MURPHY STATEMENT NO. 2

C-2015-2475725
PL JR 12/5/16

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

Laura Sunstein Murphy	:	
	:	
v.	:	Docket No. C-2015-2475726
	:	
PECO Energy Company	:	

DIRECT TESTIMONY
COMPLAINANT
LAURA SUNSTEIN MURPHY

April 29, 2016

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DIRECT TESTIMONY
OF
LAURA SUNSTEIN MURPHY

I. INTRODUCTION AND PURPOSE OF TESTIMONY

1. Q. Please state your full name and address.

A. My name is Laura Sunstein Murphy. My address is 1191 Telegraph Road, West Chester, PA 19380.

2. Q. How long have you lived at your current address?

A. I have lived at my current address since 1990.

3. Q. Are you a customer of PECO Energy Company?

A. Yes. I receive residential electric service from PECO under account number 2346901005.

4. Q. How long have you been a PECO customer?

A. I have been a PECO customer since 1967.

5. Q. What have you alleged in your complaint?

A. My complaint, and the amended complaint filed on July 28, 2015, allege that PECO's installation of a smart meter that emits harmful EMFs on my house would create an unsafe and unhealthy condition in my home because I suffer from a number of rare and serious medical conditions which are exacerbated by exposure to such emissions. I am asking the Commission to order PECO

to provide me with safe and reasonable service in compliance with the Public Utility Code.

6. Q. Would you please tell us why you are here today?

A. I am here today because Pennsylvania has no opt out for Smart Meters, and I have genetic medical conditions which require me to limit all forms of excessive oxidative stress in my life. PECO's smart meters, including the current AMR meter which has been on my house since May 2002 (I recently found out pursuant to PECO's Answers to Murphy Interrogatories, Set I, Appendix A) and the proposed AMI smart meter which PECO has demanded it install under threat of termination of my electricity (Appendix B) both emit far too many bursts of EMF for my body to handle safely. I want to register first both my public embarrassment and my indignation on account of the ordeals that PECO has forced upon me with its Gestapo tactics regarding universal deployment of smart meters to its customers, with no regard for deleterious health effects of those meters on sensitive individuals. I know a few other individuals in PECO's territory who have been adversely affected by smart meters installed on their homes, but they have been intimidated by PECO into submission. I have overcome my embarrassment and I have moved forward with demanding respect for my individual rights under the law and my health and wellbeing. My own grandmother was not permitted to vote solely because she was a woman. I will never forget her telling me that she became a suffragette, along with her sister, demanding their rights to vote along with their husbands.

If Pennsylvania did have an opt out, I merely would have opted out of having any sort of wireless meter on my home, and that would be the end of it. I would not be wasting the Commission's time, my retirement money, my experts' time, and PECO's time. I would not have to reveal to my utility company very private information about my genetic diseases which have been greatly aggravated by the EMF emitted by the wireless transmission of the AMR meter on my home. I would not have to be forced to reveal very private health issues to my utility company which I have only revealed to my health care providers. I would simply fade into the woodwork.

But Pennsylvania does not offer an opt-out to smart meters. The Pennsylvania PUC has interpreted Act 129 to require universal deployment of smart meters to all affected electric utility customers, apparently even if deployment of those meters would be harmful to the health of certain of those customers. This is unacceptable to me.

7. Q. What advice has PECO and the Commission given to Complainants who have expressed the desire not to have a smart meter installed on their property?

A. PECO and some PUC judges have advised PECO smart meter health issue Complainants, in filings and in rulings, that PECO customers who wish to opt out of smart meters should seek a legislative solution.[1]

8. Q. Is a legislative solution a feasible alternative for you?

A. No. Unfortunately, Robert Godshall has been the Chairman of the House Consumer Affairs Committee of the Pennsylvania House of Representatives for many years. Mr. Godshall's son works for PECO. In fact, until recently, Mr. Godshall's son worked for PECO's smart meter roll out initiative. Although it has been reported that Mr. Godshall does not have a smart meter on either his office or his residence, Mr. Godshall has been a vehement opponent of any customer who alleges deleterious health effects from a smart meter having her day in Court. See, Appendix C, Robert Godshall letter to the PUC regarding the initial PUC decision of September 2015, on PECO's interlocutory appeal of Judge Heep's ruling on the Kreider Complaint in July 2015.

[1] See, e.g., *Povacz v. PECO*, Docket No. C-2012-2317176, 2012 Pa. PUC LEXIS 1579 (Order issued September 28, 2012); *Tucker v. PECO*, Docket No. C-2015-2515592 (Oder issued April 5, 2016); Respondent's Preliminary Objections in this Docket.

Mr. Godshall has steadfastedly refused to allow any opt out bills to smart meter deployment to be debated and called for a vote. The only smart meter bill which Robert Godshall has allowed to be debated and called for a vote, is the bill which seeks to protect the privacy of electrical consumption of the customer who owns smart appliances, gleaned by intrusion into the private life of the customer.

Those customers who have legitimate health concerns over the safety of the wireless smart meters deployed by PECO as to us, have no reason for optimism that the congressmen who have introduced opt out bills in the Pennsylvania legislature will be able to see any of those bills passed during their lifetimes. Such is the ability of any PECO customer to "seek a legislative solution" to the health effects of smart meters on vulnerable individuals.

9. Q. Are you aware of any other possible alternatives available to you?

A. I understand PECO has considered having sensitive individual customers who claim deleterious health effects from smart meters that the customer move the meter socket (at the customer's expense), to a location farther from the customer's living space.

10. Q. Is moving the meter socket a viable alternative in your situation?

A. No. Moving the meter would not be a viable alternative for me. As explained more fully below and in the testimony of Dr. Pall (Murphy St. 1),

the harmful effects of EMFs for sensitive individuals like me are not ameliorated by simply moving the meter a short distance. Perhaps PECO believes that moving the meter socket of a customer's residence would protect the customer from the harmful effects of EMF emitted by the smart meter. Of course, PECO is not prepared to accept the premise that any customer could be harmed by EMF emitted by PECO's smart meter in the first place, or by any other effect of the smart meter on a customer's home.

11. Q. What actions of PECO have led you to believe that PECO is either uninformed or willfully blind in this respect?

A. That was evident from the hearing of the Complaint of Susan Kreider vs. PECO. I attended Ms. Kreider's hearing in early March 2016, and I recall Dr. Mark Israel, PECO's expert medical witness, testifying that Ms. Kreider's symptoms (which Ms. Kreider had testified had first appeared shortly after the smart meter was placed on her home, and that most of those symptoms had started to fade shortly after she was forced to remove the smart meter from her home and replace it with an analog meter due to health reasons) could not have been caused by the smart meter. Dr. Israel testified that there was no credible evidence that smart meters can cause health effects. Dr. Davis, PECO's expert witness, testified in the Susan Kreider case that there was no mechanism by which low level EMF which was too low to heat up the tissues could cause harm, because there was no mechanism to explain how it could happen. See, Appendix D (Excerpts of Transcript from March 7, 2015 hearing).

I am a very religious person. I believe in God, but I cannot explain the mechanism as to why I believe in God.

Fortunately, we do not have to accept on faith the fact that low level intermittent bursts of EMF do have biological effects on living organisms without heating the skin. There is an elegant electro biological mechanism for this to occur which has been explained by Dr. Martin Pall in many scientific papers. I consider myself very privileged that Dr. Pall agreed to act as my expert witness, because Dr. Pall understands, I believe better than anyone else in the United States or perhaps the world, the mechanisms by which low level EMF bursts act upon the voltage gaited calcium channels in the cells of the body, leading to oxidative stress and a whole cascade of usually nasty consequences in vulnerable individuals.

And Dr. Palls' understanding regarding the voltage gaited calcium channels also explains quite succinctly how low level bursts of EMF can also, in the appropriate medical settings, be used for healing.

I was the beneficiary of those healing qualities of pulsed EMF back in 1999. I will explain my story presently.

12. Q. In offering to allow the customer to move the smart meter socket to another place farther from the customer's living space, what specific

health issues regarding the smart meter technology does PECO choose to ignore?

A. PECO does not explain to the customer that moving a socket for the smart meter farther from the customer's living space does absolutely nothing to shield a sensitive customer from the whole-house Wi-Fi that the Zigbee radio contained in the smart itself bathes the home with EMF, with pulses being emitted at least every 30 seconds with no reprieve for the customer and the customer's family or pets to get away from the Wi-Fi. In fact, PECO answered my Interrogatory Set I question that PECO did not offer any customers any shielding from its smart meter emissions. See, Appendix E. PECO obviously believes or is willfully blind to the notion that sensitive people need no shielding from EMF and can be inundated with these emissions with no recourse, with no ill health effects.

With no shielding available, and no ability of the customer to shut the zigbee down, the customer and her family and pets have no escape, because the zigbee Wi-Fi has to continually search for smart appliances in every customer's home, because that is what Act 129 requires, and because there can be no harm ever caused to anyone by the EMF emitted by PECO installed equipment, because that EMF is too weak to burn the customers. That is the party line espoused by PECO and its experts. I know differently.

13. Q. Do you have other concerns with regard to PECO's smart meters?

A. Yes. The Flex Net module contained in the AMI meter is equally problematic for sensitive customers. It emits huge bursts of EMF, on a constant basis, and The Flexnet can be remotely programmed by PECO to transmit once every 15 minutes and every transmission is 70 milliseconds in duration. See, Appendix F - Murphy Interrogatory Set I at I-16 and I-14.

Additionally, the Switched Mode Power Supply contained in all digital meters creates dirty electricity and harmonics on the whole household current which are harmful to me. PECO has admitted in its answers to the Murphy Interrogatory Set I that the smart meter technology cannot be turned off by the customer, and it continues unabated even if the customer turns off all power on her circuits. See, Appendix F.

14. Q. Were you aware that the AMR meter PECO used to replace the analog meter that had been on your home since you purchased your farm was emitting EMF?

A. No.

15. Q. Before you first filed your Complaint against PECO in March 2015, were you consciously aware that the AMR meter that PECO placed on your home in 2002 was harming you?

A. The AMR meter which PECO placed on my home in May 2002 has caused me considerable harm which I will detail below. I was not aware that the

AMR meter placed on my home has been constantly zapping my living space and my yard with EMF for the last 14 years until PECO answered my Interrogatories, Set I.

Back in 2002, I was never informed by PECO that the meter they wanted to install was doing that. I was only informed back in 2002 by PECO that they wanted to install a new meter so they didn't have to come up my very long driveway every month to read the meter. I thought, "That's nice. Glad you have the technology to do that".

I never knew until PECO answered my first set of interrogatories that PECO had never tested my AMR meter on humans, or that it was a powerful source of injury to my body and my cognitive abilities.

PECO and the PECO experts would like to discredit the whole notion of electrosensitive individuals' existence, with studies of self defined electrosensitive individuals who were not able to distinguish consciously when they were being exposed to EMF and when they were being subjected to sham exposures. This misses the point. People have different thresholds for consciousness of what is happening to their bodies.

For example, I have developed bouts of atrial fibrillation after the installation of the AMR meter on my home. Before installation of the AMR meter, I never experienced any heart irregularities. I have little control over its inception. I know now that certain triggers may start my heart arrhythmia. Triggers such as watching suspenseful movies, a few sips of wine,

epinephrine administered by a surgeon, ramping up my desiccated thyroid medication too high or allowing it to get too low, caffeine consumption, etc. I have learned to avoid those triggers. But oftentimes now, I go into afib without any known triggers. When my heart goes into atrial fibrillation, I feel horrendous, agitated, and distraught. I immediately have to lie down. I cannot move without tremendous effort. I feel like I am dying. My heart is out of control. I have to drag myself to my bottle of magnesium, down several pills, drink some milk, run a bathtub of hot water filled with epson salts, try to relax and pray that the atrial fibrillation will end quickly.

I was amazed that my cardiologist, a doctor I never needed before the installation of the AMR meter, informed me that many of his patients have no idea when their heart is in abnormal rhythm. This is astonishing to me, because I am very painfully aware when my heart goes out of regular rhythm. But it explains that someone may be hypersensitive to EMF, yet not be consciously aware when the exposure happens. Dr. Pall has pointed me to a study of EMF hypersensitive individuals who may or may not have been consciously aware when they were irradiated with low level EMF, but their EKGs showed clear indications of changes when they were exposed to EMFs.

II. PERSONAL AND FAMILY BACKGROUND

16. Q. What is your personal background and how did you come to believe that the AMR meter has been harming you?

A. Because of my genetic abnormalities, I am particularly sensitive to many environmental factors which may cause no harm to the average individual.

I grew up in Bala-Cynwyd in the 1950s. I have five siblings. My father was an electronics engineer, a graduate of MIT, a fellow of the IEEE and of its predecessor, the IRE, and he served on Jimmy Carter's scientific advisory panel. He was also quite a prolific inventor, holding more than a hundred patents, and having invented hundreds more innovations that he never bothered to patent.

We children were taught to respect the environment, to always use our brains, to keep our bodies healthy, and to understand innovation. The adage, "if you are not part of the solution, you are part of the problem" was a mantra in the family I grew up in.

My father spent much of his career working on top secret military projects. He did inform us of some of his inventions which were not classified.

My father had been working on the development of radar during World War II. He realized that radar used on ships at the time was not able to distinguish between another vessel and the choppy seas. He thought about the problem, and he invented and patented a type of radar in the mid 1940s by means of

which viewers could differentiate between choppy ocean and a solid mass such as another ship. He named his invention "color radar", and his patent is still cited in recent patent applications today.

My father died of leukemia at the age of 60, after having been diagnosed five years previously, far too young. His brain was still working full speed, but his body succumbed to leukemia. He told his children when he was diagnosed that his unprotected work with microwaves throughout much of his career had caused him to develop leukemia.

I am sure that my father is rolling over in his grave at the thought that low level EMF cannot cause biological effects, both from a scientific perspective and from the cause of his early death.

17. Q. What physical activities were you engaged in before PECO's deployment of the AMR meter on your home?

A. I should explain that I was born with two genetic diseases of the connective tissue: Ehlers Danlos Syndrome Type III, and lipedema. I was incredibly flexible as a child and young adult. I was extremely athletic. Even though my ankles twisted all the time and I tore ligaments all the time in my ankles due to loose and poorly formed connective tissue, and my legs were always covered in bruises, I still was very active in sports. Because of my hyperflexibility, I excelled in gymnastics and diving. I went to public school and played on the lacrosse teams and hockey teams all through junior high and high school.

18. Q. What is your connection with horses and farmland?

A. Even though I was born in Bala-Cynwyd, I always longed for the country. My passion was horses. I had never actually seen a real live horse until I was nine, but I begged for riding lessons from the time I was five. As a teenager, I spent all my babysitting money on riding lessons and renting horses for a few hours at a time. I wrote all my class projects in elementary school on equine topics. I drew pictures of horses all the time. I watched Fury on TV on Saturday mornings. I watched the Lone Ranger and his horse Silver on TV. I was obsessed. I adore the feeling of sitting on a horse, being one with his movements, walking or gliding through the woods or over Andrew Wyeth countryside on horseback. There is nothing that can compare to the tranquility, the beauty or the sheer pleasure of that experience. I thought I would never own a horse. I thought you had to be rich to own a horse.

19. Q. When did you notice you could not tolerate fluorescent lighting?

A. My first job after graduating from the University of Pennsylvania was teaching Latin and French in Cheltenham School District. The school I was assigned to was brand new. It had fluorescent lights in the ceiling. I could not stand to teach in that classroom with the lights on. They quickly gave me a headache. I brought in an incandescent lamp from home to my classroom and placed it on my desk. That was in 1972 or 73, way before the ADA. My principal never asked me to turn the overhead lights on, and I never had a problem teaching with only my desk lamp for illumination of the classroom.

20. Q. Do you have other sensitivities because of your genetics?

A. Because of my genetic abnormalities, I am particularly sensitive to many environmental factors which may cause no harm to the average individual.

I can smell a lit or smoldering cigar or a lit cigarette a mile ahead of me being smoked in a car with all the windows rolled up. That smell nauseates me, and I have often thrown up in the past upon smelling a cigar. I have always been designated the official "smeller" to make sure food is safe. My sense of smell is highly developed.

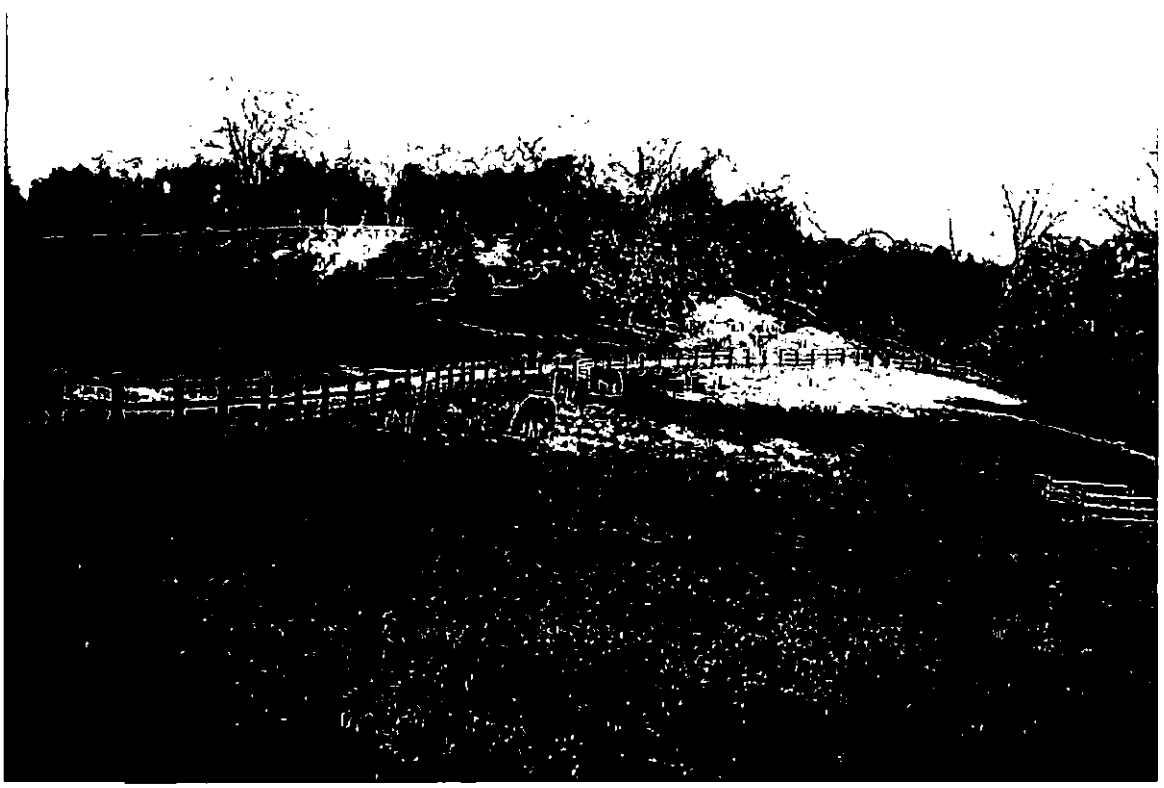
I cannot tolerate most prescription drugs.

Many of them do not work on me at all, such as Percocet, codeine, lidocaine, ibuprofen, Tylenol, acetominaphen, oxyodone, warfarin, gadolinium dye, flagyl, keflex, statin drugs, quinapril, all antibiotics ending in -cycline, versed, levaquin, demerol, all general anesthetics except propafol, and many others, or if they do work on me, they cause me bodily harm, especially to my liver which cannot detox them. Up until recently when I underwent genetic testing, I learned my adverse drug reactions the hard way. I was the guinea pig.

21. Q. When did you move to the farm you currently live on?

A. I decided in 1986 to go to law school. My goal was to graduate in the top of my class, get a good paying job at a large law firm, and buy a home with at least 10 acres where I could have a horse to ride on the trails. I achieved all those goals. I worked hard. I graduated from Villanova Law School in 1989

Order of the Coif and started to work at a large law firm in Chester County. John and I were married in 1989, and we purchased our dream home in Chester County in the spring of 1990. We own 11.88 acres, and our home is quite far from the road, which was still unpaved when we bought our farm.



View of the Murphy lower pasture field as seen from the top of the lower field pasture, in autumn. My four horses appear tiny at the far end of the pasture. Part of my hay field appears across the driveway to the left of the fencing. The well which provides water to the Murphy household and barn is located in the lower field pasture near where the horses are grazing, and it is boxed in with post and rail fencing.

My horse Magic Millennium at my barn in 2012. Magic is a Tennessee Walker whom I have had since 2002. He is now 16. One of our barn cats, Molly, can be seen on the right.

It took several years for us to save up the money to convert the outbuilding into a barn, and to buy fencing. But we accomplished that and I bought a horse in the mid-1990s. I took in a boarder at first to keep Mr. Ed company and to afford my riding friends a mount to go out on the trails with me. I was in heaven. I rose early every morning to feed the horses and change for work. I hired a helper to do the afternoon feed when I was still at work, but I had my horse to come home to at night. And all weekend I took complete care of the horses and the barn and garden, and all entertaining of friends and family. I rode every single weekend, and I was full of energy. Life was good, if not hectic, as an associate at a large law firm. My farm was always a haven of

tranquility for me each morning, each evening and each weekend after putting in long days at the office. There is something about the smell of hay and horses that I have always loved. They bring me peace, even though they can be a lot of work to take care of and to tack up and untack after a ride. Feeding and caring for horses in the winter with bitter wind and heavy snows can be a challenge, and there is no break from responsibility if you own a horse. Appendix I contains additional photographs of the farm and the animals referenced above.

III. HEALTH AND MEDICAL HISTORY

22. Q. How was your health from the time you bought the farm in 1990 until the AMR meter was installed?

A. The years 1990 to 1999 were years of productivity and robust health for me. I had a few minor medical procedures over the years, such as a recommended colonoscopy when I turned 50, which was normal, no findings, a meniscus repair of one knee which I had torn using an exercise machine in law school, and little else. My health was fine. I worked tirelessly. I had a very demanding job as a health law attorney, and I took care of the gardening myself, and of my autistic son, also entertaining friends and traveling to see our children and traveling on vacations, despite having been born with two inherited potentially debilitating genetic abnormalities. I was rarely sick. I rarely picked up even a cold. I had never experienced debilitating pain.

23. Q. What changed in regards to your health?

A. My current state of health is quite poor, and has been poor for many years. I became disabled and I remain disabled.

Up until 2002, at age 54, I had no significant health problems. The one interruption in my health for my entire life before 2002, was a year long convalescence from a badly broken leg sustained in a horse back riding incident in 1999. I was galloping my horse in a field across from my home. I fell off at a gallop when he shied at a barking dog, and I landed with all my weight on my right heel, shattering my tibia into many shards and snapping my fibula in half. My tibia bone protruded through the skin. My foot hung at a strange odd angle as my posterior tibial tendon was ripped by the break. I had been riding since I was nine years old, and we horseback riders are used to having accidents and getting right back up on the horse, but this one was a doozy. There was no getting right back up in the saddle this time.

From 1999 to 2001 I had to take a year off from work as I was recovering from the compound comminuted pylon fracture of the right leg, which resulted in osteomyelitis, and being bedbound and wheelchair bound with no weight bearing on that leg from 1999-2000.

I underwent three surgeries with internal fixation devices to plate and screw the shards of bone together and to close the surface wound; several picc lines were inserted seriatim for IV vancomycin antibiotic administration to heal the bone infection (My genetic abnormalities caused my body to close (sclerose)

each picc line within 2 days). During this time, I underwent a nuclear bone scan test to see if the infection was gone after the IV therapy, resulting in radiation poisoning symptoms lasting at least 5 days.

24. Q. What medical device was prescribed for you during your healing from the badly broken leg incident?

A. I was prescribed a bone stimulator which I placed on my leg for a short time each day to stimulate bone growth in my non unionizing bone. The bone stimulator was FDA approved, prescribed by my orthopedic surgeon, and paid for by my insurance company. That bone stimulator worked by emitting pulsed EMF in very low doses. I was carefully instructed by the device manufacturer representative how many minutes to place the stimulator on my leg each morning and each evening and no more. I could feel the bone stimulator tingling on my broken leg. The bone stimulator worked so well, in fact, that when I had subsequent surgery in 2001 to remove the plates and screws in my leg, the surgeon who removed the hardware had a really tough time jack hammering the plate off my fibula because I had grown so much bone over the plate. She thought perhaps the fibula plate had been on for at least three years.

I doubt that the PECO experts have an explanation of the efficacy of bone stimulators on non union fractures, if it were true, as they insist, that EMF that are too weak to cause heat can have no biological effects. Dr. Pall, of

course, explains how the bone stimulator works via EMF effects on the voltage gaited calcium channels.

25. Q. Were you able to resume your previous level of activity following the successful healing of your broken bones?

A. Yes, I was. I started to weight bear and walk and ride my horses again and resume all my former activities in 2000 shortly after a revision surgery in Baltimore to cut out a piece of bone from my heel to slide my leg more vertically over my foot, and a tendon transfer of one of my toe tendons to tie into the severed posterior tibial tendon to help keep my ankle from collapsing. The surgeon used my heel bone tissue he had removed to form a graft onto my still unhealed fibula, which had been set crooked by the local surgeon the previous year. I also went back to work full time to my former position as the head of Morris James health law practice in Wilmington, Delaware, and I resumed all my previous leisure activities.

In retrospect, the speed at which I healed from all the surgeries I underwent before the deployment of the AMR meter speaks volumes, in comparison to the lack of complete healing and incomplete restoration of health my body has suffered from since deployment of the AMR meter on my house in May 2002.

26. Q. What happened, then, to your health following the deployment of the AMR meter on your house?

A. In May 2002: PECO removed my analog meter and replaced it with a constant emitting AMR meter, which I measured recently to be emitting far more EMF than even PECO's interrogatory answers indicate. This constant bath of EMF has caused me unremitting bodily injury, robbed me of my golden years, exacerbated all my genetic disease and has prevented me from healing from any of these insults.

I will summarize my deteriorating conditions, likely caused mostly by the AMR meter, that have led to my current disabled state of health.

2002: Immediate and continual constipation. I began to suffer from constant constipation and concomitant abdominal pain. I was followed by GI doctors. Constipation quickly led to diverticulitis (see below). Severe constipation has continued now more than a decade later, despite all possible interventions. However, when I first placed a smart meter guard faraday cage on my AMR meter a few days ago, I was immediately blessed with a day of diarrhea. Most people would not feel blessed to have diarrhea, but if you have suffered severe constipation for 14 years, you would be happy to experience the opposite. I believe that complete absence of EMF in my home would go far to regulate my entire body and all its systems, which have been terribly skewed since the advent of the AMR meter on my home.

It is interesting to me to reflect on the symptoms that those who suffer from electromagnetic sensitivity suffer from. Many of them report immediate constipation which does not abate until the source of EMF is removed. Dr. Pall explains in his expert opinion why this is so.

In my case, the severe constipation brought about by the installation of the AMR meter quickly led to diverticulitis the following year. This is surprisingly fast growth, since just in 1997, when I had turned fifty, there was no sign of any diverticulosis, and I had no symptoms of constant constipation or abdominal pain at that time..

2002: Hypothyroidism. I started to suffer from extreme fatigue. I was first diagnosed with hypothyroidism in 2002. I have been treated with desiccated thyroid. Hypothyroidism persists to this writing, and has to be monitored carefully at least every six months. Hypothyroidism and changes to the endocrine system are common among those who are adversely affected by low irregular emissions of EMF.

2002: I first started to gain weight despite no change in diet or exercise. Excess weight of 35 pounds cannot be removed to this day, despite extremely careful healthy food intake.

2003: First attack of diverticulitis, sending me the ER. Many more attacks of diverticulitis would follow, sending me the Chester County ER and Paoli Hospital ER; I finally had to undergo a colon resection in 2011 to remove the diseased section of the colon near the sigmoid colon (see below).

2004: I was first diagnosed with uterine fibroids, which were treated via D and C operation.

2004: Atrial fibrillation was first diagnosed. A-fib recurs from time to time to this day. My attacks of afib are incapacitating, and require me immediately to lie down quietly and rest, soak in Epsom salts bath, take massive doses of magnesium and hope and pray that they will go away in a few hours. Heart arrhythmias are common symptoms of those who suffer from electromagnetic sensitivities.

2004: ptosis, my eyelids were overhanging my eyes and had become so limiting of peripheral vision that I had to undergo blepharoplasty surgery. I suspect that the AMR meter had little contribution to the development of ptosis, and Ehlers Danlos had more to do with the development of ptosis. But the EMF added an additional burden of oxidative stress to my body as I was healing from the surgery.

2005: aortic valve regurgitation and mitral valve prolapse were first diagnosed. I am followed by a cardiologist. Heart abnormalities are common complaints in those suffering from electromagnetic sensitivity.

2007: Spontaneous detached retina of the right eye, surgically repaired on an emergency basis. I am followed by a retinal specialist. Those who have suffered ill effects from smart meters have reported seeing flashes and floaters in front of their eyes. This is the first sign of the retina pulling away from the eye, and it is a warning sign of the medical emergency that a detached retina

presents. I was unfortunate in that I was not able to prevent my retina from detaching, not the first time and not the second time (see below). My retinal specialist has told me that once one retina detaches, I am at higher risk for the other retina in the other eye detaching.

Having to undergo laser surgery for the repair of a detached retina without any anesthesia was akin to and nothing short of Samson's eyes being put out with a red hot poker by the Philistines.

After the surgery to repair the detachment, the surgeon places a bubble of oil, I believe, to act as a sort of compression bandage, inside the eye, and the patient has to hold her head cocked at a strange angle, day and night, for weeks to hold the repair in place until the body resorbs the bubble. I had to be driven to the eye doctor every day for three weeks to have her check my progress. It was sheer torture. I have diminished vision in that eye and I still have remaining black floaters that persist.

2007: I also began to suffer from severe leg pain. I went to vascular surgeons who diagnosed me with venous insufficiency. I underwent several vein ablations in both legs and one arm between 2007 and 2010.

However, my severe leg pain continued to increase, rendering me an invalid by 2013. This was lipedema pain, caused by inflammation and the growth of abnormal lipedema fat in my legs, and then arms, which was finally relieved by three surgeries in 2013.

2008: I was first diagnosed with cataracts. This is unusual because I was only sixty years old and have never gone into the sun without a hat due to my very pale skin from Ehlers Danlos. It turns out that low level EMF also can cause cataracts. Dr. Pall has written on cataracts being caused by low level EMF in his expert opinion.

By 2008, I was unable to maintain my law practice because of the severe pain I was suffering from. I sold my practice to another law firm and started to work part time.

2010: My second spontaneous retinal detachment in my right eye. I underwent surgical repair again by a retinal specialist. She mentioned that it was very unusual for a repaired retinal detachment to recur after the first year. I now know that the AMR meter combined with my genetics likely caused my retina to detach a second time. All the more reason that I should never be subject to EMF in my home.

2011: My abdominal pain had become constant. My bouts of diverticulitis were unbearable. I underwent a sigmoid colon resection for severe constipation and many years of diverticulitis attacks. This proved to be a very difficult operation for me to recuperate from. My surgeon remarked that the portion of colon she removed was like cement. The lumen was greatly reduced. No wonder I had been in so much pain! It took me over six months to recuperate from the effects of the surgery. I cannot metabolize general

anesthesia. I developed adhesions. I was so fatigued for many months after this surgery that it was as if I were hung over, and I don't drink.

2011: I developed a severe sinus infection leading to a perforated eardrum. Before the deployment of the AMR meter, I rarely got sick from contagious diseases. I had never had an ear infection before in my entire life. Immune system disorders are common among those who are affected by EMF.

27. Q. What happened to your state of health in 2012? Did your health improve following the colon resection recuperation?

A. To the contrary. I did not improve. Although my abdomen was not in dire pain all the time, presumably because the lumen of my colon had been enlarged by cutting out the most diseased section of my colon, I still experienced abdominal pain from moderate constipation a few times per week. But my leg pain had become more and more severe over time, starting in 2008. By the spring of 2012, I was unable to walk more than a few blocks because of the pain. I was not able to ride my horses without extreme effort because of the leg pain. It took me hours to feed my horses in the morning, having to sit down for frequent breaks, whereas before deployment of the AMR meter, I could accomplish all my morning horse duties in 40 minutes. EMF can fan the flames of inflammation, and I was going down the inflammation path steadily and surely ever since the deployment of the AMR meter on my house.

The worse of this is, the sicker I became from the AMR meter, the more I was forced to remain at home in bed, instead of spending my days largely outside, enjoying the countryside and my horses. And it turns out, the headboard of my bed sits directly above the AMR meter, spewing its poison on my head all the while I was lying down, trying to recuperate!

In the fall of 2012, I was first diagnosed with lipedema Stage II. While I was born with lipedema, which is a rare adipose tissue disorder which is somehow related to Ehlers Danlos Syndrome, the only manifestations of lipedema I had suffered from before 2008 was heavy thighs. My healthy lifestyle had kept my inflammation at a minimum level, and I was only Stage I well into my fifties. But by the fall of 2012, with the onslaught of constant EMF for ten years, my disease had progressed into Stage II, or, the peas in a bag stage, when macrophages have infiltrated the abnormal fat layer and the fat has already hypertrophied. I became more and more disabled due to constant leg pain. I could no longer walk more than a half a block without searing pain. I had to lie down for at least an hour after rising from a prone position for the pain to dissipate. As soon as I sat in a chair with my legs down, or stood up, the pain returned.

In previous years, I had undergone many months of manual lymphatic drainage (MLD) and wrapping on my legs, so that I looked like the Michelin tire woman. MLD was performed by Vodder trained PTs, with no lasting results. I had had many veins in my legs closed or ablated. This only served to increase my leg pain.

I started to research why my legs hurt so much, why my legs bruised easily, why my legs could not be touched without pain, why they continued to get fatter and fatter despite a very healthy diet, plenty of exercise before the pain became unbearable, and what treatments were available.

I had already incorporated all the conservative lipedema treatments into my life: MLD, wrapping, dietary changes, specific targeted supplements, medical grade support hose, epsom salts baths, whole body vibration machine, and sequential pneumatic pump for my legs and abdomen. My therapies alone took up at least four hours a day, and yet my lipedema symptoms continued to increase in intensity and severity.

28. Q. What surgical interventions did you undergo in 2013 and why?

A. 2013: In February 2013, I had to undergo pelvic organ prolapse repair surgery, both anterior and posterior, despite my possessing excellent muscle tone in that area, due to my bladder sinking from its moorings and my descending colon pushing into my pelvic area. This required an overnight stay in the hospital and many months of recuperation. This, too, was a complicated and protracted surgery from which to recuperate.

Because I was lying in bed most of the time, trying to get better, I was having great difficulty answering emails or getting on line, because sitting in front of the computer was painful. My children purchased an iPad for me so I could send and answer emails from my prone position. The iPad required a Wi-Fi to operate. My computer had been hard wired and so had my husband's, so

there was no need for a Wi-Fi except to operate my iPad. I had no idea then that my head was being flooded constantly with EMF from the AMR meter as I lay in bed for hours and hours, and I had no idea that the new gadget my children had purchased for me was also radiating me with EMF. So, I was unwittingly getting sicker and sicker because of low level EMF emitted by the AMR meter for nine years, and now an iPad and Wi-Fi router necessitated by the increasingly diseased state I was in due to the AMR meter effects as the single most contributing factors in my disease.

2013: By the summer of 2013, my lipedema pain had become unbearable if I had my legs in any position but prone. **I had became a complete invalid.** I had to lie down all day long or I experienced severe pain. It appeared that my condition was hopeless.

As I lay in bed, or in my flexitouch machine in the den, I continued to Google lipedema. I found newly started facebook groups for those suffering from lipedema. I was the 100th member of the USA group. I also joined a British lipedema facebook group. Lipedema is an inherited, autosomal dominant adipose tissue disorder that is rarely diagnosed; it affects women almost 100 percent of the time, not men, and its cause and genetic underpinnings are not clearly understood yet.

Lipedema aggravation seems to be triggered by hormonal changes, stress, and abdominal surgeries. Lipedema etiology seems to arise with leaky capillaries and leaky lymphatics causing a huge amount of inflammation, and fat

hypertrophy, which is followed by macrophage infiltration, and stagnation of the lymphatic system, which had been working before the lipedema fat overwhelmed the lymphatic system. Which causes yet more inflammation.

29. Q. What physicians are researching lipedema?

A. Dr. Karen Herbst, an endocrinologist at the University of Arizona, is the world's leading expert in rare adipose tissue disorders, including lipedema. Her videos on the subject of lipedema are the best resource for patients to learn about the disease. I watched all of them. I researched constantly, reading all the medical literature and watching all the lipedema videos I could find.

30. Q. Did you discover physicians who could offer hope for your lipedema pain in your research?

A. Yes.

31. Q. Where were they located?

A. I discovered that German doctors had been successfully treating lipedema patients with liposuction for over twenty years. But in 2013, there was no one in the US who treated lipedema surgically. Rare diseases get short shrift in the US, especially those for whom there is no pharmaceutical treatment. I corresponded with the top five German doctors and also a British doctor who had not treated lipedema patients, but at least he spoke English. Among the German doctors I corresponded with, was Dr. Josef Stutz, who had invented

water jet assisted liposuction (WAL) for the treatment of lipedema patients. Although WAL was FDA approved in the US, it was used in the United States for cosmetic surgery only, and no one in the United States was trained in treating lipedema patients surgically in 2013. I even drove to the nearest cosmetic surgeon I could find who used WAL and asked him if he would operate on my legs. He declined, saying he was not qualified to do the surgery, and I would be better off going to Germany.

32. Q. When did you first travel to Germany and for what reason?

A. I arranged with Dr. Stutz to operate on my legs in June and July 2013. With much apprehension, my husband and I left for Germany in June 2013; I was in so much pain I could not stand in line at the airport to check in. I had to have wheelchair assistance to go through security and to get to the plane and off. On the three hour trip from the airport in Frankfurt to the hotel we were staying in, I kept my feet on the dashboard to try to get some relief from the horrendous leg pain I was suffering from due to the flight.

I underwent three WAL lymph sparing liposuction surgeries in Germany by Dr. Stutz to remove lipedema fat: two in June and July 2013 on my legs, three weeks apart, and one on my arms in the fall of 2013. Each leg surgery lasted three hours. My arm surgery did not take as much time, only an hour and a half. (My arms, which had only fairly recently begun to grow lipedema fat, had doubled in size between the summer and the fall, due to the arm lipedema fat growing intensely after the lipedema fat had been removed from

my legs.). PECO may obtain all pertinent medical records from all my health care providers who treated me for each and every one of these ailments, pursuant to PECO's Interrogatory Set II, after PECO executes a Protective Agreement to safeguard my private healthcare information.

33. Q. **How have you used your own experiences with lipedema to help others who are afflicted with the disease?**

A. I have helped other lipedema patients in two respects. I used social media to get the word out that lipedema pain can be successfully mitigated through lymph sparing liposuction, and that US insurance companies can be educated to pay for these surgeries which are not cosmetic at all, even though the US CPT codes for liposuction trigger automatic categorizing of the surgeries as cosmetic and therefore not payable by insurance.

I posted on the American and British lipedema facebook groups in the summer of 2013, about the success of my surgeries with Dr. Stutz, that he had taken away my lipedema pain, *and I was able to walk two miles, twice a day, which was nothing short of a miracle, post surgery.*

Dr. Stutz has focused his practice on treating lipedema patients for the past 24 years, and patients travel to him to be operated on from around the world. I was his second American patient, but as the word has spread through social media, more and more English speaking lipedema patients are traveling to Germany for liposuction, as well as now, traveling to the US and British surgeons whom Dr. Stutz has trained. Several American lippy patients who

saw my Facebook posts or met me in person in Tucson in 2013 (see below) later traveled to Germany to be operated on by Dr. Stutz. Upon my entreaty in 2013, Dr. Stutz agreed to travel to the US the following summer (2014) to teach American doctors how to operate on lipedema patients. I am very gratified that there are now five or six surgeons in the US who operate on lipedema patients; these experienced surgeons were first identified by lippy patients as candidates for training by Dr. Stutz, and were subsequently trained by Dr. Stutz.

Secondly, after my success at getting Blue Cross Blue Shield to pay me back for my surgeries in December of 2013 (which required two levels of appeals, a 200 page brief and extensive research of the medical literature), many US patients are getting insurance precertification for payment of their liposuction surgeries. I have shared my reasoning, my research and my brief with all lipedema patients who ask for it, and followers in my path have added more recent research to the mix in getting insurance approval.

In fact, despite my worsening health in 2014 following an automobile accident (discussed below), I rallied my strength momentarily because I was invited to speak on methods to obtain insurance coverage for lipedema treatments at the Washington DC lipedema conference held in September 2014. The conference speakers' presentations were videotaped, and a video of my presentation is available at: http://www.fatdisorders.org/fat-disorders/videos. It was quite an honor to be invited to speak along with world renowned lipedema luminaries such as Dr. Stutz and Dr. Karen Herbst.

The support groups for lipedema patients in the US have mushroomed on facebook since 2013, and there are probably nine or ten of them now, with focuses on education, exercise, surgery, and many splinter subgroups.

34. Q. Why did you travel to Arizona in 2013?

A. I was able to travel to Tucson, Arizona on my own in October 2013, to meet with Dr. Karen Herbst and attend the largest ever lipedema conference in the world held to date--80 lipedema patients were in attendance. Dr. Herbst confirmed my diagnosis of lipedema stage 2, a diagnosis which Dr. Stutz had diagnosed me with a few months previously when I met him in person for my first WAL surgery. (PECO may obtain all pertinent medical records from all my health care providers who treated me for each and every one of these ailments, pursuant to PECO's Interrogatory Set II, after PECO executes a Protective Agreement to safeguard my private healthcare information)

35. Q. So, after your liposuction surgeries, describe your state of health. Were you able to return immediately to your former state of health before the AMR meter was placed on your home?

A. I traveled to Germany by myself in November 2013 for Dr. Stutz to remove the lipedema fat from my arms. I was able to resume horseback riding and gardening. And I was able to entertain friends again. I was still rather weak, but I was out of lipedema pain. I still had to undergo lymphatic drainage therapy two or three times per week and I was doing my utmost to allow my body to heal.

36. Q. What additional medical procedures did you have to undergo in 2013?

A. Dr. Stutz had advised me to seek treatment from a vascular surgeon in the US to close a perforator vein in my right calf which had been bothering me with heat and inflammation ever since I broke my leg. Dr. Stutz explained that the lipedema fat on my calf had obscured the perforator vein as the cause of the inflammation.

The US vascular surgeon I consulted did not close the perforator vein, but instead performed additional vein closure surgery bilateral legs due to venous insufficiency. Again, I will furnish PECO medical records of this surgery pursuant to PECO's interrogatory Set II upon PECO's signature to the confidentiality and non disclosure agreement.

In the spring of 2014, I returned to Germany for Dr. Stutz to close my perforator vein. After a relatively short healing period, that area of my body has never bothered me since.

37. Q. What happened in your life in early 2014 which was beyond your control, which increased your oxidative stress levels greatly?

A. In the spring of 2014, while I was crossing an intersection on a country road, driving to my PT appointment, my car was broadsided by a car which was driving full speed through a red light. This motor vehicle accident caused post concussion syndrome and multiple areas of torn ligaments and tendons and other soft tissue, for which I am still undergoing treatment. I have not

progressed at all in the areas of cognitive functioning in the last year, according to my speech therapist at Bryn Mawr Rehabilitation Post Concussion Neuro Unit I have also undergone many PRP and stem cell treatments to repair damage to my soft tissue. Because I have Ehlers Danlos Syndrome and am elderly, I am not a candidate for joint replacement. Autologous PRP and stem cell treatment in the areas that have been injured are the only possible treatment which can offer me hope of recovery of the soft tissue injury caused and exacerbated by the accident.

My neck was hurting so much that I was unable to bend down to pick up anything from the floor for over a year. I could not turn my neck to drive safely. My back was in spasm for many months. I had difficulty in carrying anything heavier than five pounds. I could no longer entertain friends or go out to events with my husband. I was diagnosed with post concussion syndrome following the MVA. I continue to suffer from forward and sideways sheering forces in the concussion. I suffer from ongoing brain dysfunctional issues from the MVA, despite multiple treatments and therapies, and despite the passage of more than two years since the accident. I started with therapy at the Bryn Mawr Rehab Post Concussion Syndrome Neuro treatment center in January 2015, but I had to interrupt treatment at Bryn Mawr Rehab due to life threatening developments. I restarted treatment at Bryn Mawr Rehab this year.

My life had once more, after a very brief reprieve following my lipedema surgeries, been severely circumscribed and restricted. I became unable to

cook and clean up the kitchen. I was unable to go with John to visit friends. I was unable to engage in leisure activities in general, and I have been relegated to lying on the sofa and watching Netflix. (Thank goodness for Netflix).

I have been unable to travel to see my grandchildren by myself. And I certainly have been in no shape to garden. We have had to hire a full time gardener to take care of the garden. And since last fall, we have had to hire caregivers to come and cook dinner and clean up several days a week.

38. Q. Please describe your post concussion syndrome symptoms.

A. My post concussive symptoms I have suffered from are surprisingly similar to many symptoms suffered by those who are injured by EMFs.

They include: easy fatigue and lethargy, mental confusion and fog when information is fed at a normal rate, difficulty in paying attention to tasks, constant headache, pain on entering a brightly lit room especially one with bright lights in the ceiling or highly contrasting light and dark environments, irritability on the slightest provocation with minimal anger or aggression control, easy frustration, and other similar symptoms.

Perhaps most cruel of all, I can no longer take care of my horses. I rarely even venture outside. I have had to hire a full time caregiver for my horses.

It breaks my heart to be so disabled, and I miss spending time with my horses, even though their paddock and large field are right in front of my home.

PECO will be afforded an opportunity to examine my records for post accident treatment pursuant to PECO's interrogatory Set II, upon execution of the confidentiality and non disclosure agreement.

39. Q. What is your progress and prognosis to achieving a full recovery? What are your feelings as to the AMR effects on your lack of healing?

A. Since I have made very little progress in resolving most of injuries post accident, (the sole exception being that of the twisted out of place neck facets caused by the MVA, which plagued me for over a year, being successfully realigned by my PT after months of adjustments twice per week) despite massive amounts of treatment, there is little doubt in my mind, since I have researched possible causes of my lack of healing, and since I received PECO's answers to my Interrogatory Set I, and since I have purchased an accurate EMF meter and performed my own measurements on the EMF emitted by the AMR meter, that my lack of progress with healing completely from the MVA is almost solely due to the AMR meter effects on me.

40. Q. What additional body dysfunction did you experience as time went on and you had not healed from the MVA?

A. As my fatigue post motor vehicle accident increased, Dr. Prociuk increased my thyroid replacement hormone because my endocrine system was malfunctioning again, after it had been fairly stable for many years.

I was also found to have developed a new symptom: anemia.

Dr. Prociuk ordered a fecal occult blood test in late 2014 which turned up positive. He, therefore, referred me for an endoscopy and colonoscopy, even though I had been given a clean bill of health as to polyps upon colonoscopic examination as recently as 2011. The January 2015 colonoscopy revealed a very large (5 cm) villous adenomous sessile colon polyp. I was fortunate that I did not have to undergo the second colon resection which my gastroenterologist urged me to undergo, and the polyp in my ascending colon was successfully removed via endomucosal resection in two surgeries eight months apart at the University of Pennsylvania. Pathology revealed many areas of high dysplasia, formerly called carcinoma in situ. PECO will be afforded an opportunity to examine my records for these treatments pursuant to PECO's interrogatory Set II, upon execution of the confidentiality and non disclosure agreement

41. Q. What threats did PECO pose to you while you were trying to recuperate from your first EMR procedure in March 2015?

A. It was while I was lying flat on my back resting at home following my first EMR procedure in March 2015 that PECO sent me a 10 day shut off notice for refusing to allow PECO to install an AMI meter on my home. See Appendix B. *This, while I was in dire pain from ongoing colon spasms, and I pled that I was disabled and could not move.* I had not researched smart meters before this time, but I had read of several houses burning down soon after smart meters were installed on residences, and all I could picture was a smart meter being put on the my house, the house catching on fire, and I was

unable to move to rescue myself, my two dogs, the housecat, or any of our possessions. Because we live out in the country, there are no fire hydrants available. My house would surely burn to the ground with me and my dogs and cat in it if a smart meter sparked and caught the house on fire. The PECO representatives I spoke with were "just following orders". Everyone got a smart meter, no matter how sick they were, or they would have their electricity shut off.

I was horrified and dumbfounded by PECO's Gestapo actions. They reminded me of the Nazis rounding up all the Jews, the priests, the sick and the disabled, and throwing them into death work camps.

The PECO ten day shut off notice said if I had a doctor's note, they would not shut off my service during the duration of my illness. Dr. Prociuk wrote a letter on my behalf, and faxed it to PECO. See, Appendix G. PECO replied to me that the stay of shut off was only for customers who did not pay their bills, not for those who were up to date with their payments, and my only option was to file a formal complaint with the PUC.

Again, I was shocked to learn that I, an elderly disabled customer who has been faithfully paying my full bill from PECO every month for over 40 years, got less deference in terms of electricity shut off than a scofflaw who did not pay her bill.

PECO cared not that I lived on a farm and I and my husband and horses and dogs and cats would have no water if the electricity were shut off. PECO

cared not that I could barely walk due to disability. PECO cared not that without electricity I would have no refrigeration or heat or air conditioning. PECO cared not that I lived in an all electric house and there is no natural gas line in our area of Chester County. PECO cared not that without electricity, I would have no access to the internet with which to download the complaint form to the PUC, and I would have no means to answer all correspondence, or research all cases that PECO threw at me as obstacles, just to try to regain my health. PECO cared not that I and my husband were dependent on electronic medical equipment. PECO cared not one iota about me. PECO only cared to execute what it felt was its God given mandate to force smart meters on all its customer service area, regardless of the health of its customers.

I was forced to file a Complaint with the PUC after pleading for help with every elected official for my district, to no avail.

My filling out a simple form that I found on the PUC website to keep my electricity on, led to this hearing for which I am writing this prefiled testimony, and led to the amicus briefs I filed for the Susan Kreider Complaint proceedings, because, although each case must be judged on its own merits, Susan Kreider is also disabled, and she suffered horrendous health deterioration from the smart meter PECO slapped on her home.

And because, it appeared that PECO and the PUC were lumping together all the customer complaints relating to deleterious health effects from smart meters, and treating them as if they were one and the same: dismissing them

on PECO motions on the pleadings, and not allowing them to go to full blown discovery and hearing as to the deleterious health effects of smart meters, since there was no opt in Pennsylvania, and customers who were harmed by smart meters should go to the House of Representatives to get a legislative solution to their ill health effects from smart meters.

42. Q. Were you then able to rest and recuperate from your EMR and return to activities of daily living?

I was not. Unfortunately. I continued to be increasingly disabled, if that were possible. Because of my continuing colon spasm that left me unable to eat any solid food post EMR, I was referred to a new gastroenterologist who prescribed that I undergo pelvic and abdominal MRIs. These studies did not reveal the cause of my colon spasms, but they did reveal an endometrial growth, which had to be surgically removed. Yet another surgery and another overnight in the hospital in the early summer of 2015.

43. Q. What further medical issues occurred in the fall of 2015?

A. I scheduled to return to the University of Pennsylvania for my six month follow up regarding the sessile polyp removal. The procedure itself went uneventfully. However, I had an allergic reaction to the feather pillows in the hotel I stayed in the night before for my colonoscopy prep, which quickly developed into sinus congestion leading to middle ear vestibular problems, dizziness, headaches, vertigo and fever. The fever persists still today.

It was during this time that I was diagnosed with malabsorption of Vitamin D, B vitamins, and iron deficiency anemia, despite oral supplementation. Genetic testing revealed genetic problems with ONOO cycles in which Dr. Pall is an expert. He has submitted his expert opinion regarding my genetic issues with inflammation and oxidative stress. See, Murphy St. 1. Blood work and urine analysis revealed many methylation issues despite massive supplementation efforts.

44. Q. How about 2016?

I underwent a series of five weekly iron infusions in January and February 2016, to address my iron deficiency anemia. Recent blood work indicates that proper iron levels have been restored. I have follow ups scheduled every three months now with a hematologist to make sure I am treated promptly if my iron levels start to sink again.

In March 2016, I underwent surgery to repair a 5 cm ventral hernia which had been caused by a poorly executed colonoscopy in January of the previous year which did not consider my Ehlers Danlos or previous colon resection muscle wall weakness. It had taken my almost a year to locate a surgeon who was willing to operate on me without mesh and without general anesthesia except for propofal. Unfortunately, the anesthesia team administered a drug called versed, a contraindication for me which I had previously advised the surgical and anesthesia team about. I had a long lasting adverse reaction to the versed because of my genetic defects.

45. Q. How are you feeling now, as of this writing?

I am feeling quite fatigued, and weak. In addition to all the symptoms I have described above, I have been experiencing trouble falling to sleep at night and staying asleep for the last several years. I often wake up early with heart palpitations and high blood pressure.

I long for the days when I no longer feel ill and can enjoy life once again.

Before I started to investigate PECO's AMR meter and PECO's AMI meter EMF emissions and their effects in depth, I had become very depressed. Just when my life seemed to be turning around with relief from lipedema pain after my WAL surgeries in Germany in 2013, the MVA sent me back down to the abyss; even farther, because now my cognitive functioning had become impaired, as well as my body. I could not imagine ever climbing out of this hell hole that my physical and mental life had become.

But, ironically, PECO's Gestapo tactics had forced me to investigate the effects of EMF on my health. I now have some hope for the first time in two years, that I can recover my health back, with the removal of all EMF from my home environment.

IV. REMEDIATION AND RELIEF

46. Q. What steps have you gone to eliminate EMF in your home environment after PECO sent you its Interrogatory Set I and answered your Interrogatory Set I?

I had not realized that much of my inability to lead a normal life in the last 14 years had been caused, in great part, by the interaction of EMF on my overly delicate and sensitive electrochemical body systems.

Although I am aware that my own created EMF exposures for a few hours a day pale in comparison to the EMF exposures emitted by both the AMR and the AMI meters constantly without stop, I have eliminated all sources of EMF that belonged to me in my home. My husband never owned a cell phone, so that was not a source of EMF. The iPad which my children had given me in 2013 to help me get on line when I was unable to sit at my computer was giving off EMF, and I was not previously aware of that. I gave it away. My husband's and my computers, along with their respective printers were always hard wired, so they were not sources of EMF. I had a friend convert our Wi-Fi to a modem, and I purchased a tablet which is hard wired for use on the first floor. I keep my cell phone in airplane mode and only check it a few times a day. I only use it in case of emergency when I am away from home and have to deal with doctors and scheduling issues.

I purchased, for $300.00, an HF 35C high frequency EMF monitoring meter, manufactured in German, which is the recommended meter for monitoring

smart meter emissions. See Appendix H, for my purchased monitoring meter specifications. I have checked every room in the house, along with the immediate vicinity of my house, all around. The gigahertz meter antenna senses those EMF pulses all through the same side of my house where the AMR meter is located, and it's very upsetting for me to learn what has been going on in my home for the last 14 years without my knowledge. I no longer can stay in the living room at all, because a great deal of EMF comes through the wall and the window which is next to the AMR meter.

I have to avoid the master bedroom which I had furnished and designed with so much care, too, at all times as are practical, because it is directly above the living room and the AMR meter, and the EMF readings coming though the wall and into the bedroom, especially as the EMF comes in through the bedroom window are very high.

There are only three bedrooms in the house. The bedroom with the lowest levels of EMF is the guest bedroom with readings below 1.0, unless the AMR meter is emitting, in which case the readings spike up over 2000 microwatts per meter squared. The guest bedroom sits as far back from the AMR meter as the house extends on that wall. There are no bedrooms in the house which are located on the opposite side of the house from the AMR meter, which undoubtedly would be safer for me. I have been forced to sleep in the guest bedroom, as it is safest for me. Actually, the safest place in the whole house is in the basement, with an EMF reading of 0, but it does not have any

bathroom, or heat, and navigating the stairs has become difficult for me, in any case.

47. Q. **What relief would you like the Commission to grant in your case, given Pennsylvania Act 129 and Section 1501 of the Public Utility Code constraints?**

1. I would like the Commission to recognize that I have rare genetic defects which require my body to be removed from dirty electricity, non linear electrical devices, low level bursts of EMF and other environmental toxins.

2. I would like the Commission to recognize that I have suffered enough over the last 14 years, and that I deserve to have my home serve as a safe haven for me to enjoy the rest of my life free from EMF which has prevented me from healing, and which has contributed mightily to my accelerated nosedive into poor health and inability to heal from inflammation and oxidative stress.

3. I would like the Commission to grant me an accomodation under the Americans with Disabilities Act and/ or alternatively under Section 1501 of the Public Utilities Act from deployment of any EMF emitting device by any utility which emits anywhere on my property.

4. I would like the Commission to direct PECO to request an accomodation under the Americans with Disabilities Act from forced deployment of any EMF emitting device which radiates any EMF onto any of my property.

5. I would like the Commission to direct PECO to explore alternative methods of complying with Act 129 in my case while still delivering safe, consistent and healthy electrical power to me, be it via fiber optics, wired communications, or via an analog meter.

