar company Duke Energy for a 230 kv transmission line that would cut our property in half. When we received the informational hearing letters last november we immediately planned to attend and express our concerns and objection to the placement of the line.

When notified our route was picked we began the process of contacting Duke for more information on how our route was chosen and made contact with our county commissioners and house of reps.

on May 4 2017 Duke visited us and many of our neighbors at our request and they heard the same thing from us all, running parallel with Parkertown Rd. weaving in and out of our homes ~~makes no sense~~.

*doesn't seem logical when the properties run perpendicular to the road. No where else along the line*

We had many questions for Drew Gilmore, Marty Clayton, Timothy Same, and Phil williams that day.

*does this run parallel this long*

We expressed what our family and neighbors had been through in 2016 with the proposed CSX rail hub. I asked if that project would have moved forward would the line still have come through or would it have to have been moved. Timothy Same replied that they followed the developments closely and in fact it "rail and industry work well together".

*My take* that is that it would have potentially made it easier for duke to acquire the right of ways needed. I find these statements and

others made by properties owners on blog posts from communities up near cleveland matthews rd such as they were told this southern route was the "no brainer" by duke reps at the public hearings to imply this route was already being squeezed as the route chosen and make an unfair bias placed open our properties.

I also asked about why this southern route <sup>39</sup> vs. the other path <sup>38</sup> through four oaks was chosen and replied by drew gilmore that the other route had 1 more home. we asked if those homes counted where livable properties which we were told if they had a tax id that is all that mattered. One home on the other route is uninhabitable with trees growing through roof.

I asked them to confirm that number, because using the provided map on their website, my preliminary findings found route 39 to have more homes.

The text response if received, i provided to utilities <sup>that</sup> confirming duke found <sup>route 38</sup> 12 versus 9 homes on route 39. i recalculated each home using the measurement tool on their map and i found the exact opposite 9 vs 12. i have provided those measurements to the <sup>commission</sup> as well as your public staff.

\*\*\*\* may leave out\*\*\*\*

I would also like to mention that at every turn whether it be a meeting with our house of representatives or <sup>court commissioners</sup> your public staff, duke meet with them immediately before or after us. I find this concerning, to the point that one of our house of reps even was going to cancel meeting with us property owners siting his meeting with duke yielded he could do nothing for us.

with that being said i also agree with my husband that neither southern route should <sup>not</sup> even be in question. this line should stay in the area of need. we have or will not receive any benefit from the placement of this line.

it will ruin our daughters inheritance and also place limitations on my husbands farming operations which is income we use to save for our daughters college funds.

I am also concerned about numerous articles i have read about increased childhood cancers from EMF waves. I am in the medical field and know things can be manipulated to provide the response a certain entity needs for public perception. *Research studies can be structured and backed by mg \$ to fail to find health effects*

The amount of property owners against this project in our area should *Area of need* carry the same weight as the cleveland area. the point of less *in the dock* complaints to me is a mute point because we as landowners own more property than a subdivision property owner yielding less complaints only because we live on a farm not neighborhood.

I have also found articles discussing cows not breeding back because of emf waves our family also depends on our cow operations. reference article here *also issues w/ stray voltage*

I researched properties close to transmission lines and found a home in neighboring benson that has been on the market for more than 365 days. the realtor provided information that the chief complaint from potential buyers was that they did not want to purchase because line behind home. this home is a \$250000 property that has sat on the market for over 1 year. that being said this line will depreciate my family home and property gravely. show pictures of property

In Closing I ask you please keep the project in the area of need and not affect properties and families who have no benefit from the placement of the line.

I ask you make Duke Energy and county developers be more accountable for providing renewable energy sources. Our land and future for our girls will be ruined. The past two years my husband and myself have had to give up too much of our time on researching and trying to prepare ourselves for the worst case scenario. It has caused us many wasted hours that could have been spent with our girls. I would love to look my oldest in the eyes and tell her everything is gonna be ok because she has had many questions and had to be explained things about property rights she should not have to worry about.

When Duke says to me they can adjust the line on properties proposed but not go on properties they don't have permission. let me remind ~~you and~~ them that they never had my permission either. Nothing is set in stone until you make your decision i urge you to please make them find a better way. thank you for your time.