6. I would like the Commission to understand that the AMR meter and the AMI meter cannot be rendered safe when deployed using wireless technology in my case.

7. I would like the Commission to understand that the intricacies of the technology contained in both the AMR meter which has been deployed on my home for 14 years, and the AMI meter which PECO is attempting to deploy universally can have serious negative health consequences in certain highly sensitive individuals.

This technology includes, but is not limited to:

(1) the Switched Mode Power Supply contained in all digital meters supplied by PECO which acts like a dimmer switch on the whole house wiring at all times;

(2) the meters themselves and their continual flooding of the customer's property with EMF, which cannot be deactivated by the customer even if the customer turns off all the power to her property

(3) The AMI meters do not contain surge arrestors, to prevent arcing and sparking, whereas the analog meters do contain surge arrestors. PECO expert

Mr. Pritchard informed the Commission in the Kreider hearing that analog meters had not been available since the 1970s, (Appendix D, Kreider Tr. at 107), but I have been recently informed that analog meters are now being manufactured again due to high demand.

(4) the zigbee whole house Wi-Fi: I would like the judges to appreciate the lack of logic and humanity involved in the proposed administration by PECO in its AMI meter deployment of constant and unremitting torture to a sensitive customer to be flooding that customer with EMF through the use of the zigbee radio whole house Wi-Fi, which emits EMF at least every thirty seconds day and night, looking for a link up with a Smart appliance. I believe that this is especially inhumane and violative of Section 1501 when the customer does not possess any Smart appliances because the customer cannot tolerate non linear electrical currents in her home;

(5) I would like the Commission to understand that merely locating the customer's meter socket elsewhere on the property does not solve the EMF problems nor does it solve the SMPS dirty electricity problems.

(6) I would like the Commission to require that PECO cease and desist from all attempts to install or maintain any wireless device on my property.

(7) I believe that the Commission has the power to request accommodation for sensitive individuals in compliance with the mandates of Section 1501 of the Public Utilities Act to modify the PUC implementing regulations of Act 129 to permit (1) PECO to grant an accomodation from certain Act 129

mandates in my case, or (2) allow me to obtain my electrical distribution services at normal customer rates from a supplier other than PECO which is not constrained to supply its customers with smart meters of any kind.

48. Q. Does that conclude your direct testimony?

A. Yes. This concludes my written direct testimony. Thank you.

APPENDIX A

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-1: With regard to the AMR meter currently installed at the Murphy residence ("Murphy AMR Meter"),

a. When was the Murphy AMR Meter installed?
b. What is the make, model and specifications of the Murphy AMR Meter?
c. How many times per day does it transmit remotely?
d. How long is each transmission?
e. How far does it transmit remotely?
f. What are its peak emissions?
g. Does it transmit EMF to the electric wires inside the Murphy household?

PECO Answer to Murphy I-1:

a. May 8, 2002
b. Siemens/L+G/Duncan electro-mechanical meter with a Landis + Gyr AMR Communications Transmit Only Meter Module (TOMM)
c. 288 times per day for its scheduled transmissions (every 5 minutes)
d. 20 milliseconds. Total daily on-air time transmitting is 5.76 seconds
e. The expected useful distance is less than 1 mile
f. 1 watt or 33 dBM
g. No transmissions are made to communicate with facilities, devices, or wires inside the household

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-2: Does the Murphy AMR Meter transmit to each of the Murphy electrically powered devices, such as the HVAC system, computers, lamps, refrigerator, etc. through the airwaves or through the household wiring?

PECO Answer to Murphy I-2:

The Murphy AMR meter does not transmit to electrically powered devices within the residence.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-3: Are the transmissions from the Murphy AMR Meter sent wirelessly or via wire?

PECO Answer to Murphy I-3:

The transmissions from the Murphy AMR meter are sent wirelessly.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-4: How often does PECO collect data emitted from the Murphy AMR Meter?

PECO Answer to Murphy I-4:

Data is transmitted from the Murphy AMR meter every five minutes. The data is compiled at an intermediate facility and then delivered to PECO electronically once a day.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-5: How is the Murphy AMR Meter usage data read by PECO

a. For billing purposes?
b. For outage purposes?
c. For any other purpose?
d. Name any and all other purposes.

PECO Answer to Murphy I-5:

a. Yes, the AMR meter usage data read by PECO is used for billing purposes
b. Yes, the AMR meter usage data read by PECO is used for outage purposes
c. Yes, the AMR meter usage data read by PECO is used for other purposes
d. Primarily theft detection and related analytics.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-6: When was the last time the Murphy AMR Meter was checked by PECO on site?

PECO Answer to Murphy I-6:

The Murphy AMR meter was tested for accuracy on April 15, 2002, prior to installation. PECO has not identified any onsite checks.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-7: For what purpose was the Murphy AMR Meter checked on site by PECO?

PECO Answer to Murphy I-7:

For accuracy. See Answer to Murphy I-6.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-8: Is the Murphy AMR Meter able to be reprogrammed? If yes, what possible changes can be made to the Murphy AMR Meter programming as to its emissions?

PECO Answer to Murphy I-8:

No. It is not possible to reprogram the Murphy AMR meter to change the number of transmissions made by its communication module.

Responsible Witness: Glenn Pritchard

APPENDIX B

(AVISO DE SUSPENSION DE SERVICIO EN 10 DIAS)

Account Number: 2346901005
For Service To: 1191 TELEGRAPH RD WEST CHESTER PA 19380
Date Prepared: March 20, 2015

Past Due Amount: $_____
New Billing: $_____
Total Amount: $_____

Your Electric/Natural Gas Service May Be Shut Off!

Your electric/natural gas service will be shut off to 1191 TELEGRAPH RD WEST CHESTER PA 19380 **on or after** April 3, 2015 **because:**

_____ You have a past due amount of $_____ as of _____.

__X__ You did not give us access to our meter and your equipment.

_____ You did not pay your security deposit.

_____ You did not meet the requirements and/or complete the application for utility service.

_____ Other:_____.

We will **NOT** shut off your electric/natural gas service if you:

_____ Pay $_____ in full on or before _____, this includes any amount you owe on your payment plan. This notice is effective for 60 days.

_____ Pay the required security deposit of _____.

_____ Pay the catch up amount on your agreement if it is defaulted. Call 1-888-480-1533 for the amount.

_____ Show us a paid receipt for the past due amount.

__X__ Provide us access to our meter and your equipment. Call 215-841-5950

You may be eligible for a payment agreement or special assistance programs. Call 1-888-480-1533 right away if you dispute your bill or to provide us with household income and occupant information. To talk about your bill, please call our office at 1-888-480-1533.

WE MUST RECEIVE YOUR PAYMENT, ACCESS OR INFORMATION BEFORE THE SHUT-OFF DATE. WE WILL NOT ACCEPT PAYMENTS AT YOUR PROPERTY.

If we shut off your electric/natural gas service, you may have to pay all of the following before we can turn service on:

- Past Due Amount of _____
- Deposit Past Due Amount of _____
- Agreement Unbilled Balance of _____
- Reconnection Charge of _____
- Total _____

*If your service is shut off, you may be required to pay any additional bills that have become past due to restore your service.

**If your service is shut off, you may have to make substantial payments to have your service restored. In addition to any balance owed, you will have to pay a Reconnection Charge of between $20.00 and $1,700.00. This fee amount is set by PECO's tariff and based on how much work is needed to restore your service. You may also be required to pay a deposit equal to two times your average monthly usage.

MEDICAL EMERGENCY NOTICE

Let us know if you or anyone presently and normally living in your home is SERIOUSLY ILL. WE WILL NOT SHUT OFF YOUR SERVICE during such an illness provided you:

1. Have your licensed physician or nurse practitioner certify by phone and in writing that such an illness exists and that it may be aggravated if your service is shut off, phone certification must be followed by written certification within 7 days.

'AND'

2. Make arrangements to pay this bill. You must provide us with household income and occupant information to determine your payment terms while protected under the medical certification.

IMPORTANT TO KNOW

Before we shut off your utility service please read the back of this notice. You may be eligible for certain protections from shut off.

Attencion ! Este es en mensaje muy importante. Si usted no lo entiende, favor de llama a 1-888-480-1533

Send payment in the enclosed envelope or pay your bill at an authorized payment location or PECO's Main Office (23rd & Market Streets Philadelphia). To pay by credit card or check by phone, call 1-877-432-9384. The service provider will charge a convenience fee of $2.35

See other side for more information.

Carol Smith (215-841-4545)

APPENDIX C

ROBERT W. GODSHALL, MEMBER
150 MAIN CAPITOL BUILDING
PO BOX 202053
HARRISBURG, PENNSYLVANIA 17120-2053
PHONE: (717) 783-6428
FAX: (717) 787-7424

DISTRICT OFFICE:
1702 COWPATH ROAD
HATFIELD, PENNSYLVANIA 19440
PHONE: (215) 368-3500
FAX: (215) 361-4220
E-mail: rgodshal@pahousegop.com

House of Representatives
Commonwealth of Pennsylvania
Harrisburg

COMMITTEES

CONSUMER AFFAIRS, CHAIRMAN
INSURANCE
LEGISLATIVE BUDGET & FINANCE

October 7, 2015

Gladys M. Brown, Chairman
PA Public Utility Commission
Commonwealth Keystone Building
400 North Street
Harrisburg, PA 17120

Re: Susan Kreider v. PECO Energy Company
PUC Docket No.: C-2015-2469655 and P-2015-2495064

Dear Chairman Brown,

I am extremely troubled by the Commission's disposition of the above referenced matter as illustrated by its September 3, 2015 Order. I have communicated the same to the Commission's Director of Legislative Affairs. Upon reading all of the documents filed at these dockets and available on the Commission's website, I am particularly concerned about the statements indicating that Ms. Kreider took it upon herself to have her company installed meter removed and replaced with a different meter. If this is correct and Ms. Kreider continues to receive electric service through a rogue meter her actions jeopardize not only her personal safety but the safety and reliability of the electric distribution grid.

I'm also concerned the Order seems to open the door to considerations of fact that could create an exemption to the provisions of Act 129 of 2008. Act 129 requires electric distribution companies to deploy smart meter technology throughout their service territories. The law does not provide any circumstances under which such technology may not be deployed. PECO's deployment of meters is being conducted pursuant to its Commission approved Smart Meter Plan and various related Commission Orders. Similar to Act 129, these Commission Orders do not contain any exceptions to deployment or create circumstances under which a statutorily mandated meter may not be deployed. The law is clear and unambiguous; electric distribution companies, including PECO, must deploy smart meter technology throughout their service territories. There is no set of factual circumstances that could be presented to the Commission that would authorize it to hold that PECO is relieved of its legal obligation to deploy these meters or permitting a customer to choose not to have a meter installed at her residence. Quite simply the Commission cannot grant the relief requested by Ms. Kreider in her complaint, namely that she be permitted to have a different meter installed at her residence.

The Commission's September 3, 2015 Order opens the door for a company, acting in compliance with a legal mandate, to be found to be violating the reasonable service standard in the Public Utility Code solely for acting in accordance with the law. I'm not a lawyer but this holding is inconceivable to me. I fail to understand how compliance with one law can amount to a violation of another. Such a holding seems to be inconsistent with basic principles of statutory interpretation.

I encourage the Commission to carefully review the provisions of Act 129 when considering PECO's Petition for Reconsideration and take steps to ensure that the ruling of the Commission is consistent with the law and the regulatory authority granted by the General Assembly

Sincerely,

Bob

Robert W. Godshall, Majority Chairman
House Consumer Affairs Committee

RWG:jh

cc: John F. Coleman, Jr., Vice Chairman
Pamela A. Witmer, Commissioner
Robert F. Powelson, Commission
Andrew G. Place, Commissioner
June Perry, Director, Legislative Affairs

APPENDIX D

COMMONWEALTH OF PENNSYLVANIA
PUBLIC UTILITY COMMISSION

SUSAN KREIDER V. PECO POWER	:
COMPANY	:
Initial Hearing. Various	:Docket No. C-2015-2469655
Disputes	:

Pages 1 through 217

Hearing Room 4125
4th Floor
801 Market Street
Philadelphia, Pennsylvania

Monday; March 7, 2016

Met, pursuant to notice, at 10:00 a.m.

BEFORE:

DARLENE D. HEEP, Administrative Law Judge
CHRISTOPHER PELL, Administrative Law Judge

APPEARANCES:

SUSAN KREIDER
169 West Queen Lane
Philadelphia, Pennsylvania 19144
(Pro se)

WARD SMITH, Esquire
SHAWANE LEE, Esquire
2301 Market Street
Philadelphia, Pennsylvania 19103
(For the Respondent/PECO)

APPEARANCES (Continued):

TOM WATSON, Esquire
2442 Massachusetts Avenue NW
Washington, DC 20008
(For the Respondent/PECO)

Q. Okay. What do you do when you get the meters to ensure -- we'll just talk about accuracy.

What do you do to ensure that those are safe meters?

A. Similarly, safety is part of the testing protocol by understanding how the meter interacts with the system and then the accuracy of that meter itself both combined.

Q. Are these meters tested for compliance within underwriters laboratory standards?

A. Yes, they are.

Q. And has this meter met that compliance?

A. Which meter?

Q. I'm sorry, the AMI meter?

A. The AMI meter that PECO is using, yes, have -- are compliant.

Q. Let's talk briefly about Ms. Kreider's meter.

A. Yes.

Q. It was previously identified as a Sangamo meter. Are you familiar with that brand meter?

A. Yes, the Sangamo brand was manufactured for many years, but last manufactured in 1975.

Q. 1975. The meter that we were shown earlier had another label on it that said Hialeah Meters.

Are you familiar with Hialeah Meters?

A. Not specifically.

means.

JUDGE HEEP: There's also been some reference to magnetic fields, EMS.

Are those the same, and if they're not, how are they different?

THE WITNESS: I would believe they are relatively the same, but that goes beyond my expertise.

JUDGE HEEP: If Ms. Kreider's meter is relocated would she have to pay for that?

THE WITNESS: She would have to pay to relocate her socket itself. PECO would install the meter wherever that socket may be.

JUDGE HEEP: Is it possible to -- if the meter is located on someone's home and there's come concern regarding the health effects, is it possible for PECO to provide some sort of encasement or shield around the meter?

THE WITNESS: We have not done so, no.

JUDGE HEEP: Do you know -- given your background and expertise in this, are you familiar with that possibility?

THE WITNESS: The meter itself is designed to transmit the radio signals outside of the home and not inside, so a shield may not have any effect.

JUDGE HEEP: Referring to PECO Exhibit GP-3, the Tariff, 14.1.

A. They follow the ICNIRP recommendations.
Q. Okay. And how about New Zealand?
A. New Zealand are the same and Canada.
Q. Okay. Let's just -- make a little change in shift and ask you a question that sort of brings into play here your knowledge of biophysics and biophysics research combined with your knowledge of electromagnetics.

Is there a mechanism by which radio frequency fields can somehow damage the human body?
A. Yes, there is.
Q. What is that?
A. There's one mechanism that's generally accepted, heating, but high levels of RF, you can heat tissue to even burn it if the levels are high enough.
Q. Okay. And can you tell us how high that has to be?
A. Well the maximum permissible exposure that's set by the FCC is set to be a factor of 50 below the lowest level that's ever been detected by an animal as minor heating. So there's a very large safety factor in the FCC maximum permissible exposure.
Q. So you're saying this standard on -- over here on PECO Exhibit CD-1, the standard is -- maximum permissible exposure is 0.6 milliwatts per square centimeter?
A. Yes.

Q. And can you just explain again what the safety factor of 50 how that plays into that limit?
A. Well basically if you take the .6 and you multiply by 50, okay, then you get, you know, 30 milliwatts -- sorry, three milliwatts per square centimeter and -- I'm sorry, 30 milliwatts per square centimeter and that's the lowest level of which the animals have been observed to detect that they're feeling a little bit warm in a radio frequency field.
Q. Okay. So what you're saying is, once they found that number at 30, then they apply the 50 safety factor or AMIs dropped it by 50 times?
A. Correct.
Q. Now, you know you hear talk about various other proposed mechanisms people write about things, "Oh, we think it's this, we think it's that."

Are you familiar with all the research on mechanisms by -- people studying whether there are mechanisms by which radio frequency fields could cause harm to the human body?
A. I'm extremely familiar with it.
Q. Okay. Are you -- other than getting so much EMR that you start to heat somebody up, are there any other mechanisms that are recognized that could cause -- as a means for causing harm to the human body from exposure to

RF?

A. I've been working in this field since 1977 and I started out looking for mechanisms, and in my long career, along with most other scientists in this business, we've concluded that there is no other plausible mechanism, but we've looked very hard, but other mechanisms have been discounted, but there's no evidence for them.

Q. Okay. Can AMI -- can the AMI meters that PECO uses produce enough heat to damage tissues?

A. No.

Q. Thank you.

MR. WATSON: Your Honor, that's all the questions I have on direct. I would like to turn the witness over for cross-examination.

JUDGE HEEP: Ms. Kreider.

CROSS-EXAMINATION

BY MS. KREIDER:

Q. Mr. Watson, you're not a medical doctor? You're here as an expert witness?

A. Actually, I'm Davis not Watson, Ms. Kreider.

Q. I beg your pardon. Christopher Davis. I beg your pardon. You're not here as an expert witness who is a medical doctor?

A. No, I'm not.

Q. You said that you have a lot of knowledge on

dosimetry and brought up the subject of specific absorption rate. Could you explain what that is?

A. Yes. Specific absorption rate is another measure of exposure that's used for what's called near field exposure. In other words, when you're very, very close to the source such as putting a cell phone in your ear, in that case, the classification of the exposure is done through this thing called a specific absorption rate and it has an FCC guideline -- well stated maximum. You cannot be exposed to more than 1.6 watts per kilogram averaged over one cubic centimeter in your body.

Q. And isn't it true that babies and individuals have different specific absorption rates that some people are more vulnerable, like babies to Wifi?

A. Well again --

Q. Electromagnetic fields?

A. Again, there's no firm consensus as to whether children experience higher or lower -- it depends on the placement of the phone and other factors. Nobody is acknowledging that these are harmful levels. That's why we have the safety guidelines.

Q. But isn't it true that the FCC safety guidelines only recognize thermal effects of radiation and do not recognize non-thermal effects of radiation?

A. That's absolutely not correct. The safety

guidelines recognize the risk of exposure without specifying exactly what that exposure causes. The only mechanism that's been accepted is the thermal mechanism. There's no acceptance of these so-called thermal mechanisms because there's no mechanism for them to occur.

Q. So are you familiar with the May 2015 petition by -- it's now over 220 independent scientists from 40 different countries to the United Nations saying that these guidelines are unacceptable and these scientists -- never mind. I'm going to testify.

Are you familiar with that petition?

A. I've heard about it.

Q. Okay. So it is a real thing. Can you explain what ELF, Extreme Low Frequency Radiation, where that falls on the spectrum? Would that be a microwave for example?

A. No.

Q. Okay.

A. ELF refers to Extremely Low Frequencies.

Q. Right.

A. Typically the 60 hertz that our power generation system distributes its electricity around the country.

Q. And isn't it true that there are some concerns about ELFs as being -- causing harm?

A. There's still some controversy about whether there is a very, very tiny risk to ELF fields.

APPENDIX E

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-53. What kinds of EMF filters does PECO provide its customers from harmful EMF and harmonics caused by the AMI Smart Meters it deploys? Are any such filters available from any source, such as the filters which are available for computer cords, which filter electromagnetic static?

PECO Answer to Murphy I-53:

PECO does not accept the premise of the question that its AMI meters cause harmful EMF and harmonics. Given that, PECO does not provide any filters referred to in this question, and is not familiar with the marketplace, if any, for such filters.

Responsible Witness: Glenn Pritchard

APPENDIX F

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy 1-10: Does the Murphy Intended Smart Meter differ from the Smart Meters that PECO has installed in its other customers' residences?

PECO Answer to Murphy I-10:

PECO's residential customers receive a Landis+Gyr Focus AX-SDR meter, unless the house has unusual wiring requirements or the location of the house causes unusual communications issues for the meter. PECO has reviewed the AMI meters installed in the Murphy's neighborhood; none of the houses have unusual wiring requirements or communications issues; all have Landis+Gyr Focus AX-SDR meter.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-11: What is the mechanism by which the Murphy Intended Smart Meter communicates with PECO?

PECO Answer to Murphy I-11:

A wireless radio frequency communications module in the Murphy Intended Smart Meter.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-12: What makes and models of wireless communications modules are in the Murphy intended Smart Meter?

PECO Answer to Murphy I-12:

A Sensus FlexNet Type 51 (Sensus Model 560 Xz) communication module. In addition, the meter assembly contains a Zigbee radio that is capable of communicating with certain devices within the residence.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-13: Do the wireless communication modules in the Murphy Intended Smart Meter differ from those PECO is installing for its other customers?

PECO Answer to Murphy I-13:

See Answers to Murphy I-10.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-14: With regard to the Murphy Intended Smart Meter,

a. What is the instantaneous peak radiofrequency/microwave power output of the wireless transmitter in the Wireless Smart Meter as presently programmed, and
b. What is the instantaneous peak radiofrequency/microwave power output possible to be programmed remotely?
c. How long does each of the peaks last?

PECO Answer to Murphy I-14:

a. The instantaneous peak radio frequency power output of the wireless transmitter in the FlexNet Communication modules is 2 watts/30dBm. The peak radio frequency power output for the ZigBee Radio is 130.92 milliwatts/20dBm.
b. The instantaneous peak power output cannot be reprogrammed remotely.
c. Each transmission of the FlexNet module is 70 milliseconds in duration. Each transmission of the Zigbee radio is 0.7 milliseconds in duration.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-15: With regard to the Murphy Intended Smart Meter,

a. What is the gain of the antenna(s) of the meter in the direction of maximum gain, at each of the frequencies of current operation and
b. What is the gain of the antenna(s) of the meter in the direction of maximum gain, at each of the possible maximum frequencies of operation?

PECO Answer to Murphy I-15:

a and b: At all frequencies of operation, the gain of the antenna on the FlexNet Communication module is 0 dBi, and the gain on the ZigBee radio antenna is -6dBi.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-16: With regard to the Murphy Intended Smart Meter, what is the total number of transmissions of radiofrequency/microwave radiation per day:

a. as currently programmed and
b. as could possibly be programmed for any purpose, both on average and at a maximum, at their respective frequency of operation?

PECO Answer to Murphy I-16:

a. The FlexNet communication module is programmed by the manufacturer to transmit once every ninety minutes. The Zigbee is programmed to transmit once every thirty seconds until it pairs with an enabled device.

b. Once installed, the frequency of transmission from the FlexNet communication module will be remotely tuned to reflect how well the specific module communicates with the nearest Tower Gateway Basestation. Theoretically, the module can be tuned to transmit as frequently as once every 15 minutes or as seldom as once every 240 minutes. The system average tuning to date is approximately once every 145 minutes, or just under 10 (9.63) times a day on average. However, PECO has reviewed the communication tuning of the installed FlexNet modules in the immediate neighborhood of the Murphy residence, and they have been tuned to 240 minutes (6 times a day). PECO therefore expects that the Murphy Intended Smart Meter will be tuned to transmit once each 240 minutes (6 times a day).

   The ZigBee radio is currently programmed to transmit once every thirty seconds (2880 times per day) until it is paired to any known device(s) in the household. Under the existing programming, once the Zigbee radio is paired to any known device(s) in the household, the duty cycle will decrease.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-17: With regard to the Murphy Intended Smart Meter, what is

(a) the current average length of transmission time per day and
(b) the maximum total transmission time per day possible for any purpose from the meter's respective frequency of operation?

PECO Answer to Murphy I-17:

(a) For the FlexNet Module, the current average length of transmission time per day is 0.7 seconds (10 transmissions at 70 milliseconds per transmission). For the ZigBee radio, the current average length of transmission time per day is 2.016 seconds (2880 transmissions at 0.7 milliseconds per transmission).
(b) The maximum total transmission time per day of the Flex Net Module is 6.72 seconds per day (96 transmissions at 70 milliseconds per transmission). However, at the expected tuning of the Murphy meter, the maximum total transmission time per day will be 0.42 seconds (6 transmissions at 70 milliseconds per transmission). The maximum total transmission time per day for the Zigbee radio is the programmed level described in subpart (a) to this answer; the actual transmission time will be less once the Zigbee radio is paired with a device.

PECO notes that, during outages, events of remote connect/disconnect, or other occasional events, there may be short periods during which the communication module will make additional transmissions for the duration of those occasional events.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to
Interrogatories Complainant Laura
Sunstein Murphy, Set I

Murphy I-18: With regard to the Murphy Intended Smart Meter please describe:

(a) the pulsed EMF emitted in isolation for each pulsation, and in interval,
(b) the amplitude and duration of EMF emissions,
(c) how many pulsed EMF emissions the meter currently emits,
(d) and how many pulsed EMF emissions the meter capable of emitting.

PECO Answer to Murphy I-18:

(a) to (d): The term "pulsed EMF" is not defined in this question. As PECO understands the term, it refers to systems in which, during a given transmission interval, data is transmitted by rapidly turning the system on and off within the duration of that transmission interval. This is not how the PECO AMI system operates. In the PECO AMI system, when the communication module switches on to transmission mode, it remains on for the entire 70 millisecond transmission cycle; there is no on/off pulsing during the transmission. The data is transmitted by use of phase or frequency changes of the radiofrequencies – effectively, by transmitting a collection of sine waves.

Responsible Witness: Glenn Pritchard and Dr. Christopher Davis

PECO Energy Company's Answers to
Interrogatories Complainant Laura
Sunstein Murphy, Set I

Murphy I-19: With regard to the Murphy Intended Smart Meter, what are the peak electric field pulses which can be produced by the meter in the direction of maximum gain, and at what distance from the meter?

PECO Answer to Murphy I-19:

See Answer to Murphy I-18.

Responsible Witness: Glenn Pritchard and Dr. Christopher Davis

APPENDIX G

Peter J. Prociuk, M.D.
322 North High Street,
West Chester, PA 19380
Tel: 610-701-5702 Fax: 610-701-4225
www.drpeterprociuk.com
peterprociukmd@gmail.com

March 27, 2015

PECO
Brenda Eison, Supervisor
2301 Market St.
Philadelphia, PA 19103

Re: Acct # 234-690-1005
1191 Telegraph Rd., West Chester, PA 19382
Medical Necessity To Keep Electricity On

Dear Ms. Eison,

I understand that PECO will shut off electricity on April 3, 2015 unless a new smart meter is installed on the above mentioned property, to which Laura and John Murphy, the owners, are opposed. I have no interest in the outcome of this dispute but it is an unequivocal medical necessity for electric service to be maintained without interruption.

Both the owners are elderly and in fragile health for a number of reasons which is documented extensively by a number of health care providers. Their water comes from a well and is dependent on electricity. Any unecessary or prolonged interruption in electric service would seriously jeopardize their health.

Your attention, understanding and courtesy in resolving this important health issue is greatly appreciated.

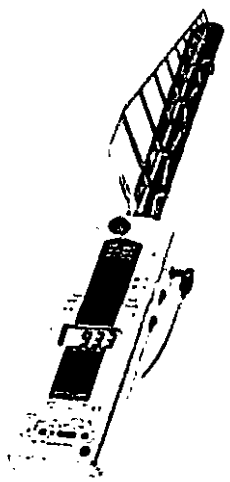
Sincerely,

Peter J Prociuk MD

APPENDIX H

HF 35C high frequency EMF monitoring meter

Calibrated, Directional, Accurate, Sensitive
This is the Recommend Meter for Checking "Smart Meter" Emissions



We like this calibrated wideband **(800 MHz – 2.5 GHz)** RF meter because it has a switch which allows a choice of peak or average indication mode. This permits accurate measurement of both digital and analog signals. Two ranges: 1 - 1999 $\mu W/m^2$ (same as **0.1 – 199.9 nW/cm²**) and 0.1 - 199.9 $\mu W/m^2$ (same as **0.01 – 19.99 nW/cm²**).

In addition, you get a choice of audio tone – none, or intensity proportional to field strength, or proportional to frequency (for analyzing pulsed signals)
- accuracy ±6 dB
- true logarithmic - periodic antenna, 800 MHz – 2.5 GHz, single polarized, included
- standard 9V battery included

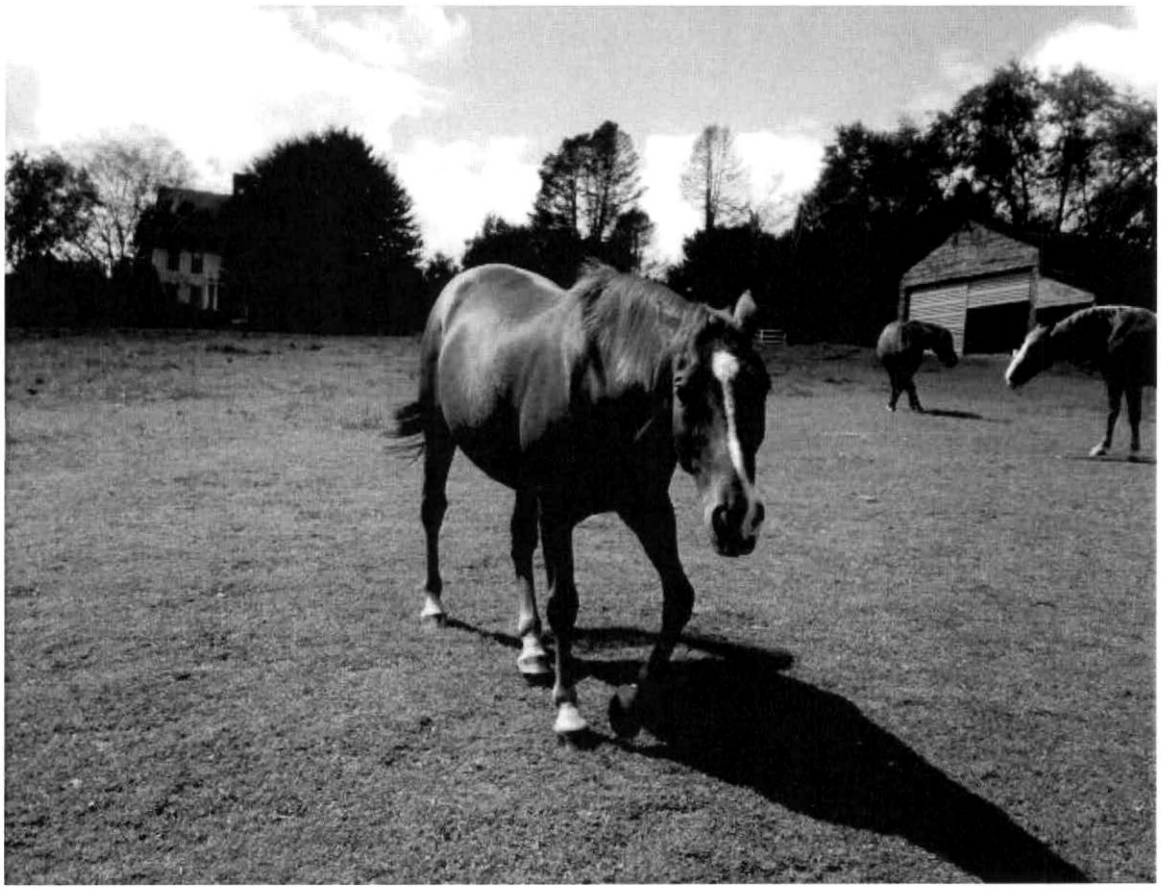
The display on the HF35C updates every 0.5 second. The fastest pulse it can detect is 0.5 micro seconds.
When in peak mode, the display averages all peaks within ½ of a second. The average peak is displayed on the display.
In RMS mode it displays the true average of all signals during that ½ second period. This reading is much lower than Peak mode when measuring a digital signal.

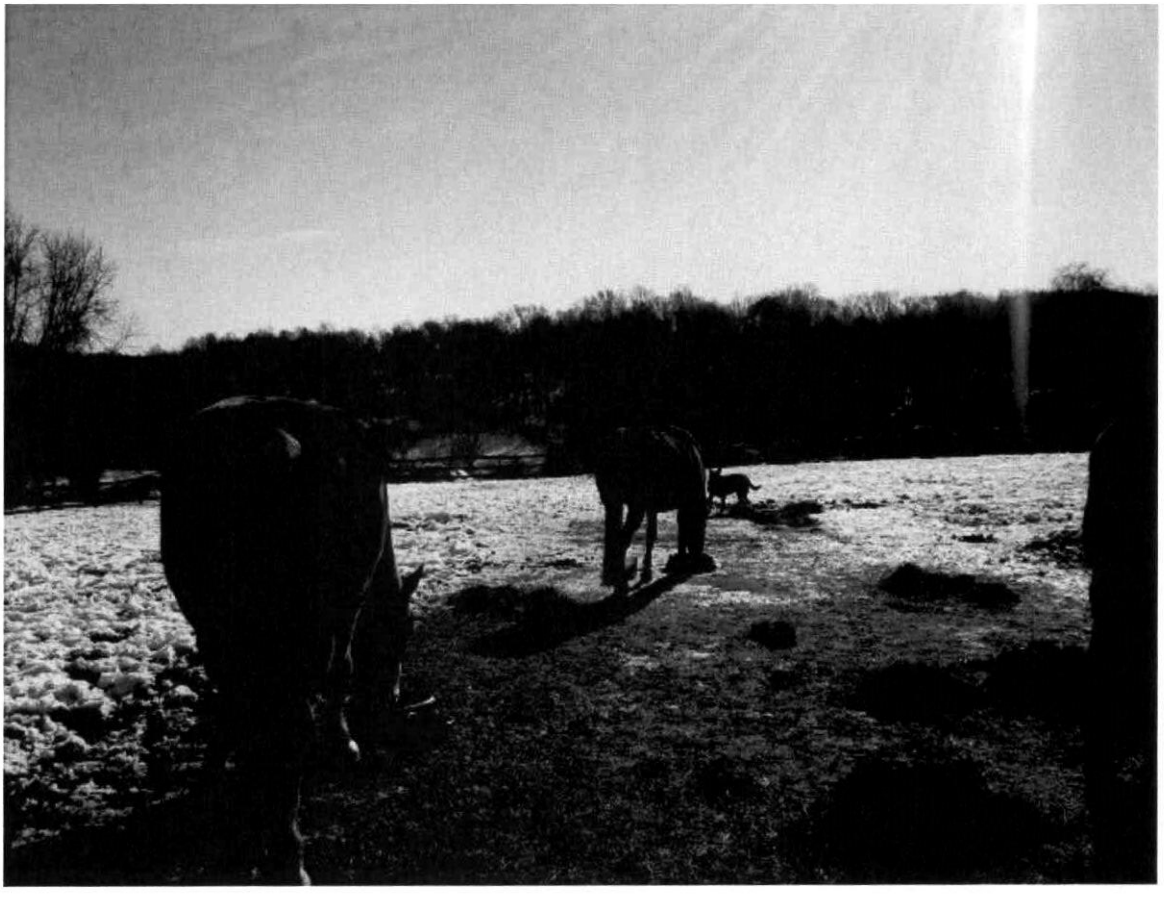
Easily detects cell phone towers, cordless phones even microwave oven leaks. Instructions in German and English. The 3 minute YouTube video at right demonstrates how the sounds from this meter help distinguish different sources of RF (video sound starts after 19 seconds). 2 year Warranty. Model HF35C.

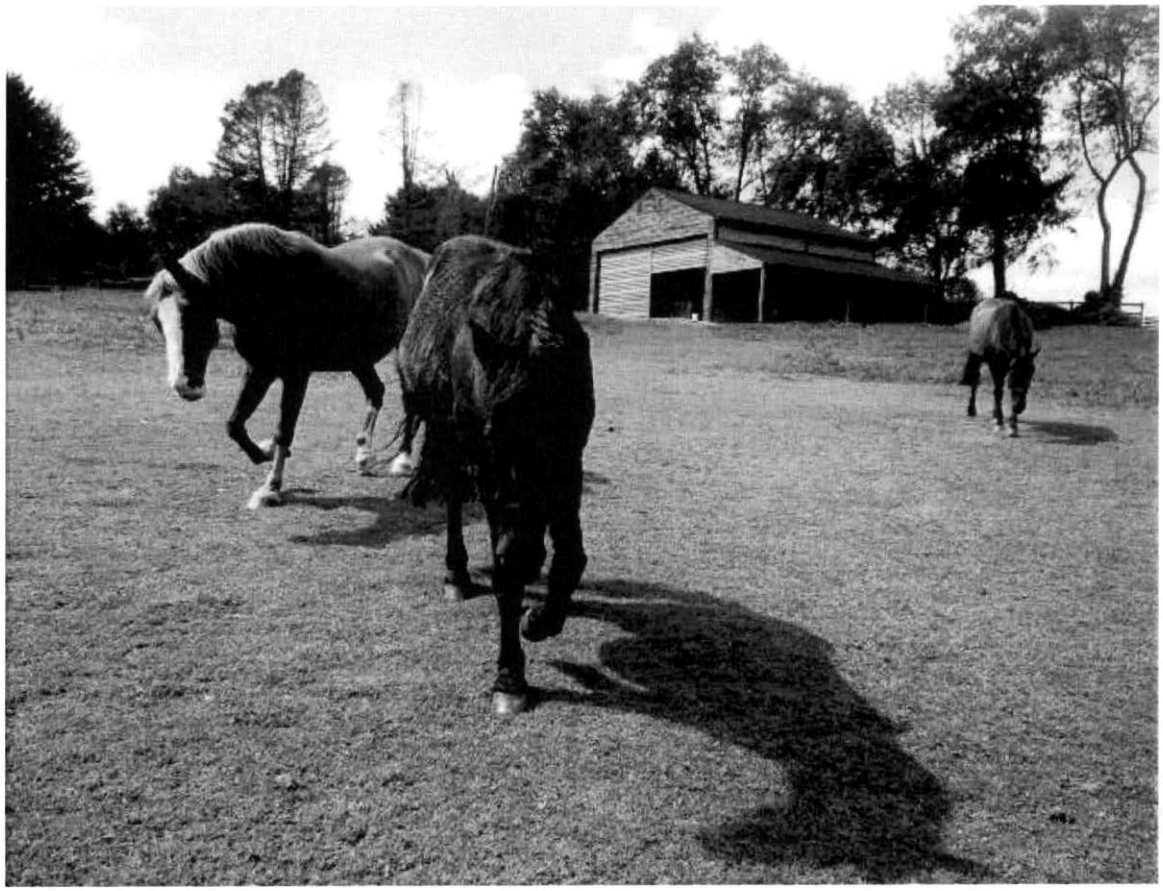
To view the short demonstration video, differentiating the differing audio sounds from various sources of EMF as displayed on the HF 35C, paste this link in your
browser: lessemf.com/rf.html

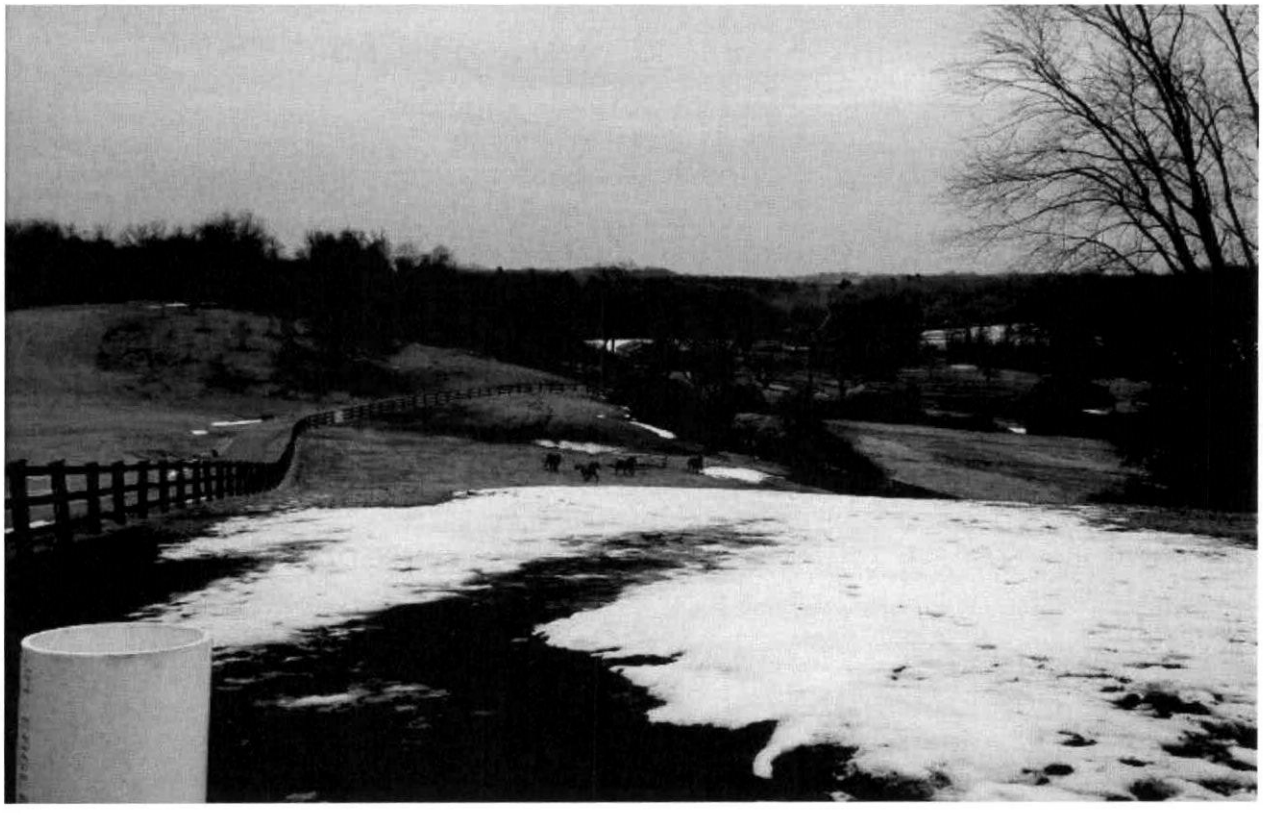
APPENDIX I

Pictures of the Murphy farm and some of my animals









MURPHY STATEMENT NO. 2S

C-2015-2475726
12/5/16 PLI TX

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

Laura Sunstein Murphy	:	
	:	
v.	:	Docket No. C-2015-2475726
	:	
PECO Energy Company	:	

SURREBUTTAL TESTIMONY
OF COMPLAINANT
LAURA SUNSTEIN MURPHY

June 3, 2016

SURREBUTTAL TESTIMONY
OF
LAURA SUNSTEIN MURPHY

1. Q. Please state your full name.

A. My name is Laura Sunstein Murphy.

2. Q. Have you submitted prior testimony in this case?

A. Yes, I have. I submitted Murphy Statement No. 2 on April 29, 2016.

3. Q. What is the purpose of your submission of Surrebuttal testimony?

A. The purpose of my testimony is to respond to the rebuttal testimony submitted by PECO witnesses on May 20, 2016. I respond to PECO's rebuttal testimony by offering evidence to contest and challenge the claims made by PECO's witnesses.

4. Q. Please tell us in a nutshell, what your Surrebuttal testimony is about.

A. My testimony in this Surrebuttal is about my health. Only about my health, which is the main issue in this case and the main substance of PECO's rebuttal. My testimony also deals with how pulsed EMF emitted by PECO's wireless meter on my home for the last 14 years has damaged my health, and my attempts to regain my health. I will mention certain inconsistencies in the PECO expert testimony, which pertain to the emissions of these wireless meters which have a direct effect on my health.

5. Q. Dr. Israel concludes that your health issues are not related to PECO's meters? What changes in your health have occurred since you filed your testimony in this case approximately one month ago?

A. My health has taken an astonishing turn for the better for the first time in over two years; in fact, for the first time since my lipedema surgeries in 2013.

6. Q. Please comment on what improvements you have noticed.

A. I will categorize my improvements into various somatic and mental systems:

Heart:

My heart rate is steady; it no longer races. I have had no incidents of atrial fibrillation. My heart rate only varies between 80 and 90 beats per minute after exercise. In prior months, I had recorded "resting" hearts rates often in the range of a 100 bpm (up to 121 bpm) and bp of 211/111; 200/114, and 179/102, with lows in the range of 87/57 and 96/54 and 109/61. My blood pressure no longer climbs above 145/87 and it doesn't fall below 114/68 when I first wake up. Usually, it remains between 145/87 and 138/83, without wide fluctuations day to day.

Sleep:

I sleep beautifully through the night. I no longer wake up at 4 am or 5:00 AM with heart palpitations at all. I go to bed between 10:00 and 11:30 PM and fall asleep immediately; whereas in the past several years, I have been unable

to get to sleep until past 1:00 AM and at that, I had to take a 5 htp to even fall asleep in the first place.

I sleep peacefully and restfully all night and wake up between 7:00 and 8:00 AM, (although for a few days I slept until 10:00 AM).

Eyes:

My eyes are no longer as dry as they had been for some time. They are not tired at night.

Cognitive abilities:

I am not so easily tired or frustrated when I go to concussion therapy. I welcome the brain challenges which had been frustrating and difficult for me in the past. I have been able to successfully complete more than twice as many problems and puzzles than I had been able to complete a year ago, or even two months ago. I no longer have to stop the problems because my brain is too tired to go on.

Joints:

I am able to walk without my knee brace for the first time in over two years. I am able to walk far longer distances than I had been able to walk in over two years. My knees no longer hurt to stand up, initially, and I am able to stand for at least ten minutes without pain, whereas at the time I filed my initial testimony, I was unable to stand up for any length of time without knee

pain, and I was so weak from the effort, I quickly sought something--anything-- to sit down on. I am still holding on to my handicapped parking hangtag, but I can park farther away from my destination, and don't have to seek out the handicapped designated parking all the time anymore.

My lower back has begun to bother me, and I have returned to PT to focus on the lower back. My PT, who has been treating me twice a week for two and half years, with short times off for surgeries (the latest time off was for my hernia surgery on March 9, 2016), remarked how improved my knee function was last week when I first saw him. He mentioned that I had some psoas issues on one side which I did not have earlier, so he recommended home exercises to perform in between appointments. I was delighted that I have the energy to pursue a home exercise program, because in the past, I was simply too fatigued to exercise at home in between appointments.

Digestion:

I am adjusting to digestive issues. I have had so many digestive problems ever since the AMR meter was deployed on my home, that my digestion may be the last somatic issue to resolve in my case. However, I am less constipated, I have fewer digestive issues in general, after some initial gerd for a few days.

Mood:

I am no longer depressed over my health. I am elated to be on the road to good health again. I am happy that I will be able to travel to see my grandsons in California. Especially my grandson who was born over Thanksgiving last year, the one whom I have never held, and never smelled his sweet baby hair.

Headaches and heat in the forehead:

I don't suffer from constant headaches in general, as I did before, although I do still get them if I enter a room, like the PT studio, which has fluorescent lights. My forehead becomes cool for longer and longer periods, not every day, but often. I usually wake up with a cool forehead.

However, I did wake up early with a headache the morning of May 30th, caused, it turned out, by my sister who was visiting from California, who had left her cell phone (which did not have an airplane mode setting) on and plugged into an outlet in the wall, in another section of my home, but which was only separated from my headboard by two plasterboard walls and ten or so feet of airspace. I woke her up as soon as I discovered her cell offending phone, and made her move it out of my house, and I have not had a return of a headache since. My other siblings and their children who were visiting over the Memorial Day weekend had all put their cell phones in airplane mode at my request, so their cell phones were not an issue for me.

Energy levels and fatigue:

Virtually every day I feel more energetic. I have had a few days of backsliding, due to vertigo or back pain, but in general, I am beginning to feel like my old self again--that old self before the AMR meter and before my concussion. I can sit for hours without feeling the need to lie down. I can go up the stairs several times a day instead of coming downstairs for the entire day, as I had to do for the last two years. I no longer have to lean on the banister to go up and down steps. I no longer have to grab a grocery cart to lean on in the grocery store when I shop. I don't have to spend my days lying on the sofa, too weak to get up. I have been able to go shopping and run errands, many in one day, instead of limiting myself to one errand. Many people have commented that my voice on the phone has a different quality to it, that it is more vibrant.

I have been able to go outside to see my horses. I have been able to groom them. I have been able to go outside and hose them off when it is really hot. Last evening, I had enough strength to feed my horses and scoop horse poop for the first time since last summer.

Contrast in energy and sustainable exercise:

My very large family came in waves to visit over Memorial Day weekend, with second and third generations arriving, also.

This is the first time in at least 10 years that all my siblings have gotten together. It is the first time in three years that I have had enough energy to entertain any company in my home.

One of my brothers has lived in Nepal for the last 30 years, so his presence in the U.S. is quite sporadic. And my brother and his wife who live close to me in Chester County are moving to Ecuador at the end of the summer. Two of my brothers live in New England and my sister lives in California; some of the family lives in Seattle, and some live in the Midwest. My daughter and her family drove up from Western Virginia.

Each of my four brothers had made special trips to see me last November 2015, because I was so weak, it had appeared to them (and me) that I was in the process of dying.

They had remarked when I had spoken with them on the phone recently as we were firming up plans for the family reunion that I had new energy in my voice, but seeing me in person, being able to stand up, walk around, go up and down the stairs, cook and clean up for two days over Memorial Day, and I had enough energy to keep up with them all--this was an amazing turnaround. We had 18 people for lunch and dinner for two days straight.

I did tire and go to bed by 10 or 11 PM, while many of them stayed up till 1 AM.

I was delighted that I had enough energy to help my granddaughters brush my mustang mare down, pick her hooves out, and go for a ride on her in my little round pen ring, which was a highlight for the horse, me and my granddaughters.

Two of my brothers celebrated their birthdays at my home, including one brother who turned 70 on May 30th, and one who turned 72 on May 27th. It was a very joyous and memorable occasion.

7. Q. **Are you completely cured of your disabilities which you wrote about in your prior testimony?**

A. No, I am not completely cured by a long shot, but I am headed in the right direction to regain many of my former activities once more.

I still suffer from vertigo, especially when I lie down at night. I still do not have the energy to take care of or ride my horses the way I used to. I still have a stiff back. I still cannot stand for hours at a time. I still cannot lift heavy bales of hay, or anything heavier than 10 or 15 pounds, for that matter.

I still cannot stand for hours at a time to wash dishes and pans (much to my husband's chagrin).

I know that I never will regain good eyesight even with correction, in my right eye, which suffered a detached retina twice; because I constantly see a wiggly flume of grey smoke-like floaters that move around in the liquid in that eye all the time, punctuated with dots of many black floaters that look like gnats. I was told years ago by my retinal specialist that I would l have to get used to the floaters, because there is nothing that could be done about them.

I still am on a yearly colonoscopy follow up with an EMR specialist for my 5 cm highly dysplasic colon polyp in my ascending colon.

8. Q. **Dr. Israel testified that your health issues are not related to PECO's meters. What evidence do you have regarding possible PECO AMR meter EMF emissions effects on your health?**

A. There are new findings of the National Toxicology Program rodent study linking certain cancers to cell phone whole body radiation have finally been partially released, whether my developing a huge, highly dysplasic colon polyp in a short period of time and my development of an endometrial polyp that had to be removed last year could have been caused in large part by PECO's AMR meter deployment on my home. (See, http://biorxiv.org/content/biorxiv/early/2016/05/26/055699.full.pdf) and Appendix N for this study preliminary result.

9. Q. **What steps had you already taken before you made the ultimate change in your environment that brought about positive changes in your health?**

A. I had discussed my health issues with Dr. Martin Pall for him to write his expert testimony in my case, and he informed me that many of the conditions I had been suffering from were actually caused by EMF.

I was, quite frankly, astonished. I began investigating EMF in earnest. I had already eliminated my home wifi and my ipad. I already had ceased using my cell phone except for emergencies. I even considered asking my physician for

a prescription for a calcium channel blocker, since Dr. Pall's writings had indicated that calcium channel blockers halted the EMF induced voltage calcium regulator ill effects. I did research what CYP 450 mechanism the calcium channel blockers used, and found that two of them were metabolized in the body by CYP 450 pathways that I had genetic defects in, so a trial on a calcium channel blocker prescription was out, anyway, in my case.

I researched the best semi affordable meter to measure the smart meter and other EMF coming into my house and outside my house. This turned out to be the HF 35C Gigahertz meter, manufactured in Germany, which I purchased for $300 plus shipping. I checked all the areas of my home with that meter, and many areas outside my home. I found high readings on the side of my home which housed the AMR meter and fairly high readings in the front of my home. I stayed out of my bedroom except to grab some clothing to put on in the morning. I started to sleep in the farthest room away from the AMR meter. I bought EMF shielding clothing which I wore whenever I went out of the house, and even in the house in rooms that were in the front of and on one side of the house.

I lined the shutters in the master bedroom with aluminum foil. I bought very expensive EMF shielding fabric for sheer window curtains, and had curtains made for the windows in the front of the house and along one side of the house. I began shunning all public gatherings, unless I could get the cooperation of the attendees to put their cell phones in airplane mode.

I rarely went outside of the house. And yet, having taken all these steps, I was still very tired and I still suffered from a great many maladies, despite increases in my thyroid medication, despite vitamin D patches and vitamin B injections, and despite iron infusions. I had no energy to do anything. I was depressed. Who wouldn't be? There was nothing more I could do to save my health. Or was there?

I took a drastic step. This step was one I did not did not enter into lightly. I knew that, technically, to take this step I would be violating PECO's rules. As a lawyer and as a moral person, I abhor even the thought of violating a regulation.

But my health was deteriorating rapidly, despite being followed by so many health care professionals that the judges told me I had to limit the number of health records that PECO should request of my providers.

If Dr. Pall was correct, that EMF combined with my genetics were the root cause of my fatigue, my constipation, my endocrine dysfunction, my cataract formation, my heart arrhythmias, my high blood pressure, my osteoarthritis, my post concussion syndrome symptoms that I could not I heal from, then, I could not take a chance on further deterioration. I was still disabled. I could not stand up without knee pain. I could not climb the stairs more than once per day. I could not go outside to see my horses. I spent most of my days lying down on the sofa because I was so tired. I could not sleep well. I woke up with heart palpitations often.

10. Q. What ultimate change in your environment were you forced to take to protect your health, and was that the only change in your environment which brought an immediate noticeable difference in your health?

A. I reasoned that PECO was violating its tariffs as to me every single minute of the day for 14 years (albeit unwittingly so, to be generous to PECO, or recklessly so, to be more accurate, for the first 13 years until I notified PECO of my disability in March of 2015). And PECO assuredly had every opportunity to avoid further violations of PUC regulations as to me when I asserted in my testimony, and Dr. Pall asserted in his testimony in my case, that the AMR meter was having deleterious health effects on me.

PECO chose not only chose not to grant my requests for relief (1) under Section 1501 of the PA PUC law, and (2) under the ADA, but also PECO chose to further persecute me for my genetics, and the disabilities which were either directly caused by or exacerbated by PECO's AMR meter.

So, on advice from two of my physicians, I had an electrician remove the PECO AMR meter and replace it with an analog meter. I fully intended at the time of removal, if I did not appreciate any improvement in my health, to have an electrician remove the analog meter and return the AMR meter in its stead. I had no expectations either way. I merely observed.

11. Q. What did you do with the PECO AMR meter?

A. I have kept the AMR meter safely and will happily return it to PECO upon their request. I have not tampered with the AMR meter in any way.

12. Q. Do you intend to steal electricity from PECO or your energy supplier?

A. I have no such intention. As I suggested in prior testimony, as an accommodation to me, who cannot safely have PECO's suggested wireless AMI meter on my property, PECO can bill me based on my historic energy consumption on a yearly basis, or PECO can accept called in meter readings from me, or I can send meter readings to PECO via the internet at any interval PECO suggests. PECO is free to check on my analog meter readings at any time, just so long as when PECO comes onto my property, PECO refrains from deploying any other meter without my fully informed consent and knowledge as to its safety to me, a disabled customer pursuant to my rights under Section 1501 of the Public Utility Code Code.

13. Q. Why did you not have the AMR meter redeployed on your home?

A. I quickly found that it would be impossible for me to return the AMR meter on my socket and maintain the health gains that I appreciated in the short time that the AMR meter has been off my house. The analog meter deployment has brought me the relief from agony I had been seeking medical solutions for unsuccessfully for years. Dr. Pall was correct in all respects.

14. Q. How do you know that Dr. Pall was correct in all respects as to the AMR meter and your health?

A. The changes in my health after removal of the AMR meter were immediate and have continued almost steadily every day since. For the first two weeks I kept a daily log of my reactions to the removal of the AMR meter which I have attached to this testimony as Appendix J. After the first two weeks with the analog meter on my home, resulting in virtually no EMF in my home environment, I became so busy with activities during the day, and had so much energy in the evening, I no longer kept a daily log, but added to it every week. You can see from reading Appendix J, that there was one evening during the first week of having the AMR meter removed, that the AMR meter was redeployed, without my knowledge, and it was only after I felt the ill effects of the meter, that I investigated as to why I felt ill again, and discovered that the electrician had redeployed the AMR meter on my home. After he removed the AMR meter late that night, and reattached my analog meter, it took me quite a while to get over the effects that I had suffered from having the AMR meter on my house again, even if the AMR redeployment was only for a few hours. So, I had just confirmed by an involuntary double blinded experiment, that the AMR meter made me sick, and the removal of the AMR meter led to my feeling better, although it took some time to get over the ill effects of a few hours of having the AMR meter on my home.

15. Q. What do you have to say to Dr. Israel when he makes comments such as he made on pages 13 and 14 of his Rebuttal testimony, summarizing his

views on government studies from the UK, the Royal Society of Canada, the New Zealand Ministry of Health, the Scientific Committee on Emerging and Newly Identified Health Risks of the European Commission, as to what Dr. Israel terms "Idiopathic Environmental Intolerance"?

A. Dr. Israel has not lived in my body. He has never met me. He can cite all kinds of studies, but those studies have been nullified by my experiences and the experiences of many hundreds of people all over the world who cannot tolerate EMF the way others can apparently tolerate EMF. My deteriorating health conditions have been monitored and studied and treated by more than 35 healthcare professionals over the last 14 years. You do not need to take my word for it. PECO has been granted access to my health care records. Every single word of my previously filed testimony is true. I was severely disabled and I had been severely disabled especially since early 2014.

After removal of the AMR meter less than one month ago, my health has improved dramatically. Again, you do not need to take my word for it. My improvements have been noted by several of my health care providers already. They have been noted by friends and family. I know that I can never regain those 14 years of poor health. Everyone ages, and everyone's health tends to decline somewhat over the years. But PECO's wireless meter combined with my genetics accelerated the process tenfold, I am convinced. It was as if I had been afflicted by an even rarer genetic disease, called

progeria, which causes the body to age prematurely, resulting in an early death.

I have made considerable progress in just four weeks since I had the AMR meter removed. But I am still far from "cured" of the deleterious effects that the AMR meter had on my health. It will take more time for my body to achieve a new healthier homeostasis, since the noxious stimulus has been removed.

16. Q. Can this be just a coincidence? What is your response to Dr. Israel's statements on page 16, lines 4 to the end of page 17?

A. Dr. Israel's statements on those two pages, which can be summarized, "Ms. Murphy's symptoms were not caused by the AMR meter: they could be caused by any number of factors, including the nocebo effect: they could be caused because she sincerely believed that the AMR meter caused them [which makes absolutely no sense whatsoever to me, by the way, or to any intelligent reader] and just because the various deterioration symptoms she suffered from following the AMR meter deployment, that does not mean there can be attributed a cause and effect relationship to the AMR meter deployment and the somatic and cognitive problems Laura Murphy has developed."

I firmly deny that I was under some nocebo illusion, that I believed that the meter was causing me to get sick. To the contrary, for 14 years I thought the meter was not a contributing factor to my illnesses. I had no thoughts

whatsoever that the meter was emitting EMF until very recently. As I mentioned before, I didn't even know what EMF was.

I could not have accomplished all that I have accomplished in life so far, despite the presence of PECO's AMR meter on my home without a very strong drive to get well and stay healthy. No, I don't have an MD degree, and no, I am not an expert in electrical engineering, but I am an expert in one area: my experience of my body and mind.

I am an intelligent, well educated, introspective. and logical individual, and I research extensively. I do admit to some residual cognitive deficits from my post concussion syndrome, but many of these defects have been ameliorated considerably over the last month since removal of the AMR meter.

I have been on an almost constant quest to become healthier not coincidentally, it turns out, ever since the deployment of the AMR meter on my home. Little did I know then, the AMR meter was what was making me sick. Sicker and sicker. There was no escaping it. I had always eaten a healthy diet and exercised. But I modified my diet even more over time, and with the help of my doctors, to eliminate all refined sugar, all processed food, and virtually all non organic food. I started to take enormous numbers of vitamin and mineral supplements plus enzymes and other nutritional supplements to aid my body in getting the nutrients it could not get from my food. See Appendix M for a list of the supplements I had been taking as of March 9, 2016 in an attempt to function more like a normal person. I no

longer am forced to take this humungous list of supplements each day. I take only a handful, and the handful I take varies somewhat from day to day, depending on how I feel. For example, I used to have to take NAC twice a day, as a sort of natural antihistamine, because I would get stuffed up, I would get bumps just under my skin and I would feel congested. I now only have to take NAC once a day, or every other day.

17. Q. What are your thoughts as to why PECO has forced you to take drastic steps to get well?

A. As I mentioned in my earlier testimony, I have a strong belief in God. I kept asking God when PECO sent me a 10 day shut off notice last spring, as I was lying down on the sofa, trying to recuperate from my first EMR procedure, "Why me? Why doesn't PECO just leave me alone? The meter I have is fine. Please just make them leave me alone." But PECO would not leave me alone. PECO kept after me. PECO filed motions for summary judgment. PECO filed motions for judgment on the pleadings. PECO filed motion after motion after motion. "Why me? Why not leave me alone? I am sick. I am disabled. Please make them leave me alone."

Now I understand why PECO could not leave me alone. I had to find out why I had become disabled. If PECO had left me alone, I never would have discovered that my current AMR meter which had been on my home for 14 years was, indeed, not fine for me. It was extremely toxic to me. If PECO had left me alone, I never would have hired Dr. Pall as an expert, and I never

would have researched EMF. And I never would have found out what had rendered me a disabled person. Never. God was showing me the way, and it certainly was not comfortable for me being harassed by PECO, it certainly was not comfortable for me to file amicus briefs in the Kreider case, while I was extremely downtrodden, but it led me to the discovery that I had been zapped with PECO EMF for 14 years, and it led me to discover to the one expert who could explain to me why I was disabled, which led me to discover what I could do further to improve my health.

I can never be properly compensated for all my pain and suffering and all my expenses PECO caused me to incur. I can never regain those 14 years, much of them riddled with pain, deprivation of activities of daily living and operation after operation. But I can regain my health to a great extent in the future, despite my genetic abnormalities. And I look forward to being able to enjoy my golden years.

18. Q. Have you looked critically at and considered carefully the evidence presented by PECO's witnesses relating to the health effects of EMFs?

A. Yes. I have been forced by PECO to take a crash course in EMF. I didn't even know what EMF was last year. I don't believe everything that has been written about EMF, pro and con on their face. I look at research parameters and I try to understand those parameters. I realize that some folks think that EMF is the bane of all existence, and that we will wipe out all life forms with

EMF, just as in the early fifties, there were warnings that nuclear bombs would wipe out all living creatures on earth.

I grew up in the fifties. I remember air raids and darkened shades in the windows. I remember the Russian Sputnik launch and how the U.S. rushed to add science and mathematics curricula in the elementary schools because we were lagging behind the Russians in those areas.

And I have learned to look critically at industry pundits such as the PECO experts and the cell phone industry, who say that EMF cannot be harmful whatsoever to any living creature if it is below the FCC standards exposure levels.

So, I take a balanced viewpoint as to EMF, the great divide as to what research is credible, and what research is not credible or applicable to my situation as to wireless devices in my presence.

My father died from microwave exposure in his work with radar and other non ionizing radiation, which had led to his developing leukemia. (As an aside, one of my brothers brought some of my father's effects to the family reunion at my home last weekend. Included in those effects was his wallet, just as it had been left when he died in January 1978. I attach as Appendix O, a photo of my father's IEEE card from that time, indicating, as I had mentioned in my previous testimony, that he had been elected a Fellow of the IEEE (designated on his membership card under: "Grade" is marked with an "F" for Fellow. My brother who followed in my father's footsteps graduating

from MIT, and is likewise an electronics engineer and inventor remarked that being elected a Fellow in the IEEE at the time our dad was elected was quite an honor)).

It turns out, unbeknownst to me until I started a dialogue with Dr. Pall, that I happen to be extremely sensitive to EMF. This is borne out by the almost miraculous improvement in my health since I had the AMR meter taken off my home, which was flooding me 24/7 with short pulses of EMF, with varying emission frequencies, anywhere from less than one minute to up to six minutes between zaps. Whether or not objective tests show that I am EHS, does not matter to me. All that matters to me is that I regain my health. I am working diligently towards that goal.

19. Q. Mr. Pritchard testified that PECO's meters do not produce pulsed emissions. How do you know that the PECO AMR meter was zapping you with pulsed EMF 24/7 since 2002, with varying emission latencies, "anywhere from less than one minute to up to six minutes between zaps?"

A. First, a short history of how I first learned that my AMR meter was toxic.

I attended Susan Kreider's hearing on March 7, 2016. This was a very difficult hearing for me to attend for several reasons. (1) It was in the City of Philadelphia, which is at least an hour train ride from my local SEPTA station. (2) I was in a very debilitated state of health when I attended the Kreider hearing; (3) the room in which the Kreider hearing was held was

illuminated with fluorescent lights in the ceiling, which gave me an instantaneous headache which lasted for many days; (4) I suspect that the ambient EMF in the City of Philadelphia made me even sicker than I was, going into the hearing but I have no direct proof of that; (5) the few block walk from the Jefferson SEPTA station and to the hearing room and back was quite exhausting for me.

However, I learned quite a bit from attending Ms. Kreider's hearing. The most important piece of knowledge I picked up from the hearing was Mr. Pritchard's testimony at (Kreider TR 97, lns. 4-7), that the AMR meter that had been deployed on Ms. Kreider's home and on every PECO customer home prior to the deployment of the AMI meters was emitting radiofrequency wireless emissions every five minutes. This certainly was news to me. I followed up on that information, in my Interrogatory Set I at I-1 and I-6, asking specifically how often the AMR meter on my home was emitting, and when it was installed, and how often it had been serviced. Murphy St. 2, Appendix A. A PECO customer either takes PECO's word for how often the PECO meters are emitting EMF or the customer has to purchase an expensive Gigahertz meter and take her own measurements. As will be shown throughout my Surrebuttal testimony and throughout Dr. Pall's Surrebuttal testimony, there are so many inconsistencies in the PECO expert witness testimonies, it is necessary for the customer to take her own measurements of PECO meter emissions, which I did in April 2016.

PECO's expert Mr. Pritchard maintained in his answers to Murphy Interrogatory Set I-6, and in the Kreider v PECO case, page 97 LL 4-7 that the AMR meters such as the meter PECO installed on my home in 2002, emit wirelessly every five minutes.

I have no way of knowing whether the AMR meter that PECO slapped on my house in 2002 ever emitted every five minutes. PECO admitted that they never serviced my AMR meter in 14 years. Murphy Interrogatory Set I at I-6. Murphy St. 2, Appendix A. Obviously, PECO had no concern whether my meter was emitting wirelessly as frequently as every two seconds, because PECO never bothered to service my meter in 14 years. It was serving its purpose as to PECO. PECO had no concern whether it could be harming me; it fact, PECO continues to allege that non thermal EMF exposures below the FCC guideline limits cannot harm any individual, despite reams and reams of scientific evidence spanning over fifty years to the contrary.

After PECO forced my hand, I bought a Gigabyte meter to measure the EMF in my surroundings. I purchased, for $300.00, an HF 35C high frequency EMF monitoring meter (Murphy St. 2, Appendix H), is the literature describing my Gigabyte meter. I have attached as Appendix K, a listing of the pulsations of EMF emitted by my AMR meter during the short time that was filmed on April 16, 2016 at my home. I was too weak to stand up when the film was made; I had to sit down. I was too weak to announce clearly what had to be announced when the video was filmed; that is why Sal LaDuca made the introductory statements about my PECO AMR meter filming, and

also about the PECO AMI meter filming on a PECO customer's home that same day, which is also attached as Appendix K. Videos of my Gigabyte meter pointed (1) at my AMR meter (two videos) and later that same day, (2) pointed at a PECO AMI meter in situ on a PECO customer's home, both sets of videos evidencing wildly differing actual EMF pulsed emissions from what Dr. Davis and Mr. Pritchard admitted to. Take, for example, the longer video taken of my AMR meter, 10 minutes and 52 seconds long. Here are the time lapses between pulsed EMF emissions:

Pulsed EMF emissions from the Murphy AMR meter ---elapsed times:

13 seconds
1 minute 58 seconds
4 minutes 47 seconds
5 minutes 31 seconds
7 minutes 12 seconds
9 minutes 55 seconds

Anyone can hear and see the pulsations coming from my PECO AMR meter which registered on my Gigabyte meter in this video, and those pulsed EMF emissions are certainly not spaced every 5 minutes, as PECO experts have testified to many times.

20. Q. Do you have doubts about the credibility of PECO experts when they testified about the emissions of the AMI meter, as well?

A. Yes, I do. The PECO experts' testimony fluctuates wildly as to time intervals of emissions from the AMI meter, and my measured emissions from a PECO

AMI meter in situ show far more frequent emissions than even the shortest interval testimony of either Mr. Pritchard or Dr. Davis.

Mr. Pritchard testified in the Kreider hearing, (Kreider TR 101, ln. 24), that the PECO AMI meters transmit a minimum of 10 to a maximum of 96 times a day or every 15 minutes.

PECO's answers to the Murphy Interrogatory Set I at I-46, state that the AMI meters transmit only once every four hours for 70 milliseconds.

On page 140 lines 11 to 15, of the Kreider testimony, Mr. Pritchard stated the PECO AMI smart meter emissions are very low because it will "typically" emit only for .07 seconds ten times per day.

Obviously, the AMI meter I filmed was not emitting the "typical" amount. That PECO AMI meter was emitting more often than ten times every five minutes, not counting the FlexNet pulsed EMF, which was far more often than once every 90 minutes as demonstrated in my videos. So, either Mr. Pritchard is not telling the truth when he testified in the Kreider hearing, or the PECO AMI meter which I filmed is so far from "typical", that it is a monster meter and should be replaced.

And Dr. Davis stated in the Kreider hearing (Kreider TR 149 lns. 22 to 24) "no, because the AMI unit only turns on for less than a tenth of a second, so it's not exposing you all the time". And Dr. Davis testified in his Rebuttal testimony, Page 13, line 8, question 30, "do PECO AMI meters produce radio

frequency fields all the time? No, only when they are sending a radio signal". Does Dr. Davis understand that the PECO AMI meters are transmitting every 30 seconds or more? Twenty-four hours a day, 7 days a week, with no break for the homeowners to sleep or rest from EMF? I think not.

Elsewhere, PECO's answers to Murphy Interrogatory Set I indicate that the Flexnet transmits approximately every 90 minutes and the Zigbee transmits every 30 seconds.

And yet, I have filmed a PECO AMI meter in situ, which was transmitting Flexnet and Zigbee EMF at much, much higher rates:

Video labeled: "AMI Flexnet first 8 second" video:

This video is 27 seconds long. It captured one Flexnet pulse in the first 8 seconds and one Zigbee pulsation.

This video was filmed as we were setting up and before the introduction could be announced, so we stopped the video, and started it again with the following video, number 2 below, approximately 3 minutes later.

2. Video labeled: "ami meter 3 flexnets in 29 minutes":

This video is 33 minutes long.

Here are the elapsed times of the Zigbee pulsed EMF emissions:

25 seconds
41 seconds
56 seconds

1 minute 11 seconds
1 minute 26 seconds
1 minute 41 seconds
1 minute 56 seconds
2 minutes 11 seconds
2 minutes 25 seconds
2 minutes 41 seconds
2 minutes 49 seconds
2 minutes 55 seconds
3 minutes 3 seconds
3 minutes 10 seconds
3 minutes 16 seconds
3 minutes 26 seconds
3 minutes 41 seconds
3 minutes 55 seconds

(at 4 minutes: You can hear a sawing sound like the Flexnet, but low level intensity, so I am not sure what that pulse was; there were no cell phones or other devices in the home or outside which might have generated that sawing sound, there was no customer owned Wifi in the home, and all devices accessing the internet in the home were hard wired)

4 minutes 11 seconds
4 minutes 26 seconds
at 4 minutes 28 seconds, the Flexnet pulsed yet another EMF emission. This is certainly less than 90 minutes from the first video Flexnet pulsed EMF emission!!! (more like 6 minutes after the previous video Flexnet pulsed emission!!)

Zigbee emissions (continued):

4 minutes 56 seconds
5 minutes 10 seconds
5 minutes 26 seconds
5 minutes 40 seconds
5 minutes 56 seconds
6 minutes 10 seconds
6 minutes 25 seconds
6 minutes 41 seconds
6 minutes 55 seconds

(The Zigbee continues to emit pulsation of wireless EMF at varying intervals for the rest of the 33 minute video)

I have painstakingly reviewed each video and created a list of pulsed EMF emissions in Appendix K, including the elapsed times between pulsed EMF from the PECO meters, in order to save the judges time in reviewing the pulsations.

My videos provide proof that PECO experts have no idea how often their deployed wireless meters are emitting pulsed EMF in customers' homes and outside their homes.

In fact, Mr. Pritchard and Dr. Davis have renamed the PECO emissions as not being "pulsed". See PECO answers to Murphy Interrogatory Set I at I-18 (Murphy St. 2, Appendix F) and Dr. Davis' Surrebuttal testimony answer to Q 46, page 21-22 at lines 22 ff. Dr. Davis even goes so far as to opine in his Rebuttal testimony that PECO meters do not emit pulsed RF signals at Q 47, pages 22 to 23, and that only lasers emit pulsed EMF!

See Davis Rebuttal testimony, Page 22, lines 1 to 4, "the only wireless communications devices that use pulses to convey information are laser communication devices. PECO's AMI meters are not laser communications devices". Please see infra, discussion regarding spin doctoring to try to avoid responsibility for certain behaviors.

21. Q. In addition to the frequency of EMF emissions errors in PECO expert testimony and interrogatory answers, do you have any other concerns

about PECO's language choices when describing its wireless meter emissions?

A. As a language major, as a PhD in psychology, and as a lawyer, who has been taught to be careful with language, it is my experience that changing the definition of one's behavior, in order to attempt to avoid having one's behavior pegged as falling into certain undesirable categories, does nothing to change the nature of the behavior itself. It is Mr. Pritchard's and Dr. Davis' thinly veiled attempts to avoid PECO's wireless smart meters' peaks of EMF emissions being pegged as falling into the category that thousands and thousands of peer reviewed articles for decades have shown, regarding non ionizing pulsed EMF radiation's deleterious health effects. Attempting to change the name of the PECO smart meter emissions does nothing to change the nature of those PECO smart meter emissions.

Similarly, Dr. Davis' attempts to recharacterize the Zigbee in his Rebuttal testimony, as simply a "radio", whereas in the Kreider testimony, Mr. Pritchard correctly described the Zigbee as a whole house WiFi, is another attempt of experts trying to function as spin doctors. The Zigbee is a whole house WiFi that the customer cannot turn off. The Zigbee transmits constantly, with pulsed EMF at a rate in excess of every 30 seconds, as I have filmed, looking for smart appliances in a household, twenty-four/seven, day and night, all the time. A rose by any other name would smell as sweet, and garbage by any other name would still stink.

22. Q. Do you have further concerns about Dr. Davis' purported calculations in his Rebuttal testimony concerning your exposures to EMF from 7 minutes a month of cell phone use?

A. I certainly do. Dr. Davis must take me for an idiot. Dr. Davis opines in his Rebuttal testimony that my 7 minutes of cell phone use in a month is 188 times greater than exposure at a distance of one meter from an AMR smart meter and 1,200 times greater than exposure from an AMI meter, or 107 years of exposure within 1 meter of an AMI meter. Davis Rebuttal at page 18 lines 5 to 12.

This is patently ridiculous. Obviously, Dr. Davis has never researched the difference between pulsed EMF spikes and continuous EMF exposure as to health effects.

Obviously, Dr. Davis is using sum total exposure times, with the AMI meter sending out EMF at fractions of milliseconds per spike, added together over time, rather than the actual number of spiked peaks of EMF which are biologically far more deleterious to the body than are constant emissions of EMF, even if the body is not aware of the fractional millisecond exposures at the time. And Dr. Davis obviously did not take into consideration, because he had no information to use for this exercise, the distance I was from the cell tower(s) and the distance that my cell phone connection had to travel to send and receive the audio when I engaged in the 7 minutes of cell phone use two

months ago, nor the distance at which I held the phone from my ear, or whether I used speakerphone mode.

It is far more instructive as a comparison exercise, to view Dr. Karl Maret's slides prepared using a dosimeter, of an iPhone cell phone caller's exposure to spiked EMF emissions and exposures to EMF spikes a short distance from a bank of 5 smart meters, which Dr. Pall has included in his Surrebuttal testimony. You can see that Dr. Davis' comparison using averaged total exposure times of EMF fraction of a second spikes of EMF emissions is ludicrous in terms of biological effects of EMF, and Dr. Davis knows it or should know it.

23. Q. Why do you defer to Dr. Pall regarding research into pulsed EMF harmful effects?

A. I have been forced to hire an expert witness in this litigation. My expert witness, Dr. Martin Pall, has read all the research on EMF and has written and spoken extensively throughout the world on the harm caused by EMF, and can speak to Dr. Israel's cited literature and Dr. Davis' uncited pronouncements far more articulately than I.

Dr. Pall and others have pursued research as to why pulsed EMF of irregular intensities and irregular pulsation patterns may be more harmful to susceptible individuals than constant non varying EMF. As I said, I am no expert in EMF; I have just begun my studies in that area.

24. Q. In addition to your own experience of harmful effects of EMF in your life, including that emitted by the PECO AMR meter, have you uncovered information regarding harmful effects of EMF in published literature?

A. There is so much research I have read stemming back before the 1940s, including compilations by Dr. Zorach (Zory) Glaser, starting in 1971, who worked for the Navy, and was commissioned by the military to research biological effects of EMF. His fascinating compilation of Navy research, entitled RF/Microwave Bioeffects Bibliography spans decades and is comprised over 6,000 research studies from around the world. Very early research in Dr. Glaser's files reported non thermal EMF exposure fatalities in mice, rats, flies, wasps and frogs and non thermal EMF exposure cataracts in rabbits and dogs. Dr. Glaser's research is posted on Dr. Magda Havas' website at http://www.magdahavas.com/ showing conclusively that low level EMF do have deleterious health effects on individuals.

I am astonished that this research can continue to be ignored by industry such as PECO experts. Just as industry ignored the harm caused by tobacco, asbestos, pesticides, and the like, until the class action law suits hit industry and those industries were forced to change their tactics. Perhaps it will take class action lawsuits to wake up the telecom and power industries to admit that low level EMF can and do harm some individuals.

It might also be of some import that a recently released partial result of a US government study undertaken by the U.S. National Toxicology Program,

under the auspices of NIH, has found, studying the effects of cell phone frequency wireless EMF whole body exposures at 900 megahertz for rats (which is very close to the frequencies for the PECO AMI meters) and 1900 megahertz for mice, a causal relationship between exposure of the male rats and both glial tumors and schwannoma tumors, and precancerous changes in the cells. Both glial and schwannoma tumors have been reported to be on the rise, and linked to the advent of widespread cell phone usage. This peer reviewed and carefully planned study was one of the most expensive and comprehensive experiments to date in the U.S., regarding health effects from cell phone EMF exposure. The NTP researchers have not released any data on the mouse statistics yet, but those results are expected to be released in the coming years. I have not seen any data regarding the pulsation patterns of the experiment, which could have a marked effect on the results.

25. Q. What's the research that contradicts PECO's claims regarding the harmful health effects of smart meters? Is there any research or research compilations in particular that you found illuminating regarding EMF harmful effects on individuals such as yourself?

A. The research I have read, and it is extensive for a layperson over a short period of time, points to epidemiological studies, including the Lamech and Conrad studies that Dr. Pall directed me to in his testimony, Dr. Pall's studies on the effects of EMF on the ability of the body to deal with oxidative stress and other chemical reactions that the voltage gated calcium channel regulators set in motion after being zapped by EMF, and also a very readable and

seminal work by Dr. Martin Blank: Overpowered, What Science tells us about the Dangers of Cell Phones and other WiFi-Age Devices, Seven Stories Press (2014). Dr. Blank summarizes his experiments and research over the last 25 years, and the research of thousands of others into the health effects of non ionizing radiation, and the politics involved in research funding and publication. Dr. Blank traces the history of how he became an unlikely activist, and how the history of harmful effects of non ionizing EMF were known in the US and Russia for decades; the US military was researching non ionizing EMF harm as early as pre World War II, and afterwards, for many decades. Dr. Blank delineates the reasons that the public was not particularly concerned initially about the findings of these studies, because the public in general was not often exposed to these microwaves earlier. That is, until cell phones became a desirable means of communications, and cell towers went up in the mid to late 1980s. And the cell phone industry in the U.S. and elsewhere in the world has driven almost all the funding for research ever since, which has obviously been biased in favor of the cell phone industry.

I have also been reading Dirty Electricity, Electrification and the Diseases of Civilization. by Samuel Milham, MD, MPH , iUniverse (2010). Dr. Milham takes the reader through not just EMF, but also many studies reporting on the severe biological harm, including death, that dirty electricity (or harmonics) can bring to the world, and some forms of remediation that can be employed to minimize dirty electricity, such as can be generated by PECO's AMI smart meters.

I was surprised to find out that butterflies and bees and even plants are negatively affected by non ionizing radiation. When we first moved to our farm in West Chester in 1990, there were tons and tons of monarch butterflies which stopped on our land on their journey south every summer. They feasted on the milkweed that grew on in my garden and on the borders of our property. The monarchs no longer stop on our farm. The milkweed is no longer plentiful here.

I had been studying the decline of the bee colonies over the years. Most experts had attributed colony collapse to the use of pesticides, like Roundup. I know beekeepers in my area who refuse to bring their bees to farms which have used Roundup. But with my new knowledge of the harm caused by EMF, through such sources as Dr. Blank's Overpowered, which devotes a whole chapter on research regarding negative effects of EMF on non humans, I cannot help but wonder what effect the blanketing of all the farmland with EMF from cell phone towers and wireless smart meters has had on our wildlife population. I miss seeing those monarchs.

26. Q. Have you noticed any parallels in other EMF studies to your condition?

A. I recently came across Dr. Magda Havas' 2013 article entitled: "Radiation from wireless technology affects the blood, the heart, and the autonomic nervous system", De Gruyper, Rev Environ Health (2013) 28:(2-3) 75-84, (APPENDIX S) which discusses electrohypersensitivy in great detail, in addition to blood changes upon exposure to cell phone radiation, and heart

effects in heart rate, irregular heartbeat, and changes in the parasympathetic and sympathetic nervous systems, also. The whole article certainly rang true to me as it described many of the deleterious health symptoms I experienced due to deployment of the AMR meter on my home. She explains on page 78 the flaws in the study cited by Dr. Israel in his Rebuttal testimony as showing that EHS is a non existent disease, and how studies to test EHS have to be properly designed and carried out.

In the Dr. Blank book discussed above, Overpowered, op. cit., his chapter, "Other Health Effects of EMF", categorizes many known degenerative health effects of EMF, including several that I experienced--a decrease in melatonin, accelerated aging, pineal gland effects, cancerous growths, depression, dizziness, headaches and cataracts, plus several other effects that I did not suffer from: Alzheimers, ALS, buzzing in the ears and male infertility.

And one final note on this section, in reading the Lamech and Conrad surveys of deleterious effects of smart meters in two different populations, works cited by both Dr. Pall in his prior testimony and by Dr. Israel in his Rebuttal testimony, I was again struck by the similarities of the participants' symptoms to many of my symptoms caused by the AMR meter, and which were relieved in great part by removal of the AMR meter. Since I had no prior knowledge of EMF or its possible deleterious effects, I could not have been influenced by either of these studies before I wrote my initial testimony in this case.

27. Q. Do you have any further comments on PECO's Rebuttal testimony by Mr. Pritchard?

A. Yes, thank you. I see many inconsistencies or half truths in Mr. Pritchard's statements in his Rebuttal testimony compared with his earlier testimony.

28. Q. What are those inconsistencies?

A. First of all, Mr. Pritchard states on page 3 lines 19 and 20 of his Rebuttal testimony that PECO's AMI system is not a "mesh" system, and it does not use "pulses". I have discussed the "pulses" spin doctoring above. But the issue of whether or not PECO has installed a mesh system is one that Mr. Pritchard has never explained. I will discuss below.

Additionally, Mr. Pritchard states that the use of fiber optics (line 22) is not a reasonable alternative to communication using RF transmissions.

There are even more inconsistencies in his testimony regarding the frequency of transmissions of the Zigbee whole house wifi on page 6, lines 3 through 9.

And Mr. Pritchard states that "Neither PECO's AMR meters nor its AMI meters transmit "constantly" or "continuously" as claimed by Ms. Murphy". (lines 2 to 3) page 4 as further expounded upon on page 7, Q11, lines 7 through 15.

I would like to first dismiss his spin doctoring regarding the lack of constantly or continuous transmission of the AMR and AMI meters, because it is simple.

Those meters do transmit all the time. There is no way to turn them off to go to sleep, and there is no way for a homeowner to escape them.

Does Mr. Pritchard think he can fool the judges into thinking that adding up many thousands of the fractional millisecond pulses of EMF spikes emitted by the PECO smart meters into one fraction of a second over a day long period makes them no longer continuous or constant?

I would hope that the judges have more understanding of the constancy and continuous nature of the PECO smart meter emissions than to accept that slight of hand.

Mr. Pritchard does not even try to explain how he comes to believe that a pause of a few seconds before each new transmission of the Zigbee and a few minutes' pause before each EMF transmission of the AMR meter takes the AMR and AMI meters out of the "constant and continuous" realm of transmission. Mr. Pritchard simply ignores that uncomfortable reality of the PECO smart meter EMF emissions.

He would rather total up all the thousands of emissions of the meters in a day into one little tiny package and be done with it.

Would a prisoner subject to so-called Chinese water torture look upon his moments when water is not trickling down his forehead as "rest"? I think not.

Likewise, PECO's AMI and AMR meters are continuously transmitting, even if there are a few seconds or a few minutes' pause between pulsations of

EMF, and even if, in toto, those slices of fractional seconds worth of transmissions only total to less than a second a day.

The bodies of sensitive individuals like myself do register those pulsed emissions, even though I may not consciously perceive them, and even though my Gigahertz meter takes some time to ratchet down after registering a pulsed EMF spike being emitted from a PECO smart meter.

29. Q. What is your concern about Mr. Pritchard's statement that PECO has not installed a mesh smart meter system?

A. Mr. Pritchard mentioned in his Surrebuttal testimony at page 6, lines 1-6, that mesh systems send out thousands of communications a day, whereas PECO's system is not a mesh system.

While I am not mathematical wizard by any stretch of the imagination, it appears that one Zigbee home area network module in one PECO AMI smart meter emits pulses every thirty seconds, especially if the PECO customer doesn't have any smart appliances for the Zigbee to link up with, if you can believe the PECO interrogatory answers referenced above, and that is 2880 emissions per day, from just one customer's home, just from the Zigbee.

Now, I have measured a PECO in situ AMI meter, as referenced above, not for a whole day, but for quite a while, as recorded in my videos attached as Appendix K to this testimony. And at the rate that that one AMI meter was

emitting, we have probably in the five or six thousand or more emissions per day range, just from that one customer's meter.

I question what Mr. Pritchard meant when he compared the PECO AMI meters favorably to a mesh system emissions frequency, when mesh system emit thousands of times per day.

I have examined Mr. Pritchard's testimony in the Kreider case, and his testimony in my case, and certain previous statements he has made before other groups.

I have broken down my comments into categories below:

Distance and strength issues of the AMI emissions:

Mr. Pritchard has stated in an undated but post October 2012 and pre February 2015 talk before the IEEE PES entitled, "PECO Delivers a Reliable and Resilient Smart Grid" (APPENDIX P), that the PECO AMI smart grid contains 179 Tower Gateway Substations, (Kreider at p 101 ff), and those Tower Gateway Substations serve the whole 1.7 million PECO customer base, which comprises 2100 square miles [sic]. See, APPENDIX P.

I have downloaded a map of the PECO territory (APPENDIX T). There seems to have been some gerrymandering to carve out from PECO's territory, the area around Hatfield in Montgomery County, oddly enough, the locus of the residence of our own Chair of the Pennsylvania Ways and Means Committee, Representative Godshall, whose scathing letter to the PUC

following the PUC decision to allow Susan Kreider to proceed to hearing as to the harm caused her by PECO's AMI meter I had attached to my previous testimony.

In Mr. Pritchard's testimony in the Povacz case, he stated on page 10, lines 10 and 11, "PECO service territory is approximately 1713 [sic] square miles, and PECO services approximately 1,700,000 meters".

I don't know if the PECO service area diminished by 500 plus square miles in the intervening time between his talk a few years ago, and his testimony in the Povacz case, or if he is unsure of the PECO service area or just playing loose with his figures.

Since Mr. Pritchard testified in the Kreider hearing at page 101ff .that PECO is using only 179 Tower Gateway Substations to collect data from all the PECO AMI smart meters, that means, depending on geometry, that each AMI meter might have to be sending wireless signals much farther than the one mile transmission distance Mr. Pritchard answered in Murphy Interrogatory Set I as to AMR meters.

These are not small distances for the PECO AMI wireless broadcasting to take place, from our homes, and the constant pinging spikes of EMF are going around and around in our home and being broadcast wirelessly from each of our homes and businesses, for a distance of many miles away, over our countryside, towns, backyards and cities, constantly emitting, from 1.7 million smart meters, to PECO's Tower Gateway Collectors.

In fact, we really have no idea exactly how far and to what extent the PECO AMI meters are broadcasting from Mr. Pritchard's sworn testimony.

Buddy Meter deployment:

Examining earlier communications of Mr. Pritchard found on the internet through searches, in his presentation to the IEEE Power and Energy Society, entitled: "PECO Delivers a Reliable and Resilient Smart Grid" of unknown date, but after October 2012 and before February 2014 (APPENDIX P), we can see on Slide 7 that the PECO Smart grid is set up for customer AMI meters to use a "single hop mesh" where needed to communicate with the Tower Gateway Substations via wireless EMF transmissions, and that PECO has installed or will install 375 miles of fiber optic cable to construct the backbone of communications from the Tower Gateway Substations to PECO administrative offices. Mr. Pritchard reiterated that PECO had installed fiber optics as a backbone for the TGBs to communicate with PECO in his Rebuttal testimony at page 8, lines 11 and 12. I will talk more about the fiber optics situation below.

Mr. Pritchard told a similar audience of the IEEE in February 2014, in a talk entitled "Ice Storm in Southeastern Pennsylvania, APPENDIX Q, that each meter is designed to see between 3 and 7 Tower Gateway Substations, at slide 14, but when a PECO AMI wireless meter cannot directly reach a Tower Gateway Substation, a "buddy meter" can be set up to use nearby AMI wireless meters to relay the data back to a Tower Gateway Substation.

Is Mr. Pritchard mincing his words once again, to state that this "Buddy Meter" set up and the single hop mesh system is not actually a "mesh" system in disguise?

How many of these "buddy meters" and "single hop mesh" configurations is PECO employing in its smart grid system? We have no idea. There was no mention of this buddy meter or single hop mesh set up in any of Mr. Pritchard's sworn testimony when he was describing the AMI system. And what effect do those "buddy meters" have on the inhabitants of the homes that they are deployed in?

We have no idea how a house is selected to get a "buddy meter" and how the homeowner is notified that her home has been selected to get a "buddy meter", and if the homeowner has any choice in the matter, or if it is simply a ministerial decision by PECO that everyone who is selected to get a "buddy meter" gets one or her electricity is shut off.

PECO has provided the court and the Complainants absolutely no information regarding the additional wireless emissions of its relay system of the "single hop mesh". In fact, Mr. Pritchard has flat out denied that PECO is using a mesh system.

Did PECO abandon the buddy meters and single hop mesh before deployment of the smart grid? Or is PECO still using the buddy meter/ single hop mesh system?

Mr. Pritchard can set up a smart grid system where one customer's Smart Meter becomes a hub for the neighborhood and it is not considered a "mesh" system, because Mr. Pritchard can define "mesh" any way he wants to. Mr. Pritchard has not informed any Complainant or the court how many of these buddy meters PECO has deployed, nor has he described in any detail whatsoever the extent to which such buddy meters may be deployed in the neighborhoods of the Complainants.

Assuming 1,700,000 PECO customers and only 1,700 square miles of territory, that makes 100 customers per square mile on average (realizing that this is not a precise calculation, since the PECO territory is not symmetrical, and customers are not uniformly spread out in distance. But, nonetheless, if there are 179 Tower Gateway Substations in PECO's service area, and each customer's AMI meter can see 3 to 7 of these (out of the 179) Tower Baseway Substations. Starting with the number 3 Tower Gateway Substations instead of 7, to be conservative, that's almost 9,500 customer meters sending out EMF per each Tower Gateway Substation, but if each customer meter can "see" 3 Tower Gateway Substations and send its EMF to each of those TGBs, then, conversely, each TGB should be able to "see" 28,500 customer meters, and 28,000-50,000 or more PECO customers' AMI meters are spewing EMF to three to seven TGBs, all the time, night and day, because "seeing" in terms of PECO, means sending fractions of a second batches of microwaves from the customer's home to 3 to 7 TGBs which are

located many miles from each other, and many miles from the customers' home, on average, all the time, all the time.

Frequency of emissions of the Zigbee whole house Wifi System

Mr. Pritchard states on page 6, lines 3 and 4 of his Rebuttal testimony, that the Zigbee will transmit every five minutes until such time as it acquires communication with a device within the home. [emphasis added]. This is an error of tremendous magnitude for anyone who is EMF sensitive. PECO's answers to Murphy Interrogatories Set I at I-16 stated that the Zigbee transmitted every 30 seconds until it was paired with a device within the home. My own investigation and filming of a PECO AMI meter in situ showed that the Zigbee transmitted on a far more frequent basis than even once every thirty seconds, as shown in Appendix K. If Mr. Pritchard does not remember how often the Zigbee is supposed to be transmitting, then Mr. Pritchard has no business opining on such matters as PECO's expert witness. If Mr. Pritchard simply makes up emissions frequency data on the spot for purposes of testimony, that is unforgiveable.

30. Q. What is disturbing to you about Mr. Pritchard's testimony concerning the use of fiber optics for smart metering?

A. Mr. Pritchard had testified in the Kreider hearing, p 102 lines 16ff, that fiber optics is not a viable alternative, because if it were, it would be considerably more costly. and he knew of no large electricity supplier who had utilized fiber optics. In Mr. Pritchard's Rebuttal testimony, he stated on page 9, lines

8 ff, that to install a fiber optics system to communicate with the TGBs would have added billions of dollars of cost to the system.

I see this an example of PECO's linear boxed in thinking. If PECO even considered for one minute that using a customer's fiber optics or cable internet connection could save "billions of dollars" to transmit customer usage data without creating an unsafe environment for sensitive customers, as is done in at least one modern country, Germany (a country comprising 137,000 square miles), perhaps sensitive Pennsylvania customers like myself would not be fighting for our lives against PECO.

I am reminded of the Mari Jensen v PECO case, where PECO argued so strenuously against paying solar power generating customers what was due them under the tariffs, because to do so, would require PECO to spend $500,000 to implement a program to pay those customers correctly under the tariffs.

A: Mr. Pritchard has spoken to other utilities in the U.S. about their smart grid systems. But Mr. Pritchard obviously has not spoken to Italian and German smart grid builders, who, I am told, have not used wireless communications whatsoever. If you go to www.yellostrom.de, you can see information regarding the YelloStrom German meter, which attaches to the customer's fiber optics cable system. PECO has installed many hundreds of miles of fiber optics from its Tower Gateway Substations to reach PECO headquarters, using fiber optics as its "backbone". as cited above. I believe that Mr.

Pritchard used to work in Exelon's fiber optics department. Why could Mr. Pritchard not see the advantages of using wired fiber optics in its metering system, to save the environment and to save EHS sensitive individuals from excessive EMF?

Additionally, a fiber optic system is far more secure and reliable than a wireless system.

In fact, PECO has installed a fiber optic system as its "backbone" along with phone lines according to Mr. Pritchard's Rebuttal testimony at Q8 page 5, lines 10 and 11.

At P 8, Q15 of his Rebuttal testimony, Mr. Pritchard discusses the Chattanooga fiber optics metering system, which he apparently familiarized himself with after the Kreider hearing, most likely from my or other Complainant's filed testimony. On Lines 5 through 7 on page 9, Mr. Pritchard states "every Chattanooga subscriber therefore has RF exposure from WiFi as well".

Fiber optics comes into the home from buried fiber optic cable. Whether or not the customer chooses to install WiFi is up to the customer. The customer may very well choose to hard wire her computers to the fiber optic cable using a modem, and not use WiFi, as I have chosen to do, and as other PECO Complainants have chosen to do to cut down on EMF exposure. Mr. Pritchard seems to be unaware that fiber optic internet and metering

technology is NOT dependent on WiFi at all; this is a very serious flaw in an expert witness who is in charge of smart grid design.

Furthermore, it is apparent that Mr. Pritchard did not read my prior testimony carefully. In it, I offered to allow PECO to connect to my already in place underground Fios fiber optic line, which I rent from Verizon every month for my internet and telephone services, just as is done in Germany via YellowStrom smart metering systems. This would not cost PECO (and PECO customers, ultimately) the billions of dollars which Mr. Pritchard mentions in his Rebuttal testimony on page 9, line 13.

I don't know a single neighbor who does not have internet access in their home. PECO could have easily and seamlessly used the customer's internet access to transmit usage of electricity data. All it would take would be a contract between the customer's internet provider and PECO. Done deal.

Just because Chattanooga installed all new fiber optics to implement its smart grid and provide internet services to its population, does not mean that PECO would be forced to install a complete single use fiber optic grid de novo. PECO could have used fiber optics systems already in place for its smart grid application, but PECO chose not to.

PECO, unfortunately, did not consider its mandates under Section 1501 of the PUC code and potential health effects on sensitive customers when it installed its AMI meter system. PECO only considered whether or not the system worked for its purposes. And, unfortunately, PECO individual customer

Complainants probably do not have standing in this type of procedure to force PECO to protect wildlife and the environment in delivering electricity to them.

31. Q. **What other reasons do you have for believing that Mr. Pritchard has no concept of your health issues and the accommodations you have to make for them, which you raised in your prior testimony?**

A. Mr. Pritchard does not understand that I cannot tolerate continuous exposure to EMF. That I cannot tolerate WiFi; and for that reason, as I have testified in prior testimony, I have hard wired all my electronics which use the internet, and I possess no smart appliances despite my desire to conserve electricity, because I cannot tolerate non linear electricity.

Perhaps because Mr. Pritchard is not an electrical engineer, he cannot understand these concepts, but as the designer of PECO's smart grid, he should be aware of these concepts, especially in light of PECO's mandates under Section 1501 of the Public Utility Code.

In any case, Mr. Pritchard states in his Rebuttal testimony on page 9, at lines 20 and ff, continued to the top of page 10 lines 1 and 2, that even if an analog meter were used as part of an AMI meter, the meter assembly would still have a FlexNet radio and a Zigbee radio. This statement shows that Mr. Pritchard has no understanding whatsoever of my requests, nor of his mandate under Section 1501.

I cannot tolerate a whole house WiFi, which is what the Zigbee radio is. Therefore, Zigbee cannot be safely deployed on my residence. No matter how far away the meter socket is located, the Zigbee is a whole house WiFi. I am far too sensitive to EMF to tolerate a Zigbee which penetrates my house structure at all. And I possess no smart appliances, for the reasons stated in my prior testimony. So the Zigbee would be useless for purposes of pairing with a smart appliance in my case, and extremely harmful to my health. The forced installation of a whole house WiFi by PECO in my home is not permitted under Section 1501 of the Public Utility Code.

On the other hand, If the Flexnet pulses could be transmitted via a different type of meter, instead of wirelessly, using fiber optic cable connections which are already existing in my residence, without interfering with my internet connectivity or my telephone line, and without generating additional harmonics, I would me amenable to that resolution.

PECO and its experts including Mr. Pritchard seem to be stuck in a "one size fits all customers" mode of thinking. Since the wireless AMI system fits PECO's needs, and since PECO gained a huge amount of federal stimulus moneys to install its smart grid at an accelerated pace, PECO cannot fathom that some of its customers cannot tolerate wireless transmissions emitted by its smart meters.

But I know and have testified that I cannot tolerate those wireless transmissions, as has my expert Dr. Martin Pall, and PECO has to realize that

fact and grant me appropriate relief under both the ADA and Section 1501 of the PA Public Utility Code.

32. Q. What has Mr. Pritchard stated about PECO's smart meter grid system and your disability that solidifies your confidence that Mr. Pritchard does not understand nor wish to accommodate your disabilities whatsoever?

A. Mr. Pritchard attached a map of my immediate neighborhood, and gloatingly stated that "Ms. Murphy is going to regularly encounter PECO AMIs anywhere that she travels in PECO's service territory. Indeed, as shown in Exhibit GP-5 (Map showing AMI Meters Near Murphy Residence), AMI meters have been installed at all buildings in her neighborhood." p 12, lines 15 to 18.

In Mr. Pritchard's opinion, it would be useless for me to not be furnished an AMI meter because there was no place in my neighborhood I could go which was not flooded with AMI wireless meters. p 12, lines 15 to 18.

If I were PECO, I would not be proud of that accomplishment. I would be ashamed.

This comment shows how callous PECO and its experts are to my disabilities. They are so proud of their accomplishments that the smart grid they have forced down the throats of sensitive PECO customers like foie gras geese before the slaughter, that they have lost sight of how, if what they say is true,

they have rendered the countryside where I live uninhabitable for me and others like myself.

33. Q. Do you take exception to Mr. Pritchard's statement that there is no place in your neighborhood to escape AMI meter emissions?

A. If Mr. Pritchard is telling the truth about its wireless AMI system, I can only hope that the still rural area that I live in is not completely blanketed by microwaves emitted by PECO wireless smart meters. When I did have a wifi in my home, I could not pick up any neighboring wifi signals, so my home's relative isolation may be somewhat of a protective factor. I have attached a Google map of my immediate vicinity. APPENDIX U. My home is elevated far above the road. My hayfield is also elevated, with my driveway being lower than the rest of my land. I know that microwaves are affected by elevation. Chester County where I live is known for its undulating hills and valleys. I don't have the expertise to figure out exactly how AMI smart meter microwave emissions are affected by the topography of my land in particular. The only neighbor's smart meter that is pointed at my property that I know of, is that of my immediate neighbor, whose house is the second on our shared driveway. My neighbor's house holds an AMI smart meter, directly across my horse paddock from my front door.

34. Q. Explain why PECO's expert testimony statement, roughly paraphrased as: "It is useless to refuse an AMI meter because your whole neighborhood is infested with EMF caused by AMI wireless

communications" misses the entire point of why you have filed a formal complaint against PECO.

A. PECO's argument above offends my intelligence. If a person cannot tolerate pulsed EMF, and has been harmed by a PECO AMR meter which pulses EMF, why in the world would that person want to subject herself to additional inescapable EMF in her own home?

One's home should be one's castle. We revere the sanctity of our homes so much that the federal government has established "Do not call lists".

The "castle doctrine" has developed in criminal law cases. Where is the castle doctrine in PUC law?

I have the right under case law to defend myself from an intruder who uses a deadly weapon in my home. I do not believe in killing others, in general, but if a stranger approaches me in my own home with a deadly weapon aimed at me, I will not hesitate one second to try to disarm that person and use his weapon against him.

PECO has done just that. PECO is not invited into my home as a guest. PECO provides a necessary service to me for which I pay a fee every month depending on how much of that service I use that particular month. I have no choice as to who my electric distribution company is, but I do have a choice as to who my electric supplier is. PECO has brought a deadly weapon into my own home and taken aim at me. PECO has been shooting at me with a

deadly weapon for 14 years, hitting me over and over and over, every one to six minutes, night and day.

35. Q. Now, is this weapon that PECO has aimed at you as deadly to others as it is to you?

A. Obviously not, because otherwise, there would be no doctors to treat me. Everyone would be as disabled as I was before I had the AMR meter removed. Dr. Pall has explained in his Surrebuttal testimony (see Id), that EHS affects different individuals differently, and that there are certain commonalities as there are many differences. If EMF affected all individuals the same, then all studies would show development of all the same ill health effects from EMF in all study cell lines and all study participants. This is obviously not the case, and it is not surprising. Just as certain drugs have certain ill effects on test subjects and on patients after drugs have been approved by the FDA, certain EHS sensitive individuals develop symptoms and others do not.

For example, I cannot tolerate fluorescent lights. I have never been able to tolerate them. I develop an immediate headache from them, which increases in intensity the longer I am exposed to them. If no one on earth could tolerate fluorescent lights, there would be no market for them, and very quickly, they would no longer be manufactured.

Just because I cannot tolerate wireless smart meters, does not mean that everyone cannot tolerate wireless smart meters. But it does mean that PECO

has no right to give me the choice of getting a wireless smart meter on my home or doing without electricity. This flies in the face of PECO's responsibilities delineated in the very language of Section 1501 of the Public Utility Code.

36. Q. What are your thoughts on PECO's answer to the Murphy Interrogatory Set I-35 question about the number of customer complaints regarding health effects of the AMI system meter?

A. PECO has responded to Murphy Interrogatory Set I-35 that it has received only 8 complaints regarding health effects of the AMI system, and two formal complaints were dismissed on preliminary objections, and two informal complaints were dismissed, leaving 4 formal complaints to proceed to hearing. APPENDIX R. Although I am not willing to accept that answer as true, since a quick search of the PUC website reveals many more potentially applicable formal complaints, and since PECO did not reveal any customer contacts with PECO representatives who might not have filed formal or informal complaints (we know from her testimony that Brenda Eisen keeps very detailed records of all customer contacts and complaints regarding the AMI system, but PECO has not included any such customer contacts in its response to my Interrogatory. Only 8 complaints out of a customer base that includes 1.7 million customers must be some kind of record. A search of the PUC website reveals far more individuals who have had their electricity shut off for non payment. But accepting, arguendo, that PECO is telling the truth in answering this Interrogatory question, then it would stand to reason that

PECO would be bending over backwards to settle these remaining 4 smart meter health effects cases to the sensitive customers' satisfaction, so that PECO would not run the risk of being judged to have violated Section 1501 and harming customers. That is not the path that PECO has chosen.

37. Q. Do you have any additional comment regarding Brenda Eison's testimony?

A. Only this: that it was quite difficult for me to maneuver through the PECO customer service personnel, to find out where Dr. Prociuk was to fax my medical excuse letter pursuant to the PECO ten day shut off notice I had received. When I finally did obtain a fax number, after being disconnected and redirected to various personnel for over an hour, and with at least four different telephone calls, and Dr. Prociuk did fax the medical excuse letter which PECO has in its possession, Brenda Eisen did not send me or Dr. Prociuk any follow up letters whatsoever, which she referenced in her Povacz Rebuttal testimony (but not in mine) as being essential under the PECO rules.

But I later learned that a medical excuse letter was only permitted to stay the threatened disconnection of electricity for customers who did not pay their bills, and everyone in PECO's territory was required to accept a wireless smart meter in their homes because Act 129 required PECO to install smart meters in every customer's home. Again, I find it incredible that paying customers who are disabled get far less deference paid to them than scofflaw disabled customers in PECO's eyes.

38. Q. Does that conclude your surrebuttal testimony?

A. Yes. This concludes my written surrebuttal testimony. Thank you.

APPENDIX J

APPENDIX J:

DAILY LOG:
LAURA SUNSTEIN MURPHY
HEALTH EFFECTS FROM REMOVAL OF AMR METER

Day One:

AMR meter was removed by an electrician and replaced with an analog meter, against legal advice from my attorney, but as a means of saving my life only. I did not take this action lightly. I have been an attorney since 1989, and if there were any method of escaping PECO's wireless transmissions from the AMR meter in my own home, I would have gladly pursued it.
In fact, I have pursued this course of action with PECO and have been met with legal resistance from PECO at every turn, instead of a reasonable accommodation under the ADA as requested.

Immediately, I felt a sense of pervasive calmness outside my home where the meter was located, and also inside my home.

Within thirty minutes, I developed acid reflux.
Then shortly after that, I developed strong stomach pains. So strong I took a dose of a homeopathic remedy for stomach upset: nux vomica.
I took an Epsom salts and vita c powder hot bath, but upon getting out of the tub I felt seriously faint and dizzy but mostly faint. Seriously.
I wrapped myself in a towel and staggered to the bed.
I lay down for 45 minutes and then was well enough to get dressed.

I believe my body is purging toxins after 14 years of EMF damage.

That afternoon, I became very tired again. I fell asleep on the den sofa for a nap. I could not keep my eyes open. But it was a peaceful nap. Not troubled by heart palpitations or anxiety at all.

Later that evening, I still experienced some stomach pains, but not as strong as earlier.

Already, before 24 hours had elapsed, that evening, my knees, and especially my left knee which had required an offloader knee brace for a year or two no longer hurt to walk. Even without the brace.

That first night I went to bed around 10 pm, which was early for me, since I had not been able to get to sleep until midnight or so in the last several years, and I did not have to take a 5htp to get to sleep. I slept soundly until 7:45 am which was quite late for me, because in the past say 5 years, I would wake up at 6 or 6:30 AM. This time, I woke up with no heart palpitations, my sleep had not been disturbed during the night, and I awoke to no high blood pressure, no atrial fib, no heart palpitations, no anxiety, just restful sound sleep, for the first time in years.

Day Two

The following day, I felt more energized. My stomach no longer hurt. My lower back started to hurt less. My knees definitely had lost their crepitation when I weight bore or walked.
I had probably the most productive day in years, my thought processes had begun to clear somewhat for the first time in years, and my depressed mood started to lift.

I was able to accomplish three errands in that one day, whereas before I only had enough energy to accomplish one errand, then I was exhausted.

I felt that I would be able to live and enjoy life again. I went outside and picked some flowers. I helped to make dinner.

That night, I fell asleep with no problems, with no 5 htp and I slept soundly, waking up refreshed at 7:30 am, with no midnight wake ups, no agitation, no racing heart beats, no afib and no high blood pressure.

Day Three

That next morning, I had a scheduled lymphatic drainage appointment. My therapist of more than four years noted that I had a lot of outgassing in my legs and kidneys, liver and spleen and axilla lymph nodes and arms.

After lymphatic drainage therapy, I felt even better.

I continue to have no pain in the knee joints upon weight bearing, even though I still have edema in my knee joints. I still experience a lot of lower back pain and stiffness, but it seems more like muscle pain now, rather than spinal bone pain.

My stomach is still no longer hurting me. I find I have more patience, more like my old self. I was able to drive into West Chester twice today for appointments, accomplishing four errands in the process. I was able to park my car and walk into the bank without pain. I was able to park in the supermarket parking lot and walk into the supermarket and shop with no pain and without having to lean on the cart.

Day 4:
I felt so good that I walked half way down our ½ mile long very steep driveway and up again, with no ill effects. In previous years, I was able to walk the whole way down the driveway and back up again with ease. But I have been pretty much confined to the house with no walking at all for the past several years. Again, my knees were not hurting and I wasn't wearing my knee brace.
I even had enough energy to groom my horses (cursory but it was grooming), and fed them some carrots, walking more swiftly than I have been able to walk for several years. Since the past year, I had been unable to groom them or stand for any length of time needed to perform these functions. My heart has responded with no extremely high blood pressure when awakening in

the morning (134/79), but my heartbeat was 102, which was very high. I still have quite a ways to go to go back to normal body regulation.

Day 5

A friend drove me to another friend's house which was 60 miles away for me to test her home with my HF135C meter.
I felt fine on the drive to her house in the car and on the drive leaving her house, but in her house, I did not feel comfortable at all. It was a stifling sensation and I was beginning to get heart palpitations, except in the basement. True enough, her entire house except the basement was filled with high levels of EMF on my meter. Even her patio showed levels on the 20s spiking up to the 100s and 200s. Most of her house showed readings of at least 18 or more on the meter at all times, spiking up beyond 2000, with lesser spikes in the 200 to 400 range, almost constantly.
The only spot in her house with readings similar to my whole house since the analog meter was placed on my home, was in her basement.
I was very glad to return to my own house. However, I was only home for a short time when my heart started to race. I did not understand why. I checked and found that:

(1 the replacement analog meter that was sent to me, (because I had complained that the first analog meter I had purchased did not rotate to register electricity used), was not on the meter socket, and
(2 neither was the first analog meter I had purchased.

I called my electrician who informed me that my meter socket was 6 jaw, not the normal 4 jaw, and that was why he had replaced the AMR meter on my home.
I was fit to be tied!

My heart was acting up and I felt like panic was about to set in. I retreated to the basement where my HF 135C had measured zero when the AMR meter was on my house, the only place in the house I was able to retreat to safely. My heart has settled down quite a bit after I have been in the basement for twenty minutes, but I cannot remain here.
There is no heat in the basement, no water and no toilet.
I know now that I must avoid EMF exposure consistently or I risk heart ailments almost instantaneously.
I am hoping that my electrician will return this evening to remove the AMR meter or I will contemplate trying to sleep in my car tonight.

I understand now what it means when they say "there is no going back". In just the few hours that I was in the house with that AMR meter back on my socket, even though I spent most of the time in the basement, my heart was already beginning to race and my agitation level was going through the roof.
Within 10 minutes of no EMF after my AMR meter was removed, my mood has lifted considerably and I am once more calmer.

There is no going back for me. I cannot tolerate wireless. I can now feel my body bracing when I am close to someone who is using his or cell phone. If they are in my house, I ask them to put their phone in airplane mode. I can now feel when the airplane mode button is turned on. My body immediately relaxes out of bracing mode, I then take a deep breath.