----- Forwarded message -----

From: "Shirley Macy" <[smacy6@gmail.com](mailto:smacy6@gmail.com)>

Date: Oct 25, 2017 9:10 AM

Subject: Re: 201 lake shore drive benson--2095686

To: "Randy Johnson" <[randy.johnson@vermeermidatlantic.com](mailto:randy.johnson@vermeermidatlantic.com)>

Cc:

Hi Randy,

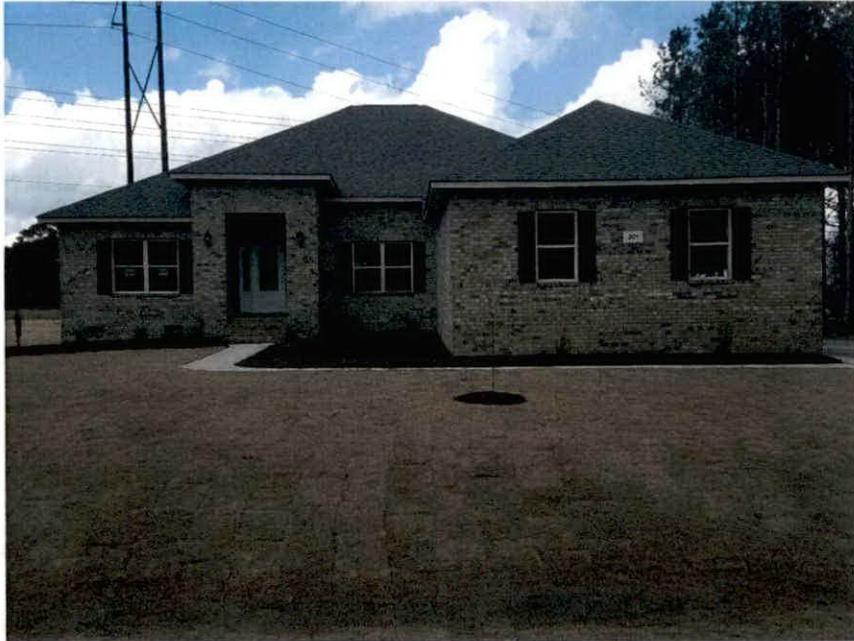
I am a licensed realtor in North Carolina since 1993 and I am a on site agent for Adams Homes. I have a few homes for sale in the community of Johnson Place in Benson. We have power lines very close to these homes. I have shown the homes many times and the same comment by the potential buyers and their agents is " I would not buy a home that close to power lines". Some of these homes have been on the market for a year. The power lines have definitely hurt the sale of these homes.

Thank you

***Shirley Macy, CSP***  
***Broker On Site***



**HOMES**



**207 Lakeshore Drive, Benson, NC 27504**

All Brick Exterior

3 Car Side Load Garage

.71 Acre Lot.

Gas Fire Place

Decorative Glass Front Door

Stainless Steel Appliances

Separate Garden Tub/Shower

Door from Master to Deck

Hardwood—Kitchen, Nook,

Hall

**\$273,200**

\$1000 Deposit

Builder Paid Closing Costs w/ Preferred Lender's

MLS# 2095686



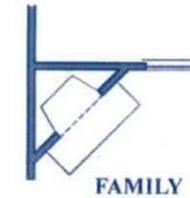
ELEVATION A

ELEVATION C

### 2508 PLAN

FEATURES:

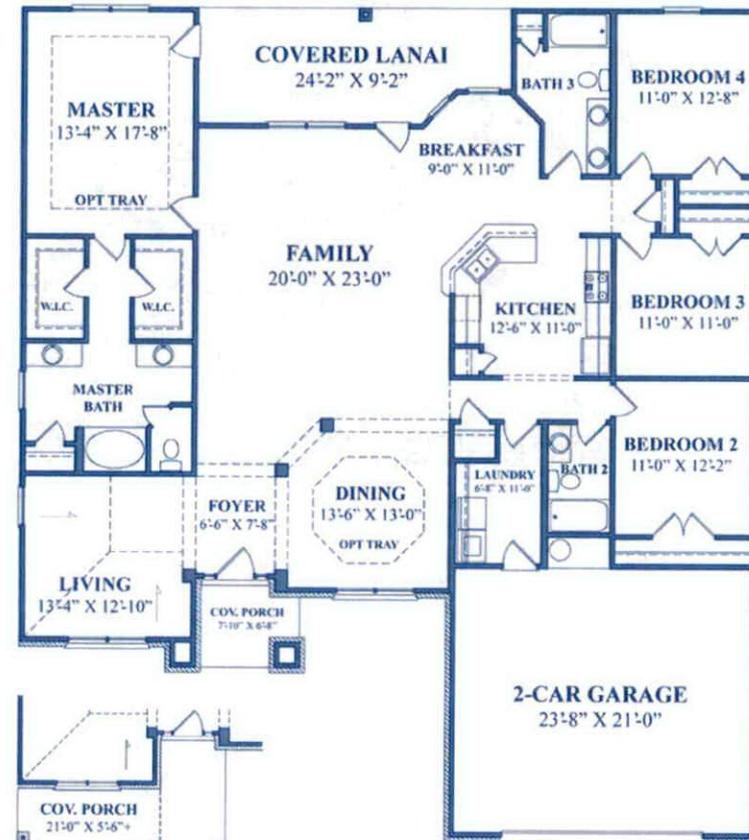
- 4 BEDROOMS / 3 BATHS
- 2-CAR GARAGE
- BREAKFAST NOOK
- FORMAL DINING
- FORMAL LIVING
- COVERED LANAI



OPTIONAL  
FIREPLACE



OPTIONAL  
MASTER BATH



ELEVATION C



Continuing a policy of constant research and improvement, Adams Homes reserves the right of price, plan, or specification change without notice or prior obligation. Optional features may be shown; dimensions may vary. Plans copyright 1998 Adams Homes.