Day 6

I woke up at 6:00 am with a too fast heart rate, but I had not checked with bp cuff, but fell back to sleep and checked when I woke up again at 7:45 am, first bp taken was 155 over 84 with heart rate of 89, then second bp reading a few minutes later was 134 over 79 with heart rate of 89.

I was able to go to the barn this am and help with the horses. I sure was glad to see them. I then went to my speech therapy at bryn mawr rehab, (which is not speech therapy per se, but cognitive processing therapy.)
Upon her inquiry, I told the therapist that I was feeling much better, but made her wait for the reason when she asked why, because I wanted her test me before I told her why I was feeling better.
She gave me several tests, and I did quite well on all of them, and was not fatigued as I was previously.
She had measured that I had not improved in cognitive function between (1) the performance tests that I had taken a year previously, and (2) the tests that I had taken last month, but she noted that my performance showed improvement today in several aspects since last month and last year.
She was very curious as to the reason. When I told her it was only a week since the EMF emitting meter had been removed, she was astonished. She said I looked like a weight had been taken off my shoulders, and she was happy to see me smile, because I had been unable to smile before. She noticed that I was walking faster too, and that I did not report any headache as I had previously.

I do not have any digestive discomfort today, either.

Day 7

This was another productive day. I ran several errands, and drove to two drs. Appts.
When I was in the local independent pharmacy to pay for purchases, when the other person in line's credit card was inserted into a brand new chip reader, I felt a stabbing pain in my chest, like a bolt of lightning.
This is the first time I have experienced this pain.
I can only assume that the independent pharmacy was using a wireless card reader and I was standing right in front of its transmission.
I had to lie down after I returned home to try to calm my heart down.
After a few hours of rest, I had enough energy to drive myself to new Bolton center at night for a riding club meeting, the first time in over two years.
I had the club president make an announcement to turn off all cell phones or put them in airplane mode for a member's health needs, and the members were very obliging. I was fine sitting and standing among twenty or so riding club members, but I became increasingly agitated after about

45 minutes, and found, upon looking around that a member had arrived late was working on her cell phone a few seats in front of me, so I was forced to get up and leave the meeting.
I have become more aware of what my body is telling me, and I must remove myself from the negative stimulus.

That night, I went to sleep ok, but I woke up at 3:30 am with an attack of gerd, which almost made me vomit. I had to prop myself up in bed for a half an hour until the gerd dissipated and I slept soundly until 7:30 AM.

Day 8

I went for a walk outside on my property for the first time in over two years; I had enough energy to do this.
I took my HF 35C gigahertz meter with me. I measured in my home, .6 or so in most of the downstairs, to up to 10 microhertz per square meter very high readings in the front and northern side of the house, especially by the northern windows, and on the front porch.
The readings in the neighbor's field riding easement area that I have ridden in for 26 years, adjacent to my property on the northern end were extremely high, causing me heart palpitations, and shortness of breath, with readings consistently in the 70s, 80s, 90s and above, especially with my antenna pointed to the west towards sugar bridge road, presumably where peco's collection nearest collection site is on a telephone pole on sugar bridge road, for its smart meter customer emissions.
If this is the location of peco's collection site on sugar's bridge road, as I suspect, peco has caused a constructive eviction to me, depriving me of the enjoyment of my property, then it is obvious that peco has caused a constructive eviction to me, depriving me of the enjoyment of my property.

Day 9

I woke up at 3:30 am with gerd and had to sit up in bed for a half hour until it went away but then, I was able to sleep well until 7:30. I woke up for the day with a normal heart beat and without high blood pressure. It was a busy day for me, which involved driving down to Delaware to pick up a new pair of glasses since the puppy had chewed up a good pair of glasses. That evening I accompanied my husband to villanova law school for a retirement dinner for several of his colleagues. I was fine until the table we were assigned to became filled. I had asked that the people sitting at the table turn their cell phone to airplane mode, and the people on either side of me did, but obviously others did not, and after ten minutes, I felt the sharp uneasiness in my chest and trouble breathing I had experienced in the friend's house with smart meters on Day 5, I had to leave the room for a while. I was glad to leave the reception and head home. I fell asleep in the car on the way home, even though it was only 10 pm. The following morning I again woke up at 3:30 am with gerd, and sat up in bed for a half an hour before being able to lie down and sleep again.

Day 10

I awoke for the day at 7:00 am with blood pressure of 134/75 with pulse of 89. I felt good. A friend picked me up for a visit to a mutual friend's house in Chester County and we enjoyed the day, went out for lunch in St. Peter's Village, and returned home without incident. That evening, I had enough energy to go out to the barn and groom all the horses. This is a first in a very long time. I even had enough energy to cook a partial dinner that evening and clean up.

Day 11

Work up today feeling happy. Bp is stable at 145/74 with pulse of 84. Ran a few errands. Worked on a few client matters on my tablet. Started hemming an EMF scarf I was making out of EMF shielding silk material. One of my eyes felt like I had scratched my cornea. Don't know if some tiny fragments of the metal from the fabric had migrated to my eye despite my wearing glasses all the time. That was painful. I was constantly irrigating that eye for a few hours until the pain subsided. That evening John and I went to a local restaurant for dinner; I wore my emf shielding vest, and the patrons at adjoining tables were kind enough to accommodate my request to turn their cell phones onto airplane mode. After returning home and watching Netflix with my husband, I felt sleepy at 10 pm and went to bed without taking a 5 htp. I fell asleep immediately.

Day 12

I slept like a baby the night before, never waking up at all, and awoke at 8:15 am. No heart palpitations. No gerd. I was in a great mood. Bp of 134/84 with pulse of 82. I was able to empty the dishwasher for the first time in absolute ages and wash the pans in the sink, without taking a break and without fatigue. I did a huge amount of legal work today for many, many hours, drafting changes to an employment agreement and sale of practice agreement, while I was parked in front of my computer. Work which had been impossible for me to accomplish just three weeks ago.

Day 13

I slept well again. Woke up with normal bp of 145/84 pulse of 89. My digestive system seems to be working much more normally. I went on several driving errands today without tiring and spoke with opposing counsel; was able to follow the contract revisions I had drafted and his points, too, without fatigue. I spoke with many others on the phone and felt organized, instead of tiring easily and getting a headache. I fell asleep immediately at 10:30 pm.

Day 14

Two weeks today! A celebration. I woke up at 8:15. Bp normal with 134/84 pulse of 82. Had a lot of work to accomplish today, legal work and preparing for hosting my siblings and their children and grandchildren next weekend. I went to therapy again at bryn mawr rehab. My therapist retested me with a test I was only able to complete a little over a third of a month ago. This time I was able to finish it completely. I did almost the same score as before on the part I had finished before, (about average, taking into consideration that I usually get almost all the

questions correct, but it takes me longer than the average patient to finish), and I did about average on the remaining parts.
This is a huge improvement. My therapist was very pleased that I have started to make such progress.
She did ask whether I experienced any headache after performing the timed test, and I had think about it, and to tell her yes, I was getting a headache about 1.5 or 2 on a scale of 1 to 10, which I did not have when I walked in to therapy. My headache was located in the right temple area. I have no idea what that means. I was not paying particular attention to my headache, and had not noticed that it had appeared. I did remark on starting therapy that I did not have a headache at all, (which is a new since the AMR meter was removed). Before the meter was removed, I had almost constant headaches ever since the concussion.
I also successfully completed several other tests she gave me, and took home several worksheets to help in the areas that I am still deficient in since the concussion, especially recalling names and working quickly under time pressure.
I went to the supermarket after therapy, bought meat and fish, came home, emptied the car and cooked most of the dinner. This is huge. I had not been able to do this for two years!
I spent a lot of time after dinner doing legal work and organizing my files.
Whew, banner day today. I can hardly wait to experience continued healing and improvement. I long for the day I can once more tend to my horses in the morning and ride them again. I am already planning to visit my son and his family in California late this summer or early fall, to meet my baby grandson who was born over thanksgiving. (The one I have been too weak to travel to meet and hold before. Breaks my heart).

I stopped keeping daily logs since removal of the AMR meter because I got so caught up with doing so many more activities each day.

I restarted PT, this time for my back, and after being away from PT for 2 and half months due to my hernia surgery on March 9 and the need to recuperate from that surgery, I was surprised that I suffered adverse health effects just entering the PT studio, which I had been going to for two a half years previously, twice a week.
I never noticed all the florescent lights in the ceiling, and the whole studio wifi, plus each therapist uses the wifi with a laptop to take patient notes at the time of treatment.
I immediately felt attacked by all the emf and dirty electricity, which I had avoided ever since the removal of the AMR meter from my home.
I told the receptionist that I had to wait in my car until my therapist was ready to see me, and I was fine in my car, but as soon as I had to lie on the table for therapy, the fluorescent lights really bothered me, and the wifi and computers (and probably cell phones of therapists and patients) bothered me too.
Although I was quickly developing a headache, I went through the 45 minute long PT evaluation. My PT remarked that my knees were much better, I was able to position my knees with no problems, the swelling was down in my knees, I was able to stand for long periods of time without pain, and I had more energy than the last time he saw me.
He expressed appreciation for my having discovered what was making me so sick. I explained rudimentarily what Dr. Pall had discovered about EMF affecting the voltage gated regulated

calcium channels, and he told me the voltage gated calcium channels was basic biology 101 and that he had been a biology major in college.

Week of May 23 through Memorial Day
I continue to improve as to heart regularity, and digestion and energy. I had one dizzy spell and sometimes I feel dizzy when I lie down, so I know there is something still going on with my vestibular system but in general, the progress is steady. My forehead becomes cool for longer and longer periods, not every day, but often. I usually wake up with a cool forehead.
My very large family was coming in waves to visit over memorial day weekend, with second and third generations arriving also.
This is the first time in at least 10 years that all my siblings have gotten together. One of my brothers lives in Nepal, so his presence in the US is quite sporadic. And my brother and his wife who live close to me in Chester County are moving to Ecuador at the end of the summer. Two of my brothers live in New England and my sister lives in California; some of the family lives in Seattle, and some live in the Midwest. My daughter and her family drove up from Western Virginia.
Each of my four brothers had made special trips to see me last November, because I was so weak, it had appeared to them (and me) that I was in the process of dying.
And they were so happy to see me after the removal of the AMR meter. They had remarked when I had spoken with them on the phone (as had many others of my friends) hat I had new energy in my voice, but seeing me in person, being able to stand up, walk around, go up and down the stairs, cook and clean up, help my granddaughters ride a horse… this was remarkable progress. We had 18 people for lunch and dinner for two days straight. And many folks sleeping over in various rooms with blow up beds. Two of my brothers celebrated their birthdays at my home, including one brother who turned 70 on May 30, and one who turned 72 on May 27. It was a very joyous and memorable occasion.

I did have one set back though, which was the morning of May 30. I woke up early with a headache. My headache lasted much of the day. Initially, I didn't know what caused it because all the family knew to put their phones in airplane mode. And to go outside if they had to use their cell phones.
I found out that my sister's old flip phone cell phone does not have airplane mode, and she had left it on all night charging in an outlet which was only 12 feet from where my head was positioned, as I lay in bed, through several plasterboard walls, but still, I was affected.
She moved her cell phone out of the house for the remainder of her stay at my house and I had no further headache incidents.

APPENDIX K

APPENDIX K:

Listing of the Murphy AMR meter emission times in videos taken on April __, 2016, and of a PECO in situ AMI meter emission times in videos taken on April __, 2016

PECO meter measured emissions from AMR meter and AMI meter

Measured and videotaped pulsed emissions in situ on two PECO customer homes:

1. NOTE: This PECO AMI meter was installed by PECO in November 2015. All videos of the PECO AMI meter included in this Appendix B were filmed at the same PECO customer's home on the same afternoon on April __, 2016. The homeowners where this PECO AMI meter is located do not have any "smart" appliances for the Zigbee to link up with, and several of the family members have been adversely affected by EMF since the installation of the AMI meter. The customer is trying to work with PECO to resolve the issues of EMF and dirty electricity since the installation of the AMI meters in the neighborhood without having to file a formal complaint:

Video labeled: "AMI Flexnet first 8 second" video:

This video is 27 seconds long. It captured one Flexnet pulse in the first 8 seconds and one Zigbee pulsation.

This video was filmed as we were setting up and before the introduction could be announced, **so we stopped the video, and started it again with the following video, number 2 below, approximately 3 minutes later.**

2. **Video labeled: "ami meter 3 flexnets in 29 minutes" video:**

This video is 33 minutes long.

Here are the elapsed times of the Zigbee pulsed EMF emissions:

25 seconds
41 seconds
56 seconds
1 minute 11 seconds
1 minute 26 seconds
1 minute 41 seconds
1 minute 56 seconds
2 minutes 11 seconds
2 minutes 25 seconds
2 minutes 41 seconds
2 minutes 49 seconds

2 minutes 55 seconds
3 minutes 3 seconds
3 minutes 10 seconds
3 minutes 16 seconds
3 minutes 26 seconds
3 minutes 41 seconds
3 minutes 55 seconds
(at 4 minutes: You can hear a sawing sound like the Flexnet, but low level intensity, so I am not sure what that pulse was)
4 minutes 11 seconds
4 minutes 26 seconds

at 4 minutes 28 seconds, the Flexnet pulsed yet another EMF emission. This is certainly less than 90 minutes from the first video Flexnet pulsed EMF emission!!! (more like 6 minutes after the previous video Flexnet pulsed emission!!)

Zigbee emissions (continued):

4 minutes 56 seconds
5 minutes 10 seconds
5 minutes 26 seconds
5 minutes 40 seconds
5 minutes 56 seconds
6 minutes 10 seconds
6 minutes 25 seconds
6 minutes 41 seconds
6 minutes 55 seconds

(The Zigbee continues to emit pulsation of wireless EMF at varying intervals for the rest of the 33 minute video)

Summary of Flexnet pulsed EMF emissions in videos 1 and 2:

1. One Flexnet pulsed emission in the previous video
One unquantifiable pulsed emission with saw-like sound at 4 minutes

2. One Flexnet pulsed emission at 4 minutes 28 seconds (approximately 6 minutes after the Flexnet emission in previous video)

3. One Flexnet pulsed emission at 32 minutes 15 seconds

4. Last Flexnet pulsed emission at 32 minutes 54 seconds

3. Video labeled: "AMI meter 3 or 4 zigbees in one minute"

Zigbee pulsed EMF emissions lapsed times:

13 seconds
28 seconds
32 seconds
58 seconds

4. Video labeled: AMI meter 14 zigbee 2 flexnets 3 minutes" video:

This video may have been misnamed. The sawlike buzzing noises similar to Flexnet emissions were not of great intensity. Still, they are noted below:

Zigbee pulsed EMF emissions;

3 seconds
18 seconds
32 seconds

At 40 seconds: low level intensity saw like buzzing similar to Flexnet but not as intense

48 or 50 seconds
1 minute and 3 or 4 seconds
1 minute and 9 seconds
1 minute and 18 seconds
1 minute and 33 or 34 seconds
1 minute and 48 seconds
2 minutes and 2 seconds
2 minutes and 9 seconds
2 minutes and 18 seconds
2 minutes and 33 seconds
2 minutes and 48 seconds

At 2 minutes and 56 seconds, again, a low intensity buzzing sound similar to the Flexnet but not as intense.

3 minutes and 3 seconds

4. The following two videos were taken at the Murphy residence on April _____, 2016.

Video labeled: "lsm amr meter 6 spikes in 11 minutes":

Note: This is the first video taken at the Murphy home. It is 10 minutes and 52 seconds long. It is difficult to see my gigabyte meter from the placement of the chair, but the viewer

can make out the gigabyte meter reading, and also can hear the pulsed EMF emissions coming from the PECO AMR meter.

Pulsed EMF emissions from the Murphy AMR meter elapsed times:

13 seconds
1 minute 58 seconds
4 minutes 47 seconds
5 minutes 31 seconds
7 minutes 12 seconds
9 minutes 55 seconds

5. Video labeled: **"lsm amr meter 21 minutes 4 zaps":**

Note: This is the second video taken at the Murphy residence on April ___, 2016. It is 21 minutes long. We had duck taped the door of the meter enclosure open and rearranged the gigabyte support, so that the gigabyte meter face is more easily seen than in the first AMR video.

Pulsed EMF emissions from the Murphy AMR meter elapsed times:

3 minutes and 29 seconds
7 minutes and 57 seconds
12 minutes and 4 or 5 seconds
15 minutes and 8 seconds

Full videos at:
https://onedrive.live.com/redir?resid=56AA90065E6ED459!651&authkey=!AF65pvK7YEXA0Nw&ithint=folder%2cm4v

APPENDIX L

APPENDIX M

APPENDIX M

LIST OF SUPPLEMENTS AS OF MARCH 9, 2016

Vitamin C 1000 mg 2 to 3 times a day
MSN
L glutamine
Protandim complex
vitamins A&D
acetyl l-carnitine
Vitamin B injections once a week
Magnesium citrate three times a day
Beta carotene
Zinc
Potassium
NAC twice a day
L glutamine
Vitamin B 6
Vitamin B 12
B vitamin injections once per week
Inositol
Taurine
Pycnogenol
Glucosamine and condroitin
Choline
Selenium
Swanson's leg vein essentials
Iron infusions
Vit d transdermal patches with vit k
Lumbrokinase
Chromium
Collagen
Krill oil
Probiotics three times a day
Digestive enzymes
Pancreatic enzymes
Aloe vera juice
5 htp
L tyrosine

APPENDIX N

Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation in Hsd: Sprague Dawley® SD rats (Whole Body Exposures)

Draft 5-19-2016

Table of Contents

Abstract

The US National Toxicology Program (NTP) has carried out extensive rodent toxicology and carcinogenesis studies of radiofrequency radiation (RFR) at frequencies and modulations used in the US telecommunications industry. This report presents partial findings from these studies. The occurrences of two tumor types in male Harlan Sprague Dawley rats exposed to RFR, malignant gliomas in the brain and schwannomas of the heart, were considered of particular interest, and are the subject of this report. The findings in this report were reviewed by expert peer reviewers selected by the NTP and National Institutes of Health (NIH). These reviews and responses to comments are included as appendices to this report, and revisions to the current document have incorporated and addressed these comments. Supplemental information in the form of 4 additional manuscripts has or will soon be submitted for publication. These manuscripts describe in detail the designs and performance of the RFR exposure system, the dosimetry of RFR exposures in rats and mice, the results to a series of pilot studies establishing the ability of the animals to thermoregulate during RFR exposures, and studies of DNA damage.

Capstick M, Kuster N, Kühn S, Berdinas-Torres V, Wilson P, Ladbury J, Koepke G, McCormick D, Gauger J, Melnick R. A radio frequency radiation reverberation chamber exposure system for rodents

Yijian G, Capstick M, McCormick D, Gauger J, Horn T, Wilson P, Melnick RL and Kuster N. Life time dosimetric assessment for mice and rats exposed to cell phone radiation

Wyde ME, Horn TL, Capstick M, Ladbury J, Koepke G, Wilson P, Stout MD, Kuster N, Melnick R, Bucher JR, and McCormick D. Pilot studies of the National Toxicology Program's cell phone radiofrequency radiation reverberation chamber exposure system

Smith-Roe SL, Wyde ME, Stout MD, Winters J, Hobbs CA, Shepard KG, Green A, Kissling GE, Tice RR, Bucher JR, Witt KL. Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure

Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation in Hsd: Sprague Dawley® SD rats (Whole Body Exposures)

Draft 5-19-2016

SUMMARY

The purpose of this communication is to report partial findings from a series of radiofrequency radiation (RFR) cancer studies in rats performed under the auspices of the U.S. National Toxicology Program (NTP).[1] This report contains peer-reviewed, neoplastic and hyperplastic findings only in the brain and heart of Hsd:Sprague Dawley® SD® (HSD) rats exposed to RFR starting *in utero* and continuing throughout their lifetimes. These studies found low incidences of malignant gliomas in the brain and schwannomas in the heart of male rats exposed to RFR of the two types [Code Division Multiple Access (CDMA) and Global System for Mobile Communications (GSM)] currently used in U.S. wireless networks. Potentially preneoplastic lesions were also observed in the brain and heart of male rats exposed to RFR.

The review of partial study data in this report has been prompted by several factors. Given the widespread global usage of mobile communications among users of all ages, even a very small increase in the incidence of disease resulting from exposure to RFR could have broad implications for public health. There is a high level of public and media interest regarding the safety of cell phone RFR and the specific results of these NTP studies.

[1] NTP is a federal, interagency program, headquartered at the National Institute of Environmental Health Sciences, part of the National Institutes of Health, whose goal is to safeguard the public by identifying substances in the environment that may affect human health. For more information about NTP and its programs, visit http://ntp.niehs.nih.gov

Lastly, the tumors in the brain and heart observed at low incidence in male rats exposed to GSM- and CDMA-modulated cell phone RFR in this study are of a type similar to tumors observed in some epidemiology studies of cell phone use. These findings appear to support the International Agency for Research on Cancer (IARC) conclusions regarding the possible carcinogenic potential of RFR.[2]

It is important to note that this document reviews only the findings from the brain and heart and is not a complete report of all findings from the NTP's studies. Additional data from these studies in Hsd:Sprague Dawley® SD® (Harlan) rats and similar studies conducted in B6C3F$_1$/N mice are currently under evaluation and will be reported together with the current findings in two forthcoming NTP Technical Reports.

STUDY RATIONALE

Cell phones and other commonly used wireless communication devices transmit information via non-ionizing radiofrequency radiation (RFR). In 2013, IARC classified RFR as a *possible human carcinogen* based on "limited evidence" of an association between exposure to RFR from heavy wireless phone use and glioma and acoustic neuroma (vestibular schwannoma) in human epidemiology studies, and "limited evidence" for the carcinogenicity of RFR in experimental animals. While ionizing radiation is a well-accepted human carcinogen, theoretical arguments have been raised against the possibility that non-ionizing radiation could induce tumors (discussed in IARC, 2013). Given the extremely large number of people who use wireless

[2] IARC (International Agency for Research on Cancer). 2013. Non-Ionizing Radiation, Part 2: Radiofrequency Electromagnetic Fields. IARC Monogr Eval Carcinog Risk Hum 102. Available: http://monographs.iarc.fr/ENG/Monographs/vol102/mono102.pdf [accessed 26 May 2016].

communication devices, even a very small increase in the incidence of disease resulting from exposure to the RFR generated by those devices could have broad implications for public health.

DESCRIPTION OF THE NTP CELL PHONE RFR PROGRAM

RFR emitted by wireless communication devices, especially cell phones, was nominated to the NTP for toxicology and carcinogenicity testing by the U.S. Food and Drug Administration (FDA). After careful and extensive evaluation of the published literature and experimental efforts already underway at that time, the NTP concluded that additional studies were warranted to more clearly define any potential health hazard to the U.S. population. Due to the technical complexity of such studies, NTP staff worked closely with RFR experts from the National Institute of Standards and Technology (NIST). With support from NTP, engineers at NIST evaluated various types of RFR exposure systems and demonstrated the feasibility of using a specially designed exposure system (reverberation chambers), which resolved the inherent limitations identified in existing systems.

In general, NTP chronic toxicity/carcinogenicity studies expose laboratory rodents to a test article for up to 2 years and are designed to determine the potential for the agent tested to be hazardous and/or carcinogenic to humans.[3] For cell phone RFR, a program of study was designed to evaluate potential, long-term health effects of whole-body exposures. These studies were conducted in three phases: (1) a series of pilot studies to establish field strengths that do not raise body temperature, (2) 28-day toxicology studies in rodents exposed to various low-level field strengths, and (3) chronic toxicology and carcinogenicity studies. The studies were carried out under contract at IIT Research Institute (IITRI) in Chicago, IL following Good Laboratory

[3] Specifications for the Conduct of NTP Studies. http://ntp.niehs.nih.gov/ntp/test_info/finalntp_toxcarspecsjan2011.pdf

Practices (GLP). These studies were conducted in rats and mice using a reverberation chamber exposure system with two signal modulations [Code Division Multiple Access (CDMA) and Global System for Mobile Communications (GSM)] at two frequencies (900 MHz for rats and 1900 MHz for mice), the modulations and frequency bands that are primarily used in the United States.

STUDY DESIGN

Hsd:Sprague Dawley® SD® (Harlan) rats were housed in custom-designed reverberation chambers and exposed to cell phone RFR. Experimentally generated 900 MHz RF fields with either GSM or CDMA modulation were continuously monitored in real-time during all exposure periods via RF sensors located in each exposure chamber that recorded RF field strength (V/m). Animal exposure levels are reported as whole-body specific absorption rate (SAR), a biological measure of exposure based on the deposition of RF energy into an absorbing organism or tissue. SAR is defined as the energy (watts) absorbed per mass of tissue (kilograms). Rats were exposed to GSM- or CDMA-modulated RFR at 900 MHz with whole-body SAR exposures of 0, 1.5, 3, or 6 W/kg. RFR field strengths were frequently adjusted based on changes in body weight to maintain desired SAR levels.

Exposures to RFR were initiated *in utero* beginning with the exposure of pregnant dams (approximately 11-14 weeks of age) on Gestation Day (GD) 5 and continuing throughout gestation. After birth, dams and pups were exposed in the same cage through weaning on postnatal day (PND) 21, at which point the dams were removed and exposure of 90 pups per sex per group was continued for up to 106 weeks. Pups remained group-housed from PND 21 until they were individually housed on PND 35. Control and treatment groups were populated with no

more than 3 pups per sex per litter. All RF exposures were conducted over a period of approximately 18 hours using a continuous cycle of 10 minutes on (exposed) and 10 minutes off (not exposed), for a total daily exposure time of approximately 9 hours a day, 7 days/week. A single, common group of unexposed animals of each sex served as controls for both RFR modulations. These control rats were housed in identical reverberation chambers with no RF signal generation. Each chamber was maintained on a 12-hour light/dark cycle, within a temperature range of 72 ± 3°F, a humidity range of 50 ± 15%, and with at least 10 air changes per hour. Throughout the studies, all animals were provided *ad libitum* access to feed and water.

RESULTS

In pregnant rats exposed to 900 MHz GSM- or CDMA-modulated RFR, no exposure-related effects were observed on the percent of dams littering, litter size, or sex distribution of pups. Small, exposure-level-dependent reductions (up to 7%) in body weights compared to controls were observed throughout gestation and lactation in dams exposed to GSM- or CDMA-modulated RFR. In the offspring, litter weights tended to be lower (up to 9%) in GSM and CDMA RFR-exposed groups compared to controls. Early in the lactation phase, body weights of male and female pups were lower in the GSM-modulated (8%) and CDMA-modulated (15%) RFR groups at 6 W/kg compared to controls. These weight differences in the offspring for both GSM and CDMA exposures tended to lessen (6% and 10%, respectively) as lactation progressed. Throughout the remainder of the chronic study, no RFR exposure-related effects on body weights were observed in male and female rats exposed to RFR, regardless of modulation.

At the end of the 2-year study, survival was lower in the control group of males than in all groups of male rats exposed to GSM-modulated RFR. Survival was also slightly lower in control females than in females exposed to 1.5 or 6 W/kg GSM-modulated RFR. In rats exposed to CDMA-modulated RFR, survival was higher in all groups of exposed males and in the 6 W/kg females compared to controls.

Brain

A low incidence of malignant gliomas and glial cell hyperplasia was observed in all groups of male rats exposed to GSM-modulated RFR (Table 1). In males exposed to CDMA-modulated RFR, a low incidence of malignant gliomas occurred in rats exposed to 6 W/kg (Table 1). Glial cell hyperplasia was also observed in the 1.5 W/kg and 6 W/kg CDMA-modulated exposure groups. No malignant gliomas or glial cell hyperplasias were observed in controls. There was not a statistically significant difference between the incidences of lesions in exposed male rats compared to control males for any of the GSM- or CDMA-modulated RFR groups. However, there was a statistically significant positive trend in the incidence of malignant glioma ($p < 0.05$) for CDMA-modulated RFR exposures.

Table 1. Incidence of brain lesions in male Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Malignant glioma [†‡]	0[*]	3 (3.3%)	3 (3.3%)	2 (2.2%)	0	0	3 (3.3%)
Glial cell hyperplasia	0	2 (2.2%)	3 (3.3%)	1 (1.1%)	2 (2.2%)	0	2 (2.2%)

[§] Data presented as number of animals per group with lesions (percentage of animals per group with lesions).
[*] Significant SAR-dependent trend for CDMA exposures by poly-6 ($p < 0.05$). See appendix B
[†] Poly-6 survival adjusted rates for malignant gliomas were 0/53.48 in controls; GSM: 3/67.96 (4.4%), 3/72.10 (4.2%), and 2/72.65 (2.8%) in the 1.5, 3, and 6 W/kg groups, respectively; CDMA: 0/65.94, 0/73.08, and 3/57.49 (5.2%) for the 1.5, 3, and 6 W/kg groups, respectively.
[‡] Historical control incidence in NTP studies: 11/550 (2.0%), range 0-8%

In females exposed to GSM-modulated RFR, a malignant glioma was observed in a single rat exposed to 6 W/kg, and glial cell hyperplasia was observed in a single rat exposed to 3 W/kg (Table 2). In females exposed to CDMA-modulated RFR, malignant gliomas were observed in two rats exposed to 1.5 W/kg. Glial cell hyperplasia was observed in one female in each of the CDMA-modulation exposure groups (1.5, 3, and 6 W/kg). There was no glial cell hyperplasia or malignant glioma observed in any of the control females. Detailed descriptions of the malignant gliomas and glial cell hyperplasias are presented in Appendix C.

Table 2. Incidence of brain lesions in female Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Malignant glioma [‡]	0	0	0	1 (1.1%)	2 (2.2%)	0	0
Glial cell hyperplasia	0	0	1 (1.1%)	0	1 (1.1%)	1 (1.1%)	1 (1.1%)

[§] Data presented as number of animals per group with lesions (percentage of animals per group with lesions).
[‡] Historical control incidence in NTP studies: 1/540 (0.18%), range 0-2%

Heart

Cardiac schwannomas were observed in male rats in all exposed groups of both GSM- and CDMA-modulated RFR, while none were observed in controls (Table 3). For both modulations (GSM and CDMA), there was a significant positive trend in the incidence of schwannomas of the heart with respect to exposure SAR. Additionally, the incidence of schwannomas in the 6 W/kg males was significantly higher in CDMA-modulated RFR-exposed males compared to controls. The incidence of schwannomas in the 6 W/kg GSM-modulated RFR-exposed males was higher, but not statistically significant ($p = 0.052$) compared to controls. Schwann cell

hyperplasia of the heart was also observed in three males exposed to 6 W/kg CDMA-modulated RFR. In the GSM-modulation exposure groups, a single incidence of Schwann cell hyperplasia was observed in a 1.5 W/kg male.

Table 3. Incidence of heart lesions in male Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated cell phone RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Schwannoma [†‡]	0*	2 (2.2%)	1 (1.1%)	5 (5.5%)	2 (2.2%)	3 (3.3%)	6 (6.6%)**
Schwann cell hyperplasia	0	1 (1.1%)	0	2 (2.2%)	0	0	3 (3.3%)

§ Data presented as number of animals per group with lesions (percentage of animals per group with lesions).
* Significant SAR level-dependent trend for GSM and CDMA by poly-3 ($p < 0.05$). See appendix B
** Significantly higher than controls by poly-3 ($p < 0.05$)
† Poly-3 survival adjusted rates for schwannomas were 0/65.47 in controls; GSM: 2/74.87 (2.7%), 1/77.89 (1.3%), and 5/78.48 (6.4%) in the 1.5, 3, and 6 W/kg groups, respectively; CDMA: 2/74.05 (2.7%), 3/78.67 (3.8%), and 6/67.94 (8.8%) for the 1.5, 3, and 6 W/kg groups, respectively.
‡ Historical control incidence in NTP studies: 9/699 (1.3%) range 0-6%

In females, schwannomas of the heart were also observed at 3 W/kg GSM-modulated RFR and 1.5 and 6 W/kg CDMA-modulated RFR. Schwann cell hyperplasia was observed in one female in each of the CDMA-modulation exposure groups (1.5, 3, and 6 W/kg).

Table 4. Incidence of heart lesions in female Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated cell phone RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Schwannoma[‡]	0	0	2 (2.2%)	0	2 (2.2%)	0	2 (2.2%)
Schwann cell hyperplasia	0	0	0	0	1 (1.1%)	1 (1.1%)	1 (1.1%)

§ Data presented as number of animals per group with tumors (percentage of animals per group with tumors).
‡ Historical control incidence in NTP studies: 4/699 (0.6 %), range 0-4%

Schwann cells are present in the peripheral nervous system and are distributed throughout the whole body, not just in the heart. Therefore, organs other than the heart were examined for schwannomas and Schwann cell hyperplasia. Several occurrences of schwannomas were observed in the head, neck, and other sites throughout the body of control and GSM and CDMA RFR-exposed male rats. In contrast to the significant increase in the incidence of schwannomas in the heart of exposed males, the incidence of schwannomas observed in other tissue sites of exposed males (GSM and CDMA modulations) was not significantly different than in controls (Table 5). Additionally, Schwann cell hyperplasia was not observed in any tissues other than the heart. The combined incidence of schwannomas from all sites was generally higher in GSM- and CDMA-modulated RFR exposed males, but not significantly different than in controls. The Schwann cell response to RFR appears to be specific to the heart of male rats.

Table 5. Incidence of schwannomas in male Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Heart[‡]	0[*]	2 (2.2%)	1 (1.1%)	5 (5.5%)	2 (2.2%)	3 (3.3%)	6 (6.6%)[**]
Other sites[†]	3 (3.3%)	1 (1.1%)	4 (4.4%)	2 (2.2%)	2 (2.2%)	1 (1.1%)	1 (1.1%)
All sites (total)	3 (3.3%)	3 (3.3%)	5 (5.5%)	7 (7.7%)	4 (4.4%)	4 (4.4%)	7 (7.7%)

[§] Data presented as number of animals per group with tumors (percentage of animals per group with tumors).
[*] Significant SAR level-dependent trend for GSM and CDMA, poly 3 test ($p < 0.05$)
[**] Significantly higher than controls, poly-3 test ($p < 0.05$)
[‡] Historical control incidence in NTP studies: 9/699 (1.3%), range 0-6%
[†] Mediastinum, thymus, and fat

In female rats, there was no statistically significant or apparent exposure-related effect on the incidence of schwannomas in the heart or the combined incidence in the heart or other sites (Table 6).

Table 6. Incidence of schwannomas in female Hsd:Sprague Dawley® SD® (Harlan) rats exposed to GSM- or CDMA-modulated RFR[§]

Schwannoma site	Control	GSM			CDMA		
	0 W/kg	1.5 W/kg	3 W/kg	6 W/kg	1.5 W/kg	3 W/kg	6 W/kg
Number examined	90	90	90	90	90	90	90
Heart[‡]	0	0	2 (2.2%)	0	2 (2.2%)	0	2 (2.2%)
Other sites[†]	4 (4.4%)	1 (1.1%)	3 (3.3%)	1 (1.1%)	0	2 (2.2%)	2 (2.2%)
All sites (total)	4 (4.4%)	1 (1.1%)	5 (5.5%)	2 (2.2%)	2 (2.2%)	2 (2.2%)	4 (4.4%)

[§] Data presented as number of animals per group with tumors (percentage of animals per group with tumors).
[‡] Historical control incidence in NTP studies: 4/699 (0.6%), range 0-4%
[†] Ovary, uterus, vagina, thymus, abdomen, and clitoral gland

DISCUSSION

The two tumor types, which are the focus of this report, are malignant gliomas of the brain and schwannomas of the heart. Glial cells are a collection of specialized, non-neuronal, support cells whose functions include maintenance of homeostasis, formation of myelin, and providing support and protection for neurons of the peripheral nervous system (PNS) and the central nervous system (CNS). In the CNS, glial cells include astrocytes, oligodendrogliocytes, microglial cells, and ependymal cells. Schwann cells are classified as glial cells of the PNS. In the PNS, Schwann cells produce myelin and are analogous to oligodendrocytes of the CNS. Generally, glial neoplasms in the rat are aggressive, poorly differentiated, and usually classified as malignant.

In the heart, exposure to GSM or CDMA modulations of RFR in male rats resulted in a statistically significant, positive trend in the incidence of schwannomas. There was also a statistically significant, pairwise increase at the highest CDMA exposure level tested compared to controls. Schwann cell hyperplasias also occurred at the highest exposure level of CDMA-

modulated RFR. Schwann cell hyperplasia in the heart may progress to cardiac schwannomas. No Schwann cell hyperplasias or schwannomas of the heart were observed in the single, common control group of male rats. The historical control rate of schwannomas of the heart in male Harlan Sprague Dawley rats is 1.30% (7/539) and ranges from 0-6% for individual NTP studies (Table D2, Appendix D). The 5.5-6.6% observed in the 6 W/kg GSM- and CDMA-modulated RFR groups exceeds the historical incidence, and approaches or exceeds the highest rate observed in a single study (6%). The increase in the incidence of schwannomas in the heart of male rats in this study is likely the result of whole-body exposures to GSM- or CDMA-modulated RFR.

In the brain, there was a significant, positive trend in the incidences of malignant gliomas in males exposed to CDMA-modulated RFR, and a low incidence was observed in males at all exposure levels of GSM-modulated RFR that was not statistically different than in control males. Glial cell hyperplasia, a preneoplastic lesion distinctly different from gliosis, was also observed at low incidences in rats exposed to either GSM or CDMA modulation. Glial cell hyperplasia may progress to malignant glioma. Neither of these lesions was observed in the control group of male rats. Although not observed in the current control group, malignant gliomas have been observed in control male Harlan Sprague Dawley rats from other completed NTP studies. Currently in males, the historical control rate of malignant glioma for those studies is 2.0% (11/550) and ranges from 0-8% for individual studies (Table D1, Appendix D). The 2.2-3.3% observed in all of the GSM-modulation groups and in the 6 W/kg CDMA-modulated group only slightly exceeds the mean historical control rate and falls within the observed range.

The survival of the control group of male rats in the current study (28%) was relatively low compared to other recent NTP studies in Hsd:Sprague Dawley® SD® (Harlan) rats (average 47%, range 24-72%). If malignant gliomas or schwannomas are late-developing tumors, the absence of these lesions in control males in the current study could conceivably be related to the shorter longevity of control rats in this study. Appendix E lists the time on study for each animal with a malignant glioma or heart schwannoma. Most of the gliomas were observed in animals that died late in the study, or at the terminal sacrifice. However, a relatively high number of the heart schwannomas in exposed groups were observed by 90 weeks into the study, a time when approximately 60 of the 90 control male rats remained alive and at risk for developing a tumor.

CONCLUSIONS

Under the conditions of these 2-year studies, the hyperplastic lesions and glial cell neoplasms of the heart and brain observed in male rats are considered likely the result of whole-body exposures to GSM- or CDMA-modulated RFR. There is higher confidence in the association between RFR exposure and the neoplastic lesions in the heart than in the brain. No biologically significant effects were observed in the brain or heart of female rats regardless of modulation.

NEXT STEPS

The results reported here are limited to select findings of concern in the brain and heart and do not represent a complete reporting of all findings from these studies of cell phone RFR. The complete results for all NTP studies on the toxicity and carcinogenicity of GSM and CDMA-modulated RFR are currently being reviewed and evaluated according to the established NTP process and will be reported together with the current findings in two forthcoming NTP

Technical Reports. Given the large scale and scope of these studies, completion of this process is anticipated by fall 2017, and the draft NTP Technical Reports are expected to be available for peer review and public comment by the end of 2017. We anticipate that the results from a series of initial studies investigating the tolerance to various power levels of RFR, including measurements of body temperatures in both sexes of young and old rats and mice and in pregnant female rats, will be published in the peer-reviewed literature later in 2016.

APPENDIX A – CONTRIBUTORS

NTP CONTRIBUTORS

Participated in the evaluation and interpretation of results and the reporting of findings.

M.E. Wyde, Ph.D. (NTP study scientist)
M.F. Cesta, D.V.M., Ph.D. (NTP pathologist)
C.R. Blystone, Ph.D.
J.R. Bucher, Ph.D.
S.A. Elmore, D.V.M., M.S.
P.M. Foster, Ph.D.
M.J. Hooth, Ph.D.
G.E. Kissling, Ph.D.
D.E. Malarkey, D.V.M., Ph.D.
R.C. Sills, D.V.M., Ph.D.
M.D. Stout, Ph.D.
N.J. Walker, Ph.D.
K.L. Witt, M.S.
M.S. Wolfe, Ph.D.

APPENDIX B – STATISTICAL ANALYSIS

The Poly-k test (Bailer and Portier, 1988; Portier and Bailer, 1989; Piegorsch and Bailer, 1997) was used to assess neoplasm prevalence. This test is a survival-adjusted quantal-response procedure that modifies the Cochran-Armitage linear trend test to take survival differences into account. More specifically, this method modifies the denominator in the quantal estimate of lesion incidence to approximate more closely the total number of animal years at risk. For analysis of lesion incidence at a given site, each animal is assigned a risk weight. This value is one if the animal had a lesion at that site or if it survived until terminal sacrifice; if the animal died prior to terminal sacrifice and did not have a lesion at that site, its risk weight is the fraction of the entire study time that it survived, raised to the kth power. This method yields a lesion prevalence rate that depends only upon the choice of a shape parameter, k, for a Weibull hazard function describing cumulative lesion incidence over time (Bailer and Portier, 1988). A further advantage of the Poly-k method is that it does not require lesion lethality assumptions.

Unless otherwise specified, the NTP uses a value of k=3 in the analysis of site-specific lesions (Portier et al., 1986). Bailer and Portier (1988) showed that the Poly-3 test gives valid results if the true value of k is anywhere in the range from 1 to 5. In addition, Portier et al. (1986) modeled a collection of relatively common tumors observed in control animals from two-year NTP rodent carcinogenicity studies, showing that the Weibull distribution with values of k ranging between 1 and 5 was a reasonable fit to tumor incidence in most cases. In cases of early tumor onset or late tumor onset, however, k=3 may not be the optimal choice. Tumors with early onset would require a value of k much less than 3, while tumors with late onset would require a value of k much greater than 3. In the current studies, malignant brain gliomas occurred only in animals surviving more than 88% of the length of the study. For these brain tumors, a Weibull distribution with k=6 is a better fit to survival time than with k=3 (Portier, 1986). Malignant schwannomas of the heart occurred in animals surviving at least 65% of the length of the study; a Weibull distribution with k=3 adequately fits these heart tumor incidences. Therefore, poly-6 tests were used for analyses of brain tumors and poly-3 tests were used for schwannomas.

Variation introduced by the use of risk weights, which reflect differential mortality, was accommodated by adjusting the variance of the Poly-k statistic as recommended by Bieler and Williams (1993) and a continuity correction modified from Thomas et al. (1977) was applied.

Tests of significance for tumors and nonneoplastic lesions included pairwise comparisons of each dosed group with controls and a test for an overall dose-related trend. Continuity-corrected Poly-k tests were used in the analysis of lesion incidence, and reported P values are one sided.

Body weights and litter weights were compared to the control group using analysis of variance and Dunnett's test (1955). The probability of survival was estimated by the product-limit procedure of Kaplan and Meier (1958). Statistical analyses for possible exposure-related effects on survival used Cox's (1972) method for testing two groups for equality and Tarone's (1975) life table test to identify exposure-related trends. Survival analysis p-values are two-sided.

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APPENDIX C – PATHOLOGY

Pathology data presented in this report on cell phone RFR were subjected to a rigorous peer review process. The primary goal of the NTP peer-review process is to reach consensus agreement on treatment-related findings, confirm the diagnosis of all neoplasms, and confirm any unusual lesions. At study termination, a complete necropsy and histopathology evaluation was conducted on every animal. The initial pathology examination was performed by a veterinary pathologist, who recorded all neoplastic and nonneoplastic lesions. This examination identified several potential treatment-related lesions in target organs of concern (brain and heart), which were chosen for immediate review.[1] The initial findings of glial cell tumors and hyperplasias in the brain and schwannomas, Schwann call hyperplasia, and schwannomas from all sites were subjected to an expedited, multilevel NTP pathology peer-review process. The data were locked[2] prior to receipt of the finalized, study-laboratory reports to ensure that the raw data did not change during the review.

The pathology peer review consisted of a quality assessment (QA) review of all slides with tissues from the central nervous system (7 sections of brain and 3 sections of spinal cord), trigeminal nerve and ganglion, and heart. Additionally, the schwannomas of the head and neck region were reviewed. The QA review of the central nervous system and head and neck schwannomas was performed by Dr. Margarita Gruebbel of Experimental Pathology Laboratories, Inc. (EPL), and the QA review of the hearts and trigeminal nerves and ganglia was performed by Dr. Cynthia Shackelford, EPL.

The QA review pathologists then met with Dr. Mark Cesta, NTP pathologist for these studies, and Dr. David Malarkey, head of the NTP Pathology Group, to review lesions and select slides for the Pathology Working Group (PWG) reviews. All PWG reviews were conducted blinded with respect to treatment group and only identified the test articles as "test agent A" or "test

[1] Pathology peer review of remaining lesions from the cell phone RFR studies continues and is not addressed in this report.

[2] Locking data refers to restricting access to the computer database so the data for a particular study cannot be changed.

agent B". Due to the large number of slides for review, the PWG was held in three separate sessions:

- January 29, 2016, for review of glial lesions in the brain and Schwann cell lesions in the heart
- February 11, 2016, for review of schwannomas of the head and neck
- February 12, 2016, for review of granular cell lesions of the brain

The reviewing PWG pathologists largely agreed on the diagnostic criteria for the lesions and on the diagnoses of schwannomas in the head and neck, and granular cell lesions in the brain. However, there was much discussion on the criteria for differentiating glial cell hyperplasia from malignant glioma and Schwann cell hyperplasia from schwannoma. The lack of PWG agreement on definitive criteria for the glial cell and Schwann cell lesions, and the requirement for a high level of confidence in the diagnoses prompted NTP to convene two additional PWGs (organized and conducted by the NTP pathologist, Dr. Mark Cesta) with selected experts in the organ under review. These second level PWG reviews were also conducted as noted above and held in two separate sessions:

- February 25, 2016, for review of glial lesions in the brain
- March 3, 2016, for review of cardiac schwannomas, schwannomas in other organs (except the head and neck), and right ventricular degeneration

In both PWGs, the participants came to consensus on the diagnoses of the lesions and the criteria used for those diagnoses. Participants of the individual PWGs are listed below.

Table C-1. NTP Pathology Working Group (PWG) Attendees

PWG member	Affiliation
January 29, 2016 - Evaluated glial lesions in the brain and Schwann cell lesions in the heart	
A.E. Brix, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences
G.P. Flake, M.D.	National Institute of Environmental Health Sciences
R.H. Garman, D.V.M.	Consultants in Veterinary Pathology, Inc. Monroeville, PA
M.M. Gruebbel, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC (observer)
R.A. Herbert, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
J.S. Hoane, D.V.M.	Charles River Laboratories, Inc. Durham, NC (contract study pathologist)
K.S. Janardhan, BVSc, MVSc, Ph.D.	Integrated Laboratory System
R. Kovi, BVSc, MVSc, Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC (observer)
D.E. Malarkey, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
R.A. Miller, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
J.P. Morrison, D.V.M.	Charles River Laboratories, Inc. Durham, NC

PWG member	Affiliation
A.R. Pandiri, BVSc & AH, Ph.D.	National Institute of Environmental Health Sciences
C.C. Shackelford, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC (observer)
J.A. Swenberg, D.V.M., Ph.D.	University of North Carolina – Chapel Hill, NC
G. Willson, BVMS, Dip RC Path, FRC Path, MRCVS	Experimental Pathology Laboratories, Inc. RTP, NC (PWG coordinator)
February 11, 2016 - Evaluated schwannomas of the head and neck	
A.E. Brix, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences
G.P. Flake, M.D.	National Institute of Environmental Health Sciences
M.M. Gruebbel, D.V.M., Ph.D.,	Experimental Pathology Laboratories, Inc. RTP, NC (PWG coordinator)
K.S. Janardhan, BVSc, MVSc, Ph.D.	Integrated Laboratory System RTP, NC
D.E. Malarkey, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
A.R. Pandiri, BVSc & AH, Ph.D.	National Institute of Environmental Health Sciences
R.R. Maronpot, D.V.M.	Experimental Pathology Laboratories, Inc. RTP, NC
February 12, 2016 - Evaluated granular cell lesions of the brain	
A.E. Brix, D.V.M., Ph.D.	Experimental Pathology Laboratories, Inc. RTP, NC
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences
M.M. Gruebbel, D.V.M., Ph.D.,	Experimental Pathology Laboratories, Inc. RTP, NC (PWG coordinator)
J.S. Hoane, D.V.M.	Charles River Laboratories, Inc. Durham, NC (contract study pathologist)
K.S. Janardhan, BVSc, MVSc, Ph.D.	Integrated Laboratory System RTP, NC
A.R. Pandiri, BVSc. & AH, Ph.D.	National Institute of Environmental Health Sciences
R.R. Moore, D.V.M.	Integrated Laboratory System RTP, NC
February 25, 2016 - Evaluated glial lesions in the brain	
D. Bigner, M.D., Ph.D.	Duke University Durham, NC
B. Bolon, D.V.M., MS, Ph.D.	GEMpath, Inc. Longmont, CO
V. Chen, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (observer)
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (PWG coordinator, NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences (observer)
G.P. Flake, M.D.	National Institute of Environmental Health Sciences (observer)
J.S. Hardisty, D.V.M.	Experimental Pathology Laboratories, Inc. RTP, NC
R.A. Herbert, D.V.M., Ph.D.,	National Institute of Environmental Health Sciences (observer)
R. Kovi, BVSc, MVSc, Ph.D.	Experimental Pathology Laboratories, Inc. (observer)
P.B. Little, D.V.M.	Experimental Pathology Laboratories, Inc.
D.E. Malarkey, D.V.M., Ph.D.	National Institute of Environmental Health Sciences
J.P. Morrison, D.V.M., Ph.D.	Charles River Laboratories, Inc.
A. Sharma, BVSc, MVSc, MS, Ph.D.	Covance
March 3, 2016 - Evaluated heart lesions, and schwannomas in other organs (except head and neck)	
B. Berridge, D.V.M., Ph.D.	GlaxoSmithKline RTP, NC
M.C. Boyle, D.V.M., Ph.D.	Amgen Thousand Oaks, CA
V. Chen, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (observer)
M.F. Cesta, D.V.M., Ph.D.	National Institute of Environmental Health Sciences (PWG coordinator, NTP study pathologist)
S.A. Elmore, D.V.M., MS	National Institute of Environmental Health Sciences (observer)
M. Elwell, D.V.M., Ph.D.	Covance Chantilly, VA

PWG member	Affiliation
J.R. Hailey, D.V.M.	Covance Chantilly, VA
M. Novilla, D.V.M., MS, Ph.D.	SNBL Everett, WA

LESION DESCRIPTIONS

Brain

Malignant gliomas were infiltrative lesions, usually of modest size, with indistinct tumor margins. The neoplastic cells were typically very densely packed with more cells than neuropil. The cells were typically small and had round to oval, hyperchromatic nuclei. Mitoses were infrequent. In some of the neoplasms, invasion of the meninges, areas of necrosis surrounded by palisading neoplastic cells, cuffing of blood vessels, and neuronal satellitosis were observed. The malignant gliomas did not appear to arise from any specific anatomic subsite of the brain.

Glial cell hyperplasia consisted of small, proliferative, and poorly demarcated foci of poorly differentiated glial cells that accumulated and invaded into the surrounding parenchyma. In some cases, there was a small amount of perivascular cuffing. The hyperplastic cells appeared morphologically identical to those in the gliomas but were typically less dense with more neuropil than glial cells. There were no necrotic or degenerative elements present, so there was no evidence that the increased number of glial cells was a reaction to brain injury.

Heart

The intracardiac schwannomas were either endocardial or myocardial (intramural). The endocardial schwannomas lined the ventricles and atria and invaded into the myocardium. Two morphologic cell types were observed, but indistinct cell margins and eosinophilic cytoplasm were common to both types. Groups of cells with widely spaced small, round nuclei and moderate amounts of cytoplasm were interspersed among bands or sheets of parallel, elongated cells with thin, spindle-shaped, hyperchromatic nuclei. The myocardial schwannomas were typically less densely cellular and infiltrated amid, sometimes replacing, the cardiomyocytes. The cell types described for the endocardial neoplasms were both present, but in fewer numbers. In both subtypes of schwannomas, there was a minimal amount of cellular pleomorphism. In some larger neoplasms, Antoni type A and B patterns were present.

The Schwann cell hyperplasias were similar in appearance to the schwannomas, but were smaller and had less pleomorphism of the cells. In the case of the endocardial Schwann cell hyperplasia, there was no invasion of the myocardium.

APPENDIX D – HISTORICAL CONTROLS

Table D1. Incidence of astrocytoma, glioma, and/or oligodendroglioma in brains of male Harlan Sprague Dawley rats in NTP studies

Chemical	First dose	N	Control incidence
Dibutylphthalate	8/30/2010	49	4%
2-Hydroxy-4-methoxybenzophenone	11/8/2010	50	0%
p-Chloro-a,a,a-trifluorotoluene	1/17/2011	50	4%
Di-(2-ethylhexyl)phthalate	2/17/2011	50	8%
Di-(2-ethylhexyl)phthalate (perinatal)	6/27/2011	50	0%
Tris (chloroisopropyl) phosphate	12/12/2011	50	0%
Sodium tungstate	12/23/2011	50	4%
Resveratrol	5/7/2012	50	0%
Black cohosh	7/2/2012	50	2%
Radiofrequency radiation (GSM/CDMA)	9/16/2012	90	0%

Historical control rate: 11/550 (2.0%)

Table D2. Incidence of schwannoma in the heart of male Harlan Sprague Dawley rats in NTP studies

Chemical	First dose	N	Control incidence
Indole-3-carbinol	3/14/2007	50	2%
Perfluorooctanoic acid	6/19/2009	50	0%
Dietary zinc	9/3/2009	50	0%
Dibutylphthalate	8/30/2010	49	4%
2-Hydroxy-4-methoxybenzophenone	11/8/2010	50	2%
p-Chloro-a,a,a-trifluorotoluene	1/17/2011	50	0%
Di-(2-ethylhexyl)phthalate	2/17/2011	50	6%
Di-(2-ethylhexyl)phthalate (perinatal)	6/27/2011	50	4%
Tris (chloroisopropyl) phosphate	12/12/2011	50	0%
Sodium tungstate	12/23/2011	50	0%
Resveratrol	5/7/2012	50	0%
Black Cohosh	7/2/2012	50	0%
Radiofrequency radiation (GSM/CDMA)	9/16/2012	90	0%

Historical control rate: 9/699 (1.30%)

APPENDIX E – TIME ON STUDY TO APPEARANCE OF TUMORS

Malignant Glioma

SAR (W/kg)	Animal ID number	Time on study (weeks)
GSM-modulated exposed males		
1.5	717	105
	735	102
	786	104
3.0	924	101
	943	105
	1014	93
6.0	1135	104
	1137	102
CDMA-modulated exposed males		
6.0	1795	105
	1799	104
	1852	105
GSM-modulated exposed females		
6.0	1246	96
CDMA-modulated exposed females		
1.5	1463	105
	1474	105

Time to Malignant Schwannoma in Heart

SAR (W/kg)	Animal ID number	Length of survival (weeks)
GSM-modulated exposed males		
1.5	758	104
	801	105
3.0	931	105
6.0	1149	83
	1155	105
	1187	104
	1206	104
	1230	91
CDMA-modulated exposed males		
1.5	1364	105
	1352	105
3.0	1559	92
	1617	105
	1622	104
6.0	1801	76
	1821	70
	1829	104
	1833	89
	1849	104
	1860	105
GSM-modulated exposed females		
3.0	1037	105
	1077	83
CDMA-modulated exposed females		
1.5	1461	106
	1480	93
6.0	1888	105
	1965	106

APPENDIX F – REVIEWER'S COMMENTS

National Toxicology Program

Peer Review Charge and Summary Comments

Purpose: To provide independent peer review of an initial draft of this partial report. The peer reviewers were blind to the test agents under study. Introductory materials on RFR and details of the methods dealing with the field generation and animal housing were redacted from the version sent to the reviewers. The reviewers were provided a study data package, also blinded to test agents, containing basic in life study information such as body weight and survival curves and information concerning the generation of pups from the *in utero* exposures.

Report Title: Draft Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Test Articles A and B (and associated Study Data Package)

Reviewers' Names:

David Dorman, D.V.M., Ph.D., North Carolina State University
Russell Cattley, D.V.M., Ph.D., Auburn University
Michael Pino, D.V.M., Ph.D., Pathology consultant

Charge: To peer review the draft report and comment on whether the scientific evidence supports NTP's conclusion(s) for the study findings.

1. Scientific criticisms:
   a. Please comment on whether the information presented in the draft report, including presentation of data in any tables, is clearly and objectively presented. Please suggest any improvements.

   All three reviewers found the results to be clearly and objectively presented, although there were suggestions to provide historical control information for brain and heart lesions for female Harlan Sprague Dawley rats, clarify statements about the specific statistical tests used and the presence or lack of statistical significance of the brain

gliomas in the Results, and expand the conclusions statements to clarify the basis for the conclusions.

b. Please comment on whether NTP's scientific interpretations of the data are objective and reasonable. Please explain why or why not.

The reviewers stated that the NTP had performed an adequate and objective peer review of the pathology data, and the statistical approaches used were consistent with other NTP studies. The methods were described as objective and reasonable. The interpretations of the data, including the limitations, were also reasonable and objective. One reviewer found the data on schwannomas of the heart to be more compelling with respect to an association with treatment than the brain gliomas. This reviewer summarized the findings as:

> "In the heart the evidence for a carcinogenic effect can be based on 1) the presence of the tumors in all six of the test article groups versus none in the controls 2) the statistically significant trend for schwannomas with both compounds and the statistically significant increase in incidence in the 4X (top) dose for test article B; 3) the fact that the incidence of the tumors in both 4X dose groups approaches or exceeds the high end of the historical control range; and 4) the tumors in the 4X group of test article B are accompanied by a higher incidence of Schwann cell hyperplasia. Using the NTP's guide for levels of evidence for carcinogenic activity, I would consider the heart schwannomas as 'Some Evidence' of carcinogenic activity.
>
> The proliferative lesions in the brain are more difficult to interpret because 1) their low incidence that was well within the historical control range, 2) lack of clear dose response; and 3) lack of statistical significance (except for the significant exposure-dependent trend for test article B. . . . However, the presence of malignant gliomas and/or foci of glial cell hyperplasia in 5 of 6 test article groups for both sexes vs none in controls of either sex is suggestive of a test

article effect. . . .I would consider the malignant gliomas as 'Equivocal Evidence' of carcinogenic activity."

2. Please identify any Information that should be added or deleted:

One reviewer suggested that more information be given on the time when tumors were observed (e.g., at terminal necropsy, or early in the study) to help assess the possible impact of the decreased survival times in the control animals on tumor incidence. This reviewer also suggested a discussion of how the survival of control male rats in this study compared to the historical control data. There was also concern that the diagnostic criteria developed by the PWG and used in the current study would impact the historical control incidence rates reported in Table D.

3. The scientific evidence supports NTP's conclusion(s) for the study findings:

The NTP's overall draft conclusion was as follows: "Under the conditions of these studies, the observed hyperplastic lesions and neoplasms outlined in this partial report are considered likely the result of exposures to test article A and test article B. The findings in the heart were statistically stronger than the findings in the brain."

The reviewers had the option of agreeing, agreeing in principle, or disagreeing with the draft conclusions. All three reviewers agreed in principle, reiterating issues discussed above.

APPENDIX G – NIH REVIEWER'S COMMENTS

National Institutes of Health

Peer Review Charge and Reviewer's Comments

Purpose: To provide independent peer review of the pathology diagnoses and statistical evaluation of the partial findings from NTP's studies. Background materials included the draft NTP report, introductory materials on RFR, and details on the methods dealing with the field generation and statistical analyses references and guidance. The reviewers were provided a study data package, containing basic in life study information such as body weight and survival curves, information concerning the generation of pups from the *in utero* exposures, and raw pathology data.

Report Title: Draft Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Test Articles A and B (and associated Study Data Package)

Reviewers' Names:

Diana C. Haines, D.V.M., Frederick National Laboratory
Michael S. Lauer, M.D., Office of Extramural Research, NIH
Maxwell P. Lee, Ph.D., Laboratory of Cancer Biology and Genetics, NCI,
Aleksandra M. Michalowski, M.Sc., Ph.D., Laboratory of Cancer Biology and Genetics, NCI
R. Mark Simpson, D.V.M., Ph.D., Laboratory of Cancer Biology and Genetics, NCI
[Sixth reviewer's name and comments are withheld.]

Charge: To peer review the draft report, statistical analyses, and pathology data and comment on whether the scientific evidence supports NTP's conclusion(s) for the study findings.

Reviewer's comments and NTP responses to the comments are provided.

- Appendix G1: Reviewer's comments
- Appendix G2: NTP's responses to NIH reviewer's comments

Appendix G1: Reviewer's comments

Reviewer: Diana C. Haines, D.V.M., Frederick National Laboratory

April 5, 2016

Dr. Tabak,

I've always relied on experts, not myself, for statistical analysis, and so do not feel qualified to address the statistical methods used. My training and experience has been in veterinary pathology, including QA review of NTP studies, and serving on PWGs, so will give my opinion on the pathology interpretation (biological significance rather than statistical significance).

Having perused the 3 RFR Draft Report and the raw data, all appears to be in order, including QA of the histopathology (technique) as well as PWG review (diagnosis). Looking at the data, I agree with the report's conclusion: *Under the conditions of these studies, the hyperplastic lesions and neoplasms observed in male rats are considered likely the result of exposures to GSM- an CDMA-modulated RFR. The findings in the heart were statistically stronger than the findings in the brain.* But note, it is "considered likely" not "definitely is".

There may be also several caveats relating to "under the conditions of these studies", including how well the conditions recapitulate actual human exposure: whole body exposure from in utero to old age; 18.5 hours/day (10 min on/10 min off, for total of 9hr actual exposure); and dose[A]. I'm not physicist, so have to presume experts analyzed and accepted concept of the reverberation chamber, including "doses"[A] as being relevant to human exposure.

[A] Dosimetric Assessment paper: "As could be expected in a study following NTP protocols, the exposure levels for the rodents in this project exceed the limits for the wbSAR and psSAR defined in the IEEE Std C95.1-2005 safety standard for human exposure to mobile phone radiation. In the low dose exposure group the exposure level in the organs exceeds or is close to the localized SAR limit for the general public, except for a few low-water content tissues. More specifically, the psSAR over 1 g in the human head, is limited by the safety standards to <2W/kg, whereas, in the low dose rodents the SAR averaged over the whole brain is >2.4 W/kg for mice, and >1.3 W/kg for rats, hence similar to the limit. Furthermore, the psSAR and oSAR have larger uncertainty compared to the wbSAR. Deviations of the exposure level from the target dose, especially during the early exposure period, should be carefully evaluated in the interpretation of the final biological studies.

Results from the companion mouse study will hopefully add some insight.

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Appendix G1: Reviewer's comments

Reviewer: Michael S. Lauer, M.D., Office of Extramural Research, NIH

Michael S Lauer, MD (OER)

Review of NTP paper: "Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation (Whole Body Exposures)"
March 20, 2016

Summary of findings:

This is a partial report, a report which is presumably part of a larger set of studies involving 2 species (mice and rats), 2 sexes (male, female), and multiple tissue types, all based on 90-week studies of two different types (GSM and CDMA) of cell phone radiofrequency radiation (RFR). In this partial report, we are given findings regarding brain gliomas and heart schwannomas in male and female Harlan Sprague Dawley rats which were exposed exposed to control or 3 different levels (1.5, 3.0, 6.0) of two types (GSM and CDMA) of RFR. There were 90 rats in each group. Using the poly-3 test with the Bieler-Williams variance adjustment, the authors found a statistically significant increase in the rate of brain gliomas in males exposed to CDMA RFR. Using the poly-6 test, the authors found a statistically significant increase in the rates of heart schwannomas in males exposed to GSM and CDMA. There were no statistically significant differences in rates of gliomas or schwannomas in females; also there was no statistically significant increase in rates of gliomas in males exposed to GSM RFR.

Comments:

1) Why aren't we being told, at least at a high level, of the results of other experiments (i.e., male and female mice, tissues other than heart and brain, tumors other than glioma and schwannoma)? Given the multiple comparisons inherent in this kind of work (see pages 27-30 and Table 13 of the FDA guidance document), there is a high risk of false positive discoveries. In the absence of knowing other findings, we must worry about selective reporting bias.
2) I was able to reproduce the authors' positive P-value findings (see Appendix 1, R code) using the MCPAN R package. However, I'm getting slightly different values for adjusted denominators (also in Appendix 1).
3) I was able to reproduce the authors' findings of longer survival with RFR (see Appendix 1, R code).
4) I have a number of questions about the study design:
    a. Were control rats selected in utero like the exposed rats were?
    b. Were pregnant dams assigned to different groups by formal randomization? If not, why not?
    c. Why were pups in the same litter included? Did the authors take any steps in their analyses to account for the resulting absence of i.i.d?
    d. The authors state that at most 3 pups were chosen per litter. How were the 3 pups chosen (and the others presumably not used for this experiment)? Were the 3 pups that were chosen selected by formal randomization? If not, why not?

e. Were all analyses based on the intent-to-treat principle? Were there any crossovers? Were all rats accounted for by the end of the experiment and were all rats who started in the experiment included in the final analyses?
f. Blinding: The authors state that "All PWG reviewer were conducted blinded with respect to treatment group," but in the very next phrase write "only identifying the test articles as 'test agent A' or 'test agent B.'" Why was this information (test agent A or B) given? The blinding was not complete.

5) Sample size:
   a. Did the authors perform a prospective (that is before initiation of the work) sample size calculation? If so, what were the prior assumptions? In other words, why did the authors choose to study 90 rats in each group and why did they set the maximum duration to 90 weeks (instead of 104 weeks)?
   b. I used a publicly available simulation package[1] to calculate the study power for male rats based on the following (see Appendix 2, power calculation simulation studies):
      i. Control tumor rate of ~1.5%.
      ii. Risk ratio 2.5 in the group receiving the highest dose
      iii. 2-sided Alpha = 0.005 (based on Table 13 of the FDA guidance document). Note this low alpha of 0.005 for poly-k trend tests is recommended to minimize the risk of false positive discoveries.
      iv. Sample size of 90 for each group with one planned sacrifice.
      v. Low lethality with lethality parameters set according to study duration and Weibull shape parameter (see Table 3 of Moon et al[1]). When I re-ran the simulations using intermediate lethality, results were not materially changed.
      vi. Study duration 90 weeks
      vii. 5000 simulations
      viii. Note – I used dose levels of 0,1,2, and 4 because I was unable to adjust these on the web site (despite trying 3 different browsers).
   c. Based on these inputs, the recommendations in Table 13 of the FDA guidance document, and a sample size of 90 rats in each group, I find very low power (<5%, see Appendix 2). Even allowing for a risk ratio of 5.0 (a level that is clinically unlikely), the power for 2-sided alpha=0.005, k=3 and low lethality is only ~14% (see Appendix 2).
   d. The low power implies that there is a high risk of false positive findings[2], especially since the epidemiological literature questions the purported association between cell phone exposure and cancer.[3]

6) Summary: I am unable to accept the authors' conclusions:
   a. We need to know all other findings of these experiments (mice, other tumor types) given the risk of false positive findings and reporting bias. It would be helpful to have a copy of the authors' statistical code.
   b. We need to know whether randomization was employed to assign dams to specific groups (control and intervention).

c. We need to know whether randomization was employed to determine which pups from each litter were chosen for continued participation in the experiment.
d. We need to know whether there was a formal power/sample size calculation performed prior to initiation of the experiment. If not, why not? If yes, we need to see the details. In particular, we need to know whether the authors followed the recommendations of the FDA guidance document (in particular Table 13).
e. I suspect that this experiment is substantially underpowered and that the few positive results found reflect false positive findings.[2] The higher survival with RFR, along with the prior epidemiological literature, leaves me even more skeptical of the authors' claims.

References:

1. Moon H, Lee JJ, Ahn H, Nikolova RG. A Web-based Simulator for Sample Size and Power Estimation in Animal Carcinogenicity Studies. *J Stat Software; Vol 1, Issue 13* . 2002. doi:10.18637/jss.v007.i13.
2. Ioannidis JPA. Why most published research findings are false. Jantsch W, Schaffler F, eds. *PLoS Med*. 2005;2(8):e124. doi:10.1371/journal.pmed.0020124.
3. Frei P, Poulsen AH, Johansen C, Olsen JH, Steding-Jessen M, Schüz J. Use of mobile phones and risk of brain tumours: update of Danish cohort study. *BMJ*. 2011;343.

Appendix 1: Attempted replication of positive findings

```
# Review of NTP paper on cell phone RFR and certain cancers
# Attempt to reproduce the positive findings
# Data from Larry Tabak
# Code by Mike Lauer

setwd("~/Desktop/Files to save")

library(MCPAN)
library(rms)
library(Hmisc)

# Read in CDMA NTP data

CDMA <- read.csv("~/Desktop/Files to save/NTP CDMA Raw Tumor Data.csv")

# Survival and treatment group, adjusting for sex, by Cox proportional hazards

CDMA$status<-1
CDMA$S<-Surv(CDMA$Removal.Day, CDMA$status)
f<-cph(S~Treatment+Sex, data=CDMA)
f

# Survival greater (better) for 3.0W, P=0.0157, for 6.0W, P=0.0260

# Table 1 -- Poly-3 test for malignant glioma in males CDMA

males_CDMA<-subset(CDMA, Sex=='M')

poly3test(time=males_CDMA$Removal.Day, status=males_CDMA$Brain.Glioma.Malignant,
      f=males_CDMA$Dose, k=3, type='Williams', method='BW', alternative='greater')

# P=0.039

poly3ci(time=males_CDMA$Removal.Day, status=males_CDMA$Brain.Glioma.Malignant,
      f=males_CDMA$Dose, k=3, type='Williams', method='BW', alternative='greater')
```

Call result:

```
Sample estimates, using poly- 3 -adjustment
                    0       1.5     3     6
x                   0.0000  0.0000  0.0000  3.0000
n                   90.0000 90.0000 90.0000 90.0000
adjusted n          63.8258 72.3688 76.6821 64.8154
adjusted estimate  0.0000  0.0000  0.0000  0.0463

# Table 3 -- Poly-6 test for malignant Schwannoma in males CDMA

poly3test(time=males_CDMA$Removal.Day,
        status=males_CDMA$Heart.Schwannoma.Malignant, f=males_CDMA$Dose, k=6,
        type='Williams', method='BW', alternative='greater')

# P=0.0005

poly3ci(time=males_CDMA$Removal.Day,
        status=males_CDMA$Heart.Schwannoma.Malignant,f=males_CDMA$Dose,
        k=3,type='Williams', method='BW')

Call result:

Sample estimates, using poly- 3 -adjustment
                  0    1.5     3     6
x                 0.0000  2.0000  3.0000  6.0000
n                 90.0000 90.0000 90.0000 90.0000
adjusted n        63.8258 72.3971 77.0575 66.5582
adjusted estimate  0.0000  0.0276  0.0389  0.0901

# Read in GSM NTP data

GSM <- read.csv("~/Desktop/Files to save/NTP GSM Raw Tumor data.csv")

# Survival and treatment group, adjusting for sex, by Cox proportional hazards

GSM$status<-1
GSM$S<-Surv(GSM$Removal.Day, GSM$status)
f<-cph(S~Treatment+Sex, data=GSM)
f

# Survival greater (better) for 6.0W, P=0.0048

males_GSM<-subset(GSM, Sex=='M')

# Table 3 -- Poly-6 test for malignant Schwannomas in males GSM
```

```
poly3test(time=males_GSM$Removal.Day, status=males_GSM$Heart.Schwannoma.Malignant,
      f=males_CDMA$Dose, k=6, type='Williams', method='BW', alternative='greater')

# P=0.004

poly3ci(time=males_GSM$Removal.Day, status=males_GSM$Heart.Schwannoma.Malignant,
      f=males_CDMA$Dose, k=3, type='Williams', method='BW', alternative='greater')
```

Call result:

Sample estimates, using poly- 3 -adjustment

	0	1.5	3	6
x	0.0000	2.0000	1.0000	5.0000
n	90.0000	90.0000	90.0000	90.0000
adjusted n	63.8258	73.1547	76.1127	77.0723
adjusted estimate	0.0000	0.0273	0.0131	0.0649

Appendix 2: Simulations for power calculations

Power Simulations for NTP Cell Phone RFR paper (from https://biostatistics.mdanderson.org/acss/Login.aspx and https://www.jstatsoft.org/article/view/v007i13)[1]
Michael Lauer, MD (OER)
March 19, 2016

1) For malignant gliomas (Table 1), P = 0.005, HR = 2.5, k=3

The University of Texas M. D. Anderson Cancer Center
Sample Size and Power Estimation for Animal Carcinogenicity Studies

Reference: "A Web-based Simulator for Sample Size and Power Estimation in Animal Carcinogenicity Studies."
Hojin Moon, J. Jack Lee, Hongshik Ahn and Rumiana G. Nikolova,
Journal of Statistical Software. (2002)[1]

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 3.00
Hazard ratio(s) of dose vs. control group
1.50 2.00 2.50
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 23.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990
average lethality = 0.0816

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0060	0.0000	0.0000
67	0.0002	0.0002	0.0334	0.0000	0.0000
78	0.0003	0.0005	0.0729	0.0000	0.0000
90	0.0005	0.0023	0.1855	0.0094	0.6887

dose group 1:
average tumor rate = 0.0225
average competing risks survival rate = 0.7000
average lethality = 0.0784

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0003	0.0002	0.0325	0.0000	0.0000
78	0.0004	0.0008	0.0720	0.0000	0.0000
90	0.0007	0.0034	0.1851	0.0145	0.6842

dose group 2:
average tumor rate = 0.0297
average competing risks survival rate = 0.6997
average lethality = 0.0772

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0004	0.0003	0.0331	0.0000	0.0000
78	0.0005	0.0012	0.0721	0.0000	0.0000
90	0.0010	0.0045	0.1829	0.0191	0.6790

dose group 3:
average tumor rate = 0.0366
average competing risks survival rate = 0.7007
average lethality = 0.0772

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0005	0.0003	0.0330	0.0000	0.0000

78	0.0006	0.0013	0.0716	0.0000	0.0000
90	0.0012	0.0054	0.1812	0.0238	0.6749

Positive Trend (Power): 0.0238

2) For malignant Schwannomas (Table 3), P = 0.005, HR = 2.5, k=6

The University of Texas M. D. Anderson Cancer Center
Sample Size and Power Estimation for Animal Carcinogenicity Studies

Reference: "A Web-based Simulator for Sample Size and Power Estimation in Animal Carcinogenicity Studies."
Hojin Moon, J. Jack Lee, Hongshik Ahn and Rumiana G. Nikolova,
Journal of Statistical Software. (2002)[1]

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 6.00
Hazard ratio(s) of dose vs. control group
1.50 2.00 2.50
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 45.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990
average lethality = 0.0631

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0060	0.0000	0.0000
67	0.0001	0.0001	0.0335	0.0000	0.0000
78	0.0002	0.0003	0.0732	0.0000	0.0000
90	0.0005	0.0019	0.1859	0.0096	0.6887

dose group 1:
average tumor rate = 0.0225
average competing risks survival rate = 0.7000
average lethality = 0.0602

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0059	0.0000	0.0000
67	0.0001	0.0001	0.0326	0.0000	0.0000
78	0.0003	0.0005	0.0723	0.0000	0.0000
90	0.0006	0.0029	0.1856	0.0148	0.6842

dose group 2:
average tumor rate = 0.0297
average competing risks survival rate = 0.6997
average lethality = 0.0582

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0059	0.0000	0.0000
67	0.0002	0.0001	0.0333	0.0000	0.0000
78	0.0004	0.0007	0.0726	0.0000	0.0000
90	0.0009	0.0038	0.1837	0.0195	0.6790

dose group 3:
average tumor rate = 0.0366
average competing risks survival rate = 0.7007
average lethality = 0.0588

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0059	0.0000	0.0000
67	0.0003	0.0001	0.0332	0.0000	0.0000
78	0.0005	0.0007	0.0722	0.0000	0.0000
90	0.0011	0.0046	0.1821	0.0243	0.6749

Positive Trend (Power): 0.0230

3) For further consideration, P = 0.005, HR = 5, k=3

The University of Texas M. D. Anderson Cancer Center
Sample Size and Power Estimation for Animal Carcinogenicity Studies

Reference: "A Web-based Simulator for Sample Size and Power
Estimation in Animal Carcinogenicity Studies."
Hojin Moon, J. Jack Lee, Hongshik Ahn and Rumiana G. Nikolova,
Journal of Statistical Software. (2002) In Press.

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 3.00
Hazard ratio(s) of dose vs. control group
2.00 3.50 5.00
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 23.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990

average lethality = 0.0816

sacrifice time	d	a1	b1	a2	b2
45	0.0000	0.0000	0.0060	0.0000	0.0000
67	0.0002	0.0002	0.0334	0.0000	0.0000
78	0.0003	0.0005	0.0729	0.0000	0.0000
90	0.0005	0.0023	0.1855	0.0094	0.6887

dose group 1:
average tumor rate = 0.0301
average competing risks survival rate = 0.7000
average lethality = 0.0743

sacrifice time	d	a1	b1	a2	b2
45	0.0001	0.0000	0.0059	0.0000	0.0000
67	0.0004	0.0003	0.0324	0.0000	0.0000
78	0.0005	0.0011	0.0717	0.0000	0.0000
90	0.0009	0.0045	0.1839	0.0194	0.6789

dose group 2:
average tumor rate = 0.0515
average competing risks survival rate = 0.6997
average lethality = 0.0774

sacrifice time	d	a1	b1	a2	b2
45	0.0002	0.0000	0.0058	0.0000	0.0000
67	0.0007	0.0006	0.0328	0.0000	0.0000
78	0.0009	0.0020	0.0713	0.0000	0.0000
90	0.0017	0.0076	0.1795	0.0331	0.6638

dose group 3:
average tumor rate = 0.0727
average competing risks survival rate = 0.7007
average lethality = 0.0804

sacrifice time	d	a1	b1	a2	b2
45	0.0003	0.0000	0.0059	0.0000	0.0000
67	0.0010	0.0006	0.0327	0.0000	0.0000
78	0.0013	0.0028	0.0701	0.0000	0.0000
90	0.0025	0.0107	0.1755	0.0470	0.6496

Positive Trend (Power): 0.1420

4) For further consideration, same as in baseline (1) but with intermediate lethality

*** Input Parameters ***

Selected Seed = 3000
Number of Groups = 4
Dose metric of each group:
0.00 1.00 2.00 4.00
Number of animals in each group
90 90 90 90
Number of sacrifices including a terminal sacrifice = 1
Sacrifice time points in weeks:

Study duration = 90 weeks
Number of INTERIM sacrificed animals in each interval:
Background tumor onset probability at the end of the study = 0.01
Tumor onset distribution assumed: Weibull with a shape parameter 3.00
Hazard ratio(s) of dose vs. control group
1.50 2.00 2.50
Competing Risks Survival Rate (CRSR) for each group:
0.70 0.70 0.70 0.70
Tumor lethality parameter entered = 225.00
Level of the test = 0.01
One-sided or two-sided test = 2 sided test
Number of simulation runs = 5000

*** Simulation Results ***

dose group 0:
average tumor rate = 0.0149
average competing risks survival rate = 0.6990
average lethality = 0.3936

sacrifice time	d	a1	b1	a2	b2
45	0.0004	0.0000	0.0060	0.0000	0.0000
67	0.0014	0.0001	0.0334	0.0000	0.0000
78	0.0014	0.0004	0.0729	0.0000	0.0000
90	0.0019	0.0015	0.1855	0.0063	0.6887

dose group 1:
average tumor rate = 0.0225
average competing risks survival rate = 0.7000
average lethality = 0.3852

sacrifice time	d	a1	b1	a2	b2
45	0.0006	0.0000	0.0059	0.0000	0.0000
67	0.0022	0.0001	0.0325	0.0000	0.0000
78	0.0020	0.0006	0.0720	0.0000	0.0000
90	0.0029	0.0023	0.1851	0.0097	0.6842

dose group 2:
average tumor rate = 0.0297
average competing risks survival rate = 0.6997
average lethality = 0.3839

sacrifice time	d	a1	b1	a2	b2
45	0.0008	0.0000	0.0059	0.0000	0.0000
67	0.0029	0.0003	0.0331	0.0000	0.0000
78	0.0027	0.0008	0.0721	0.0000	0.0000
90	0.0039	0.0031	0.1829	0.0127	0.6790

dose group 3:
average tumor rate = 0.0366
average competing risks survival rate = 0.7007
average lethality = 0.3897

sacrifice time	d	a1	b1	a2	b2
45	0.0009	0.0000	0.0059	0.0000	0.0000
67	0.0037	0.0003	0.0330	0.0000	0.0000
78	0.0033	0.0009	0.0716	0.0000	0.0000
90	0.0048	0.0037	0.1812	0.0157	0.6749

Positive Trend (Power): 0.0219

References:

1. Moon H, Lee JJ, Ahn H, Nikolova RG. A Web-based Simulator for Sample Size and Power Estimation in Animal Carcinogenicity Studies. *J Stat Software; Vol 1, Issue 13* . 2002. doi:10.18637/jss.v007.i13.
2. Ioannidis JPA. Why most published research findings are false. Jantsch W, Schaffler F, eds. *PLoS Med*. 2005;2(8):e124. doi:10.1371/journal.pmed.0020124.
3. Frei P, Poulsen AH, Johansen C, Olsen JH, Steding-Jessen M, Schüz J. Use of mobile phones and risk of brain tumours: update of Danish cohort study. *BMJ*. 2011;343.

Appendix G1: Reviewer's comments

Reviewer: Maxwell P. Lee, Ph.D., Laboratory of Cancer Biology and Genetics, NCI

I think the study was well designed and the analyses and results were clearly presented.

My main concern is the control data. Since the main finding was the increased incidence rates of heart schwannomas and brain gliomas in male Harlan Spragu Dawley rats exposed to GSM- or CDMA-modulated cell phone RFR, my analyses and evaluation below were focused on the male rats.

My concern regarding the control data came from the following two considerations. First, we need to consider sample variation. The incidence rates of the current controls for brain gliomas and heart schwannomas were 0. However, the historical controls were 1.67% for gliomas (range 0-8%) and 1.30% for schwannomas (0-6%). Given that there were substantial variations among the historical controls and the concurrent control is at the lowest end of the range, it is important to evaluate how different estimates of control incidence rates may impact the results of analyses. Supplementary Table S1 shows that for gliomas with 1.7% incidence rate we have 40%, 37%, 17%, and 6% of chance to observe 0 tumor, 1 tumor, 2 tumors, and greater than 2 tumors, respectively; heart schwannomas has similar distribution. Given the low incidence rate and moderate sample size of the control, even after observing 0 tumor in the current study, the 'true' incidence rate may be higher than 0. If we were repeating the experiment, we may see some control studies have 1 or more tumors. Second, it is puzzling why the control had short survival rate. Given that most of the gliomas and heart schwannomas are late-developing tumors, it is possible that if the controls were living longer some tumors might develop. Although the use of poly-3 (or poly-6) test intended to adjust the number of rats used in the study, it is still important to re-evaluate the analysis by considering the incidence rate in controls not being 0.

Therefore I have performed the analyses using the original data as well as the data modified by adding 1 tumor to the control. I implemented the poly-3 (or poly-6) trend test in R using the formula described in the file, Poly3 correction factor[1].docx.

The results are summarized in Table 1 for brain gliomas

Table 1. Incidence of brain gliomas in male rats exposed to GSM- or CDMA-modulated RFR, comparing control data with 0 vs. 1 tumor.

RFR	W/kg				pvalue
	0	1.5	3	6	
GSM	0	3	3	2	0.9771
GSM	1	3	3	2	0.8668
CDMA	0	0	0	3	0.0233
CDMA	1	0	0	3	0.1077

Poly-6 adjusted rates were used in the chi-square trend test. The 1st and 3rd rows correspond to the original data with 0 tumor observed in the control group (Th numbers in Table 1 here are identical to those in Table 1 in the original report). The test is significant for CDMA exposures (pvalue = 0.0233). However, it is not significant after adding 1 tumor to the control group (pvalue = 0.1077, the 4th row).

Similar analysis was performed for heart schwannomas. The results are summarized in Table 2.

Table 2. Incidence of heart schwannomas in male rats exposed to GSM- or CDMA-modulated RFR, comparing control data with 0 vs. 1 tumor.

RFR	W/kg				pvalue
	0	1.5	3	6	
GSM	0	2	1	5	0.0431
GSM	1	2	1	5	0.1079
CDMA	0	2	3	6	0.0144
CDMA	1	2	3	6	0.0365

Poly-3 adjusted rates were used in the chi-square trend test. The 1st and 3rd rows correspond to the original data with 0 tumor observed in the control group (Th numbers in Table 2 here are identical to those in Table 3 in the original report). The tests are significant for both GSM (pvalue = 0.0431) and CDMA (pvalue = 0.0144) exposures. However, only CDMA exposure remains significant after adding 1 tumor to the control group (pvalue = 0.0365, the 4th row).

Since the incidence of heart schwannomas in the 6 W/kg males was significantly higher in CDMA exposed males than the control group in the original report, I also analyzed the impact of adding 1 tumor to the control group

Table 3. Incidence of heart schwannomas in male rats exposed to 6 W/kg CDMA-modulated RFR, comparing control data with 0 vs. 1 tumor.

RFR	W/kg		pvalue
	0	6	
CDMA	0	6	0.0381
CDMA	1	6	0.0986

Poly-3 adjusted rates were used in the chi-square trend test. The 1st row corresponds to the original data with 0 tumor observed in the control group. The test was significant for CDMA exposures (pvalue = 0.0381). However, it was not significant after adding 1 tumor to the control group (pvalue = 0.0986, the 2nd row).

Conclusions

Increased incidence of heart schwannomas in male rats exposed to GSM- or CDMA-modulated RFR is statistically significant by the chi-square trend test. The evidence is better for CDMA exposure than GSM exposure. I think additional experiments are needed to assess if the incidence of brain gliomas in male rats exposed to GSM- or CDMA-modulated RFR is significantly higher than the control group or not.

My additional comments are summarized below.

1. I compared poly-3 adjusted number from Table 3 in the original repor versus the poly-3 adjusted number that I calculated using the raw data from the excel files. Supplementary Figure S1 shows that these two sets of numbers agre with each other in general. This is in contrast to the comparison for poly-6 adjusted number from Table 1 in the original report versus the poly-6 adjusted number that I calculated using the raw data from the excel files (Supplementary Figure S2). In fact, the adjusted rat numbers from Table 1 and Table 3 of the original report look quite similar (Supplementary Figure S3). This suggests that the poly-3 adjusted number was used in the footnotes in both Table 1 and Table 3 in the original report.

2. I noted that in Table S2 the adjusted numbers in from.original.report and poly3 are identical at Dose 0 and 1.5 for both CDMA and GSM as well as at Dose 3 for GSM but differ slightly in the other treatment doses for heart schwannomas. One possible cause of the difference is that the version of the raw data in the excel files differs from that used to generate the original report. The second possibility is typ in the footnote in Table 3. I also generated Table S3 that has the poly-6 adjusted numbers for brain gliomas. The two sets of the poly-6 adjusted numbers are ver different.

3. There are a couple of errors in the footnote of Table in the original report. 2/74.05 (5%) should be 2/74.05 (2.7%). 3/78.67 (4%) should be 3/78.67 (3.8%).

Supplementary Information

Table S1. Expected percentage of observing different numbers of tumors in the controls based on binomial distribution.

	0 tumor	1 tumor	2 tumors	>2 tumors
control for glioma	40%	37%	17%	6%
control for heart schwannoma	43%	37%	15%	5%

The percentage was calculated with 1.7% historical control rate for male rats (gliomas) and with poly-6 adjusted animal number, 53. Similarly, the percentage was calculated with 1.3% historical control rate for male (heart schwannoma) and with poly-3 adjusted animal number, 65.

Table S2. The poly-3 adjusted rat numbers in Table in the original report and those calculated from the raw data.

RFR	Dose	from.original.report	poly3
CDMA	0	65.47	65.47
CDMA	1.5	74.05	74.05
CDMA	3	78.67	78.35
CDMA	6	67.94	66.24
GSM	0	65.47	65.47
GSM	1.5	74.87	74.87
GSM	3	77.89	77.89
GSM	6	78.48	77.66

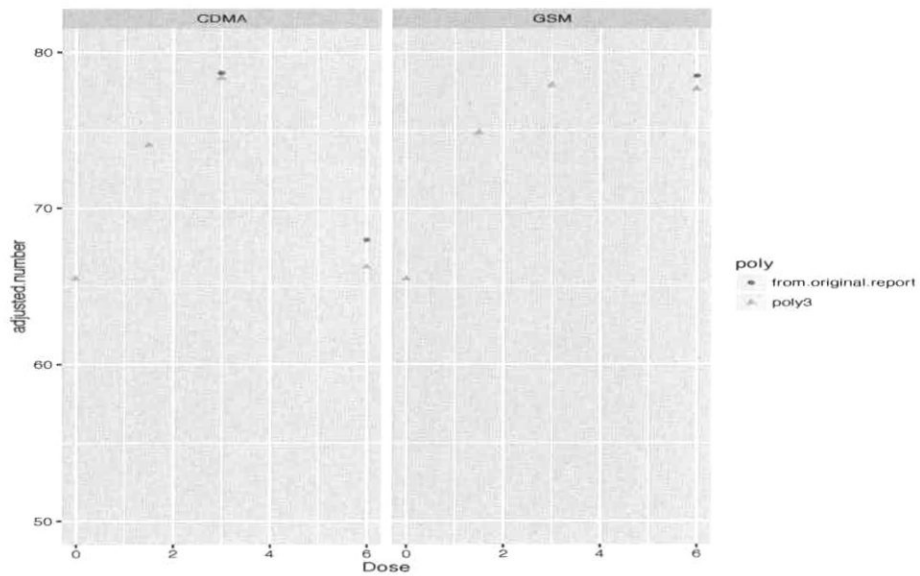
The numbers in from.original.report refers to the poly-3 adjusted rat number from Table 3 in the original report. The numbers in poly3 refers to the poly-3 adjusted rat numbers that I calculated from the raw data for heart schwannoma.

Table S3. The poly-6 adjusted rat numbers in Table in the original report and those calculated from the raw data.

RFR	Dose	from.original.report	poly6
CDMA	0	65.47	53.48
CDMA	1.5	74.05	65.94
CDMA	3	78.35	73.08
CDMA	6	66.24	57.5
GSM	0	65.47	53.48
GSM	1.5	74.93	67.84
GSM	3	78.27	71.43
GSM	6	77.1	72.55

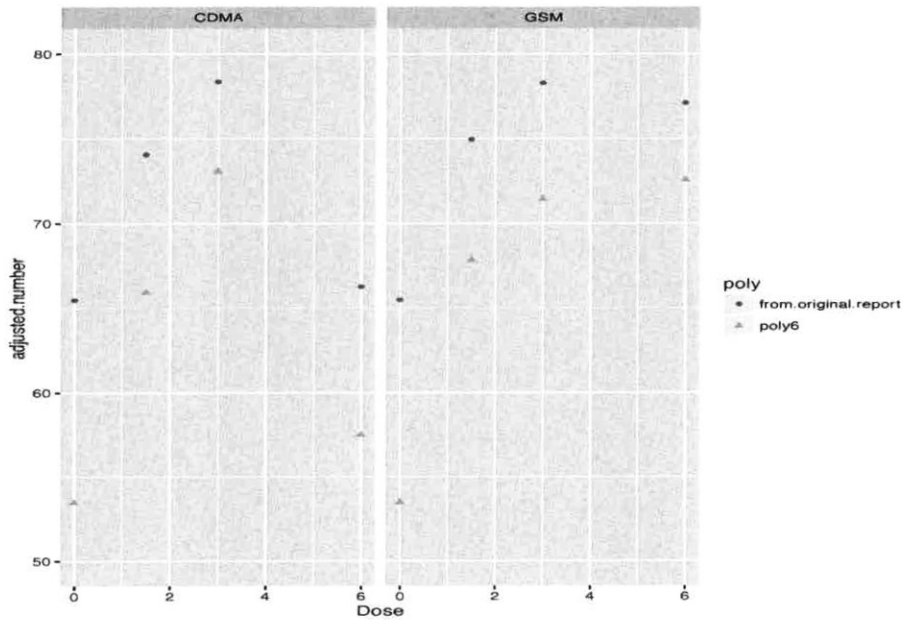
The numbers in from.original.report refers to the poly-6 adjusted rat number from Table 1 in the original report. The numbers in poly6 refers to the poly-6 adjusted rat numbers that I calculated from the raw data for brain gliomas.

Figure S1. Comparison of poly-3 adjusted rat numbers between those from the original report versus those calculated from the raw data.



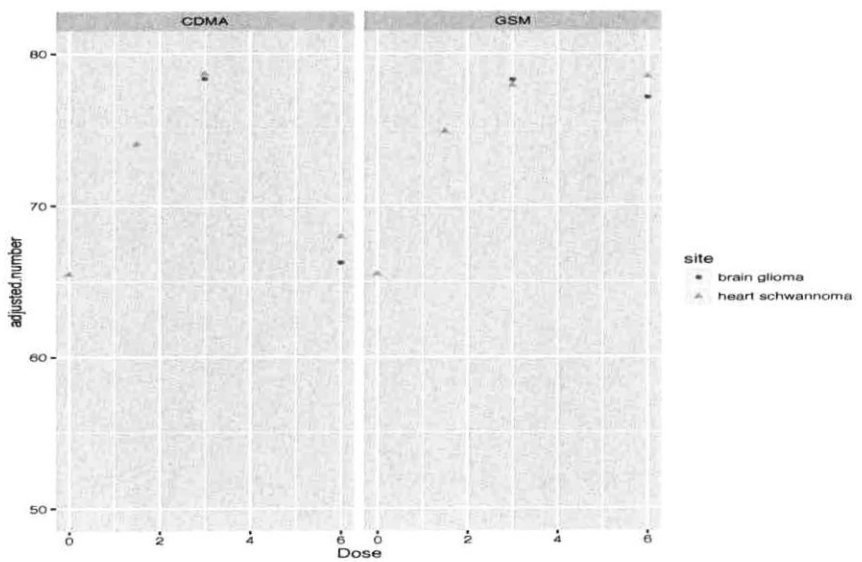
The poly-3 adjusted rat number from Table 3 of the original report is compare with the poly-3 adjusted rat number that I calculated from the raw data for heart schwannomas experiment

Figure S2. Comparison of poly-6 adjusted rat numbers between those from the original report versus those calculated from the raw data.



The poly-6 adjusted rat number from Table 1 of the original report is compare with the poly-6 adjusted rat number that I calculated from the raw data for brain gliomas experiment

Figure S3. Comparison of poly-6 adjusted rat numbers between those from the original report versus those calculated from the raw data.



The adjusted rat numbers from Table 1 and Table 3 of the original report are compared with each other.

Appendix G1: Reviewer's comments

Reviewer: Aleksandra M. Michalowski, M.Sc., Ph.D., Laboratory of Cancer Biology and Genetics, NCI

REVIEWER COMMENTS

Reviewer's Name:
Aleksandra M. Michalowski, Ph.D., M.Sc., National Cancer Institute/LCBG

Report Title:
Report of Partial Findings from the National Toxicology Program Carcinogenesis Studies of Cell Phone Radiofrequency Radiation (Whole Body Exposures); Draft 3-16-2016

Charge: To peer review the draft report and comment on whether the scientific evidence supports NTP's conclusion(s) for the study findings.

1. Scientific criticisms:

   a. *Please comment on whether the information presented in the draft report, including presentation of data in any tables, is clearly and objectively presented. Please suggest any improvements.*

Overall, the information included in the report is presented in a comprehensive and accurate manner. Specifically, the experimental design and conditions are sufficiently documented and the choice of statistical approaches is explained; the results are well organized and necessary details are provided.

Nevertheless, a few additions could be suggested:

(1) Appendix tables for all poly-k tests performed could be added. I believe this would enhance the presentation of the adjusted rates and the strength of the statistical evidence. As a possible example I prepared the below table using the R package *MCPAN* and its *poly3test()* function.

poly-3	Heart Schwannoma Malignant, Male				Heart Schwannoma Malignant, Female			
CDMA exposure	0	1.5	3	6	0	1.5	3	6
X	0	2	3	6	0	2	0	2
N	90	90	90	90	90	90	90	90
adjusted n	63.8	72.4	77.1	66.6	67.9	71.8	70.3	78.0
Dunnett contrast	–	1.5 - 0	3 - 0	6 - 0	–	1.5 - 0	3 - 0	6 - 0
Estimate	0	0.03	0.04	0.09	0	0.03	0	0.03
Statistic	–	1.24	1.58	2.45	–	1.26	0	1.24
p-value	–	0.2704	0.1542	**0.0209**	–	0.2466	0.7992	0.2562
Williams contrast	–	(6,3,1.5) - 0	(6,3) - 0	6 - 0	–	(6,3,1.5) - 0	(6,3) - 0	6 - 0
Estimate	0	0.05	0.06	0.09	0	0.02	0.01	0.03
Statistic	–	2.78	2.75	2.45	–	1.27	0.88	1.24
p-value	–	**0.0056**	**0.0060**	**0.0138**	–	0.1661	0.2871	0.1744

(2) In the portion of the text describing poly-k test results, p-values are given for significant pairwise comparisons; I would also give the p-values estimated for the significant trends (maximum test).

(3) Information could be included regarding the software or programming environment used for the computations.

(4) In the portion of the text describing differences in survival at the end of the study between control and RFR-exposed animals (page 5§2) the compared characteristic is not named (median survival, TSAC?) and also no numerical values of the estimates or the range of differences are given. I would add numbers in the text or an Appendix table showing the group survival estimates described in this paragraph.

Median survival

CDMA	Female	Male	GSM	Female	Male
0	737	662.5	0	737	662.5
1.5	734	719	1.50	738	729
3	737	731	3	737	730
6	738.5	717	6	738	731

TSAC percentage

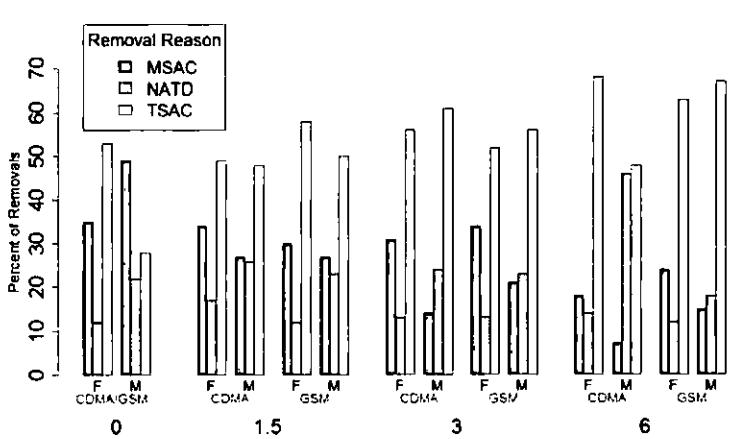
CDMA	Female	Male	GSM	Female	Male
0	53	28	0	53	28
1.5	49	48	1.5	58	50
3	56	61	3	52	56
6	68	48	6	63	67

b. *Please comment on whether NTP's scientific interpretations of the data are objective and reasonable. Please explain why or why not.*

Appropriate statistical design and methods were applied in accord with the FDA/NTP guidelines for conducting long-term rodent carcinogenicity studies and analyses. The results and limiting issues were objectively discussed. The critical issue of shorter survival in the male control group was addressed with regard to the percentage of animals surviving to terminal sacrifice in historical control data (avg. 47%, range 24% to 72%) and the possible impact of the observed age of tumor occurrence on the statistical inference.

I believe detailed information about animal selection and randomization procedures should be given so that the potential for allocation bias could be judged. As shown in the figure below, the lower survival rate to terminal sacrifice (28%) in the male control is accompanied by the higher rate of moribund sacrifice (49%); in the male group exposed to CDMA with 6 W/kg, a higher rate of natural death was observed (46%).

It has been reported that insufficient randomization can lead to differences in survival rates. As an example, in a carcinogenicity study on aspartame it was suggested that lack of randomization to different rooms may have possibly been the cause of low survival rates (27%) in the control female group due to a high background infection rate (EFSA, 2006; Magnuson, B., Williams, G.M., 2008).



2. Please identify any information that should be added or deleted:

A statement of the required statistical significance level should be added. FDA guidance suggests the use of significance levels of 0.025 and 0.005 for tests for positive trends in incidence rates of rare tumors and common tumors, respectively; for testing pairwise differences in tumor incidence the use of significance levels of 0.05 and 0.01 is recommended for rare and common tumors, respectively. If power calculations to determine the required sample size were performed, the results should also be included.

3. The scientific evidence supports NTP's conclusion(s) for the study findings:

The NTP's overall draft conclusion was as follows: "Under the conditions of these studies, the observed hyperplastic lesions and neoplasms outlined in this partial report are considered likely the result of exposures to test article A and test article B. The findings in the heart were statistically stronger than the findings in the brain."

In my view, the results support the conclusion of likely carcinogenic effect of the RFR-exposure on Schwannoma heart lesions in male Harlan Sprague Dawley rats.

Possible carcinogenic effects in the brain are marginal and are not sufficiently supported by statistical evidence in the male Harlan Sprague Dawley rats.

In the female Harlan Sprague Dawley rats very few lesions were observed in either site and statistical significance was not reached at all.

Appendix G1: Reviewer's comments

Reviewer: R. Mark Simpson, D.V.M., Ph.D., Laboratory of Cancer Biology and Genetics, NCI

Analysis of National Toxicology Program (NTP) study evaluating risk in rat lifetime exposure to GSM or CDMA RFR.

Notes:

The NTP study document acknowledges several study limitations [page 10, discussion section]. Potential limitations should prominently factor into considerations regarding the context of the findings, as well as their interpretation and application.

Working list of limitations potentially impacting NTP study interpretations

- Difficulty in achieving diagnostic consensus in lesions classifications of rare, unusual, and incompletely understood lesion association
- Document appears to indicate that the second Pathology Working Group (PWG) empaneled to review and obtain lesion classification consensus, following the inability of the initial PWG to do so, may have reviewed different lesions sets
- No record of clinical disease manifestations due to lesions involving heart and brain [note lesions in heart and brain are mutually exclusive; affected rats have either one or the other and do not appear to have the involvement of both organs together (appendix E)]
- Lesions, including malignancies, do not appear to materially shorten lifespan, except for a subgroup of rats (less than 1/3 of affected rats) with malignant Schwannomas in heart
- Lack of shortened lifespan as a consequence of malignancy for the majority of affected rats contrasts with shortened lifespan of male control rats for which there is absence of attributable cause of death. The survival of the control group of male rats in the current study (28%) was relatively low compared to other recent NTP studies (avg 47%, range 24 to 72%).
    - Creates greater reliance on statistical controlling for survival disparities and reliance on historical controls
- Reliance on historical controls made up of rats of different genetic strain background, held under different environmental conditions
- Absence of data on incidence of more frequently expected tumor occurrences in rats (background lesions)

Documenting the nature of the brain and cardiac lesions observed in RFR exposed rats and placing them into test article exposure-related context, in contrast to potential for their occurring spontaneously, are important and challenging goals. The NTP study limitations make the interpretation of reasonable risk more complicated. NTP acknowledgements of study limitations appear factored into one of NTP's reviewer's study conclusion, i.e., findings represent "some evidence" for a test article effect in statistically significant trend for Schwannomas; an opinion which is coupled with a conclusion for "equivocal evidence" of an effect in relation to malignant gliomas of the brain [NTP Appendix F, Reviewer Comments].

The summation from Appendix F reviewers regarding existence of test article effect is less than conclusive. The NTP study documents a series of cytoproliferative changes

in heart and brain. The nature of some of the changes is challenging diagnostically and appears to be incompletely understood. These findings are presented in the absence of complete analysis of the entire consequences of the study effects. For example, no potential significance for test article effect context is given to any of granular cell proliferative lesions of the brain, a finding mentioned only as a contrast to what was less well understood pathologically (NTP Appendix C, Pathology). It is noteworthy that the lesion types analyzed in the NTP RFR study under review are uncommon historically in rats, in the organs discussed. Furthermore, the malignancies of neuroglia appear to be paired with the occurrence of poorly understood changes involving neuroglial cell hyperplasias in the central and peripheral nervous systems. Little information can be gleaned from the literature about the nature and significance of these latter proliferative changes, interpreted by NTP as nonneoplastic and non-inflammation-reactive neuroglial cell in nature. Although unclear in the NTP study document, it is plausible that the particular lesion constellation, along with the relative novelty of some lesions, contributed to the lack of consensus regarding the nature of the lesions on the part of the initial PWG study pathologists. Concern raised by one of the reviewers (Appendix F, Reviewer Comments) regarding how this difficulty in ability to classify lesions might impact comparisons to historical control lesion incidence data (NTP Table D) is certainly principled.

The extraordinary PWG process, presumably posed by the difficult diagnostic interpretations, has the potential to influence the reliance on historical controls. In this regard, study limitations concerning determination of whether or not there is a test article effect include the substantially poor survival of male rats in the control group. The survival of the control group of male rats in the study under review (28%) was relatively low compared to other recent NTP studies (avg 47%, range 24 to 72%). This apparently led to greater statistical construction to account for the impact of study matched controls, and created increased reliance upon historical data of rare tumor incidences in control animals taken from other chronic carcinogenicity studies. NTP acknowledges a limitation in using the historical incident data and a small study match control group due to poor survivability. There are potential sources of variability when using historical controls of different rat strains and fluctuating study conditions (environment, vehicle, route of exposure, etc.), as is the case here. It seems less than clear what appropriate background lesion incidence is, as NTP indicates some data involve other strains of rats. The range of lesion incidence in historical controls could mean that the true incidence of some lesions varies considerably and might be considered rare or more common depending upon the incidence rate.

The guidance manual on Statistical Aspects of the Design, Analysis and Interpretation of Chronic Rodent Carcinogenicity Studies of Pharmaceuticals by the FDA provided for this review discusses applying comparisons using historical control lesion incidences at some length [beginning page 27, line 996]. Considering lesions as being rare or more common appears to influence selection of the level of statistical significance for comparisons. It appears that analysis for significant differences in tumor incidence between the control and the dose groups for these NTP studies has been established at the 0.05 level (NTP Tables 1,3,5). Interpretations of trend tests may be influenced by the choice of decision rule applied. Such choices can result in

about twice as large overall false positive error as that associated with control-high pairwise comparison tests [page 28, line 1012-1026]. The FDA guidance manual [page 31, line 1136] highlights concern regarding reliance upon historical control incidence data, stating that using historical control data in the interpretation of statistical test results is not very satisfactory because the range of historical control rates is usually too wide. This is especially true in situations in which the historical tumor rates of most studies used are clustered together, but a few other studies give rates far away from the cluster. When the range of historical control data is simply calculated as the difference between the maximum and the minimum of the historical control rates, the range does not consider the shape of the distribution of the rates. These circumstances may impose some limitations on optimal risk assessment designs.

Somewhat paradoxically then, NTP study limitations including that imposed due to reliance upon less than optimal historical control lesion incidence data for much of the comparisons between treated and untreated rats, is confronted by existence of a difficult to classify and incompletely understood lesion constellation interpreted to include neuroglial cell hyperplasia. Notwithstanding, this confounding proliferative lesion occurring in the context along with malignancies of apparently similar histogeneses, sustains a level of concern for a rare injury mechanism related to test article effect. Additional information about the study together with an assessment of the statistical analyses may enhance the value of this analysis.

R. Mark Simpson, D.V.M., Ph.D.

Appendix G2: NTP's Responses to NIH Reviewer's Comments

NTP Responses to Pathology Reviewer' Comments April 12, 2016

Reviewers: R. Mark Simpson, D.V.M., Ph.D. and Diana Copeland Haines, D.V.M.

Responses Relating to the Pathology Review Process

Drafts of the PWG reports are provided. As described in the PWG report, the specific task of the first PWG (January 29th 2016) was to: 1) confirm the presence of glial cell hyperplasia and malignant gliomas in the brain and Schwann cell hyperplasia and schwannomas in the heart; 2) develop specific diagnostic criteria in the brain for distinguishing glial cell hyperplasia from malignant glioma and gliosis, and in the heart for distinguishing between Schwann cell hyperplasia and schwannoma. The PWG participants confirmed the malignant gliomas and schwannomas, but the criteria for distinguishing between hyperplasia and neoplasia differed between the participants.

In order to clearly establish specific diagnostic criteria for the differentiation between hyperplastic and neoplastic lesions in the brain and heart, two additional PWGs were convened. The participants for the second (February 25, 2016) and third (March 3, 2016) PWGs were selected based on their distinguished expertise in the fields of neuropathology and cardiovascular pathology, respectively. Some of the participants were leaders in the International Harmonization of Nomenclature and Diagnostic Criteria initiative. The neuropathology experts of the second PWG confirmed the malignant gliomas in the brain, established diagnostic criteria for glial cell hyperplasia, and agreed that the hyperplastic lesions are within a continuum leading to malignant glioma. The cardiovascular pathology experts of the third PWG established specific diagnostic criteria for Schwann cell hyperplasia and schwannoma in the endocardium and myocardium, and reviewed and confirmed all cases of Schwann cell hyperplasia and schwannoma observed in these studies. The outcome of the PWG provided very high degree of confidence in the diagnoses.

The participants of the first PWG (January 29th 2016) only reviewed a subset of the glial lesions that were observed in the studies. The review for the second PWG (February 25, 2016) included all glial lesions in the studies including the subset that was reviewed in the first PWG.

Responses Relating to Considerations of Historical Control Data

For NTP toxicology and carcinogenicity studies, the concurrent controls are always the primary comparison group. However, historical control information is useful particularly in instances when there is differential survival between controls and exposed groups, as was observed in the RFR studies. Rates for glial cell neoplasms and heart schwannomas from control groups of male Harlan Sprague Dawley rats from other recently completed NTP studies are presented in Appendix D of the 3-16-2016 draft report. While Harlan Sprague Dawley rats are an outbred strain, they are considered single genetic strain in the same sense as other outbred strains, such as the Long-Evans or Wistar rat. Therefore, these historical control tumor rates are applicable to this study. However, it's important to note that the studies listed in Appendix D were carried out at laboratories other than the RFR studies, and under different housing and environmental conditions. At the time of the 3-16-201 draft report, not all of these studies had undergone a complete pathology peer review. In the past several weeks NTP pathologists have reviewed brain and heart slides from these male rat control groups, and have confirmed, with few exceptions, the low rates of hyperplastic and neoplastic lesions reported in Appendix D, applying the diagnostic criteria established during the PWGs outlined in Appendix C.

NTP Comments on Statistical Issues Raised by the Reviewers April 12, 2016

Given the multiple comparisons inherent in this kind of work, there is a high risk of false positive discoveries (Michael S. Lauer).

Although the NTP conducts statistical tests on multiple cancer endpoints in any given study, numerous authors have shown that the study-wide false positive rate does not greatly exceed 0.05 (Fears et al., 1977; Haseman, 1983; Office of Science and Technology Policy, 1985; Haseman, 1990; Haseman and Elwell, 1996; Lin and Rahman, 1998; Rahman and Lin, 2008; Kissling et al., 2014). One reason for this is that NTP's carcinogenicity decisions are not based solely on statistics and in many instances statistically significant findings are not concluded to be due to the test agent. Many factors go into this determination including whether there were pre-neoplastic lesions, whether there was a dose-response relationship, biological plausibility, background rates and variability of the tumor, etc. Additionally, with rare tumors especially, the actual false positive rate of each individual test is well below 0.05, due to the discrete nature of the data, so the cumulative false positive rate from many such tests is less than person would expect by multiplying 0.05 by the number of tests conducted (Fears et al., 1977; Haseman, 1983; Kissling et al., 2015).

I'm getting slightly different values for poly-k adjusted denominators (Michael S. Lauer).

I compared poly---3 adjusted number from Table 3 in the original report versus the poly---3 adjusted number that I calculated using the raw data from the excel files. Supplementary Figure S1 shows that these two sets of numbers agree with each other in general. This is in contrast to the comparison for poly---6 adjusted number from Table 1 in the original report versus the poly---6 adjusted number that I calculated using the raw data from the excel files (Supplementary Figure S2). In fact, the adjusted rat numbers from Table 1 and Table 3 of the original report look quite similar (Supplementary Figure S3). This suggests that the poly---3 adjusted number was used in the footnotes in both Table 1 and Table 3 in the original report. (Max Lee)

I noted that in Table S2 the adjusted numbers in from.original.report and poly3 are identical at Dose 0 and 1.5 for both CDMA and GSM as well as at Dose 3 for GSM but differ slightly in the other treatment doses for heart schwannomas. One possible cause of the difference is that the version of the raw data in the excel files differs from that used to generate the original report. The second possibility is typo in the footnote in Table 3. I also generated Table S3 that has the poly---6 adjusted numbers for brain gliomas. The two sets of the poly---6 adjusted numbers are very different. (Max Lee)

Information could be included regarding the software or programming environment used for the computations. (Aleksandra M. Michalowski)

The adjusted denominators in Table of the original report were labeled as poly-6 denominators, but were actually poly-3 denominators. This error was noted and brought to Dr Tabak's attention by Dr. Bucher in a March 22 email.