07.01.16

Contact **Sean Walker** at **919-675-5301** for more information and visit our model home at 116 Colonade Court, Benson, NC 27504.



Casey and Randy Johnson

935 Parkertown Rd.

Four Oaks NC 27524



Casey and Randy Johnson  
935 Parkertown Rd.  
Four Oaks NC 27524



Casey and Randy Johnson  
935 Parkertown Rd.  
Four Oaks NC 27524

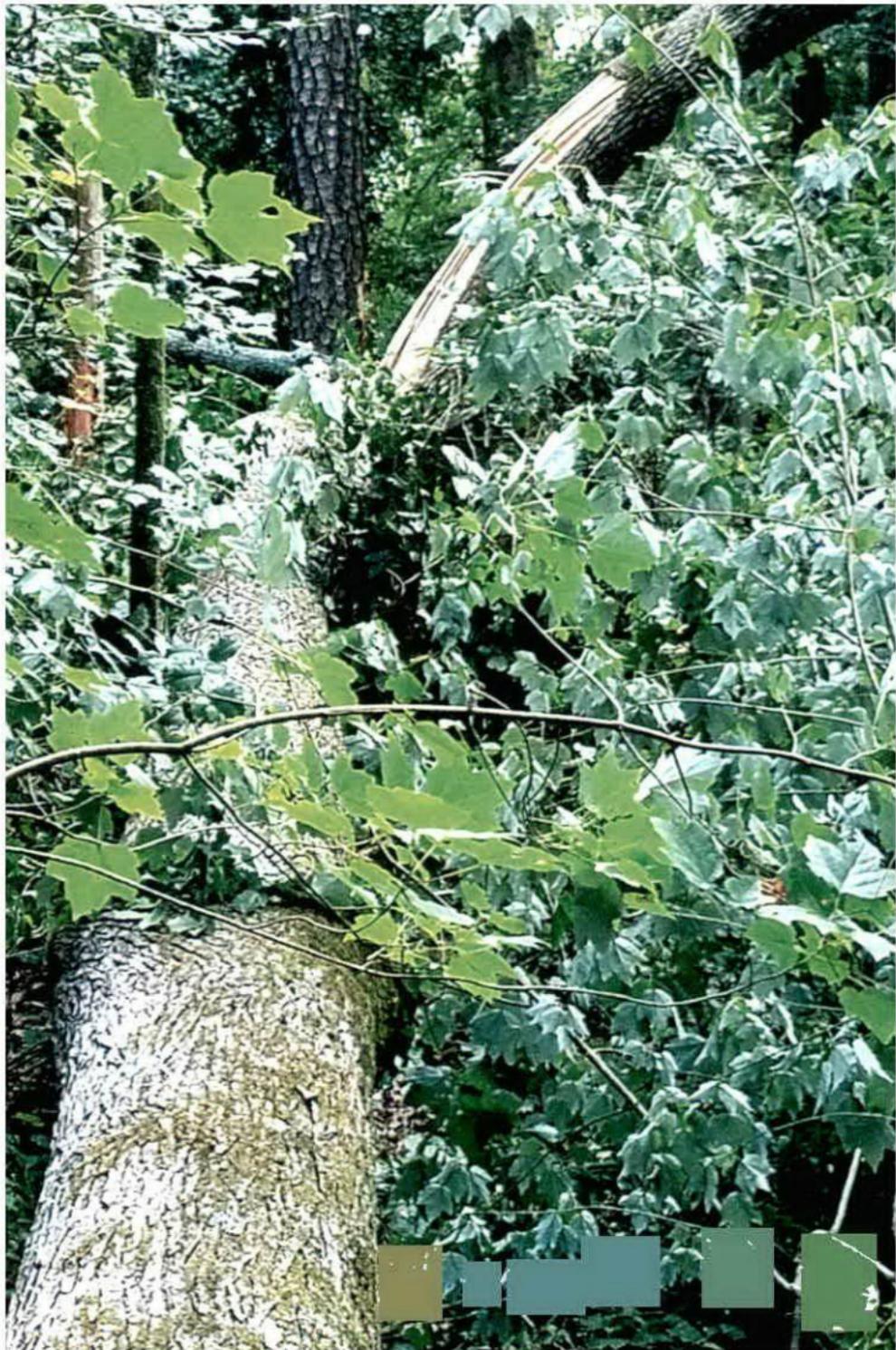


Casey and Randy Johnson

935 Parkertown Rd.  
Four Oaks NC 27524













10-30-2017  
Keen Exhibit 1 /A

Good evening. I am Linda Lassiter Keen. I grew up on our family farm on the south side of Black Creek northwest of Four Oaks in Johnston County. The proposed Cleveland Matthews Road 230-kV (kilovolt) transmission line is recommended to diagonally pass through the most valuable part of my farm. Thank you for this opportunity to speak regarding concerns I have about the selected route of the proposed line. I will speak regarding three main topics: the Lassiter family tree which tracks through nine generations that have called the north and south side of Black Creek in Johnston County home, the importance of green space to my father in particular and all of us in general, and the dramatic and long-term results that come from decisions made by those in authority.

The first permanent Lassiter of record in Johnston County was Elijah Lassiter. Born in 1762 in Northampton County, by 1800 he had served in the Revolutionary War and was included in the Johnston County Census of 1800. It is believed he made his home north of Black Creek near Lassiter Road. Through his descendants, I can track my grandfathers back to him. Although illiterate, by the time of his death in 1848, he had amassed over 1500 acres either near or bordering Black Creek. Many Lassiters, in what was known in my early years as "Lassiter Town", still call this area home. This is my ancestral home, this is the place nearest and dearest to my heart, this is the place entrusted to me to treasure and protect.

That being said, I am now a resident of Wake County and it is certainly fair to ask "Why?" In November of 1971, my only child at that time, a son, was diagnosed with acute lymphocytic leukemia. If chemotherapy and radiation treatments were successful, we could expect to have Phillip with us until about age 4. We were blessed and our son survived—but with extensive brain injury from treatment. He is now 48 years old and is totally disabled. He resides in a group home in Zebulon. Through a patchwork of government services, he receives housing and basic care. His dad and I supplement these funds. After we are no longer able to do this, the family farm is his security of last resort. If this line is approved as presented, it will go diagonally through the very best part of my farm. It is estimated the area around the 125 foot easement will decrease in value by 40 to 50%. The security that we felt we had in place for our son will no longer be there. The take away from this is that seemingly random decisions affect lives. We live in Wake County simply because more services are available for Phillip there.