The p-values and adjusted denominators calculated by NTP are correct, except as noted for Table 1, and were calculated using validated poly-k software. This software is coded in Java and is embedded within NTP's TDMSE (Toxicology Data Management System Enterprise) system. Poly-k

calculations conducted by the reviewers in R may vary slightly from the NTP's calculation due to selection of study length and the NTP's use of the Bieler-Williams variance adjustment and a continuity correction. In his calculations, Dr. Lauer used 90 weeks as the study length, whereas the actual study length was 10 weeks. It is not apparent from the R documentation that the Bieler-Williams adjustment or the continuity correction is incorporated into the poly-3 calculations in R. In his calculations, Dr. Lee used two-sided p-values. In NTP statistical tests for carcinogenicity, the expectation is that if the test article is carcinogenic, tumor rates should increase with increasing exposure; thus, the NTP employs one-sided tests and p-values are one-sided. Using one-sided p-values in Dr. Lee's Table 1, the GSM trend if there were brain glioma in the control group remains nonsignificant, but the CDMA trend approaches 0.05 (p = 0.054) if there were brain glioma in the control group. In Dr. Lee's Table 2, the one-sided p-value for the GSM trend if there were 1 heart schwannoma in the control group approaches 0.05 (p = 0.054) and the one-sided p-value for the CDMA trend in heart schwannomas remains significant at = 0.018 if there were 1 heart schwannoma in the control group. In Dr. Lee's Table 3, the one-sided p-value for the CDMA pairwise comparison is significant at p = 0.049 if there were 1 heart schwannoma in the control group.

statement of the required statistical significance level should be added. FDA guidance suggests the use of significance levels of 0.025 an 0.005 for tests for positive trends in incidence rates of rare tumors and common tumors, respectively; for testing pairwise differences in tumor incidence the use of significance levels of 0.05 and 0.01 is recommended for rare and common tumors, respectively. (Aleksandra M. Michalowski)

Although the FDA guidance suggests lowering the significance level for most tests of trend and pairwise differences, this guidance is based o a misunderstanding of findings reported by Haseman (1983). In this paper, Haseman discusses several rules proposed by others for setting the significance level lower than 0.05. *If* these rules are rigidly followed, Haseman showed that study conclusions will be consistent with the NTP's more complex decision-making process, for which 0.05 is the nominal significance level and p-values are taken into consideration along with other factors (outlined above in response to comment 1) in determining whether the tumor increase is biologically significant. The NTP does not strictly adhere to specific statistical significance level in determining whether a carcinogenic effect is present.

Appendix tables for all poly-k tests performed could be added. (Aleksandra M. Michalowski)

Dr. Michalowski proposed a sample table. The rows corresponding to X, N, adjusted n are already included in the tables or appear the footnotes in the tables. The rows corresponding to "Dunnett contrast" and "Williams contrast" are not appropriate for dichotomous tumor data. Both Dunnett's test and Williams' test assume that the data are continuous and normally distributed.

In the portion of the text describing poly-k test results, p-values are given for significant pairwise comparisons; I would also give the p-values estimated for the significant trends. (Aleksandra M. Michalowski)

Indicators of significant trends are given in the tables in the form of asterisks next to control group tumor counts.

There are a couple of errors in the footnote of Table 3 in the original report. 2/74.05 (5%) should be 2/74.05 (2.7%). 3/78.67 (4%) should be 3/78.67 (3.8%). (Max Lee)

Thank you for pointing this out. The percentages will be corrected in our final report.

Were control rats selected in utero like the exposed rats were? Were pregnant dams assigned to different groups by formal randomization? How were the pups per litter chosen? (Michael S. Lauer).

believe detailed information about animal selection and randomization procedures should be given so that the potential for allocation bias could be judged. (Aleksandra M. Michalowski)

Pregnant dams were assigned to groups, including the control group, using formal randomization that sought to also equalize mean body weights across groups. The three pups per sex per litter were selected using formal randomization, as well. Tumors in the heart and brain were not observed in littermates, indicating that there was no litter-based bias in the results.

Were all analyses based on the intent-to-treat principle? Were there any crossovers? Were all rats accounted for by the end of the experiment an were all rats who started in the experiment included in the final analyses? (Michael S. Lauer)

The intent-to-treat principle is not relevant to this animal experiment, in which all animals that were assigned to treatment group received the full and equal treatment of that group. There were no crossovers. All animals that started the experiment were accounted for by the end of the experiment and included in the final analyses.

The PWG review blinding was not complete. (Michael S. Lauer)

PWG reviewers were blinded to the identity of the test article and the level of exposure but were not blinded to the fact that there were two different, yet related, test articles (modulations of cell phone RFR), to emphasize the fact that there was a common control group.

Did the authors perform a prospective sample size calculation? (Michael S. Lauer)

If power calculations to determine the required sample size were performed, the results should also be included. (Aleksandra M. Michalowski)

Sample size calculations were conducted for this study. However, for detecting carcinogenesis, sample size and power will depend on the baseline (control) tumor rate and the expected magnitude of the increase in tumors. For example, at 80% power, sample size requirements will be quite different for detecting a 2-fold increase in a rare tumor having a spontaneous occurrence of 0.5% compared to 2-fold increase in a more common tumor having a spontaneous occurrence of 10%. Because many different tumor types having wide range of spontaneous occurrence are involved in these studies, there is no "one-size-fits-all" sample size; rather, the sample size is a

compromise among several factors, including obtaining reasonable power to detect moderate to large increases for most tumor types, while staying within budgets of time, space, and funding. A sample of 90 animals per sex per group was selected as providing as much statistical power as possible across the spectrum of tumors, under the constraints imposed by the exposure system.

The NTP's carcinogenicity studies are similar in structure to the OECD's 45 Guideline for carcinogenicity studies and the FDA's guidance for rodent carcinogenicity studies of pharmaceuticals. These guidelines recommend at least 50 animals of each sex per group, but also mention that an increase in group size provides relatively little increase in statistical power. In the NTP's RFR studies, the group sizes were 90 animals of each sex per group, nearly twice as many as the minimum recommendation. Increasing the group sizes further provides diminishing returns, for which additional animals do not substantially increase power.

The low power implies that there is high risk of false positive findings (citing Ioannidis, 2005). ... suspect that this experiment is substantially underpowered and that the few positive results found reflect false positive findings (citing Ioannidis, 2005). (Michael S. Lauer)

It is true that the power is low for detecting moderate increases above a low background tumor rate of approximately – %, as was seen in the brain and heart tumors. However, this low power does not correspond to high risk of false positive findings. The paper by Ioannidis that was cited correctly states that when studies are small or effect sizes are small (i.e., statistical power is low), "the less likely the research findings are to be true." Research findings can be "not true" if the result is a false positive or a false negative. With low statistical power, false negatives are much more likely than false positives. Therefore, the vast majority of false research findings in a low power situation will result from the failure to detect an effect when it exists. The false positive rate on any properly constructed statistical test will not exceed its significance level, alpha. By definition, the significance level of a statistical test is its false positive rate, and it is typically selected by the researcher, often at a low fixed value such as 0.05 or 5%.

If we were repeating the experiment, we may see some control studies have 1 or more tumors. (Max Lee) (Dr. Lee also presented analyses of the male rat data, inserting hypothetical data on one tumor-bearing animal in the control group.)

In light of the historical control data, Dr. Lee demonstrated that several associations became less or not significant with the insertion of a tumor data point in the control group. While we appreciate that some other studies had one or more tumors, the NTP considers the concurrent control group as the most important comparator to the treated groups. We took the historical control tumor rates into account in a more subjective manner in our interpretation of the findings. In 2010, we asked to adopt more formal method of incorporating historical control data in our statistical testing, but our Board of Scientific Counselors voted against adopting the method.

It is puzzling why the control had short survival rate. Given that most of the gliomas and heart schwannomas are late-developing tumors, it is possible that if the controls were living longer some tumors might develop. Although the use of poly-3 (or poly-6) test intended to adjust the number of rats

used in the study, it is still important to re-evaluate the analysis by considering the incidence rate in controls not being 0. (Max Lee)

We do not know why the male rat control group had a low survival rate. We generally d observe lower survival rates in studies such as the RFR studies in which animals are singly- rather than group housed. While some tumors might possibly have arisen in controls if they lived longer, it was notable that n glial cell or Schwann cell hyperplasias were found in these animals as well.

The poly-k (e.g., poly-3 or poly-6) test was developed to adjust for the fact that not all animals survive to the end of a two-year study, and survival rates may differ among groups. The test is essentially Cochran-Armitage trend test in which the denominator of the tumor rate in each group is adjusted downward to better reflect the number of animal-years at risk during the study. Each animal that develops the tumor or survives to the end of the study is counted as one animal. Each animal that does not develop the tumor and dies (or is moribund sacrificed) before the end of the study is counted as a fractional animal. The fraction is calculated as the proportion of the study that it survived, raised to the k-th power; = 3 or = 6 in this study. The survival-adjusted tumor rate in each group is then the number of animals having the tumor of interest divided by the total count of animals at risk of developing the tumor in the group. These survival-adjusted rates are used in the Cochran-Armitage formula to provide the poly-k test for dose-related trends and pairwise comparisons with the control group.

The poly-k test has been shown to yield valid inferences about tumor rates in NTP two-year rat and mouse carcinogenicity studies (Bailer and Portier, 1988; Portier and Bailer, 1989; Portier et al., 1986). Its theoretical basis is that tumor incidence, while not directly observed unless the tumor is immediately lethal, follows a Weibull distribution with a shape parameter, k. Verification using NTP studies has shown that if k is between 1 and 5, setting k = 3 yields a valid statistical test (Portier and Bailer, 1989; Portier et al, 1986). Thus, most of the time, the NTP uses the poly-3 test. If tumor type is late-occurring, as we observed with the brain gliomas, k = 6 is a better fit to the data and the poly-6 test has more validity.

In the portion of the text describing differences in survival at the end of the study between control and RFR-exposed animals the compared characteristic is not named an also n numerical values of the estimates or the range of differences are given. I would ad numbers in the text of a Appendix table showing the group survival estimates described in this paragraph. (Aleksandra M. Michalowski)

The Statistical Methods section describes the method for comparing survival distributions between the control and RFR-exposed groups, namely, Tarone's (1975) life table test to identify exposure-related trends in survival and Cox's (1972) method for testing two groups for equality of survival distributions.

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ADDITIONAL RESPONSE:

Dear All,

Thanks again for all your helpful comments on the NTP RFR studies. I did want to follow up on one remaining point of disagreement that Mike Lauer alluded to in his comments about low powered studies. Although we agree that our study design had low power to detect statistically significant neoplastic effects in the brain and heart, which occurred with both RFR modulations in male rats, we disagree over the assertion that low power in and of itself, creates false positive results. We cited a handful of publications outlining the statistical arguments against this with specific respect to the NTP rodent cancer study design in our response to comments document sent earlier. Although Mike referred to the example of positive findings in underpowered epidemiology studies that could not be replicated in larger follow up studies, there is a growing literature alluding to this problem with respect to experimental animal studies as well. An example is a relatively recent article by one of our collaborators in CAMARADES, Malcolm MacLeod.

http://www.nature.com/news/2011/110928/full/477511a.html

It's important to distinguish between low power to detect effects, and the constellation of other factors that often accompany low powered experimental animal studies in contributing to this problem. We've addressed this issue in a recent editorial, and these factors are captured in our published systematic review process for evaluating study quality in environmental health sciences (Rooney et al., 2014).

http://ehp.niehs.nih.gov/wp-content/uploads/122/7/ehp.1408671.pdf

http://ehp.niehs.nih.gov/wp-content/uploads/122/7/ehp.1307972.pdf

Table 1 in the Rooney et al. report outlines risk of bias considerations that commonly plague studies carried out by academic researchers that are accounted for in NTP studies.

I provide these examples to assure you that we are completely cognizant of these issues and take them very seriously. Again, we appreciate the help you've provided in assuring that we appropriately interpret and communicate our findings.

Best
John Bucher

APPENDIX O

TREET, NEW YORK, NE

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APPENDIX P

PECO delivers a Reliable and Resilient Smart Grid

Glenn Pritchard, PE

AGENDA

- PECO Background
- PECO's Smart Grid Project
- Reliability Benefits
  - Storm Resiliency and the Communications Network
  - Outage Management
  - Distribution Automation
  - Voltage Monitoring

PECO

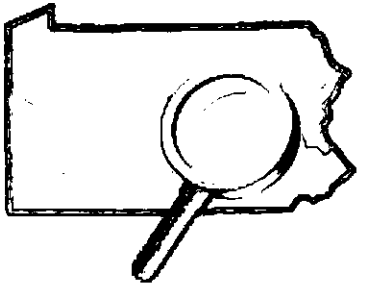
Subsidiary of Exelon Corporation

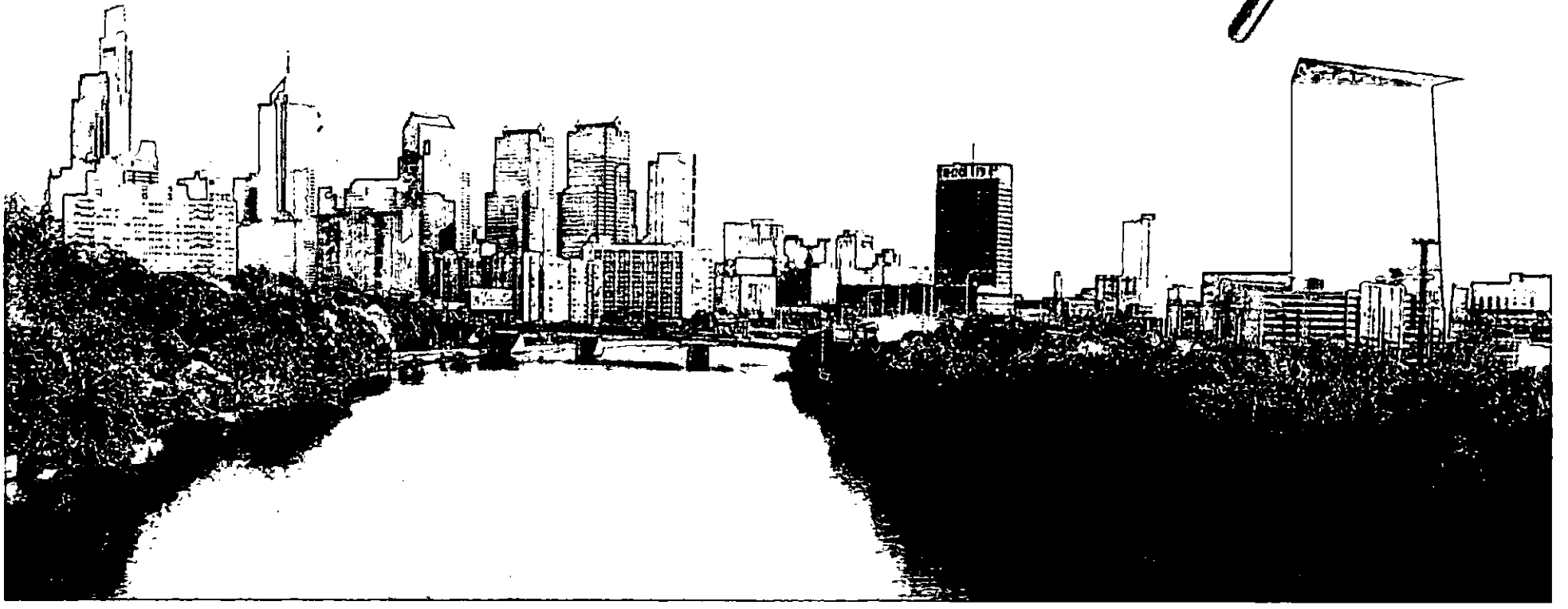
Serving the greater Philadelphia Pennsylvania area for over 100 years

2,100 square miles (5,400 km^2) service territory

Electric and Gas Utility

- 1.7M Electric Customers, 8,932 megawatt peak load
- 525K Gas Customers





PECO's Infrastructure

Transmission System

- 500, 220, 138 & 69kv Transmission Lines
- 1,067 miles (1,720 km) of high voltage lines

Substations

- 449 Primary and Unit Substations
- SCADA coverage for all primary stations

Distribution System

- 2,242 Distribution Circuits covering 21,362 miles (34,378 km)
- 34, 13.2, 4 & 2.4kv Distribution Lines

Automation

- >1,600 Distribution Automation Reclosers
  - ~75% Communicating via Telco Circuits or Private Wireless
  - Supporting 34 & 13kv Systems

- Fully Automated Meter Reading
  - Landis+Gyr/Cellnet AMR 1-Way Fixed RF System serving 2.2M electric and gas meters
  - Transitioning to a Sensus FlexNet Smart Grid and AMI Platform

- Fully integrated AMR and Outage Management Systems

The Smart Grid and PECO

PECO has been deploying and benefiting from the Smart Grid for many years, as evident in our:

- SCADA Platforms, both Transmission and Distribution SCADA
- Distribution Automation Solutions
- AMR Deployment

The State of Pennsylvania has recently passed legislation requiring all Utilities to modernize their systems, including Smart Meters

- Funding mechanisms are included in the legislation
- This program has been complemented with additional $200M US Federal funding

This combination of programs is the catalyst for PECO to create the Smart Future, Greater Philadelphia program, whose goals include:

- Promoting Innovation, Opportunity and Sustainability Through Smart Grid Technology

Smart Grid Greater Philadelphia Program

PECO's $650 million Smart Grid / Smart Meter initiative is one of the largest investments in the company's more than 100 year history and will enable us to provide electric service more reliably and efficiently and provide future new products and services to our customers

- Work is underway at PECO to deploy Smart Meters for all of our 1.6 million electric customers thanks to $200 million stimulus grant awarded by the US Department of Energy
- Currently over 950,000 Smart Meters have been installed
- We expect to complete the full system-wide deployment by the end of 2014.

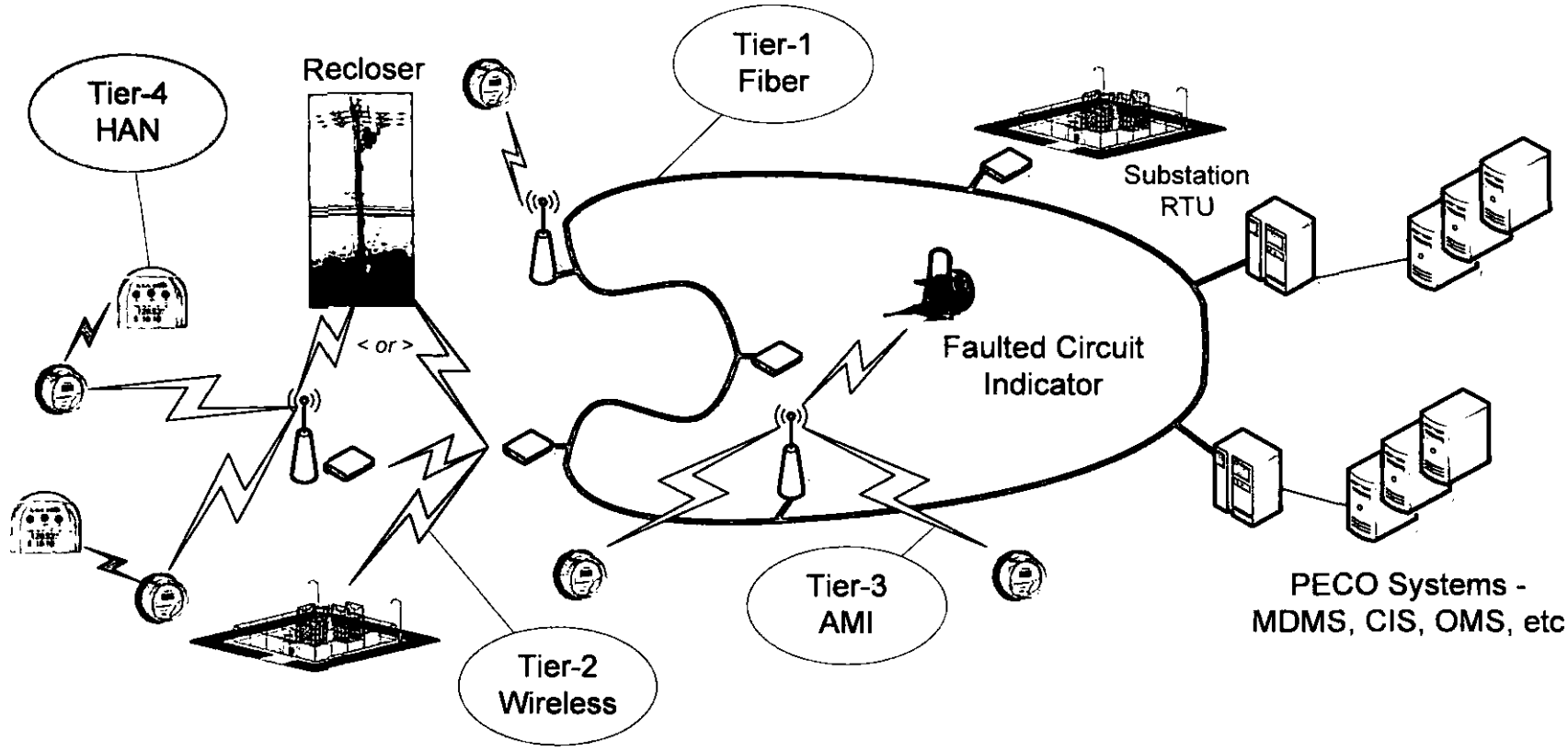
The two-way information system created by the Smart Grid / Smart Meter network is designed to improve electric service reliability and also help advance use of renewable energy sources

- Smart Meter technology will equip customers with the information they need to better understand how they use energy and how they can save energy
- With this technology PECO also will be able to offer customers enhanced rates and other 'dynamic pricing'

Key Components of PECO's Smart Grid

- To PECO, the Smart Grid is firmly rooted in a strong & resilient communication network
  - PECO designed a best-in-breed hybrid communications network
    - Backhaul with Fiber Optic, Microwave and WiMax Communications
    - RF End-Point Communications – Licensed RF
      - Principally point-to-point
      - Single hop mesh where needed
- The end-points connected to the network create operational benefits
  - Smart Meters
  - Distribution Automation Devices
    - Recloser
    - Capacitor Controllers
  - Monitoring Devices
    - Faulted Circuit Indicators
    - Voltage Sensors

PECO's Multi-Tiered Smart Grid Network



Communication Tiers:

- ✓ Tier-1: ~375 miles of high speed/high bandwidth fiber optic communications
- ✓ Tier-2: WiMax wireless communication network
- ✓ Tier-3: AMI Network, low-speed, low bandwidth network
- ✓ Tier-4: HAN In-Home Communications

Exelon.

Smart Grid Network Resiliency

- PECO's Smart Grid Network is designed to be storm tolerant with high reliability and resiliency
  - A backbone network of Sonet Fiber Optic Rings
  - Redundant communication paths were necessary
  - Each end-point communicates to numerous towers

- During Hurricane Sandy (Oct 2012), PECO's Smart Grid performed well with the following results:
  - Network Impact – Resiliency
    - During the Storm, 60 out of 163 TGB sites either lost communications or land power for an extended period of time
      - Due to the magnitude of the storm, backup batteries were depleted
      - Portable generators were dispatched to priority sites
    - Analysis indicated that only 2% of the meters that had power were unavailable

Outage Detection and Restoration Benefits

Benefit	Hurricane Irene *	Hurricane Sandy **
Total Customers Impacted	~508,000	~1,130,000
Peak Customers Impacted	480,000	608,000
Single Customer Event Cancellations	2,300	4,257
Primary Event Cancellations	350	820
Escalations – Single Events to Primary Events	700	1,042
Total Events (avoided and/or more effective truck rolls)	3350	6119
Estimated Fewer Days to Total Restoration	1-2 days	2-3 days

Cancellations – Power On verified, OMS event cancelled
Escalations – Verified neighboring meters out, job escalated from single event to multiple event

*Hurricane Irene August-September 2011; 100% AMR
**Hurricane Sandy October-November 2012; 90% AMR / 10% AMI

Avoided Generation Benefit

PECO recently completed a project to install a second transmission feed its Clay substation

- A twenty-five (25) day substation outage was required
- During the outage, over 50 distribution circuits within the southern Chester County service territory were affected and out of configuration
- This temporary configuration affected 32,779 customers

There was a need to closely monitor these circuits to ensure that voltage is maintained within the established tariff limits

- Standby generation was strategically placed at points of concern
- The AMI system was used to monitor voltage in these areas
  - ~175 AMI meters were installed to provide hourly voltage data

The AMI meter data based analysis helped avoid running the standby generation saving over $1.25M (USD)

PECO's Smart Grid Distribution Automation Vision

PECO's goal is to deploy an AMI network with the capability to support Smart Grid applications and communicate AMI and Distribution Automation data on a converged network:

- Distribution Management System (DMS)
  - Automatic Fault Detection, Isolation, and Service Restoration
- Distribution SCADA control of reclosers, regulators and capacitor banks
- Conservation Voltage Reduction (CVR) Application
- Communicating Faulted Circuit Indicators
  - Real Time Fault Locating
- Analytics

Analytics for Distribution

There are many opportunities for integrating Analytics into a Smart Grid Solution

- Connectivity Model Improvements
  - Auto-Generating Secondary Circuit Models
  - Correcting Meter Phasing
  - Detecting Transformer Connectivity Problems
- Identifying Overloaded/Stressed Assets
  - Proactive Transformer Replacement
- Locating Transformer Voltage Problems
- Using AMI Data to Detect Theft and/or Unmetered Load
  - Unbilled revenue
  - Tamper detection & Irregular Usage Patterns
- Improved Fault Locating
  - Using Substation Power Quality and Relay Data
  - Using Feeder Monitors to Locate Faults and Estimate Cause
- Reliability Analysis, Storm Analysis and Momentary Analysis

Connectivity Model Improvements

- A complete and accurate connectivity model is necessary to achieve many of the benefits.
  - Principally, the meter to transformer relationship is needed for:
    - Outage Management
    - Transformer Load Modeling and Asset Management
  - A full circuit model is important for
    - Circuit modeling
    - Phasing
    - Voltage Management
- Most model improvements are done manually when discrepancies are noticed by an operator, dispatcher or field crew
- The Smart Gird and Analytics can offer solutions to automatically identify discrepancies and errors. They include:
  - Outage & Event Analysis
  - Outlier Analysis
- 100% accuracy should always be a goal, but recognizing the current state will help drive opportunities
  - Known problem areas can be prioritized and addressed

Outlier Analysis

During a routine transformer outage, outlier customers are identified and corrections to the connectivity models are made

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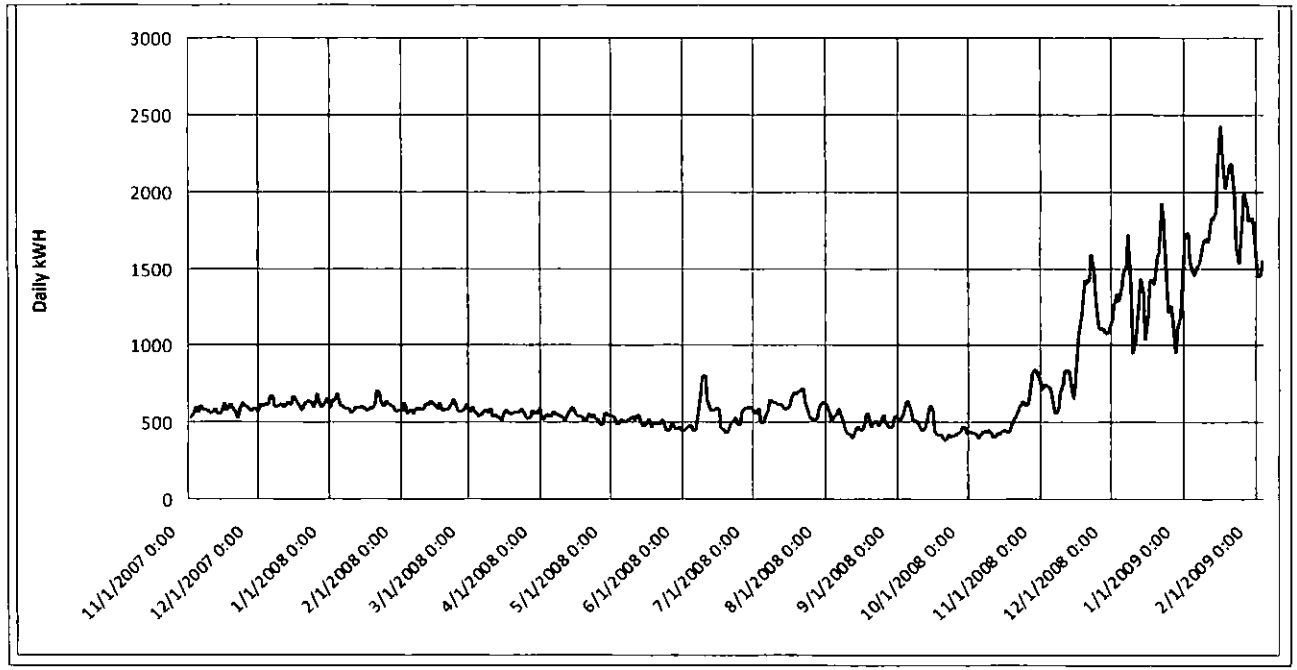
Transformer Monitoring

PECO uses analytics to determine transformer overloads and predict failures based on metering data from the AMR / AMI data

A program was initiated to investigate daily transformer consumption data (aggregated from meter data) for failures that occurred during winter peak load days

Transformers with yr-to-yr load increase of more than 25% (3,764 winter, 1,233 summer) were flagged for investigation



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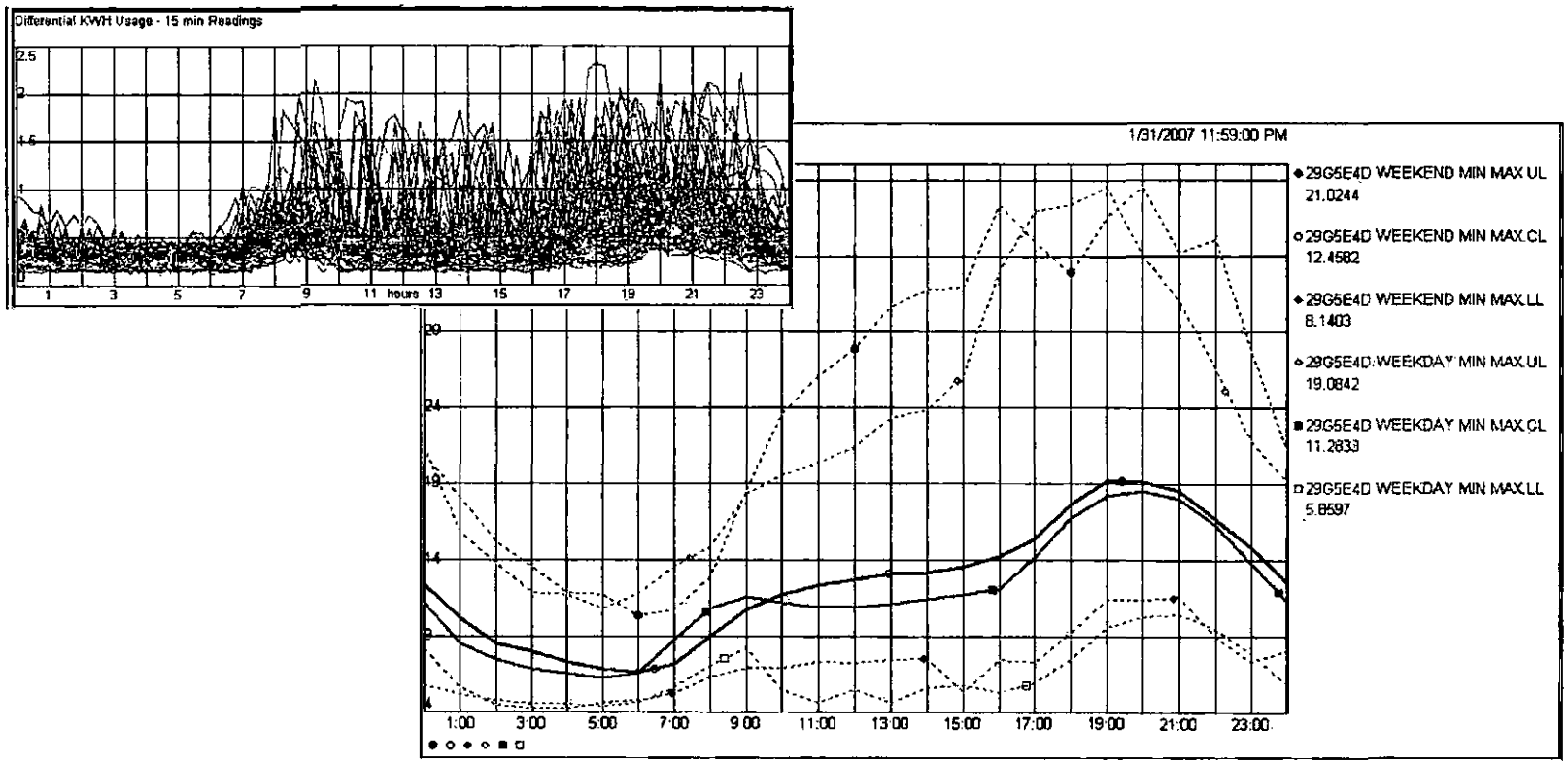
Transformer Load Profiles

- Once a daily transformer load shape is identified, it can be used to develop overall "normal" operating parameters for each device
- If the loading suddenly exceeds the normal operating envelop, an investigation can be initiated to understand why the change in load shape
  - In many cases, the equipment may need to be upsized or load relief activities must be performed



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Voltage Analysis – Feeder Voltage Profiles

While the voltage profile is within the Tariff guidelines, voltages range from red/orange = >240v to blue/gray = < 240v

Voltage Management

The Conservation Voltage Reduction Program is to realize energy savings and peak demand reduction from eligible customers in PECO's territory during the top 100 peak hours

This program incorporates voltage regulation techniques on distribution feeders that result in lower (but within regulatory requirements) service voltage levels, thereby reducing associated energy consumption and demand

Techniques deployed to achieve reductions:

- A 1% voltage reduction at the substation bus from historical levels
- The voltage set points for 13.2KV and 34KV distribution substations with automatic voltage controls (AVCs) and load tap changers (LTCs) will be recalibrated to deliver a 1% lower voltage

In the event the lower bus voltage impacts customer voltages, a mitigation strategy was deployed to improve customer voltages through the installation of capacitor banks, pole top or URD transformers and larger primary or secondary wires

Conclusion

- With sufficient planning, Smart Grids can be designed with the needed reliability and redundancy to ensure the delivery of a multitude of benefits
- Benefits such as those reviewed are real, for PECO they include:
  - Outage Management
  - Distribution Automation and Analytics
  - Voltage Mangement

Thank You

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APPENDIX Q

Ice Storm
Southeastern Pennsylvania

Last week a severe Ice Storm hit southeastern Pennsylvania. The local distribution utility PECO has experienced an unprecedented number of outages from this storm and the restoration is taking well into this week. Additional wintry weather is expected mid-week.

At the storm's peak, PECO had nearly 2/3's of its 1.6M electric customers out of power. Over the past week end, the number dropped below 400,000.

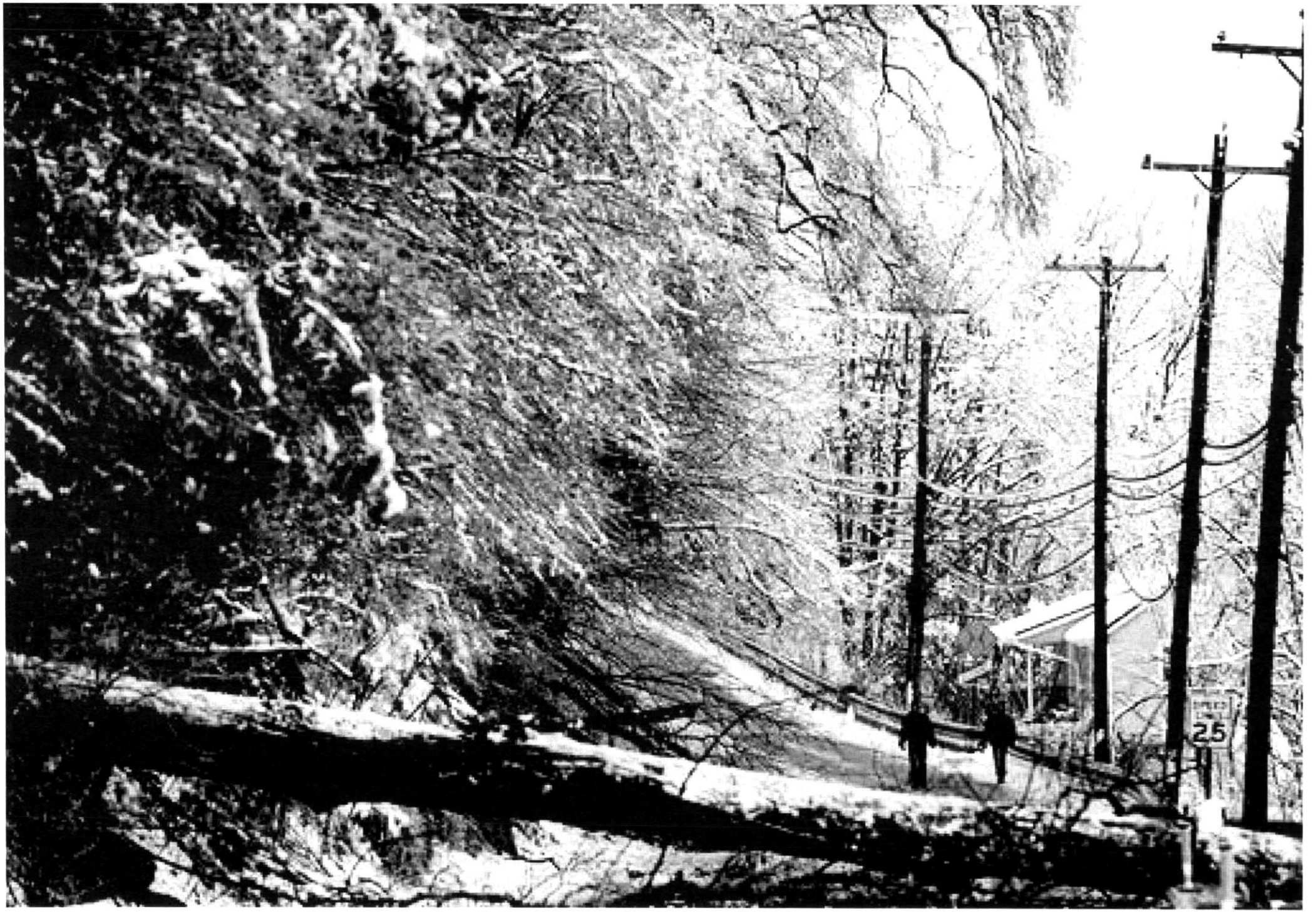
Personally, Glenn Pritchard is responsible for leading the Smart Grid Network Restoration and the Meter Pinging teams.

Glenn reports that Smart Grid continues to deliver tremendous benefit to PECO during this event. So far the Smart Meters have help avoid dispatching crews to over 900 jobs where there was not an actual power outage. This is yet another example of how the Smart Grid is able to improve customer's experiences with the power grid.

Message from Glenn Pritchard, PECO

Due to the recent Ice Storm in southeastern Pennsylvania, I must regretfully decline your invitation to participate in the upcoming Smart Grid event in Bogota Columbia so that I can continue to support our outage restoration effort. PECO has experienced an unprecedented number of outages from this storm and the restoration is expected to take well into next week. Furthermore, additional wintry weather is expected over the weekend and again mid-week. At the storm's peak, we had nearly 2/3's of PECO's 1.6M electric customer out of power. Presently, the number has dropped below 400,000. Personally, I am responsible for leading the Smart Grid Network Restoration and the Meter Pinging teams.

The good news is that the Smart Grid continues to deliver tremendous benefit to PECO during events such as this. So far the Smart Meters have help avoid dispatching crews to over 900 jobs where there was not actual power outage. This is yet another example of how the Smart Grid is able to improve customer's experiences with the power grid.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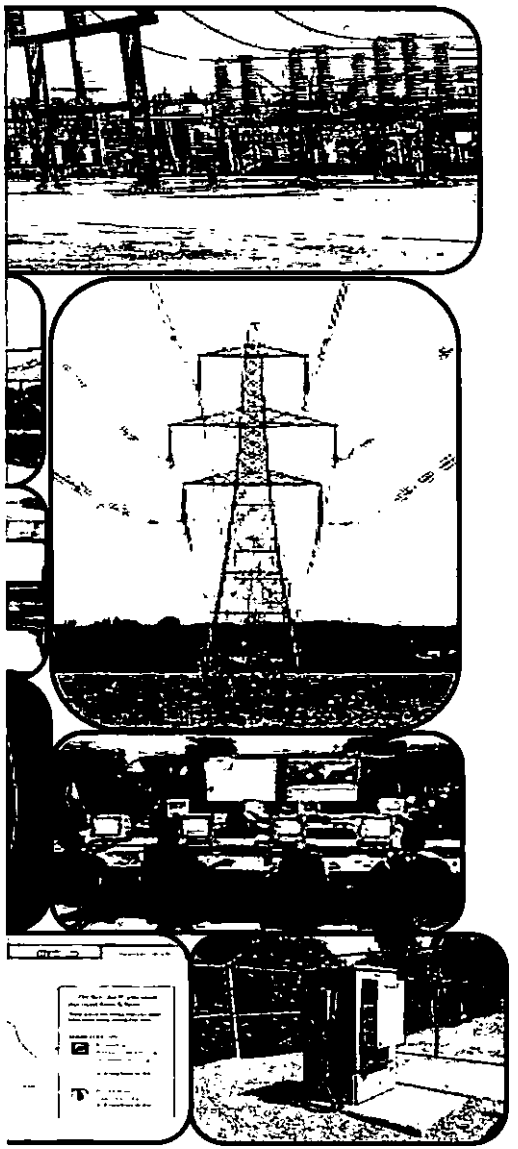


PECO's Smart Grid and Smart Meter Program

Glenn A. Pritchard, PE

February 2014

pecial Message

/ant to take this opportunity to express my regrets for not being able to attend :his event in person. Due to the ongoing restoration effort for Winter Storm Nikka, I must remain in Philadelphia to support the restoration effort, I am deep 'nvolved by ensuring that the Smart Grid remains fully operational during this :vent. Overall, the Smart Grid has once again demonstrated its ability to delive: ɔositive benefit to the Utility and our customers.

ncourage you to work closely with the USTDA team to learn and ultimately prop /our personal smart grid initiatives to meet your individual goals.

Glenn Pritchard

CO

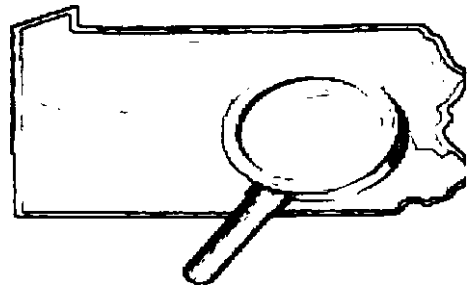
ubsidiary of Exelon Corporation

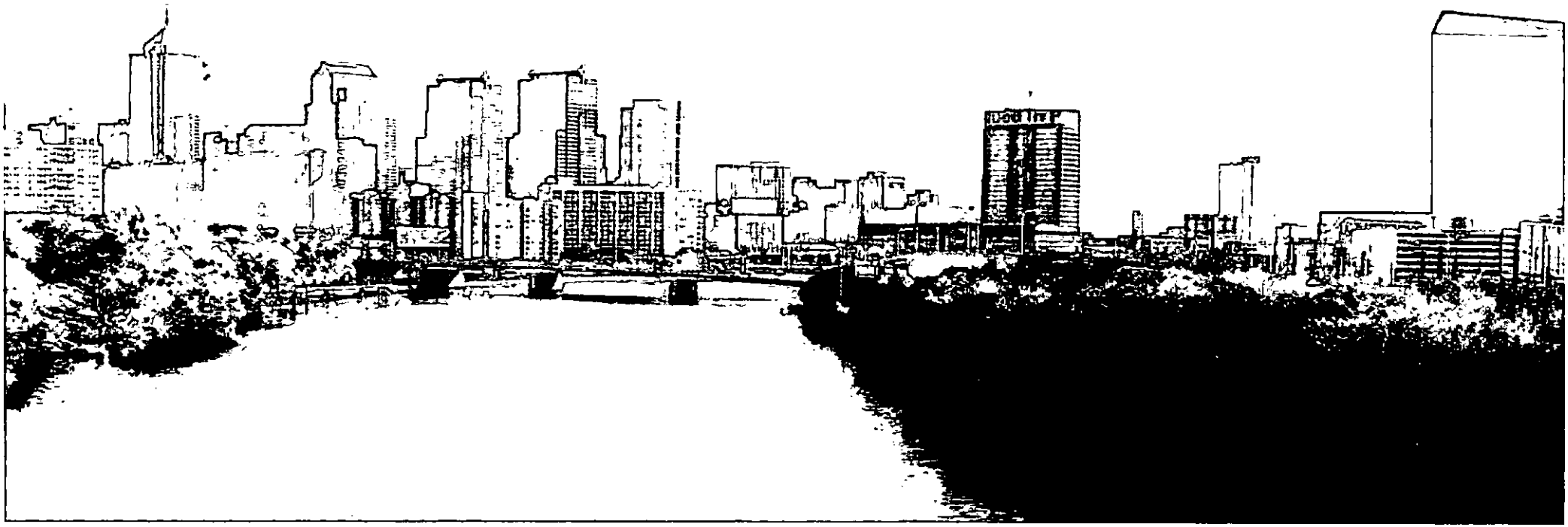
erving the greater Philadelphia Pennsylvania area for over 100 years

,100 square miles (5,400 km^2) service territory

lectric and Gas Utility

- 1.7M Electric Customers, 8,932 megawatt peak load
- 470K Gas Customers





he Smart Grid and PECO

CO has been deploying and benefiting from the Smart Grid for many years, as evident in our:

- SCADA Platforms, both Transmission and Distribution SCADA
- Distribution Automation Solutions
- AMR Deployment

e State of Pennsylvania has recently passed legislation requiring all Utilities to modernize their tems, including Smart Meters

- Funding mechanisms are included in the legislation
- This program has been complemented with additional $200M US Federal funding

is combination of programs is the catalyst for PECO to create the Smart Future, Greater Philade ogram, whose goals include:

- Promoting Innovation, Opportunity and Sustainability Through Smart Grid Technology

mart Future,
ireater Philadelphia Program

:O's $650 million Smart Grid / Smart Meter initiative is one of the largest investments in the company's m
) year history and will enable us to provide electric service more reliably and efficiently and provide future
ıducts and services to our customers

- Work is underway at PECO to deploy Smart Meters for all of our 1.6 million electric customers thanks to $200 million grant awarded by the US Department of Energy
- Currently over 700,000 Smart Meters have been installed
- We expect to complete the full system-wide deployment by the end of 2014.

: two-way information system created by the Smart Grid / Smart Meter network is designed to improve el
vice reliability and also help advance use of renewable energy sources

- Smart Meter technology will equip customers with the information they need to better understand how they use en how they can save energy
- With this technology PECO also will be able to offer customers enhanced rates and other 'dynamic pricing'

gram Scope

ters (AMI)

- ced Metering Infrastructure
- ions Network
- Data Management System,
- & Integrate Systems
- 000 Smart Meters
- ceptance testing

Communications Support Systems:

- Install 367 miles of fiber optic cable through 71 substations
- Install Tier 2 backhaul communications to support telemetry backhaul, AMI and Distribution Automation
- Update Distribution Management System & GIS (nearing end of life)

Distribution Automation

- Deploy an additional 100 Reclosers that sense problems and limit their impact
- Install 21 Underground Vacuum Circuit Breakers to modernize the network
- Communicate with 300 more existing devices to improve service

Intelligent Substatio

- Remote Terminal Unit up migration to IP centric tel substations
- Installation of substation line relays at 10 substatic
- Install disturbance monit equipment at 31 substati

Home/Business

- nonstrations
- icing Plans
- rt Buildings
- rt Campus
- ne Display Pilot
- icle pilot

Smart Meters (AMI)

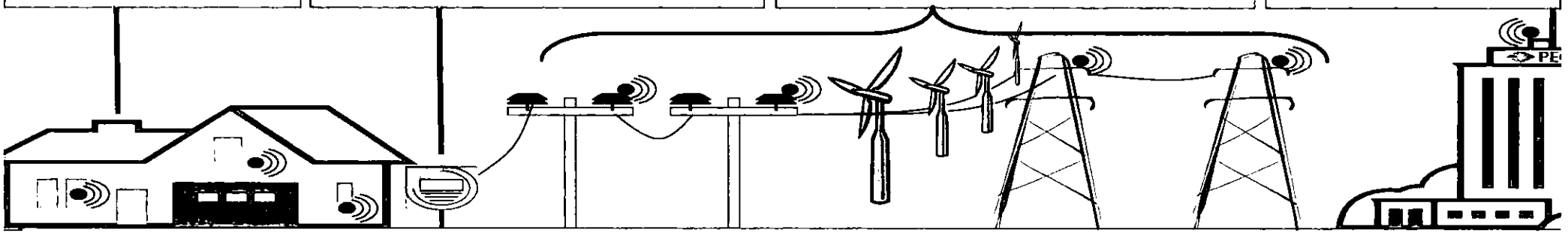
- A method to enable two-way information flow
- System status, customer outage status, usage and pricing signals delivered to and from location

Smart Distribution System

- Real-time reporting of status and outages
- Automated controls of relays and reclosers
- Efficient field force management
- Effective interconnection of renewable energy sources

Smart Utilit

- More efficient data colle processing and back off
- Enhanced operational a insight



Design Principles

ırity

Robust end-to-end, aligned with industry best practices (FIPS 140-2 compliant or certified)

/erged Communications

Smart Grid applications will share a converged shared communications infrastructure but will be logically isolated (tunn

·operable

Industry standard open protocols will be utilized preferentially end-to-end

Avoid use of proprietary protocols

ıtely owned communications

Privately owned communications enables Exelon to maintain governance and control over all aspects of the technology

Jnanalyzed Single Points of Failure (Self Healing)

The comm. architecture will be designed with no unanalyzed single points of failure

Consistent with the deterministic philosophy, failure modes and backup schemes shall be incorporated to form a "self h architecture

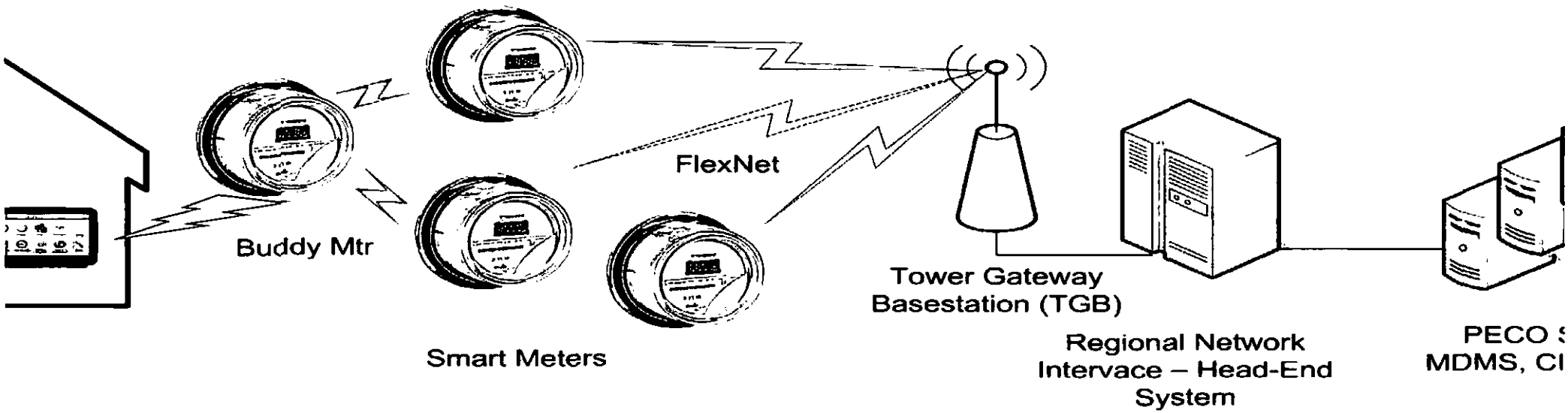
ıtenance Management & Monitoring

Inherent to the communications Architecture will be Comm. Maintenance Management & Monitoring, i.e. the ability tc monitor and control network devices

ned Standards

Architectural Design Standards will exist to embody & enumerate the details of the Fundamental Design Principles

System



s' FlexNet System is a point-to-point solution that leverages three key elements: Endpoints (meters), Tower Gateway Ba
n, and Regional Network Interface (RNI)

Meters are designed to directly communicate directly to the TGB's

The TGB Network is designed so that each meter can "see" between 3 and 7 TGB's

When a meter cannot directly reach a TGB, a buddy mode can be used to use near-by meters to relay the data back to a TGB

et uses 2 watt transmitters on Federally licensed exclusive-use frequencies

s provides both ZigBee and direct FlexNet communications into the home to meet the various deployment requiremen
onally, Sensus supports the U-SNAP modular HAN solution to provide consumer and utility flexibility

' Meter Capabilities

ınced Data Analytics – Building on current AMR practices while taking advantage of the
lata to improve business operations and create value

ote Connect/Disconnect - Will provide faster, more convenient service for customers w
oving in, out or around the neighborhoods we serve and quick support to local fire
tments and other officials during an emergency

ıge Management – Ability to monitor voltage at each premise creating new means to
ge the distribution grid

Presentment – All customers will be able to view interval usage via the web to educat
mers and lead to new usage awareness and savings

ECO's Initial Project Goals

"To provide the ability to remotely identify customer power status, to process outage messages provide restoration verification data via the AMR Network"

- 2 to 4 minutes in reduced System CAIDI through improved and reduced event analysis including better nested recognition
- ~$400,000 annual O&M Savings from reduced overtime and outside contractor requirements through better e management

After nearly 10 years of use,, the AMI outage integration has delivered an average of :

- 5 to 6 minutes of annual CAIDI savings
- In excess of $10M USD in operation savings via more efficient dispatch and more effective/productive use of fi

)utage Management Functions

Power Verification – Pinging – Using the meter to verify customer power status

- Don't need to contact customer to do verify power

Outage Detection – Last-Gasp messages from the meters when pow s lost

Restoration Verification – Power-Up messages from the meter confirming power is resotred

Blink Counts – Momentary outage logging

nter Storm Nikka

ring the week of 2/2/2014, two winter storms affected the PECO territory

2/2-3: 8-10" (20-25cm) of Snow

2/4-5: Significant Ice + Snow

- Falling trees and ice lead to massive outages.
- A total of 1.1M customers were affected by the storm
- At peak, over 800,000 customers were affected

twork Impact – *Resiliency*

During the Storm, 61 out of 165 TGB sites either lost communications or land power for an extended period of time

- Due to the magnitude of the storm, backup batteries were depleted

Portable generators were dispatched to priority sites

Analysis indicated that only 2% of the meters that had power were unavailable

rm Response – *Efficiency & Cost Savings*

A 24x7 pinging team was convened to supplement the automatic processes and screen work packages

This team pinged meters and validated work requests

It has been estimated that PECO was able to reduce the restoration effort by at least 3 days

mart Grid Benefits

Storm Response

- A 24x7 pinging team was convened to supplement the automatic processes and screen work packages
- This team pinged meters and validated work requests

During this event: (to date)

- 3912 single customer events were cancelled due to power verification pings
- 612 events were escalated from single customer events to multiple customer primary events from power verification pings
- Specific monetary benefits are still being calculated

icane Response/Benefit

Benefit	Hurricane Irene *	Hurricane Sandy **
ners Impacted	~508,000	~1,130,000
ners Impacted	480,000	608,000
omer Event Cancellations	2,300	4,257
nt Cancellations	350	820
– Single Events to Primary Events	700	1,042
(avoided and/or more effective truck rolls)	3350	6119
ewer Days to Total Restoration	1-2 days	2-3 days

ations – Power On verified, OMS event cancelled

ons – Verified neighboring meters out, job escalated from single event to multiple event

Irene August-September 2011; 100% AMR

Sandy October-November 2012; 90% AMR / 10% AMI

ded Generation

ecently completed a project to install a second transmission feed its Clay substation

wenty-five (25) day substation outage was required

ring the outage, over 50 distribution circuits within the southern Chester County service territory were aff
d out of configuration

s temporary configuration affected 32,779 customers

was a need to closely monitor these circuits to ensure that voltage is maintained within the
shed tariff limits

ndby generation was strategically placed at points of concern

e AMI system was used to monitor voltage in these areas

- ~175 AMI meters were installed to provide hourly voltage data

MI meter data based analysis helped avoid running the standby generation saving over $1M

Smart Grid
tion Automation Vision

O's goal is to deploy an AMI network with the capability to support Smart Gri
lications and communicate AMI and Distribution Automation data on a conve
work:

Distribution Management System (DMS)

- Automatic Fault Detection, Isolation, and Service Restoration

Distribution SCADA control of reclosers, regulators and capacitor banks

Conservation Voltage Reduction (CVR) Application

Communicating Faulted Circuit Indicators

- Real Time Fault Locating

Analytics

ytics for Distribution

are many opportunities for integrating Analytics into a Smart Grid Solution

entifying Overloaded/Stressed Assets

- Proactive Transformer Replacement

cating Transformer Voltage Problems

nnectivity Model Improvements

- Auto-Generating Secondary Circuit Models
- Correcting Meter Phasing
- Detecting Transformer Connectivity Problems

proved Fault Locating

- Using Substation Power Quality and Relay Data
- Using Feeder Monitors to Locate Faults and Estimate Cause

ing AMI Data to Detect Theft and/or Unmetered Load

- Unbilled revenue
- Tamper detection & Irregular Usage Patterns

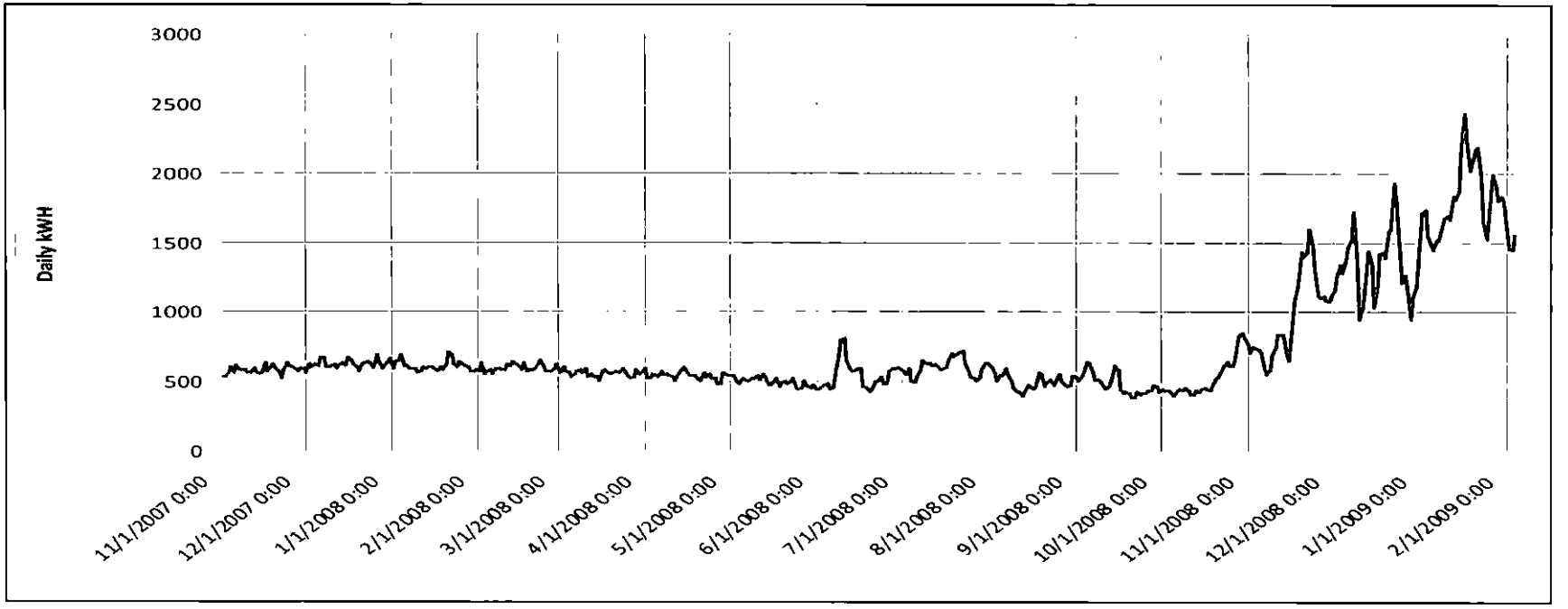
liability Analysis, Storm Analysis and Momentary Analysis

ɲsformer Monitoring

ECO uses analytics to determine transformer overloads and predict failures based on metering data from
AMI data

program was initiated to investigate daily transformer consumption data (aggregated from meter data) fc
ıat occurred during winter peak load days

ansformers with yr-to-yr load increase of more than 25% (3,764 winter, 1,233 summer) were flagged for
ıvestigation

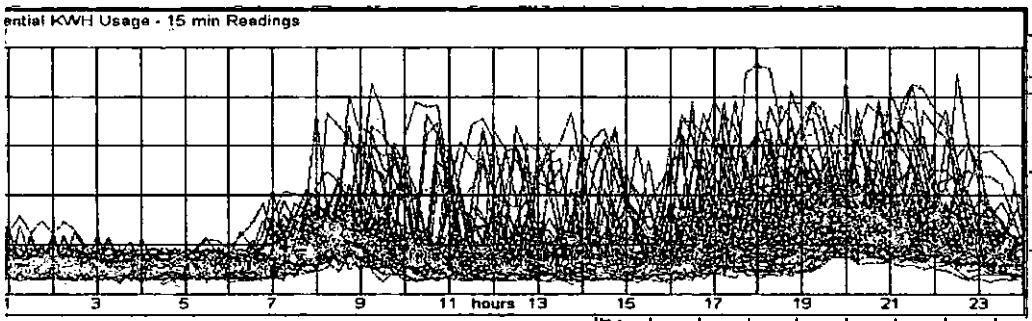


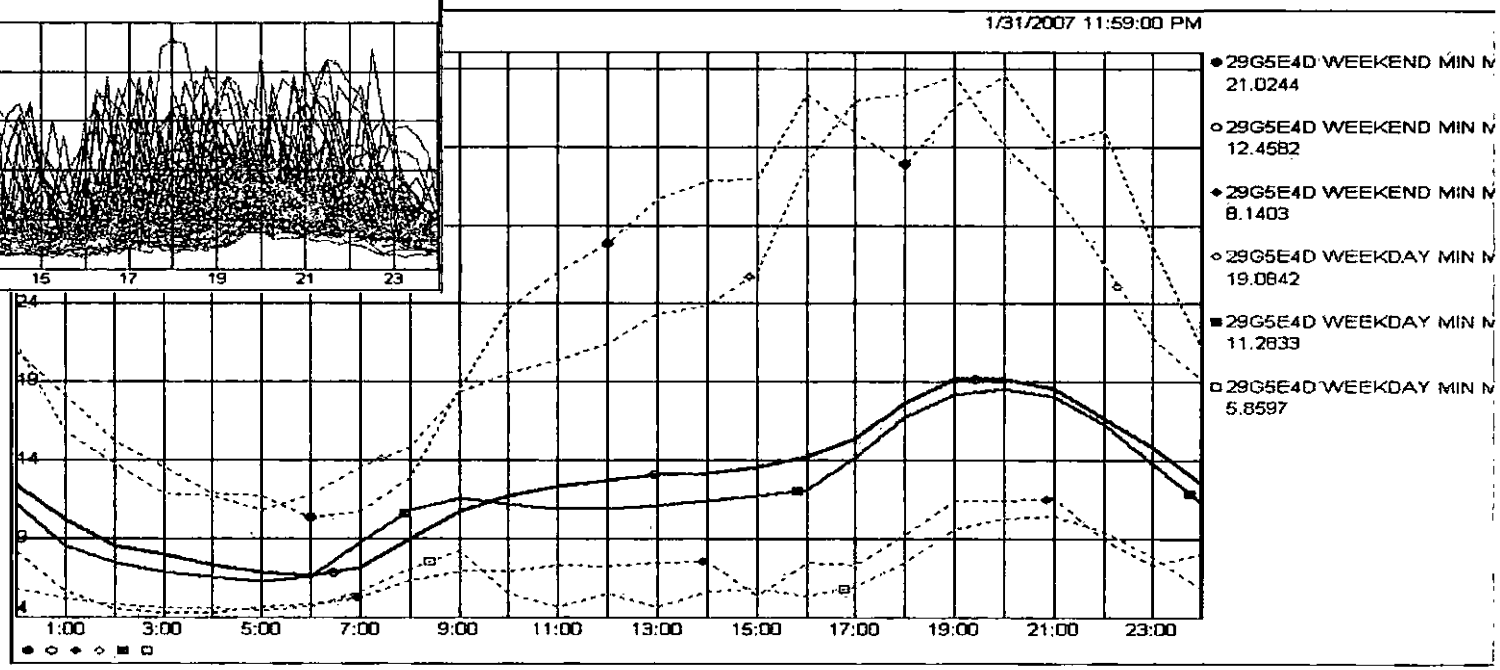
ısformer Load Profiles

Once a daily transformer load shape is identified, it can be used to develop overall "normal" op parameters for each device

If the loading suddenly exceeds the normal operating envelop, an investigation can be initiated understand why the change in load shape

- In many cases, the equipment may need to be upsized or load relief activities must be performed





onclusion

The Smart Grid and AMI is able to deliver many benefits to consume
and the Utility

Benefits include:

- Outage Management
- Voltage Profiling
- Asset/Transformer Management
- And many others . . .