And now a few words about green space. My father was a farmer who thrived being outside. He was a locally renowned fisherman who loved to drop his line in the Creek. He made his living working his land and, in his spare time, found solace, peace,

refreshment and recreation waiting for him there. After his death in 1998, I chose to honor his commitment to nature and love of green space. A big portion of what is now my farm has been in the CRP Program since 1987 or possibly before—at least 30 years. Acres of loblolly pine cover the hillside. If the suggested line route is approved, this area will be divided diagonally and the beauty and usability totally destroyed. I cannot let this happen without raising my voice in opposition.

I would like to close with a few questions that have not been adequately answered.

1. Why were proposed transmission routes rated, and ultimately the recommended route was not number 1 or 2 but the 3rd lowest rated line.
2. When a line already exists with an established right-of-way and that line is nearest to the area most impacted by population growth, why not piggyback the new line with the existing line?
3. Why choose a line significantly longer than is necessary to service an area that can be reached with a much shorter line. The Route 4 line would have required a 33,114 foot line. The Route 31 line—the route recommended—requires a 60,731 foot line—a line almost double the length of Route 4.
4. Why diagonally cross a farm, destroying the value and beauty, when other options clearly are out there.
5. Why adversely impact a homestead that has stood for some 200 years, decrease the security of a totally disabled adult, and willingly destroy green space that is being diminished in our country at an alarming rate. There has to be a better way.

In closing, I can attest to having no recollection of receiving the Duke Energy letter dated November 4, 2016. Since that letter is now deemed so important, it should have been sent certified to all land owners. A phone call or a visit would have added a personal touch. The lack of personal contact sends the impression that Duke Energy's time is more important than my fundamental right to own and enjoy personal property.

Thank you for this opportunity to share my thoughts on this matter.

Linda Keen

Mr. Christopher J. Ayers  
Executive Director Public Staff  
4326 Mail Service Center  
Raleigh, NC 27699-4300

In the matter of: State of North Carolina Utilities Commission Docket No. E-2, SUB 1150

I am a concerned property owner that the Duke Energy "preferred route", for the construction of the new 230kv transmission line for the new Cleveland Matthews Road Substation crosses. I strongly oppose the construction of the transmission line running across our property.