Thank You

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APPENDIX R

PECO Energy Company's Answers to
Interrogatories and Requests for Documents
of Complainant Laura Sunstein Murphy, Set I
C-2015-2475726

Murphy I-35:

Has PECO received any complaints from customers who claim adverse health effects from exposure to emissions from AMI Wireless Smart Meters? If so, please provide:

a. The number of customer complaints;
b. How many of the complaints received were resolved to the satisfaction of the customer;
c. How each complaint was resolved.

PECO objected to this Interrogatory. In a May 6, 2016 Order, ALJs Heep and Pell sustained the objection in part and overruled the objection in part, stating that:

Disposition: This objection is sustained in part and overruled in part. The information sought goes to the awareness of PECO and therefore the reasonableness of its response to such complaints. Relevant information would include the number of customers affected and the practices and procedures to address and prevent similar occurrences, if necessary. The question is limited, however, to those matters filed with an entity within the jurisdiction of the Commission.

PECO Answer to Murphy I-35:

a. PECO has received eight complaints (two informals filed with the Bureau of Consumer Services and six formals filed with the Office of Administrative Law Judge) from customers who claim that they have adverse health effects from exposure to emissions from AMI wireless smart meters.

b. See answer to subpart (c) for resolution of the matters. PECO does not have information regarding whether the resolutions set forth below were to the satisfaction of the customer.

c. The matters were resolved as follows:

- The two informal complaints were dismissed by the Bureau of Consumer Services; no formal complaint was filed.
- Two of the formal complaints were dismissed on preliminary objections.
- One of the formal complaints proceeded to hearing and is in the briefing stage.

- Three of the formal complaints are scheduled for evidentiary hearings.

Responsible Witness: Prepared by counsel.

APPENDIX S

DOI 10.1515/reveh-2013-0004 — Rev Environ Health 2013; 28(2-3): 75–84

Magda Havas*

Radiation from wireless technology affects the blood, the heart, and the autonomic nervous system[1)]

Abstract: Exposure to electrosmog generated by electric, electronic, and wireless technology is accelerating to the point that a portion of the population is experiencing adverse reactions when they are exposed. The symptoms of electrohypersensitivity (EHS), best described as rapid aging syndrome, experienced by adults and children resemble symptoms experienced by radar operators in the 1940s to the 1960s and are well described in the literature. An increasingly common response includes clumping (rouleau formation) of the red blood cells, heart palpitations, pain or pressure in the chest accompanied by anxiety, and an upregulation of the sympathetic nervous system coincident with a downregulation of the parasympathetic nervous system typical of the "fight-or-flight" response. Provocation studies presented in this article demonstrate that the response to electrosmog is physiologic and not psychosomatic. Those who experience prolonged and severe EHS may develop psychologic problems as a consequence of their inability to work, their limited ability to travel in our highly technologic environment, and the social stigma that their symptoms are imagined rather than real.

Keywords: electrosmog; radio-frequency radiation; rouleau; tachycardia; WiFi; Wolff-Parkinson-White Syndrome.

[1)]Presented at the Corporate Interference with Science and Health: Fracking, Food, and Wireless, Scandinavia House, New York, NY, March 13 and 14, 2013.

***Corresponding author: Magda Havas,** PhD, Environmental and Resource Studies, Trent University, Peterborough, ON, K9J 7B8 Canada, E-mail: mhavas@trentu.ca; www.magdahavas.com

Introduction

Our exposure to devices using electricity and emitting extremely low-frequency and radio-frequency electromagnetic fields has been increasing ever since Edison invented the incandescent light bulb and Tesla and Marconi discovered that radio-frequency (RF) radiation can be transmitted without wires. Radio, television, computers, cell phones, and their accompanying cell phone antennas, cordless phones, wireless routers (WiFi), wireless baby monitors, wireless games, and smart meters are increasing our exposure to RF radiation and especially to microwave radiation (300 MHz–300 GHz).

As an example of the proliferation of this technology, access to WiFi was limited in 2002 but by 2012 access was virtually ubiquitous in the USA (Figure 1). We have city-wide WiFi in some communities, WiFi at work, at home, in school, universities, and hospitals, in restaurants and coffee shops, on public transit, at airports, and on an increasing number of airplanes. As a society, we seem to be insatiable for wireless technology and the connectivity it affords.

Although the downside to this technology, namely, the potentially harmful effects of nonionizing radiation, has received relatively little attention in North America and remains controversial, it is an area that deserves proper research funding based on the sheer number of users and people exposed worldwide to RF electromagnetic fields.

In this article, the relationship between electrosmog exposure and electrohypersensitivity (EHS), with a focus on the cardiovascular system, is presented, based on provocation studies and on reports of ill health among those living near cell phone base stations or exposed to WiFi in schools.

Electrohypersensitivity

Just as some people have multiple chemical sensitivity or react to pollen, mold, and certain types of food, a growing population is becoming "sensitive" to electromagnetic radiation.

Khurana et al. (1) reviewed ten epidemiologic studies, three dealing with cancer and seven with neurobehavioral effects, that examined the putative effects of mobile phone base stations. All of the neurobehavioral studies reported more symptoms with proximity to base stations, and only

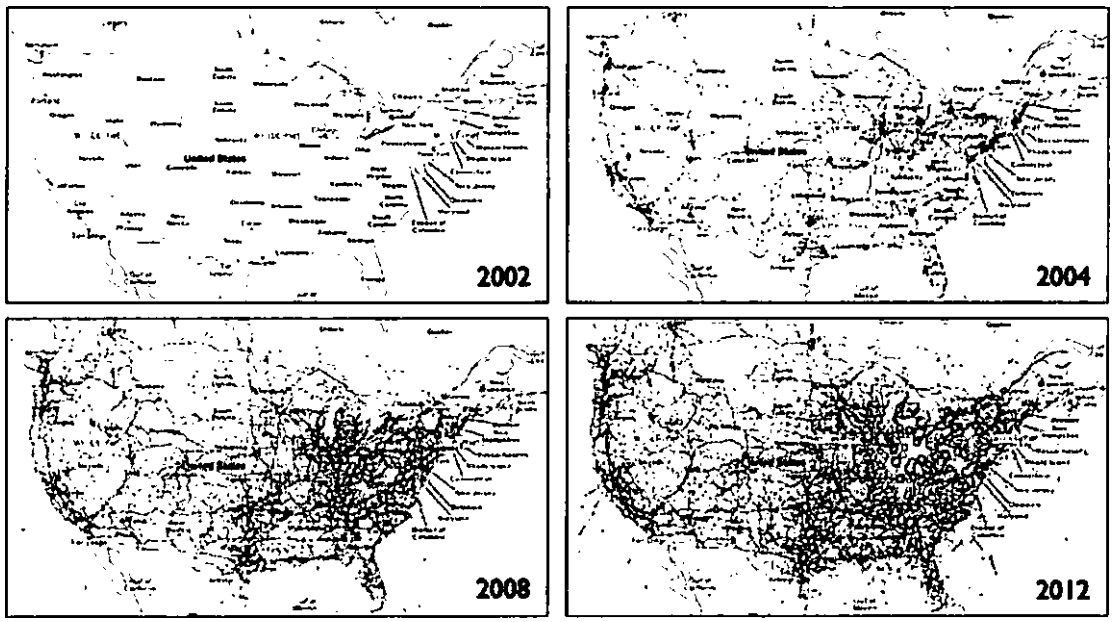

Figure 1 WiFi networks in the USA from 2002 to 2012 (source: wigle.net).

one attributed these health effects to stress rather than RF exposure.

The results from one of these studies are presented in Figure 2 (2). People who lived closest to the antennas experienced the following symptoms more often than those who lived further away: fatigue, sleep disturbance, headaches, feeling of discomfort, difficulty concentrating, depression, memory loss, visual disruptions, irritability, hearing disruptions, skin problems, cardiovascular problems, dizziness, loss of appetite, movement difficulties, and nausea. Many of these symptoms are more common as we age, thus I prefer to call this rapid aging syndrome (RAS). The difference between real aging and RAS experienced by those who are electrically hypersensitive is that when these people go into an electromagnetically clean environment, many of their symptoms diminish

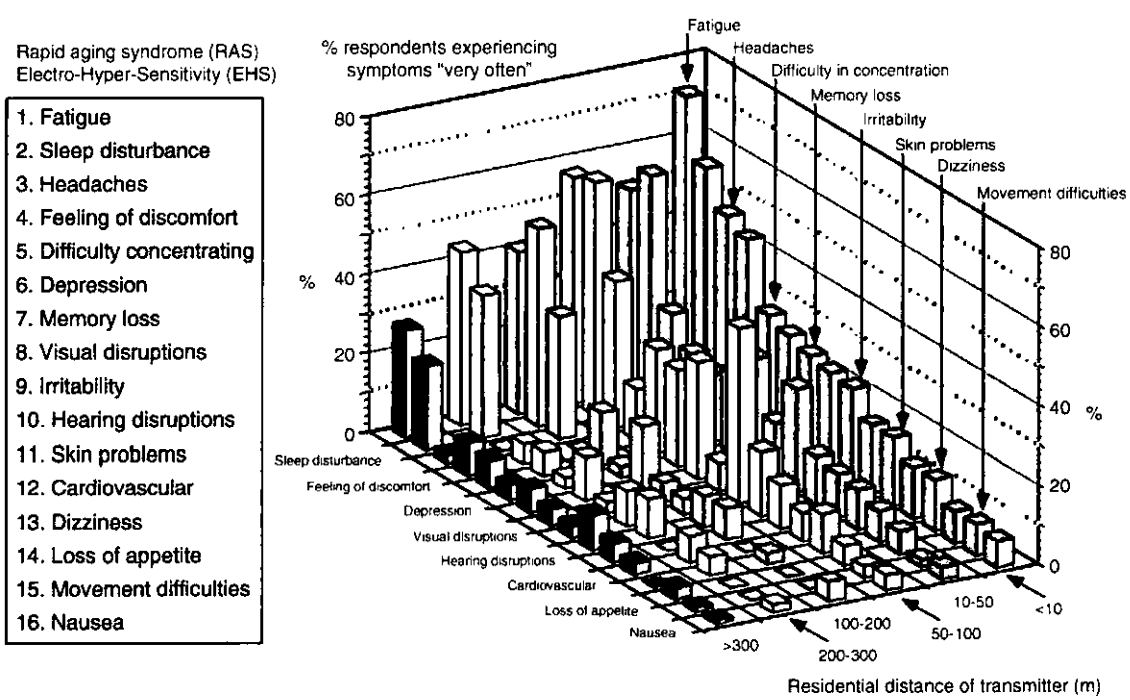

Figure 2 Symptoms experienced by people near cellular phone base stations [based on the work of Santini et al. (2)].

or disappear. Obviously, this does not happen with real aging.

Because cell towers are proliferating and difficult to avoid in both urban and rural communities and if the results of Santini et al. (2) represent what is happening to those who live near cell towers, then it is quite likely that we are going to experience (or are in the midst of experiencing) an emerging health crisis that is contributing to chronic ill health and is promoting the sale of pain medication, sleep medication, antidepressants and antianxiety medication, pills to moderate energy level and mood, and drugs for those with attention deficit hyperactivity disorder such as Ritalin® (metylfenidat).

In 2006, Hallberg and Oberfeld (3) documented the increasing prevalence of EHS. Figure 3 clearly shows that self-perceived EHS is on the rise. According to the authors, by 2017, 50% of the population is going to be complaining of this illness. Admittedly, this is a rough calculation but it demonstrates that symptoms of EHS are increasing.

It is difficult to estimate the percentage of the population that has EHS. I use a conservative estimate of 3% of the population for those who have severe symptoms, and this is based on the population in Sweden who have registered as being electrohypersensitive (4). Another 35% population may have mild to moderate symptoms of EHS when exposed to electrosmog (5). Based on these percentages, the cumulative number of people who may be adversely affected in Canada, the USA, and Europe is 25 million, for severe sensitivity (EHS), and another 300 million, for mild to moderate sensitivity (electrosensitivity). People in this latter group can function in an electrosmog environment but may develop headaches or have difficulty sleeping and are living a life compromised by increasingly poor health as a consequence of their exposure (Figure 2).

Historically, environmental contaminants have been presented as contentious issues due, in part, to the media's need for "balanced reporting" and, in part, to the economic consequences of altering our behavior as consumers. This was certainly the case with asbestos, dichloro-diphenyl-trichloroethane (DDT), lead, mercury, acid rain, and tobacco smoke and is currently the case with climate change and EHS.

EHS may be viewed as a contentious issue, yet a growing number of international experts, scientists, and medical doctors have been asking governments and international agencies for decades to lower existing guidelines for RF radiation because the current guidelines do not protect public health. Table 1 provides a list of some of these resolutions and appeals.

Some governments have heeded the warnings and have exposure guidelines that are a fraction of those recommended by the World Health Organization (WHO) and accepted by the USA, UK, and Canada.

The WHO held an international workshop on electrosensitivity in Prague in 2004 (6), and they defined EHS as follows:

> "... a phenomenon where individuals experience adverse health effects while using or being in the vicinity of devices emanating electric, magnetic, or electromagnetic fields (EMFs)."

> "Whatever its cause, EHS is a real and sometimes a debilitating problem for the affected persons.... Their exposures are generally several orders of magnitude under the limits in internationally accepted standards."

What role should the WHO and other leading health authorities play in helping these sensitive individual? Some would advocate, at the very least, lower exposure

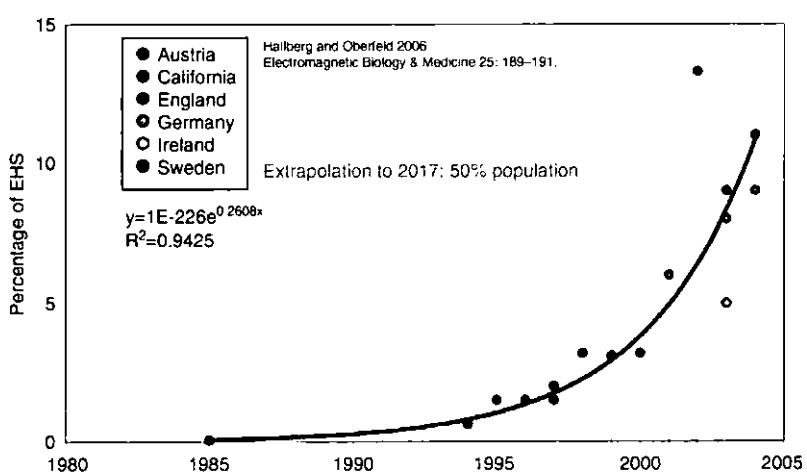

Figure 3 Estimated prevalence of self-proclaimed EHS in various countries [based on the work of Hallberg and Oberfeld (3)].

Table 1 Appeals and resolutions from international groups of scientists and medical doctors.

Resolution/group	Country	Year	Link
Salzburg Resolution	Austria	2000	http://www.magdahavas.com/international-experts-perspective-on-the-health-effects-of-electromagnetic-fields-emf-and-electromagnetic-radiation-emr/
Catania Resolution	Italy	2002	www.emrpolicy.org/faq/catania.pdf
Freiburger Appeal	Germany	2002	http://www.magdahavas.com/international-experts-perspective-on-the-health-effects-of-electromagnetic-fields-emf-and-electromagnetic-radiation-emr/
World Health Organization	Czech Republic	2004	http://www.who.int/peh-emf/meetings/hypersensitivity_prague2004/en/
Irish Doctors' Environmental Association	Ireland	2005	www.ideaireland.org
Helsinki Appeal	Finland	2005	www.emrpolicy.org/headlines/helsinki_appeal_05.pdf
Benevento Resolution	Italy	2006	http://www.icems.eu/docs/BeneventoResolution_REVISED_march2008.pdf
BioInitiative Report	USA	2007 and 2012	www.bioinitiative.org
Venice Appeal	Italy	2008	http://www.icems.eu/resolution.htm
Porto Alegre	Brazil	2009	http://www.icems.eu/docs/resolutions/Porto_Alegre_Resolution.pdf
Seletun	Norway	2011	http://www.magdahavas.com/international-experts-perspective-on-the-health-effects-of-electromagnetic-fields-emf-and-electromagnetic-radiation-emr/
International Doctors Appeal	Germany	2012	http://www.icems.eu/resolution.htm

limits and possibly places where the radiation is not allowed, similar to smoke-free environments. Instead, the WHO recommended that this illness be referred to as "idiopathic illness", which basically means the cause is unknown. By refusing to acknowledge the cause, the WHO undermines the need for governing agencies to act.

In contrast to the WHO, the Austrian Medical Association (7) came out with guidelines to help doctors diagnose and treat those who experience EHS. In that document, they recognize that there is a rise in stress-related illness and that electrosmog may play a role. They even provide a temporary code (Z58.4, exposure to radiation) under the *International Classification of Diseases, 10th Edition* to be used for EMF syndrome, which is their term for EHS.

A group of psychologists considers EHS to be entirely a psychologic illness rather than a physiologic response to electrosmog (8, 9). A number of the articles reviewed by Rubin et al. are based on flawed assumptions about (1) who is truly experiencing EHS, (2) how people with EHS respond to exposure, (3) what frequencies and intensities they respond to, (3) how quickly they respond and recover following exposure, and (3) how the data should be analyzed. These flawed assumptions lead to flawed conclusions.

For example, not everyone who believes they have EHS actually have EHS. Thus, combing the results for the self-proclaimed "EHS group" is likely to dilute the results, producing no significant effect when analyzed statistically. The question that is being tested by this type of analysis is, "Do those who believe to be electrically sensitive all respond the same way to provocation testing?" and the answer is likely to be "no".

In the study by Rea et al. (10) of 100 people who believed they were electrically hypersensitive, only 16 responded consistently to real exposure and not to sham exposure. Had the results been statistically analyzed for the entire 100 subjects tested, they would have shown no effect of EMF exposure. Objective testing is required, and people should be assessed as individuals rather than members of a group for analysis. An analogous situation is if there were 16 people with diabetes among a group of 100 people who all thought they were diabetic. Statistical analysis of blood sugar measurements before and after consuming a standard meal for the entire group would likely miss the 16 people with diabetes.

The proper way to test for EHS is to monitor and assess individual responses to electrosmog exposure in a double-blind study, as was done by Rea et al. (10).

However, it is clear that those who experience EHS and are no longer able to live a "normal" life and who are not supported by their family, friends, and physicians also experience stress leading to psychologic problems including depression and anxiety disorders. Where I disagree with Rea et al. (10) about EHS is that I believe the physiologic response precedes the psychologic problem.

In this article, examples of the effects of electrosmog on the blood, heart, and autonomic nervous system (ANS) are provided, indicating that EHS is a physiologic response to electromagnetic pollution. The only legitimate use of the term "idiopathic" (i.e., disease or disorder that has no known cause) is in reference to the trigger that initiated the electromagnetic sensitivity. In some cases, with good medical investigation, this also can be surmised.

Electrosmog affects the blood

Healthy blood consists of erythrocytes (red blood cells), which are round and which float freely in the plasma. A live blood sample, consisting of a drop of blood from a finger prick, can be viewed under the microscope, as shown in Figure 4. Changes in the size, shape, and clumping of these erythrocytes can indicate impaired health.

Figure 4 shows live blood (blood without any chemicals added to it) in an electromagnetically clean environment (A) and the blood from the same person spoke on a cordless phone for 10 min (B) and after using a wired computer for 70 min (C). The erythrocytes are sticking together and resemble a stack of coins. This is known as rouleau formation and indicates unhealthy blood.

Usually rouleau is caused by an increased fibrinogen concentration or other changes in plasma proteins as in multiple myeloma or macroglobulinemia. An alternative explanation is that the rouleau may be due to a reduction in the electrical potential at the cell membrane, which would weaken the repellent forces between cells. A third possibility is that it is a microscopic artifact, which, in this case, is unlikely because the results are repeatable. Research on the mechanisms involved in the rouleau formation is needed.

With rouleau formation, the surface area of the red blood cells is significantly reduced, and the release of nutrients and the removal of waste products are compromised. Symptoms may include headaches, difficulty concentrating, dizziness, nausea, heart and blood pressure problems as well as cold, numbness, or tingling sensation in the extremities (hands and feet).

The good news is that live blood analysis may be a useful diagnostic for EHS. How quickly the blood clumps and how quickly it recovers following exposure may be a good indicator of the degree of sensitivity.

Electrosmog affects the heart and the autonomic nervous system

Some people who are electrically hypersensitive complain of pain or pressure in the chest area, heart palpitations,

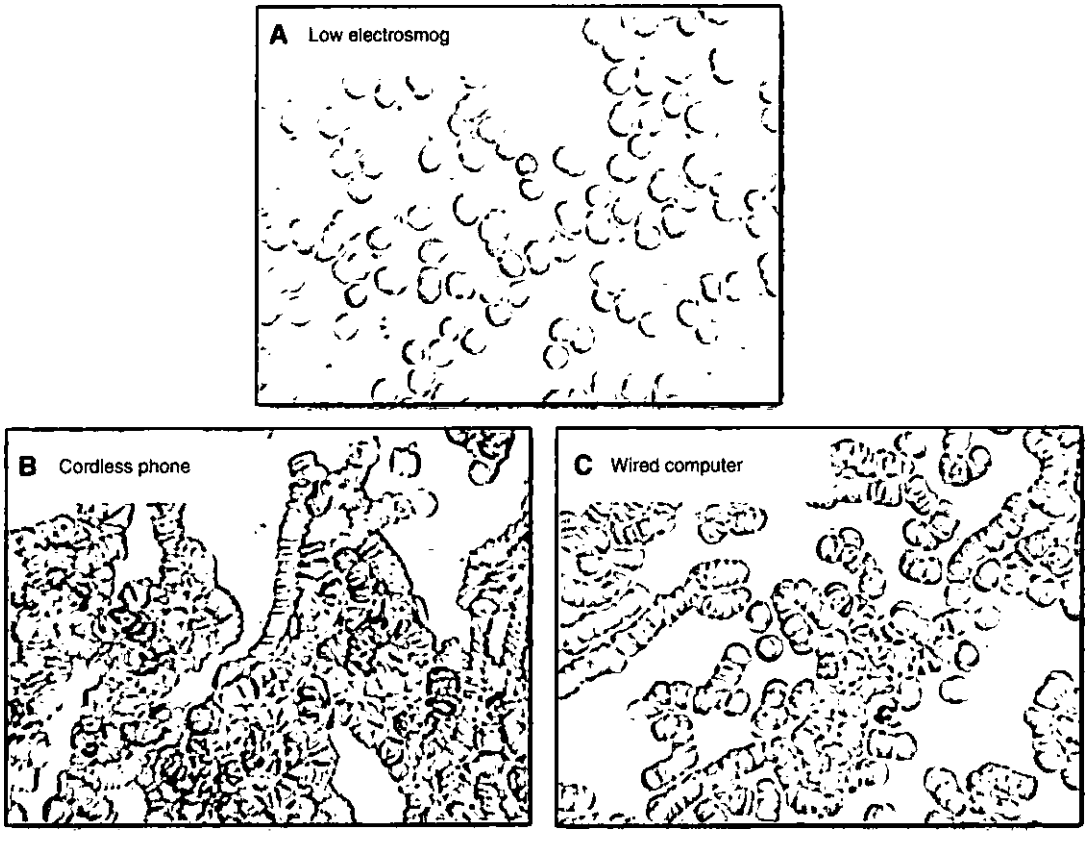

Figure 4 Live blood cells in a low-electrosmog environment (A), after using a cordless phone for 10 min (B), and after using a wired computer for 70 min (C).

and/or an irregular heartbeat, accompanied by feelings of anxiety that develop rapidly. The symptoms resemble a heart attack and thus contribute to even more anxiety.

To test the effect of electrosmog on the heart, Havas et al. (11) designed a simple experiment where subjects were exposed to electromagnetic radiation generated by the base of a cordless phone. This was a double-blind study with randomized real and sham exposure. A cordless phone base station was selected as the source of exposure because the base emits a constant beacon signal when it is plugged into an electrical outlet. The beacon signal in this case was a pulsed frequency of 2.4 GHz, the same frequency used in WiFi.

In the original study (11), 25 subjects from Colorado were tested, and although most subjects did not react adversely to the radiation from the cordless phone base station (see Figure 5, subject A), a few did react with either tachycardia (rapid heart rate) or arrhythmia (irregular heart rate) (Figure 5, subject B). The reaction was often immediate and coincided with exposure to the radiation. When the radiation ceased, the heart returned to normal.

Two examples of responsive subjects are provided. The heart rate of subject B increased from a resting heart rate of 68 beats per minute (bpm) to a rapid 122 bpm during exposure, decreased to 66 bpm as soon as the radiation was stopped, and increased to 129 bpm when it was resumed. This reaction occurred while the subject was resting in a supine position and was unaware of when he or she was or was not exposed.

During the exposure to radiation from the cordless phone base station, subject C (Figure 6) experienced a slight increase in heart rate (from 65 to 86 bpm), an irregular heartbeat, and changes in the response of the sympathetic and parasympathetic nervous system (SNS and PNS, respectively). This upregulation of the SNS and downregulation of the PNS is an example of the "fight-or-flight" response, indicating physiologic stress. During periods of this type of stress, the body redirects most of the blood and energy from the internal organs to the arms and legs to prepare the organism for fighting or fleeing a stressful situation. Intermittent exposure may not cause a problem but if the exposure is continuous and long-term, the immune system of the body will be compromised and the body will not be able to repair itself, resulting in symptoms that are commonly experienced by those who are electrically hypersensitive. This inability to heal is what then accelerates the symptoms of aging (i.e., RAS).

The level of radiation in this experiment was well below international guidelines. Subjects were exposed to 3 $\mu W/cm^2$, or 0.3% of the guidelines recommended by International Centre for Non-Ionizing Radiation Protection (ICNIRP), the Federal Communication Commission (in US) (FCC), and Health Canada for 2.4-GHz frequencies. According to these organizations, harmful biologic effects do not occur below these thermal guidelines. Both blood and heart results from these provocation experiments indicate otherwise, i.e., that biologic effects that can have serious health implications do occur at levels well below current thermal guidelines.

The cordless phone provocation study has since been repeated for a larger group of subjects and shows similar results (12).

Some suggested that the radiation from the cordless phone was interfering with the technology rather than the heart. If this were the case, then 100% of the subjects would have had similar results because the

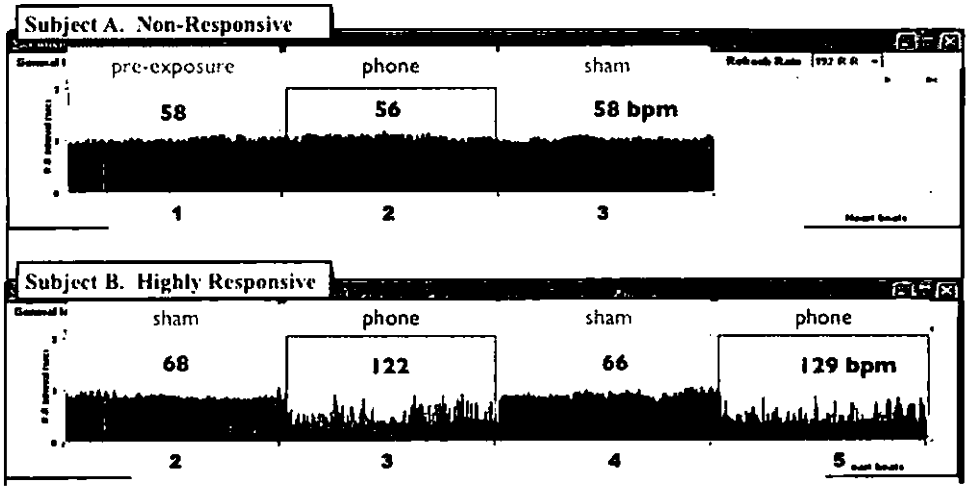

Figure 5 Rhythmograph of HRV during provocation with a digital 2.4-GHz cordless phone and sham exposure. The x-axis unit is time, with each stage lasting approximately 3 min. The y-axis is the R-R interval (in seconds).

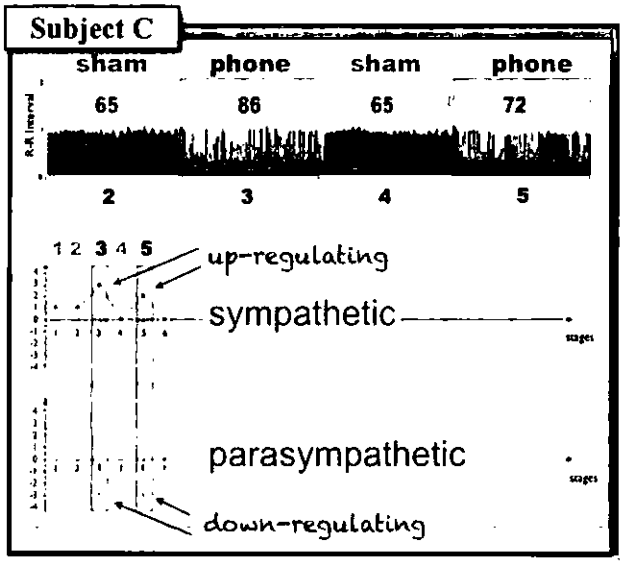

Figure 6 Rhythmograph of HRV and functioning of the SNS and PNS during provocation with digital 2.4-GHz cordless phone and sham exposure.

electromagnetic interference (EMI) would have been consistent rather than highly variable and individualistic. Additional testing of higher levels of radiation at the sensor did not affect the heart rate variability (HRV) of a subject who was nonresponsive to the original levels. Had it been EMI, then higher levels of exposure should have had a greater response, but this was not the case (12).

One subject (52-year-old man) told us that he normally experiences a delayed reaction to electrosmog exposure, and thus we monitored him for 30 min postexposure and observed the delayed response during a period of no exposure. The response included periods of short-term and intermittent irregularity in the R-R interval (HRV) as well as episodic downregulation of both the SNS and the PNS, which were both low to begin with (12). The normally low heart rate, 53–55 bpm, began to increase slightly (61 bpm) 25 min postexposure.

WiFi in schools affects student health

Students in schools with WiFi are complaining of headaches, difficulty concentrating, weakness, and heart palpitations, prompting their parents to take them to their family doctor and to their pediatric cardiologist to determine the nature of their problem.

In one Ontario school district, several students complained of heart problems. A 6-year-old girl had a "musical heart", and she experienced headaches and dizziness only at school. A 12-year-old boy had tachycardia (rapid heart rate). A 12-year-old girl experienced nausea, vomiting, no fever, insomnia, blurred vision, and tachycardia only at school. A 13-year-old boy had a pounding heart, insomnia, and headaches. His family moved to a different school district, and his symptoms disappeared.

In the same area, 4 students had sudden cardiac arrests (SCA) during exercise class within a 2-year period. Two of these students were resuscitated. The annual rate for SCA among young people in Canada is approximately 7 per year; hence, 4 in a small community is unusual.

According to Sinatra (13), a cardiologist, Wolff-Parkinson-White (WPW) syndrome, which is a disorder of the conduction system of the heart, is present in 1 out of 700 students. In a school district with 50,000 students, as many as 70 may have this generally undiagnosed condition. According to Sinatra (13), when students with WPW syndrome are exercising and are exposed to microwave radiation, the combined stress on the heart can lead to supraventricular tachycardia, thus creating the "perfect storm".

Fortunately, due to the Defibrillator Access Act, schools and other public buildings are installing defibrillators. What they should also be doing is trying to determine what is causing SCA and why students are complaining of headaches and heart palpitations at school. A key question that needs to be asked is, "What role does RF radiation from a school's WiFi system and from nearby cell phone base stations play in these symptoms?"

The effects of microwave radiation on the heart have been known for decades (14). In a 1969 symposium on the biological effects and health implications of microwave radiation, the authors clearly state that, "In the interest of occupational hygiene...researchers have recommended that cardiovascular abnormalities be used as screening criteria to exclude people from occupations involving radio-frequency exposures". Perhaps students need to be screened at school to ensure that they do not have an underlying heart condition that may be exacerbated with WiFi microwave exposure.

According to Drezner et al. (15), out-of-hospital SCA among young people is on the rise in the USA, although doctors do not know the reason. The increasing exposure to electrosmog may be to blame for at least part of this increase. More research is urgently needed in this area.

Children are much more sensitive to environmental toxins than are adults, and as such, there should be stricter guidelines for exposure. To date, at least nine countries have issued warnings that children should limit their use of cell phones. These countries include the UK (2000), Germany

(2007), France (2008), Russia (2008), India (2008), Belgium (2008), Finland (2009), the USA (2009), and Canada (2012). The same warning should be issued for children exposed to wireless games and WiFi routers, depending on the amount of time students are exposed to these emitters.

WiFi routers emit a beacon signal that is continuous as long as the device is activated. In other words, you do not have to be connected to the Internet to be exposed to the radiation generated by the wireless router. When information is either uploaded or download, the radiation levels increase both at the router and at the computer. The same is true for cordless phones and wireless baby monitors. Voice-activated baby monitors and cordless phones that radiate only when in use are available in Europe but are not currently available in North America.

Historic research on microwave illness resembles current research on electrohypersensitivity

The information provided in this article is not new. Reviews as far back as 1969 summarized the effects of microwave radiation and identified many of the same symptoms. Dodge (16) reviewed the Soviet and Eastern European literature and reported that microwave radiation affects the central nervous system, ANS (as shown here), neurohumoral systems, endocrine glands and functions, eye and ocular function, blood and hematopoietic system (as shown here), and miscellaneous organs.

Dodge (16) identified general subjective complaints resulting from exposure to electromagnetic radiation (Table 2) that are similar to the symptoms experienced by those who live near cell phone base stations (Figure 2). The major difference is that Dodge was reviewing symptoms for men who were occupationally exposed, whereas Santini et al. (2) was documenting symptoms for those who lived near cell phone antennas and were exposed to radiation in their own homes and as such were unable to avoid exposure.

Glaser (17) reviewed the literature on the biologic effects of microwave radiation and provided more than 2000 references in 1972. Although many of these studies were conducted at levels above existing guidelines, we are getting similar results at levels of microwave radiation that are well below these guidelines.

Most revealing are the "psychophysiologic disorders" based on human behavioral studies. These disorders include the following and are similar to those reported by Santini et al. (2): neurasthenia (general "bad" feeling), depression, impotence, anxiety, lack of concentration, hypochondria, dizziness, hallucinations, sleepiness, insomnia, increased irritability, decreased appetite, loss of memory, scalp sensations, increased fatigability, chest pain, and tremor of the hands.

Both Glaser and Dodge worked for the US Navy and had access to information that was later declassified. In one limited-edition (only 15 copies were produced) document, Pollack and Healer (18) recommended that the power density guideline in the USA be reduced from 10,000 $\mu W/cm^2$ to the same level used in the Soviet Union (10 $\mu W/cm^2$), but little attention was paid to this recommendation.

Table 2 Subjective symptoms associated with RF and microwave radiation.

General subjective complaints resulting from exposure to electromagnetic radiation (16)	Symptoms experienced "very often" by those who live within 300 m of a cell phone base station (2)
Similar symptoms	
Pain in head and eyes	Headaches and visual disruptions
Weakness, weariness, and dizziness	Dizziness and fatigue
Depression, antisocial tendencies, and general irritability	Depression and irritability
Impairment of memory and general mental function	Memory loss
Adenoma and inability to make decisions	Difficulty concentrating
Chest pain and heart palpitation	Cardiovascular
Dyspepsia, epigastric pain, and loss of appetite	Loss of appetite
Sensitivity of mechanical stimulation and dermagraphism	Skin problems
Different symptoms	
Lacrimation	Irritability
Hypochondria, sense of fear, and general tension	Nausea
Inhibition of sex life (male)	Movement difficulties
Scalp sensations and hair loss	Hearing disruption
Trembling of eyelids, tongue, and fingers	Sleep disturbance
Asthma	Feeling of discomfort
Brittle fingernails	

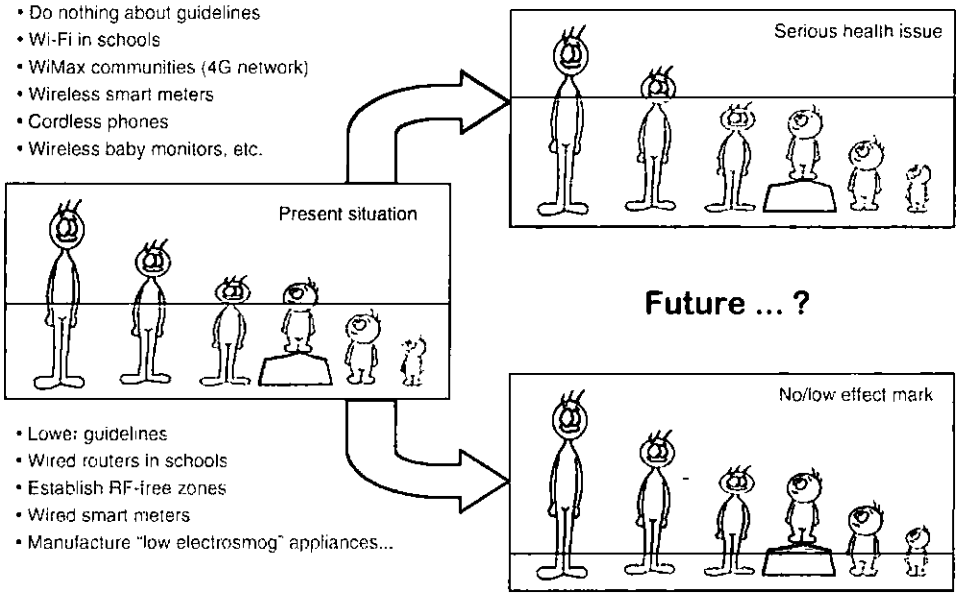

Figure 7 Two future health scenarios based on the steps we take or fail to take to reduce electrosmog exposure.

Years later, the power density guideline in the USA was reduced from 10,000 to 1000 $\mu W/cm^2$, although this was still based on thermal effects.

Where do we go from here?

If we do nothing about guidelines and allow WiFi to be installed in schools, if we allow WiMax to come into neighborhoods as part of the 4G network, if we allow wireless smart meters to be installed on homes, and if we fail to regulate the technology in a way that minimizes microwave exposure, then many more people are likely to become ill and some will die (Figure 7).

If we choose to minimize exposure by establishing biologically based guidelines rather than the current thermal guidelines, by encouraging wired Internet access in schools, universities, hospitals, workplaces, and homes, by installing wired smart meters, and by establishing RF-free zones for those who are highly sensitive, then we can reverse much of the damage that has been inflicted (Figure 7).

The choice is ours, and the real question is, "Do we have the foresight and courage to make the right decision or will we require a health tsunami before we act?"

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APPENDIX T

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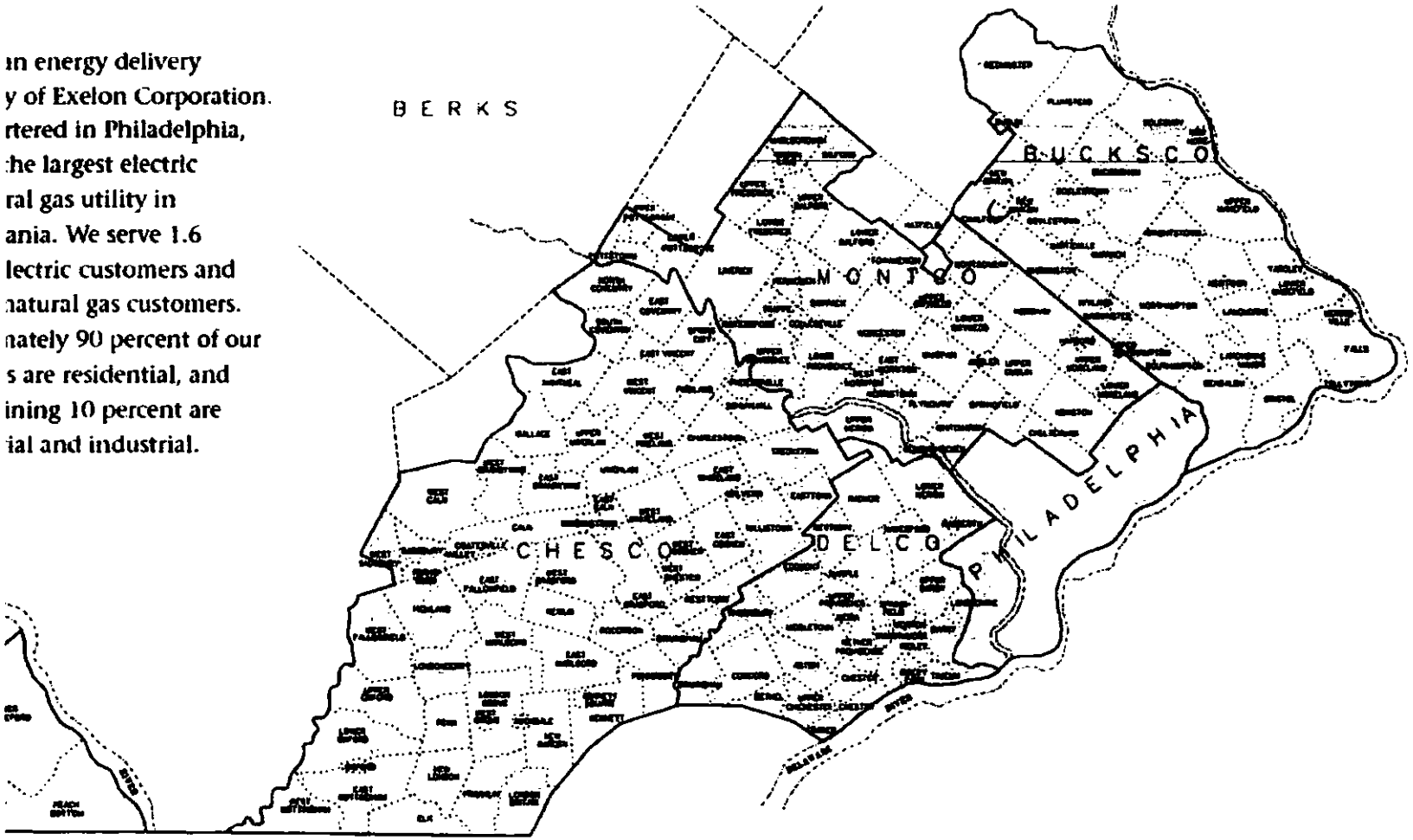

APPENDIX U


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MURPHY STATEMENT NO. 3
C-2015-2475726 12/5/16
PLI DC

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

Laura Sunstein Murphy	:	
	:	
v.	:	Docket No. C-2015-2475726
	:	
PECO Energy Company	:	

DIRECT TESTIMONY PETER J. PROCIUK, M.D.
ON BEHALF OF COMPLAINANT
LAURA SUNSTEIN MURPHY

April 29, 2016

TABLE OF CONTENTS

DIRECT TESTIMONY
OF
PETER J. PROCIUK, M.D.

I. INTRODUCTION AND PURPOSE OF TESTIMONY

1. Q. Please state your full name, occupation and business address.

A. My name is Peter J. Prociuk, MD. I am a physician who has been in private practice since 1992 in the Greater Philadelphia area. Before establishing my private practice, I served as Attending Physician for many years in Emergency Departments at large hospitals: first at Presbyterian Medical Center in Philadelphia and then at Paoli Memorial Hospital. My office is located at 225 North High Street, West Chester, Pennsylvania 19380.

2. Q. Please state your professional and educational background and experience.

A. My professional qualifications and experience are set forth in my CV, attached hereto as Appendix A.

3. Q. Are you a medical doctor?

A. Yes. I graduated from the University of Saskatchewan College of Medicine in 1981. I have been licensed to practice medicine in Pennsylvania since 1983.

4. Q. Are you board certified?

A. Yes. I am board certified in Internal Medicine, and I am a Diplomate of the American Board of Internal Medicine.

5. Q. How would you describe your practice?

A. I treat both adult and children patients who present with a whole panoply of disease. Often, patients come to me as a last resort, after they have consulted many other physicians who have not helped them to heal their diseases. I treat the whole patient, not just one aspect which might be revealed in a test result.

Since 1992, when I decided to leave the large hospital setting, I have focused on treating individual patients in my office. I realized that conventional medicine offered nothing substantial to help people recover their health from chronic illnesses. I realized that the pharmaceutical model often does not recognize the natural healing instinct present in all living organisms, and that this model of writing prescriptions solely to suppress symptoms temporarily, and not to effect a more permanent cure for the patient, forced me to focus my acumen elsewhere.

To date, I have treated over 14,000 patients with a combination of homeopathy and traditional medicine where indicated. My patients come to me from every age group and socioeconomic status: I even treat patients who visit me from foreign countries, presenting with a wide variety of conditions. This experience with homeopathy, combined with a medical background as an internist, has broadened my abilities to truly understand patients' medical conditions and what prescription drugs can or cannot do. My homeopathic experience allows me to see what other alternative approaches are possible. I have a background in nutrition, the use of supplements, and preventative lifestyle management, and I also offer advice in this regard.

Where appropriate, I write prescriptions for prescription drugs and blood and urine diagnostic testing, and imaging studies. I refer patients to specialists where indicated. I like to follow patients closely, and often patients have been seeing me for many years, because I continue to help them, and not just treat their test results.

6. Q. What is the purpose of your testimony?

A. I am submitting this testimony to provide information regarding my treatment of Laura Sunstein Murphy for her many and ongoing diverse set of health issues since her first consultation with me in 2005. Her medical conditions are complex and unique, and unlikely to be replicated in the PECO customer base. She has been diagnosed with Ehlers Danlos Syndrome Type III, and lipedema Stage II, and has cytochrome p450 genetic abnormalities resulting in oversensitivity to multiple drugs and chemicals. Ms. Murphy has never been able to tolerate fluorescent lights or loud noises. Axiomatically, her entire body is tremendously overburdened with excessive free radical formation and oxidative injury. Ms. Murphy suffers from a very low threshold for premature and accelerated degenerative processes in multiple manifestations. Her injuries and illnesses are always more extensive than a person who does not suffer from Ehlers Danlos Syndrome, with delayed and complicated healing, a primary genetic condition present in her case.

Further, I set forth my recommendation that PECO remove her current AMR meter and refrain from installing an AMI meter at Ms. Murphy's residence. My recommendation is based on my view that continued exposure to EMF and RF

radiation from these types of meters is unsafe and harmful to the fragile health of a patient with a unique array of devastating and incapacitating medical conditions.

II. LAURA MURPHY'S MEDICAL HISTORY

7. Q. When did you first see Laura Sunstein Murphy as a patient?

A. I first saw Laura Murphy as a patient in September 2005.

8. Q. What is Ehlers Danlos Syndrome Type III?

A. Ehlers Danlos Syndrome Type III is a rare genetic disease that is passed on through families. The form of Ehlers Danlos Syndrome (EDS) Ms. Murphy has been diagnosed with manifests itself throughout all the connective tissues in the body. Although there is variation in the manifestation of symptoms of EDS type III even among family members, it is diagnosed clinically, because there is no genetic test that has been isolated for Type III EDS. It usually causes pale, translucent and fragile skin, astigmatism and myopia, hyperflexibility and instability of many joints, weak and easily bruised veins and lymphatics, gut motility issues, easily torn tendons and ligaments, and delayed injury healing, among other manifestations. I have attached as Appendix B some of the literature by Dr, Marco Castori describing EDS Type III. Dr. Castori is a leading EDS researcher from Rome, Italy.

9. Q. What health complaints did Ms. Murphy have when she first consulted with you?

A. Ms. Murphy had been suffering from chronic constipation since 2002, with concomitant severe abdominal pain daily, and had had several bouts of diverticulitis,

starting in 2003, which had necessitated several trips to local hospital Emergency Departments. Recently, she was told by a specialist who performed a colonoscopy that she should undergo a colon resection as soon as possible. Although Ms. Murphy was born with Ehlers Danlos Syndrome, a genetic mutation which interferes with proper collagen synthesis, up until 2002 when she was 55 years old, she had not been seriously inconvenienced by her underlying Ehlers Danlos Syndrome condition.

Ms. Murphy was hoping that there might be an alternative treatment available which might avoid the invasive colon surgery, with its long recovery period and lifelong potential problems. At the time, Ms. Murphy was a very busy lawyer, having established her own boutique health law practice in West Chester in 2003, and tending to her farm and horses. She did not want to have to be laid up for months on end, and she had had a severe adverse reaction to general anesthesia in the past.

10. Q. What treatments have you recommended for Ms. Murphy?

A. It is difficult to condense my various medical treatment recommendations of Ms. Murphy for this testimony. Ms. Murphy has come to me for medical advice for her differing medical symptoms almost every month for the past 11 years.

I want to emphasize at the outset that Ms. Murphy is not a hypochondriac, but her list of symptoms and the sheer number of specialists she has been forced to consult, especially in the last ten years, would lead one to believe that she is obsessively involved with her body. That is not the case. When I treat hypochondriacs, I refer them to specialists, and the specialists report back to me that they could find nothing wrong with my patient. Every single time I have referred Ms. Murphy to a specialist,

the specialist has confirmed that there was definitely something wrong with Ms. Murphy, and they have reported back the treatment that they have prescribed.