This property is my inheritance from my Daddy that I value very much. It was also his inheritance from his Mother. This is a part of him that I have left in his memory and my ancestors. To me it's not just a piece of land. This is where I grew up and we played and worked. I have wonderful memories here. My Grandparents worked hard for this land and they valued it a lot. They had to sell some of their land when I-95 came through years ago. I am writing because we are against this power line coming through our land and losing more of our land. The start with we were not notified until April 20, 2017 about any of it and then it was a certified letter. All the meetings had already taken place the prior year and we could not voice our opinions. We were left out of it all until the surveying crew came and cut down trees on our property without warning. That's when I voiced my opinion. I was upset that they just come onto our property without informing us and started making a path through it. If it wasn't for caring friends and neighbors we would still be in the dark. We have a lot of concerns. First, we can't get an answer for how they come up with this route and why it would be the best. We have more land that we farm than the other route because it is residential houses on the other route. We choose to live in the country where it is quiet. The only noise I hear is crickets and frogs at night. We raise animals and raise our own food on our land. Our land is profitable and if these lines come through that's going to make it less profitable and our land value will decrease. We wouldn't be able to sell if we wanted to which we don't.

It has me worried about the amount of voltage that is going to be coming off them. That's 230KV which is equal to 230,000 volts and that's a lot. I also have researched the health dangers that come from such a high voltage. There is Electromagnetic radiation that comes from these power lines. Electromagnetic radiation from high voltage power lines is something that can affect the health of people. Variety of studies suggests living close to high-voltage power lines can increase the incidence of several kinds of cancer, as well as other diseases. It is dangerous for our children and for us. We don't need any more sickness in our family. When we bought our land where our house is we thought we would grow old and have a peaceful life. Someday this will be left to our daughter to live a good life in the country. There's a reason we live in the country and we don't live in the city. Now Duke Energy wants us to sit around and hear humming noises from power lines all the time along with health issues. Another concern we have is the communication that has come from Duke Energy. It has not been what you think would come from a professional company. There has been a lack of communication ever since we found out about this. Another thing is we do not have Duke Energy for our power company. We get our power from South River EMC. I feel there could be another route that would benefit them better than taking our farm land. We have not heard anything positive about this project, only negativities. If you would please consider our concerns and feeling on these power lines. If there is another public hearing-I would like to attend to voice my concerns. Thank you for your time and your consideration of this power lines.

Tracy Adams  
1703E Parkertown Rd.  
Four Oaks, NC 27524

**To:** Members of the NC Public Utilities Commission

**Subject:** Public hearing regarding a Motion to Intervene (E-2 sub 1150) regarding the Cleveland Matthews Project

**Date:** October 30, 2017

**Place:** Johnston County Courthouse, Courtroom 4, Smithfield, NC

Dear Commission members,

My name is John Webster and I am a resident of rural Johnston County as well as a close and concerned friend of one particular family (Kim, Jeffrey, Gus and Lillie Canady) who would be directly impacted by the installation of the transmission lines currently proposed by Duke Progress Energy. As commission members, I am certain that you often encounter resistance when the issue of eminent domain is proposed or perceived. And, to be sure, much of the resistance is often due to fear, lack of understanding of the impact of the proposed project, etc. However, I'm certain there are many other times when the resistance and objections are based in fact and best interest of the community surrounding the proposed project. In addition, there are times when you, as a commission, must weigh the pros and cons of such a project and just do what is "right" for those impacted.

In the case of the Canady family, it is my belief that their resistance is based on a number of valid and significant factors and concerns and I would like to address each of them briefly.

- (1) The overall purpose of this project is to improve the infrastructure of the ever-growing Cleveland community, most of which is clearly not included in the area of the proposed transmission lines. Those living in the proposed site are residents of Four Oaks, Parkertown community, etc. What do they stand to gain from this project? It is my contention that they stand to gain nothing other than a loss of property value, multiple health risks due to high levels of radiation transmitted, damage to wildlife in the area, etc. There is no doubt that community growth comes with some degree of pain for all residents, but surely the bulk of the negative impact should not be forced upon those residents who stand to gain the least from the process.
- (2) As mentioned above, health risks to the Canady family and the countless other families impacted by such a project cannot be ignored. Kim Canady has a long history of cancer in her family and Kim herself is at a high risk of developing breast cancer due to a genetic mutation found in only .2-.3% of the general population. Kim, her husband, and their two children currently live within 700 feet of the proposed transmission lines. Kim fears negative repercussions for her overall health and longevity should the project gain approval. More importantly to Kim, she is concerned for the health of her two minor children both now and in the future as the land is passed down from one generation to another. And yes, this genetic mutation will have to be explored as it relates to Kim's

17 year old daughter, who quite possibly could build a home on the property in the future. All of these plans that have been in the works for years would likely fall through if the project is approved as the Canady family will not want to assume the risk of developing cancer due, in part, to the radiation generated by the transmission lines.

- (3) The mere thought of having an 150 foot easement split the property that is co-owned by Kim and her brother, Marty, is unimaginable. After all, who wants to sit back and watch the negative effects that would come with this project? The loss of trees, loss of wildlife, and loss of beautiful rural landscapes would be devastating to the tranquil beauty of the land in its present state as well as to the posterity of the Canady family, along with the many additional families in the Four Oaks area.

While the concerns that are listed above are measurable and objective in nature, there is something that I would like for this commission to consider that is even more powerful and impactful to the Canady family and others in the community: the impact of this project on such intangibles as family ties, heritage, and what defines those who have chosen to live in a less populated area of the county. The Canady family is a family of faith and a family who has never tried to steal from or harm a fellow member of their community. They are not trying to stand in the way of progress. Instead, they are trying to do everything in their power to pass down to their children and future generations the age-old values and traditions of their ancestors such as land ownership, quiet and good living, and protecting the value and natural resources of the land on which they live. The land in question has been passed down for generation after generation in this quiet rural area of Johnston County and this tradition is in grave danger of being lost if this project is approved.

As I close, I would beg this commission to consider who is being forced to pay for this project. Is it really fair and right to force the weight of this project onto the backs of those who stand to gain the least or should it be moved to a site that is less impactful and more beneficial to those affected? Your decision is not one that I envy but I do trust that each of you are reasonable people who want what is best for the communities in and around Johnston County. In the name of progress, please do not step on those who stand to lose the most including the rights guaranteed to them by the Founding Fathers of this country. Perhaps Jefferson said it best when he wrote these words in the Declaration of Independence: "We hold these truths to be self-evident, that all men are created equal, that they are endowed by their Creator with certain unalienable Rights, that among these are Life, Liberty and the pursuit of Happiness."

Thank you for time and attention.

John Webster

Hello my name is Jeffrey Canady and I am requesting DEP's application for the Cleveland Matthews Road Project be denied. My family and I live at 950 Gum Swamp Rd Four Oaks, NC. I have lived at this address since March 1994. This is the only home I have ever known. During my childhood we moved from place to place never living at the same place very long. Our residence joins the farm my wife, Kimberly Canady inherited from her grandfather, Albin Lassiter. Our residence/farm is in segment 33 of Duke Energy Progress "preferred route" for the Cleveland Matthews Transmission Line. Take note that I live in the Four Oaks Community not the Cleveland Community. In fact the majority of the approximately 11.5 mile "preferred route" is located in the Four Oaks Community not the Cleveland Community.

During the route selection DEP stated the primary goals regarding routing were to: Minimize overall impacts by paralleling existing ROW's, including transmission lines, highways, and roads where possible. Maximize the distance of the line from existing residences, and minimize the overall length of the route. Based on the route selected, none of these goals were met. The route selected is the longest route at approximately 11.5 miles long. The route selected does not utilize any existing ROW's. The route selected crosses Middle Creek 3 separate times and also crosses Black Creek. This will have a negative impact on the environment.

According to the study conducted for route selection the western route 4 was the overall lowest scoring route as well as the shortest route at approximately 6.3 miles. Western route 1 was the second overall lowest scoring route and is approximately 7 miles long. Both of these routes are considerably shorter than the southern routes. Why conduct the study if you are not going to use the overall lowest scoring and shortest route. Common sense tells you the shortest route will have the least amount of impact. In DEP's application it states that longer routes have higher overall impacts because the increased length provides greater chances to affect all measured criteria. The application also states that in this case criteria such as parcels crossed, cropland crossed and wetland crossed favored the western routes due to their shorter length. The application states engineering factors were considered for the route analysis. **Total Length** is a general indicator of the overall presence of the project. Length is also an indicator of construction costs. The longer the proposed route, the more expensive the project would be, so why chose the longest route?

DEP stated that public comment was used to determine the preferred route chosen. Only one letter was mailed out by DEP about the Cleveland Matthews Project. Several land owners along the chosen route have said they did not receive this letter about the public meeting. In fact some of the land owners have said they did not receive the certified letter from DEP informing that their land had been chosen for the route. During this public meeting DEP passed out questionnaires for people to voice concerns. Would it have been too much ask for this questionnaire to have been mailed out to all the landowners along the proposed routes? I receive a monthly statement from DEP telling how much electricity I have used compared to other residences in the area. This monthly statement looks much like junk mail and the letter sent out in November 2016 looked much like these monthly statements. We also receive lots of emails from DEP but none contained information about the proposed line. We receive lots of useless information from DEP but no information that could have potentially saved our family land. It seems DEP did not want the public informed. Would it have been too much trouble to have had multiple meetings in different locations? Not to mention the meetings held by DEP was just days prior to the Thanksgiving holiday. I have included an article from The Aegis in reference to Transource Energy was conducting public meetings to determine a route for their proposed 230 volt transmission lines.

Transource held 10 public meetings to give landowners and community members to give detailed input to the project team.

The only face to face contact I have had with DEP was initiated by my wife. On June 6, 2017, we met with representatives of DEP. Present for this meeting was DEP representatives, Joe Luis and Miranda Gregory, Marty Lassiter, Kimberly Canady and I. We were attempting to get information on what type of structure would be coming through. During this meeting Joe Luis said it best, "you would have to be crazy to want this thing". The meeting ended abruptly because Joe Luis said surveying would start the next week and we would be compensated later for the trees cut.

DEP said another reason the western route was not chosen was because this route crossed open/green spaces in subdivisions. I have looked at the map of the routes on DEP's website most of the western route crosses forest and crop lands. The western route does come near some subdivisions but does not appear to cross cul-de-sacs as stated in the application by DEP. Some of the land in the western routes may have been approved for subdivisions but have not yet been developed. If the subdivisions have not been developed how does this cause litigation problems with all the residents of the subdivisions if there are no residents/houses in the subdivisions?

What about my family's right to enjoy our open/green space? How can someone else's right to open/green space be more important than our rights especially since most of the land in the western routes subdivisions have not yet been developed? In the fall of 2016 my wife was diagnosed with a rare BRCA1 mutated cancer gene. It is likely this rare gene was passed on to one or both of our children. We enjoy hiking, hunting and fishing on our property. If the line is allowed to come through our land my family will not be able to use our land due to the increased risk of developing cancer. Fishing is one of my son's favorite hobbies. The center line of the proposed route for the transmission line crosses directly over the pond on our land. If the high voltage line comes through my son will no longer be allowed to use what is ours. Most importantly neither of our children will be able to build their dream house on our family farm if the high voltage power line comes through. We will also lose the ability to develop our land if the need/desire arises. So I say again why does my family lose the right to enjoy our open/green space to a proposed subdivision that has not yet been developed?

Upon traveling areas of North Carolina, I have discovered that 230 volt transmission lines are run on existing ROW's along roadways. I first discovered this in July of 2017 while traveling NC Hwy 17 in Brunswick County. These lines are run on a single pole with the 3 230 volt transmission wires being run at the top of the poles with the normal service lines below the transmission lines. I took photographs of these lines and have included a copy for the Utilities Commission. On July 26, 2017, at approximately 4:16 pm, my wife, Kimberly Canady spoke with Kevin Hardin. Hardin is an engineer with Brunswick Electric. Hardin confirmed that the lines run along NC Hwy 17 were in fact 230 volt transmission lines with normal service lines on a single pole. Hardin said the poles are taller and bigger but this allowed both lines to be run on a single pole using existing ROW's beside the roadway. Hardin also said that Progress Energy upgraded a 230 volt transmission line from Whiteville, NC to Florence, SC on a single pole. I have also noticed what appears to be a 230 volt transmission lines run on a single metal pole alongside NC Hwy 42 in Wilson County.

Single pole 230 volt transmission lines are run on existing ROW's beside the roadway in the Western Harnett area on NC Hwy 27. These lines are coming and going from a substation beside the roadway. I followed these lines along several roads in the area and took pictures and have included a copy for the

Commission. The 230 volt transmission lines are at the top of the metal poles with the normal service lines/transformers below the transmission lines. These lines are operated by South River Electric. I made contact with Andy Garris with South River Electric. Garris said he confirmed with his Supervisors that the lines along NC Hwy 27 were in fact single pole 336 volt transmission lines at the top of the poles with normal service lines below. I have also located single pole transmission lines running beside NC Hwy 242 in Sampson County. I have included pictures of these lines for the Commission. Garris said his Supervisor told him these lines are single pole 336 volt transmission lines with normal service lines below the transmission lines. Garris said DEP actually owns the lines along NC Hwy 242. Garris said South River Electric also has single pole 336 volt transmission lines with normal service lines along NC Hwy 13. Garris said South River Electric makes all attempts to use existing ROW's beside the roadways with condemnation of property being an absolute last resort.

With this in mind, no one has to have their most precious possession taken away. It only makes sense that DEP use existing ROW's along the shortest western routes to run single pole 230 volt transmission/service lines to the Cleveland Matthews Road Substation. This method would not change the landscape of the developed area of the Cleveland Community since the lines would replace existing service lines beside the roadway. The developers and prior landowners that sold their land for a huge profit created this need for additional power, therefore the burden of the new lines should fall on the Cleveland Community not the landowners of the Four Oaks Community.

DEP is a private for Profit Company and should not have the authority to choose the route for the high voltage power line. If there is a need for the power line DEP should be responsible for conducting route studies then providing the State Utilities Commission with several proposed routes. The State Utilities Commission should have the final decision on the chosen route and type of structure such as the single pole using existing ROW's along the highway. DEP is a private for Profit Company that is only looking out for its bottom line, not what is best for the landowner.

I respectfully ask that the Utilities Commission deny DEP's request for the route chosen for this project.

Thanks,

A handwritten signature in black ink, appearing to read "Jeffrey Canady". The signature is fluid and cursive, with a long horizontal stroke extending to the right.

Jeffrey Canady

# Final route selected for controversial power line project through part of northern Harford



By Allan Vought  
The Aegis

OCTOBER 18, 2017, 12:55 PM

**T**ransource Energy has announced the routes that it will file with state regulators in Maryland and Pennsylvania for its Independence Energy Connection high voltage overhead electric transmission line project that has sparked community opposition in Harford County and neighboring York County, Pa.

The company had been looking for several routes to connect a new electric switching substation planned near the Susquehanna River in York County with the existing Conastone switching station near Norrisville in Harford County, a distance of about 16 miles. A similar connection is planned between switching facilities in south central Pennsylvania and Washington County, Md., near Smithsburg, about 29 miles.

The final proposed Harford County route unveiled by Transource Monday parallels Route 23 to the west for about 3 miles from the state line to Conastone Station.

Transource was contracted by PJM Interconnection, the regional power grid operator for the affected the area, to build the \$320 million project.

In a news release, Transource stated that PJM identified the need for the infrastructure upgrade “to alleviate congestion on the high-voltage electric grid and benefit customers in the region, including parts of Pennsylvania and Maryland.”

Steve Herling, vice president of planning for PJM, which operates the power distribution grid for 65 million people in 13 states and the District of Columbia, stated in a recent letter to The Aegis that the grid operator “performed extensive analysis of this highly congested area where limitations to move electricity efficiently have been a chronic problem.”

**“The 10 events provided landowners and community members the opportunity to give detailed input to the project team,” the news release states. “All submitted input was incorporated into determining the final proposed routes.”**

**“Transource worked to balance the public input with a variety of factors such as existing land use, sensitive species and habitats, soils and topography, historic and cultural resources and the opportunity to parallel existing infrastructure,” the release continues.**

**The company also stated that in addition to routing options, it presented two tower structure options – lattice or monopole. The majority of comments received supported the monopole option, according to the company, and that is what it will use, “except in areas where engineering or construction needs dictate another structure type.”**

**“By including community members in the siting process, rather than engaging them after decisions were made, we were able to consider and accommodate many landowner requests,” said Todd Burns, Transource director, in a statement.**

**“The input gathered over the last few months was a critical component of our decision-making process,” Burns continued. “We are confident that the route selection strikes the balance between building the required infrastructure that powers our homes and economy, while respecting land use and the environment in these communities. We look forward to continuing to work with these communities as an engaged partner as we move forward with the regulatory approval phase of the project.”**

**Transource said it is directly notifying involved landowners, as well as people who have been part of the community input process.**

**The project and the final routes for the power lines must still be approved by the Maryland Public Service Commission and the Pennsylvania Public Utilities Commission. Transource said it plans to file applications with both by the end of the year.**

**Construction of the IEC is expected to begin in 2019, with a project in-service date of mid-2020, the company said.**

**Additional information can be found on the project website at [www.TransourceEnergy.com/Projects/Independence](http://www.TransourceEnergy.com/Projects/Independence).**

“This solution is the most reliable and cost effective and will save consumers millions in the long run,” Herling wrote of the IEC.

But opponents on both sides of the Mason-Dixon Line that forms the border between northern Harford and southern York counties, have challenged both the need for the project and why Transource is not following existing power line routes.

“There have been no published studies to determine if the energy that is to be sent over the new transmission power line towers can be accommodated by the use of existing lines and towers,” Norrisville resident Aimee C. O’Neill wrote in a letter published by The Aegis last month.

O’Neill, who could not be reached for comment Tuesday, is co-chair of a group formed this summer to oppose the project called Stop Transource Power Lines MD.

Harford County opponents who have spoken out publicly about the project, many of them landowners, say the proposed power lines will disrupt businesses and ruin the mainly rural appearance of the countryside and disrupt watersheds and wildlife habitat.

Opponents also say they believe some properties in agricultural preservation programs will be negatively affected by the new power lines.

Stop Transource Power Lines MD plan to hold an informational meeting Wednesday evening at Pond View Farm in White Hall to discuss the final power line route announcement and what steps to take next.

O’Neill and others in the organization have been critical of what they say has been a lack of interest among elected officials concerning the impact of the project, although the northern Harford area’s County Council representative, Chad Shrodes, has worked closely with the opponents all summer. The area’s state legislators also have attended one or more of the community meetings this summer.

The opposition group also has worked through the Jarrettsville/Norrisville Community Advisory Board to inform Harford County residents about the project and the reasons for its opposition.

Transource hosted two community information meetings about the project in Harford County over the summer, the last in Norrisville in August, which was attended by nearly 200 people.

The company stated in its news release that it presented more than 250 miles of route options in the east and west segments of the IEC project for review.