I have treated Ms. Murphy with nutritional advice, ordered, interpreted and advised her on blood and urine tests, referred her to specialists who have performed surgery on her or performed other diagnostic tests, referred her for therapy for various diseases, ordered medical equipment and prescription drugs to treat her illnesses, and prescribed homeopathic remedies which fit her state of disease at the time.

11. Q. **Could you please detail the various ailments that Ms. Murphy has suffered from for which she has consulted with you over the years?**

A. She has asked my help in managing her diseases as they manifest themselves, and they have appeared more and more frequently as the eleven years have gone by.

I will list all the diagnoses and symptoms not mentioned above in chronological order, although a few predated my actual start of treatment:

1. Diverticulitis, severe constipation: first reported 2003

2. Chronic fatigue, hypothyroidism first diagnosed in 2002; I took over managing her hypothyroidism condition shortly after I took her on as a patient.

3. In 2004, Ms. Murphy had been diagnosed with uterine fibroids, and had been treated by her gynecologist who performed a dilation and curettage (D & C) operation.

Also in 2004, Ms. Murphy was first diagnosed with atrial fibrillation. It has been a very delicate balancing act over the last decade to treat Ms. Murphy's hypothyroidism effectively with desiccated thyroid dosing adjustments, while avoiding causing atrial fibrillation to reappear.

Ms. Murphy had been treated for Lyme disease four times before I started seeing her. Living in Chester County, and taking care of her horses every day and gardening, it is understood that Ms. Murphy would be bitten by ticks and would develop Lyme disease. I have treated her for Lyme disease symptoms on three distinct occasions since she first consulted me.

In 2005, Ms. Murphy was first diagnosed with aortic valve regurgitation and mitral valve prolapse.

In 2006, Ms. Murphy first consulted me regarding her weight issues, which had started in 2001, but had been manageable; now she was unable to lose weight, which seemed to be piling up more and more, no matter how much exercise she engaged in and no matter how few calories she ate. She followed my advice to the letter, and was able to lose twenty pounds, but she was not able to lose any more weight, and the very strict diet protocol produced even more severe constipation in her body.

12. Q. What made Ms. Murphy's weight increase unusual in your opinion?

A. While many women tend to put on weight in menopause, due to hormonal changes, it has to be understood that Ms. Murphy led a very active and physically demanding life, despite working hard at her legal career. She tended to her horses' needs every

day, rode them every weekend, tended to the garden on her own, traveled internationally with her husband in the summertime, and visited their children and grandchildren often. She did all the cooking and cleaning up and entertaining of friends. So, it was surprising to me that she was unable to lose more weight, when most of my dedicated active patients have been able to lose as much weight as they wanted to lose and keep it off. I suspect the same endocrine and hormonal disturbances which precipitated her development of hypothyroidism in 2002, is responsible for her flare up of lipedema symptoms which followed thereafter. There may well be a connection between the replacement of the Murphy's analog meter with the PECO AMR meter in 2002, and Ms. Murphy's development of hormonal and endocrine disturbances which manifest themselves in hypothyroidism, the onset of lipedema symptoms and other bodily dysfunctions.

In 2007, Ms. Murphy was diagnosed with ptosis (drooping eyelids) which blocked her peripheral vision. She was successfully operated on by a plastic surgeon.

2007: Spontaneous detached retina of the right eye, surgically repaired on an emergency basis.

2008: She began to complain of severe leg pain. Ms. Murphy was diagnosed with venous insufficiency by two different vascular surgeons who subjected her to several vein ablations in both legs and one arm. I prescribed medical grade support hose, physical therapy, and a sequential pneumatic whole lower body lymphatic pump.

2008, Ms. Murphy was unable to continue practicing law on a full time basis because of her increasing pain levels and worsening medical conditions, and she closed her office in West Chester, transferring her client files to another law firm.

2010: Ms. Murphy suffered a second spontaneous retinal detachment in her right eye. Her retinal specialist performed a second surgical laser repair.

2011: I advised Ms. Murphy that she had postponed her colon resection long enough, and she should schedule a resection as soon as possible because her life had become so impacted by the intractable constipation and diverticulitis pain, which no amount of positive dietary and supplemental modification, or homeopathic or allopathic medication had been able to resolve.

In October 2011, Ms. Murphy underwent a sigmoid colon resection. I advised her regarding her very difficult six to eight month recuperative process, which included the growth of adhesions and protracted fatigue and weakness following general anesthesia administration.

Shortly following the colon resection, Ms. Murphy suffered from a severe sinus infection leading to a perforated eardrum. Additionally, although she had suffered from dry eyes for many years, and had undergone punctal cauterization to address the extreme dryness, she reported the onset of irregularly spaced sharp stabbing pains in both of her eyes, with no precipitating factors. Her ophthalmologist was not able to offer any explanation or treatment for those stabbing pains, but they largely resolved following her arm liposuction surgeries in 2013 (see below).

13. Q. What new symptoms developed in Ms. Murphy in 2012?

A. By 2012: Ms. Murphy's leg pain became more and more debilitating. She was unable to walk more than a few blocks because of the pain. She could not pick her feet up to clear cracks in the sidewalk. I advised her regarding supplement changes and suggested whole body vibration exercise as a method of moving the lymphatics gently, referred her to lymphatic physical therapy, and she was diagnosed with Stage II lipedema, a congenital progressive adipose tissue disorder which affects only women, characterized by whole body inflammation, easy bruising, pain to the touch, and pain in the limbs when vertical. And as the growth of abnormal fat on the limbs, which cannot be lost by diet or exercise (and in later stages, all over the body), presses on the nerves, the pain and debility becomes intense. As the disease process advances and the lymphatic system becomes increasingly ineffectual, lipedema can lead to more paralyzing pain, lipolymphedema and cellulitis, which can be life threatening. See Appendix C for medical literature on lipedema. An excellent one hour video on diagnosing lipedema by Dr. Karen Herbst, the world's leading expert on lipedema and other rare adipose tissue disorders, is available from the University of Arizona Grand Rounds website here:

https://streaming.biocom.arizona.edu/event/?id=25173&play=1&format=hd

Ms. Murphy was born with a tendency towards developing lipedema, but up until her mid-fifties, she had held been able to hold it at bay; she was Stage I up until the irregular fat on her thighs became increasingly inflamed, and she developed the

typical Stage II "peas in a bag" characteristic of Stage II lipedema which occurs when macrophages appear in the lipedema fat and the fat continues to hypertrophy.

In late 2012, Ms. Murphy was diagnosed with pelvic organ prolapse disease, anterior and posterior, despite possessing excellent muscle tone in that region, and she underwent successful surgery in February 2013, but she remained an invalid post op, unable to walk more than a half a block, with increasing fatigue and pain which rendered her almost completely disabled.

14. Q. Why did Ms. Murphy travel to Germany in 2013 and 2014 for treatment?

A. Ms. Murphy researched lipedema treatments, which were not well known or available in the United States, since lipedema is a rare disease. In 2013, there was no physician in the United States who treated lipedema surgically. She was able to locate several competent surgeons in Germany, and I advised her to travel to Germany to undergo surgery, because the German doctors had been successfully treating lipedema surgically for over twenty years.

In June 2013, Ms. Murphy and her husband traveled to Germany for six weeks, where she underwent two successful water jet assisted liposuction surgeries with Dr. Josef Stutz, the pioneer of water jet assisted liposuction for the treatment of lipedema. See Appendix D for two medical journal articles written by Dr. Stutz on his treatment of lipedema patients.

In November 2013, Ms. Murphy was able to travel alone to Germany for Dr. Stutz to perform a third lipedema surgery, this time on her arms.

Dr. Stutz had recommended that Ms. Murphy undergo a perforator vein closure, because that vein was not functioning normally, and was heating up her calf. He remarked that the vein problem had been previously hidden by lipedema fat. Later in 2013, Ms. Murphy underwent many venous injections in the United States, but the American vascular surgeon did not close the perforator vein which had been troubling Ms. Murphy, so she scheduled a return visit to Dr. Stutz in Germany the following spring.

15. Q. Why did Ms. Murphy travel to Arizona in 2013?

A. Ms. Murphy traveled to Arizona alone to consult with Dr. Karen Herbst in October 2013, a trip she could not have contemplated before her liposuction surgeries, and Dr. Herbst confirmed her diagnosis of lipedema Stage II.

16. Q. What happened to Ms. Murphy in March of 2014?

A. On March 6, 2014, while traveling to physical therapy, Ms. Murphy was involved in a motor vehicle accident, when her car was broadsided by another vehicle which had run a red light at high speed, which caused a concussion, multiple torn ligaments in her knees, lumbar spine spasms, severe neck pain, and torn rotator cuffs in both shoulders, and resulting post-concussion syndrome. I treated her for the post-concussion syndrome, referred her to Bryn Mawr Rehab Post-Concussion Syndrome Unit, and monitored her recovery process as she consulted me from time to time with increasing frequency.

Subsequent to the motor vehicle accident, in addition to the conditions enumerated above, Ms. Murphy has suffered from increasing fatigue, mental confusion, difficulty concentrating, difficulty driving, difficulty in brightly lit or highly contrasting light and dark environments, and other symptoms of post concussive syndrome.

Spring, summer and fall 2014: Ms. Murphy was receiving ongoing treatment for car accident injuries to her soft tissue, through physical therapy and platelet rich plasma and stem cell treatments by a sports medicine physician. Even though she has developed osteoarthritis in both knees over the last ten years, and has many tears in her meniscus and surrounding tissue, because of her Ehlers Danlos, she is not a candidate for knee replacement or tendon transfer. *PRP* and stem cell treatment are her best option for repair of these defects.

17. Q. How was Ms. Murphy's recuperation from the car accident?

A. Ms. Murphy was not making much progress in healing from the car accident, despite vigorous treatment. She became more and more fatigued and lacked concentration. I ordered thyroid panels and other blood work. Based on her blood work, I increased her thyroid medication, and ordered fecal occult blood tests. The occult blood tests were positive, so I referred her to have a colonoscopy and endoscopy, which were performed in January 2015.

18. Q. What disease process further plagued Ms. Murphy's remaining colon?

A. The endoscopy revealed a hiatal hernia; the colonoscopy revealed a highly dysplasic 5 cm sessile adenomous polyp, which the gastroenterologist recommended be treated immediately via colon resection.

Ms. Murphy was unwilling to undergo a second colon resection; I referred her to Dr. Gregory Ginsberg at the University of Pennsylvania, to attempt to remove the polyp via endomucosal colon resection (EMR).

March 2015: Ms. Murphy underwent an EMR procedure with Dr. Ginsberg, wherein he removed most of the 5 cm sessile polyp from her ascending colon. Pathology results confirmed that much of the polyp was highly dysplasic, formerly known as carcinoma in situ.

She was unable to perform most of her activities of daily living already, due to her increasing disabilities, and PECO's threat of terminating her electricity was increasingly her stress level exponentially.

Ms. Murphy's colon had begun to spasm immediately after the EMR, and she consulted me; the spasms were so unrelenting that she was soon unable to eat any solid food, and was subsisting on carrot juice alone. I referred her to a gastroenterologist who examined her thoroughly and ordered many studies, including pelvic and abdominal MRIs. Ms. Murphy had an adverse reaction to the gadolinium dye used in the MRI.

The colon spasms continued unabated until I was able to prescribe a few homeopathic remedies which relieved the spasms.

19. Q. What did the pelvic and abdominal MRI reveal in 2015 and how was that treated?

A. Early Summer 2015: The pelvic MRI revealed an endometrial growth, which her gynecologist removed via surgery performed in the early summer of 2015.

20. Q. What new symptoms did Ms. Murphy develop in late summer of 2015?

A. Later summer 2015: Ms. Murphy consulted me for high blood pressure verging on panic, a new symptom, plus continued fatigue. I treated her and referred her to her cardiologist for follow up.

October 2015: Ms. Murphy had postponed her six month follow up EMR with Dr. Ginsberg in August 2015 because her husband had developed sepsis in early August, and it was not clear he would survive. After spending the months of August and September in two hospitals and two nursing homes, and after several trips to the ER and many emergency surgeries, Ms. Murphy's husband finally stabilized and she was able to return for a second EMR procedure with Dr. Ginsberg.

21. Q. How were increased inflammation and oxidative stress manifested in symptoms starting in October 2015?

A. October 2015: Ms. Murphy developed sinus inflammation from the down pillows she was allergic to in the hotel she stayed in overnight in Philadelphia to undergo her

colonoscopy preparation. The EMR procedure went uneventfully. Dr. Ginsberg was able to avoid using epinephrine this time, which had pushed her colon into spasm for months earlier.

October 2015: Ms. Murphy's sinus congestion and inflammation grew much worse. She consulted me, but the inflammation had spread to her inner ear, affecting her balance. She consulted her new concierge primary care physician who prescribed a 10 day course of antibiotics, which caused her to have adverse gut reactions which she still suffers from to this day. She consulted her osteopath, who, over the course of several months, successfully treated her for the imbalances in her inner ear, but Ms. Murphy's forehead is still feverish from the incident which started in October of 2015.

22. Q. How did you treat one of the underlying causes of Ms. Murphy's increasing fatigue in November and December of 2015?

A. November and December 2015: Ms. Murphy consulted me about her increasing fatigue. I reviewed her blood panels and found her iron and vitamin D and B vitamins to be extremely low, despite her supplementation with iron and vitamin D and B vitamins. I suggested she undergo iron infusions, and referred her to a hematologist, who concurred with my diagnosis of iron deficiency anemia, and prescribed a series of five weekly iron infusions, which Ms. Murphy underwent in January and February 2016. Ms. Murphy's iron levels have returned to normal as of this writing.

23. Q. How and when did Ms. Murphy develop a hiatal hernia, and how was it treated?

A. Because of her underlying Ehlers Danlos Syndrome, an inherited connective tissue disease, and because of poor technique which did not take into consideration her Ehlers Danlos Syndrome nor her colon resection incision weakness, the colonoscopy performed on Ms. Murphy in January 2015 had resulted in an immediate ventral abdominal hernia, measured at 5 cm, along the same incision line which had been the site of her colon resection in 2011.

March 2016: Ms. Murphy had searched for almost a year to find a skilled surgeon to repair the ventral hernia without mesh which is contraindicated in Ehlers Danlos patients, and without the use of typical general anesthesia drugs. She was able to undergo a successful hernia repair in March 2016, but she had an adverse drug reaction when the anesthesia team gave her a drug which she could not tolerate, that is, one compounded with fluoride, which she is unable to metabolize, due to her genetic mutations.

24. Q. What cardiac symptoms did Ms. Murphy manifest recently?

A. April 2016: Ms. Murphy consulted me again for alternating high blood pressure with normal blood pressure and fast heat rate immediately when she wakes up in the morning.

III. HEALTH EFFECTS OF PECO'S METER

25. Q. How did you become aware of Ms. Murphy's issues with PECO's meter?

A. In March 2015, while Laura was lying down resting at home shortly following the EMR procedure described above, PECO sent Ms. Murphy the 10 day shut off notice for refusing an AMI meter on her home.

She asked me if I would write a letter to PECO, explaining that she had a medical condition that required her to have electricity. For one thing, her farm has well water, and without electricity, she has no water, her husband has no water, and her animals have no water.

I wrote the letter and faxed it to PECO. My letter is attached as Appendix E.

26. Q. What is Ms. Murphy's prognosis at this point, and why is she asking PECO to remove the AMR meter EMF?

A. Ms. Murphy continues to live as an invalid. Her weakened condition deprives her of the activities she used to enjoy with zeal. She no longer has the strength to feed her horses, or even brush them. She has not been able to ride them in over a year. She cannot cook a meal for herself or her husband. They have had to hire part time workers to cook dinner for them several nights a week and clean up. She can no longer tend to the herb and vegetable garden she used to take care of herself. They have had to hire a gardener. Ms. Murphy cannot travel to see her grandchildren by herself. She cannot drive for any distance longer than a half hour. She is too fatigued.

Ms. Murphy is a patient who is dedicated to getting well. She is overwhelmed by the deterioration of her body and her mind which has taken place at a rapidly increasing rate. She is not able to heal from one insult when she is faced with another insult.

Ms. Murphy, because of her genetic makeup, has always been highly sensitive to smells, fluorescent lights, and electrical pulsations, and she is unable to tolerate most pharmaceutical drugs. Because of her genetic makeup, her body is in a continual state of inflammation and oxidative stress. Her body is stressed just with the inability to methylate normally and derive nourishment from her food, as revealed in her genetic testing and organic acid test panels. Supplementation and anti-inflammatory dietary suggestions which I have recommended have been helpful to Ms. Murphy in reducing inflammation, but they are by no means a cure, by any measure. The sheer number of operations she has had to undergo in the last three years, added to the number of operations she has had to undergo in previous years, and her adverse drug reactions, indicate that she has a much higher load of oxidative stress and inflammation than almost any other of my patients. Ms. Murphy has been engaged in a continual struggle for survival ever since the AMR meter was placed on her home, increasingly so in the last four years.

27. Q. **Do you concur with Dr. Pall's analysis of the contributions of the PECO AMR meter to Ms. Murphy's lack of health?**

A. Dr. Martin Pall, Ms. Murphy's expert on EMF, has researched extensively how PECO's smart meters can cause oxidative stress and a whole cascade of diseases, including many of the diseases Ms. Murphy has suffered from since the PECO AMR

meter was installed on her home in 2002. I have read Dr. Pall's testimony and I concur with it.

Neither Ms. Murphy nor I were previously aware that PECO's AMR meter which was placed on her home in 2002 was emitting a constant stream of irregular bursts of low level EMF, spiking to up to 2 watts for an emission, at least every five minutes, for 20 milliseconds apiece, according to information from PECO (attached here as Appendix F), into her living room and all rooms on the same side of the house as the meter, including her bedroom, the layout of which forces the master bed to be situated with its headboard on the same wall as the AMR meter. I understand that Ms. Murphy herself has measured the spikes of EMF emitted by her PECO furnished AMR meter, and her measurements indicate far more frequent and irregular spikes of bursts of EMF: 6 times in 18 minutes, with irregularity reminiscent of the atrial fibrillation she suffers from.

28. Q. Why, in your medical opinion, after treating Ms. Murphy every month since 2005, should PECO remove the AMR meter and replace it with non EMF emitting meter?

A. The presence of any PECO EMF emitting device on the Murphy property is medically contraindicated. Ms. Murphy has to be provided the ability to heal and rest from any unnecessary increase in EMF. She has eliminated her Wi-Fi, she has eliminated her iPad, she has purchased a tablet which she has hard wired. She uses her cell phone only for emergencies. She has no cordless phones in her house. She has eliminated all sources of EMF that she is able to in her home environment. PECO

must do the same for the health of Ms. Murphy to allow her to heal and to enable her to live the rest of her life without any unnecessary increases in her oxidative stress. She is already genetically predisposed to much higher levels of oxidative stress than normal individuals, and she "just can't get a break" to allow her body to heal as long as PECO showers her home with bursts of spiked EMF, night and day.

29. Q. Does this conclude your direct testimony?

A. Yes.

APPENDIX A

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Education

- University of Saskatchewan, Canada, BS 1975
- University of Saskatchewan, Doctor of Medicine 1981
- Internship/Residency Internal Medicine Presbyterian Medical Center, Philadelphia, PA 1982-1985

Credentials

- Pennsylvania Medical License 1983-present
- Diplomat American Board of Internal Medicine (Board Certification) 1985

Experience

- 1985-1987: Assistant Director of Intensive Care Unit, Department of Medicine, Presbyterian Medical Center, Teaching Staff, Presbyterian Medical Center, Philadelphia, PA
- 1987-1989: Attending Physician, Department of Emergency Medicine, Presbyterian Medical Center; Philadelphia, PA, and establishment of private practice in Internal Medicine
- 1989-1996: Attending Physician, Department of Emergency Medicine, Paoli Memorial Hospital, Paoli, PA
- 1992: Established part time practice in classical homeopathic medicine in Chester County, PA, while working full time as Attending Physician in the Department of Emergency Medicine, Paoli Memorial Hospital, Paoli, PA
- 1993-present: Full time practice in Internal Medicine and classical homeopathic medicine, in West Chester, PA, treating children and adults
- 2002-present: Integrating Defeat Autism Now! methods into the treatment of patients with autism spectrum disorder diagnoses

Professional Outreach

- Extensive outreach throughout my career, including speaking engagements at conferences, private organizations, talk radio, and producing and hosting a call-in radio show.

APPENDIX B

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Review Article

Ehlers-Danlos Syndrome, Hypermobility Type: An Underdiagnosed Hereditary Connective Tissue Disorder with Mucocutaneous, Articular, and Systemic Manifestations

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Ehlers-Danlos syndrome, hypermobility type, constituting a phenotypic continuum with or, perhaps, corresponding to the joint hypermobility syndrome (JHS/EDS-HT), is likely the most common, though the least recognized, heritable connective tissue disorder. Known for decades as a hereditary condition with predominant rheumatologic manifestations, it is now emerging as a multisystemic disorder with widespread manifestations. Nevertheless, the practitioners' awareness of this condition is generally poor and most patients await years or, perhaps, decades before reaching the correct diagnosis. Among the various sites of disease manifestations, skin and mucosae represent a neglected organ where the dermatologist can easily spot diagnostic clues, which consistently integrate joint hypermobility and other orthopedic/neurologic manifestations at physical examination. In this paper, actual knowledge on JHS/EDS-HT is summarized in various sections. Particular attention has been posed on overlooked manifestations, including cutaneous, mucosal, and oropharyngeal features, and early diagnosis techniques, as a major point of interest for the practicing dermatologist. Actual research progresses on JH/EDS-HT envisage an unexpected link between heritable dysfunctions of the connective tissue and a wide range of functional somatic syndromes, most of them commonly diagnosed in the office of various specialists, comprising dermatologists.

1. Introduction

Ehlers-Danlos syndrome (EDS) was first recognized in the first decade of the twentieth century as a hereditary disorder with typical skin manifestations (Figure 1) [1, 2]. Over the decades, EDS emerged as a clinically and genetically heterogeneous group of disorders, including an increasing number of variants (Table 1) which share the variable combination of dermal fragility, internal organ and vessel ruptures, and joint hypermobility (JHM) [3]. After many years of nosologic confusion, a group of experts, who met in Villefranche in 1997, identified six major EDS subtypes, namely, classic, hypermobility (i.e., Ehlers-Danlos syndrome, hypermobility type—EDS-HT), vascular, kyphoscoliotic, arthrochalasis, and dermatosparaxis, recognized by specific diagnostic criteria [4]. Among them, EDS-HT is the most difficult to recognize due to the lack of clinical diagnostic handles and confirmatory laboratory/molecular tests. Nevertheless, EDS-HT is now considered the commonest EDS variant [5] with an unexpectedly high disability potential [6].

While EDS-HT is characterized by the "absence" of the typical cutaneous manifestations observed in many other EDS subtypes, skin and mucosae represent common sites of disease manifestation with a plethora of minor anomalies, whose detection still has an invaluable role in suspecting such a creeping condition. Moreover, given the protean constellation of additional features of EDS-HT, mastering the broad spectrum of subtle findings detectable at inspection is crucial for early diagnosis and management of potentially disabling complications.

2. Definition(s)

EDS-HT is a hereditary connective tissue disorder (HCTD) defined by the association of generalized JHM, joint instability complications, widespread musculoskeletal pain, and

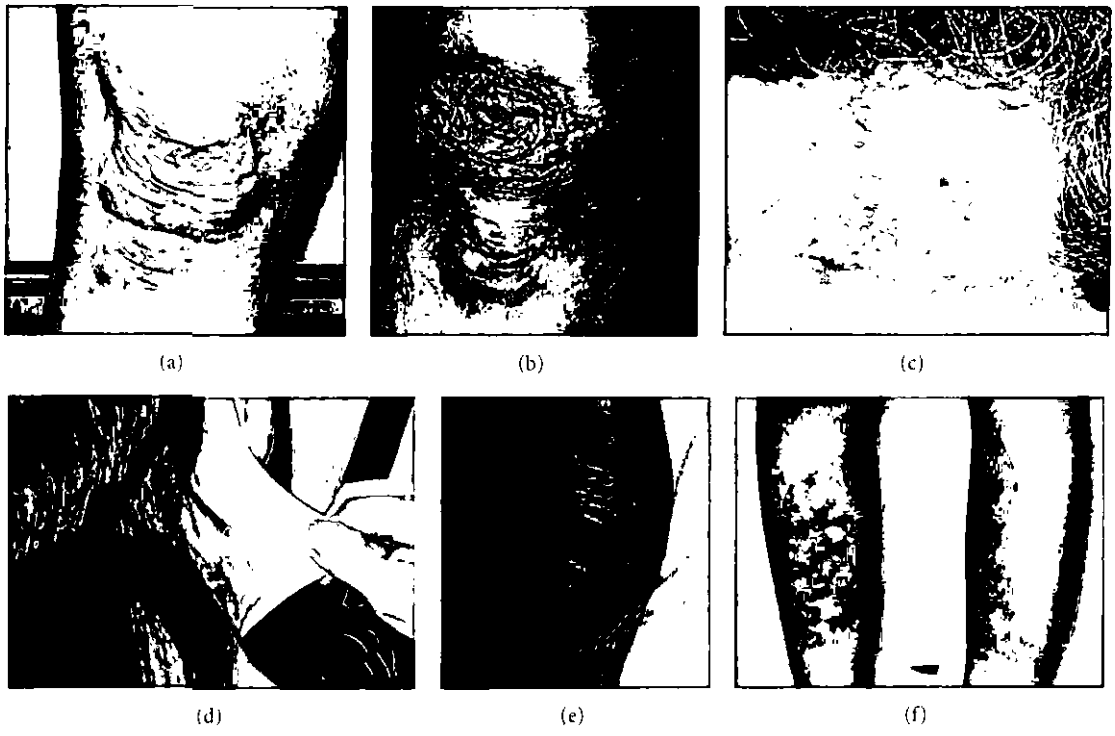

FIGURE 1: Typical cutaneous features of Ehlers-Danlos syndrome(s). Papyraceous (a), hemosiderotic and atrophic (b), and depressed (c) scars. Skin hyperextensibility (d). Molluscoid pseudotumor of the heel (e). Multiple ecchymoses with hemosiderotic depositions (f). Note that, except for skin hyperextensibility, such cutaneous changes are not observed in the hypermobility type.

(minor) skin features [4]. Table 2 summarizes the Villefranche diagnostic criteria for the six best known variants, including EDS-HT. EDS-HT shows a significant phenotypic overlap with the joint hypermobility syndrome (JHS), a rheumatologic disorder with a high disability potential [7] and strong familial aggregation. Whether such similarities reflect etiological identity remains to be established. However, many researchers are used to consider EDS-HT and JHS one and the same (i.e., JHS/EDS-HT) [8], while others do not completely agrees with this assumption [5]. In parallel with EDS-HT, JHS is actually recognized by the revised Brighton criteria (Table 3) [9]. Based on literature data and personal experience, the author of this paper agrees with the concept of considering EDS-HT and JHS the same condition.

Besides skin manifestations that are relatively unspecific in both sets of diagnostic criteria, (generalized) JHM is the most clinically relevant feature of JHS/EDS-HT. The term "joint hypermobility" refers to the ability of one or more joints to actively and/or passively move beyond normal limits. It may affect a few joints (i.e., monoarticular or localized JHM) or present in multiple body sites (i.e., generalized or general JHM). JHM is a sign, not a disease, and its occurrence varies by age, sex, and ethnicity. In fact, loose joints are more common among females and children than males and older people [10]. By ethnicity, JHM is observed in up to 35–57% Africans [11], while it shows a much lower rate (6% in females, 2% in males) among Caucasians [12]. In addition, various acquired/environmental factors, such as traumas, surgery, and regular training, may contribute in increasing range of motion at one or more joints.

Interpretation of generalized JHM is not always straightforward and needs a holistic perspective. In fact, JHM is often experienced as an asset for some occupational and sport activities, such as ballet, gymnastics, and playing instruments [13]. At the same time, generalized JHM is the physical marker of various HCTDs. Distinguishing between benign, asymptomatic JHM and an HCTD is of utmost importance for preventing potential life-threatening complications and/or early detecting and managing long-term disabilities.

3. Epidemiology

The early literature fixed to 1/5,000 the frequency of EDS as a whole [14], with EDS-HT accounting for approximately half of all registered cases. However, as JHS/EDS-HT is a neglected HCTD, its frequency is likely underestimated. Accordingly, based on registered data on the frequency of generalized JHM in various populations and the assumption of an ~10% chance of developing symptoms according to the Brighton criteria for "double-jointed" people [12], a presumed frequency of 0.75–2% has been proposed for JHS [15]. As general JHM is rarer among Caucasians compared to other populations such as Africans [16], a frequency of 0.2–0.6%, with the lowest value better fitting for men and the highest for females, appears more realistic in Europe and USA. However, no systematic study accurately investigating

Table 1: Classification of EDSs.

Subtype	Inheritance	Gene(s)
Major forms		
Classic	AD	*COL5A1*, *COL5A2*
Hypermobility/JHS	AD?	Mostly unknown
Vascular	AD	*COL3A1*
Kyphoscoliotic	AR	*PLOD1*
Arthrochalasia	AD	*COL1A1*, *COL1A2*
Dermatosparaxis	AR	*ADAMTS2*
Rare/emerging forms		
Tenascin X deficient	AR, AD?	*TNXB*
Classic with vascular rupture	AD	*COL1A1*
Cardiac-valvular	AR	*COL1A2*
EDS/OI overlap	AD	*COL1A1*, *COL1A2*
With periventricular heterotopia	XLD	*FMNA*
Musculocontractural	AR	*CHST14*
Spondylocheirodysplastic	AR	*SLC39A13*
Progeroid	AR	*B4GALT7*
Kyphoscoliotic with deafness	AR	*FKBP14*
Parodontitis	AD	Unknown
Fibronectin deficient	AR	Unknown

AD: autosomal dominant, AR: autosomal recessive, EDS/OI: Ehlers-Danlos syndrome/osteogenesis imperfect, JHS: joint hypermobility syndrome, XLD: X-linked dominant.

the real incidence of JHS/EDS-HT has been performed to date.

4. Etiology

4.1. Inheritance Pattern. Actually, JHS/EDS-HT is considered an autosomal dominant trait with complete penetrance [17]. Accordingly, an affected individual may transmit the disease to his/her children with a 50% chance, irrespectively to sex. Nevertheless, such an assumption is not always confirmed by practice. In fact, the Brighton and, perhaps, Villefranche criteria for JHS/EDS-HT are met more commonly by females with a markedly skewed sex ratio [18]. This implies that females are more commonly and, possibly, severely affected than males, and, in familial cases, the disease is more frequently transmitted by an affected mother. In addition, extended family study often shows the coexistence of different members with a clinical diagnosis of JHS, EDS-HT, or asymptomatic JHM (either present or historical), as well as asymptomatic nonhypermobile "carriers" in the same pedigree (Castori, unpublished data). Therefore, JHS/EDS-HT should be better defined as an autosomal dominant trait with incomplete penetrance, variable expressivity, and influenced by sex. On a reproductive perspective, while the "mutated gene" is transmitted from an affected parent to the conceptus with a 50% chance, the likelihood of developing the disease seems to be higher in a female fetus.

Recently, it has been introduced that in JHS/EDS-HT families asymptomatic JHM could be the core inherited trait, which eventually evolves in JHS/EDS-HT in those members in whom other independent factors converge. Among them, there are both "intrinsic" and environmental/acquired contributors, such as sex, age, somatotype/weight, sport habits, traumas, surgery, diet, and pain cognitions [19]. Therefore, what we actually call "JHS/EDS-HT" could represent the tip of an iceberg with still unclear relationships with physiology (e.g., generalized, asymptomatic JHM) and organ/tissue-specific functional somatic syndromes possibly linked to an underlying connective tissue disorder (see Section 9).

4.2. Molecular Basis of EDSs. Most EDS subtypes are caused by mutations in gene encoding collagen chains or proteins involved in their biogenesis. Biomechanical consequences of an altered collagen fiber in the expressing tissues and differential expression of the various affected collagen subtypes in the tissues are the major determinants for clinical distinction among EDS variants. Most classic EDS (EDS type I and II of the earlier classification) patients harbor heterozygous mutations in the genes encoding for two of the three chains constituting the ubiquitous collagen type V (*COL5A1* and *COL5A2*) [20]. Conversely, >95% of the cases of vascular EDS are due to mutations in the gene encoding collagen type III (*COL3A1*) [21], which is markedly expressed in vessels. Four EDS variants (i.e., arthrochalasia, classic with vascular rupture, cardiac-valvular, and EDS/osteogenesis imperfecta overlap) are caused by dominant or recessive mutations in genes encoding the two chains of collagen type I (*COL1A1* and *COL1A2*) [22–25].

Some EDS variants are caused by mutations in proteins/enzymes involved in collagen I biogenesis. Dermatosparaxis EDS is due to abnormalities in *ADAMTS2* [26], which encodes for an N-proteinase involved in the ablation of N-propeptides whose cleavage is essential for complete maturation of collagen I. Hydroxylation of lysine residues of collagens I and II depends on lysyl hydroxylase 1, encoded by *PLOD1*, which mutated causes kyphoscoliotic EDS [27].

In patients with the rare tenascin X-deficient EDS due to mutations in *TNXB* [28], immunostaining on muscle biopsies shows mildly reduced expression of collagen VI. Studies on tenascin X null mice show that deficiency of this protein decreases mRNA expression of *COL6A1*, *COL6A2* and *COL6A3* [29]. Mutations in these genes, in turn, cause the Bethlem myopathy and Ullrich congenital muscular dystrophy, which are inherited muscle disorders sharing some features, such as atrophic scars and JHM, with EDSs [30].

Finally, some rare EDS subtypes, including the progeroid and musculocontractural EDSs, are caused by mutations in genes coding for enzymes involved in the biosynthesis of proteoglycans, which are components of the CT extracellular matrix, exhibiting tight relationships with collagen fibers [31, 32]. The same cellular mechanism may be involved in the novel kyphoscoliotic EDS due to recessive mutations in *FKBP14*. In fact, this gene encodes for a FK506-binding peptidyl-prolyl cis-trans isomerase, which may act as a chaperone altering the assembly of the extracellular matrix [33].

Table 2: The Villefranche criteria for major EDS subtypes.

Subtype	Major criteria	Minor criteria
Classic	Skin hyperextensibility Widened atrophic scars Joint hypermobility	Smooth, velvety skin Molluscoid pseudotumors Subcutaneous spheroids Complications of joint hypermobility Muscle hypotonia, motor delay Easy bruising Manifestations of tissue extensibility and fragility Surgical complications Positive family history
Hypermobility	Hyperextensible and/or smooth, velvety skin Generalized joint hypermobility	Recurring joint dislocations Chronic joint/limb pain Positive family history
Vascular	Thin, translucent skin Arterial/intestinal/uterine fragility or rupture Extensive bruising Characteristic facial appearance	Acrogeria Hypermobility of small joints Tendon and muscle rupture Talipes equinovarus Early-onset varicose veins Arteriovenous, carotid-cavernous sinus fistula Pneumothorax/pneumohemothorax Gingival recessions Positive family history, sudden death in a close relative
Kyphoscoliotic	Generalized joint hypermobility Congenital hypotonia Congenital and progressive scoliosis Scleral fragility and rupture of the ocular globe	Tissue fragility, including atrophic scars Easy bruising Arterial rupture Marfanoid habitus Microcornea Osteopenia/porosis Positive family history
Arthrochalasis	Generalized joint hypermobility with recurrent subluxations Congenital bilateral hip dislocation	Skin hyperextensibility Tissue fragility, including atrophic scars Easy bruising Hypotonia Kyphoscoliosis Osteopenia/porosis
Dermatosparaxis	Severe skin fragility Sagging, redundant skin	Soft, doughy skin texture Easy bruising Premature rupture of fetal membranes Large hernias (umbilical, inguinal)

Adapted from [4].
Note 1: no clear indication for using these criteria in the establishment of a firm clinical suspect of a specific Ehlers-Danlos syndrome subtype is specified. However, the presence of at least 1 major and 1 minor criteria is usually necessary for proceeding in molecular confirmation of Ehlers-Danlos syndrome subtypes with a known, prevalent molecular cause. The presence of at least two major criteria is strongly indicative for a definite diagnosis of the specific EDS subtype.

4.3. Molecular Basis of JHS/EDS-HT. In contrast to the other EDS variants, the genetic defect underlying JHS/EDS-HT remains unknown. In the past, a handful of papers tried to clarify the conundrum. In particular, some molecular investigations suggested that *TNXB* heterozygous or homozygous mutations can be identified in ~5% of the EDS-HT patients [34, 35]. Subsequently, EDS patients harboring mutations in *TNXB* have been classified in a different EDS subtype (i.e., *TNXB*-deficient EDS) due to an apparently distinct phenotype [36]. A single family considered affected by EDS-HT was found with a mutation in the *COL3A1* gene, which, in turn, is typically mutated in the vascular EDS [37]. No subsequent study confirmed this preliminary finding. Therefore, neither *TNXB* nor *COL3A1* can be at the moment considered "the gene" of JHS/EDS-HT. Future studies are urgently needed in order to clarify this point and shed more light on the nosologic distinction between JHS and EDS-HT [5].

5. Clinical Manifestations

JHS/EDS-HT differs from other EDS variants due to the apparent paucity and nonspecificity of clinical findings. This reflects only in part the apparently minor involvement of cutaneous and musculoskeletal connective tissue in this EDS subtype. In fact, the scarcity of descriptive manifestations of

Table 3: The Brighton criteria for JHS.

The Brighton criteria
Major criteria
Beighton score ≥ 4/9
Arthralgia for >3 months in >4 joints
Minor criteria
Beighton score of 1–3
Arthralgia in 1–3 joints
History of joint dislocations
Soft-tissue lesions > 3
Marfan-like habitus
Skin striae, hyperextensibility, or scarring
Eye signs, lid laxity
History of varicose veins, hernia, visceral prolapse

Adapted from [9].
Note 1: criteria major 1 and minor 1 are mutually exclusive as are major 2 and minor 2.
Note 2: for the diagnosis of the joint hypermobility syndrome: both major, or 1 major and 2 minor, or 4 minor criteria, or 2 minor criteria and one or more first-degree affected relative(s).
Note 3: diagnosis of joint hypermobility syndrome needs previous (clinical/molecular) exclusion of other overlapping heritable connective tissue disorders, such as Marfan syndrome and other Ehlers-Danlos syndrome subtypes.

Table 4: Extra-articular disorders associated with (generalized) JHM.

Condition	Reference(s)
Anxiety	[38, 116]
Carpal tunnel syndrome	[39]
Chiari malformation type I	[40]
Chronic constipation	[41–43]
Chronic fatigue syndrome	[44–46]
Chronic regional pain syndrome	[47]
Crohn's disease	[48]
Developmental coordination disorder	[49]
Faecal incontinence	[50]
Fibromyalgia	[51–54]
Fixed dystonia	[55]
Functional gastrointestinal disorder	[56]
Headache attributed to spontaneous cerebrospinal fluid leakage	[57]
Hiatus hernia	[58]
Mitral valve prolapse	[59]
New daily persistent headache	[60]
Pelvic organ prolapse	[61]
Postural tachycardia syndrome	[62]
Psychological distress	[38]
Rectal evacuatory dysfunction	[63]
Somatosensory amplification	[38]
Urinary stress incontinence	[64]

JHS/EDS-HT in the medical literature lays on the actual lack of shared knowledge and general unawareness of the practitioners on the multifaceted manifestations of JHS/EDS-HT. The conundrum is further complicated by the increasing number of studies highlighting (generalized) JHM as a possible predisposing factor and/or noncasually associated features for a series of extra-articular disorders (Table 4) [38–64]. At the moment, whether these complaints belong to the wider picture of the JHS/EDS-HT or rather represent nonsyndromic associations needs further investigations.

5.1. Cutaneous Features (Figure 2). Skin hyperextensibility is certainly the best known cutaneous feature of the various EDSs. In JHS/EDS-HT, it may be appreciated in many patients but usually in a much minor extent compared with classic EDS. Skin hyperextensibility defines the ability of the skin to be stretched beyond normal limits and immediately returning to its original state without forming transient redundant folds. Rapid returning to the original state after traction (i.e., resilience) differentiates skin hyperelasticity from cutis laxa, which can equally be noted with an increased rate in older patients with various EDSs, including JHS/EDS-HT. Cutis laxa is indeed a late consequence of premature elastolysis due to reduced dermal resistance to extreme soft-tissue distensions, such as pregnancy and repeated gains and losses of weight. Premature blepharoptosis/chalasia and chubby checks are typical localized manifestations of cutis laxa and may represent relevant aesthetic complaints in middle-aged women. Impaired stiffness of the dermis caused by defective collagen fibrils can also facilitate the development of striae rubrae and striae distensae/atrophicae. Similarly, piezogenic papules (i.e., small spontaneous subcutaneous fat herniations through a defective dermis without appreciable dermal atrophy) may also develop on heels in orthostatism and at wrists after compression. Reduced connective tissue stiffness at abdominal fascia facilitates formation of inguinal, crural, umbilical, and epigastric hernias, especially in conjunction with increased intra-abdominal pressure (e.g., obesity, pregnancy). More rarely, small muscle herniations may form at sites of discrete areas of incontinent perimysium and be equally visible at examination. Velvety and soft skin is a further common skin texture change. Skin fragility causes increased tendency to skin lesions and lacerations, but such a feature is rarely of concern in JHS/EDS-HT. Keratosis pilaris seems more common in JHS/EDS-HT, but no systematic study assessing such an association has been carried out.

Minor wound healing defects and capillary fragility are further common features in JHS/EDS-HT. The former may present as atrophic, nonpapyraceous scars, compared to the "cigarette-paper" and crumpled scars observed in other EDSs. They are the consequence of minimally delayed wound repair combined with skin fragility at sites exposed to repeated traumas, such as knees and elbows. The dystrophic nature of such scars may be easily demonstrated by gentle squeezing between observer's fingers. Occasionally, defective wound healing after surgery or extensive/profound

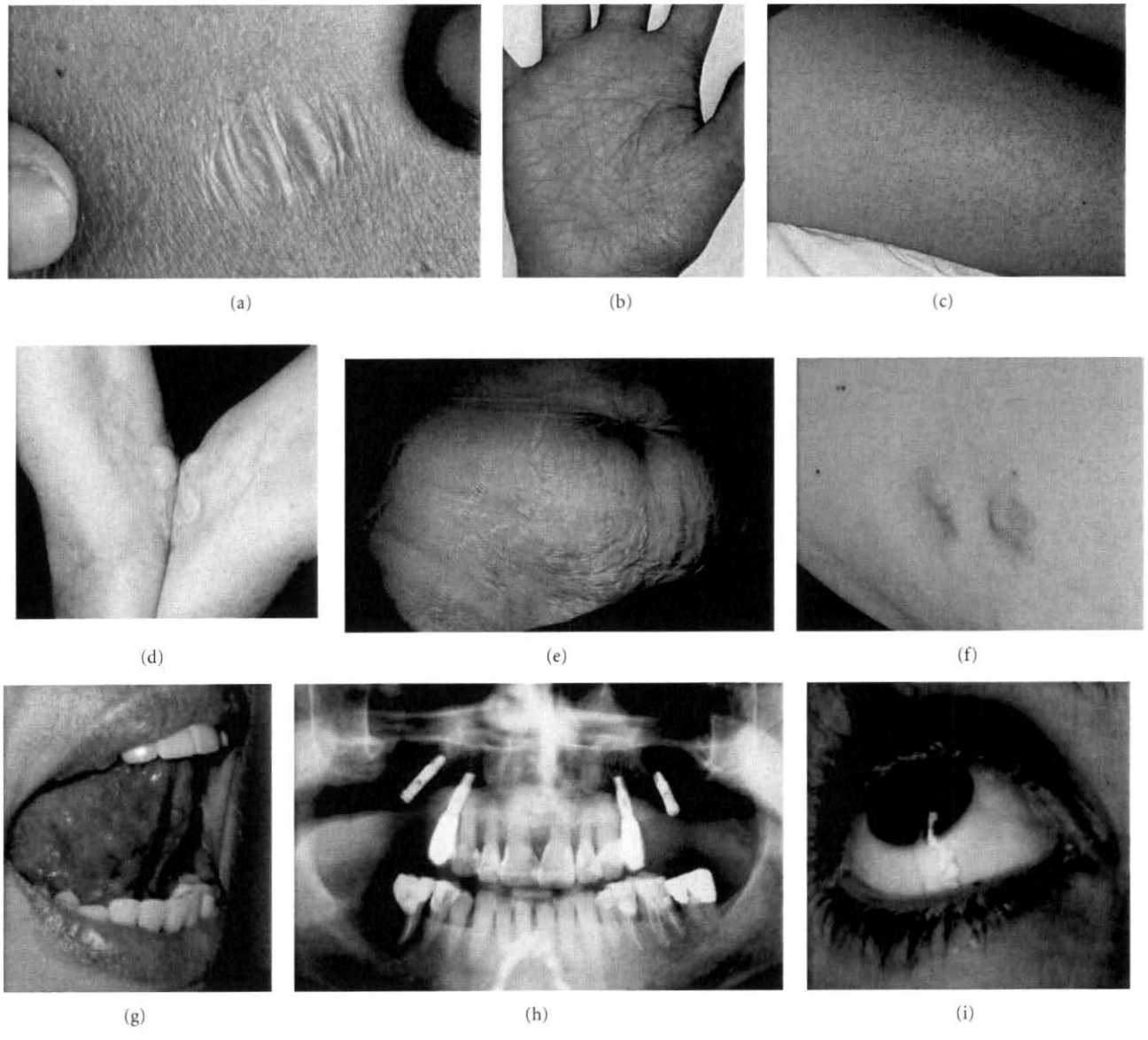

FIGURE 2: Skin and mucosal features of Ehlers-Danlos syndrome, hypermobility type. Atrophic, nonpapyraceous scar—its atrophic nature is more appreciable after gentle squeezing between examinator's fingers (a). Accentuated crease reticulum of the palm (b). Keratosis pilaris in a 26-year-old woman (c). Piezogenic papules at wrists after compression (d). Extensive abdominal striae atrophicae in a 35-year-old multipara (e). Postsurgical scar with anetoderma-like herniation of the subcutaneous fat (f). Apparent absence of the lingual frenulum (g). Radiographic orthopanoramic showing extensive tooth loss in a 50-year-old man with severe gingival involvement (h). Blue sclerae (i).

traumas may hesitate in full thickness dermal atrophy with consequent subcutaneous fat herniations with anetoderma-like features (i.e., incisional hernia). Capillary fragility causes increased tendency to and delayed resolution of ecchymoses. Spontaneous subcutaneous and intramuscular hematomas are possible rare complications of deep vessels fragility. Finally, many patients suffer from perturbed perspiration in form of diaphoresis or hypohidrosis [65]. In my experience, palmoplantar hyperhidrosis with body hypohidrosis is the most common presentation of exocrine dysregulation. This phenomenon may be neurogenic in origin and relate to the underlying dysautonomia (see Section 5.6).

5.2. Mucosal and Oropharyngeal Features (Figure 2). Mucosal involvement is common in JHS/EDS-HT. Xerostomia, xerophthalmia, and vaginal dryness are frequent accompanying complaints. Together with hypohidrosis, mucosal xerosis could be a remote consequence of autonomic dysregulation [65, 66]. Blue sclerae are overrepresented among JHS/EDS-HT patients and are likely caused by more visible uveal blood vessels through thinner sclerae [65]. Focal blue-purple discolorations of the oral mucosa are not uncommon in JHS/EDS-HT, and their origin may parallel blue sclerae. Minor pigmentation anomalies of the enamel can be observed in JHS/EDS-HT, also in the absence of environmental causes (e.g., smoking).

Increased mucosal fragility can lead to spontaneous epistaxis and, more commonly, gingival bleeding, which is often elicited by teeth brushing. Repeated gingival damage due to increased mucosal fragility may progressively cause recurrent gingival inflammations/infections, gingival retractions, and, eventually (although rarely), true parodontopathy with premature tooth loss [67]. Impaired oral cleanliness related to increased gingival bleeding and restraint of wrist and finger mobility may be the cause of the higher rate of caries in JHS/EDS-HT [67].

In the last decade, attention has been posed on the absence of the lingual and inferior labial frenulum in EDSs [68]. Subsequent reports offered contrasting results [69–72]. More recently, a functional origin for the apparent agenesis/absence of the lingual frenulum in JHS/EDS-HT has been emphasized. In fact, this feature is likely the results of multiple contributors, such as primitive (developmental) hypoplasia of the frenulum and uncoordinated tongue movements due to concomitant orofacial dyspraxia [73]. Although still unsupported by evidence-based investigations, oropharyngeal dysphagia seems common in JHS/EDS-HT and, in rare instances, may impede feeding with consequent excessive weight loss, exacerbation of fatigue, and, in children, failure to thrive.

Temporomandibular joint (TMJ) dysfunction is reported in >70% of JHS/EDS-HT patients [74, 75]. It is partly determined by TMJ hypermobility, as documented by increased mouth opening (i.e., mandibular depression over 50 mm in adults) and voluntary subluxations in asymptomatic subjects. Over the years, TMJ hypermobility becomes complicated by clicks, arthralgias, myofascial pain, masticatory dysfunction, and, eventually, articular locks. Similarly to other joints, it is likely that a primary lack of coordination (dyspraxia) of the masticatory muscles may cooperate with JHM in determining dysfunction.

5.3. Orthopedic Features (Figure 3). Congenital capsuloligamentous laxity (CLL) is the primary articular feature of JHS/EDS-HT. In both physiologic and pathologic conditions, it may determine excessive joint motion. JHM is the clinical consequence of increased joint mobility along physiologic axis(es) and may be measured by comparison with standards. Meanwhile, joint instability is used to define the capacity of a lax joint to move along nonphysiologic axes. Both contribute in generating the various EDS orthopedic complications, which comprise increased tendency to (sub)luxations, sprains, and soft-tissue lesions (e.g., bursitis, tendonitis, synovitis, tenosynovitis, and fasciitis) [13]. While, in some patients, repeated dislocations/sprains may further weaken capsuloligamentous resistance and, thus, worsen joint instability, in others, progressive joint stiffness may progressively limit such a phenomenon.

Generalized CLL also influences the late stage of morphogenesis, which starts during fetal life and extends much beyond birth. Mechanical stimuli, such as gravity, uterine constraint, and muscle contractions, on growth and molding of the skeleton are likely more effective in a body with lax joints. For this reason, a series of orthopedic dysmorphisms and minor variants usually converge in the JHS/EDS-HT patient and often depict a recognizable pattern (Table 5).

Table 5: Morphologic and orthopedic features of JHS/ED-HT.

Feature
Leptosomic built or true Marfanoid habitus
Dorsal hyperkyphosis
Lumbar hyperlordosis
Scoliosis of mild degree
Fixed subluxation of the costochondral and/or sternoclavicular joints
Fixed dorsal subluxation of the distal radioulnar joint
Fixed subluxation of the first carpometacarpal joint
Cubitus valgus
Femur anteversion[1]
Patella alta or baja
Genuum valgum
Flexible flatfoot
Hallux valgus
High-arched/narrow palate
Facial asymmetry of mild degree (likely secondary to deformational plagiocephaly)

[1] Intoeing, kissing rotulae, and "W" position of the lower limbs at sitting.

Precocious osteoarthritis, spondylosis, and lower bone mass are potential degenerative complications of generalized CLL, and all are commonly encountered in JHS/EDS-HT. However, at the moment, there are some concerns on the protective rather than predisposing factor for osteoarthritis of JHM [76], while some preliminary studies pointed out the possibility of a higher rate of osteopenia and osteoporosis among JHS/EDS-HT patients [77, 78]. Further studies urge in order to better define such relationships and identify more reliable assessment and therapeutic protocols.

Noncanonic interpretation of the effects of generalized JHM on heath and disease anticipates the existence of a wide variety of functional, developmental, and degenerative consequences, which show unexpectedly intimate and multidimensional relationships with disability and quality of life.

5.4. Neurologic Features. Neurologic implications of JHS/EDS-HT have been largely ignored in the past. More recently, much attention has been posed on nervous system involvement, as it has been recognized as a major contributor to disability in EDS [6]. In 2009, Voermans and colleagues pointed out a possible JHS/EDS-HT neurologic phenotype characterized by a high rate of myopathic electrophysiologic findings possibly combined with reduced sensation and muscle weakness, increased muscle echo intensity, and myopathic changes at biopsy [79]. Previous studies highlighted an association with neuropathies [80–82] and myalgias with cramps [83].

Chronic/recurrent pain and fatigue are, by far, the most common neurologic complaints, being reported in many

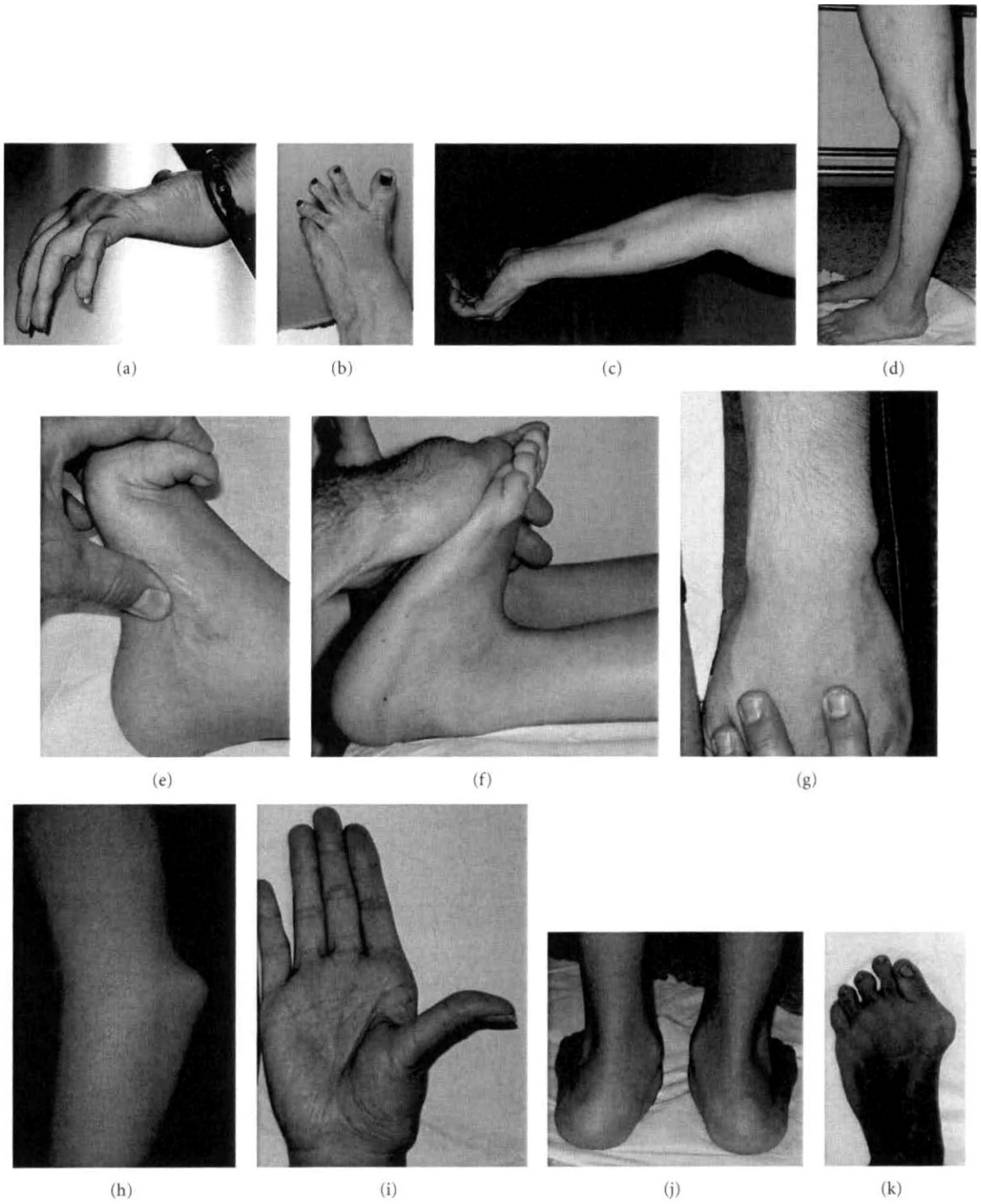

FIGURE 3: Orthopedic features of Ehlers-Danlos syndrome, hypermobility type. Active joint hypermobility at the fingers (a), toes (b), elbow (c), and knees (genu recurvatum, (d)). Passive hyperextension at great toe (e) and heel (f). Structural changes due to joint instability: fixed subluxation of the distal ulna (g), asymptomatic fixed subluxation of the elbow (h), fixed subluxation of the first metacarpal (i), hindfoot pronation and midfoot eversion in an 11-year-old boy (j), and hallux valgus in a 24-year-old woman (k).

EDS patients and in, perhaps, all adults with JHS/EDS-HT [84, 85]. Pain manifestations are widespread and involve the musculoskeletal system, as well as the nervous system and internal organs (Table 6) [19, 47, 53, 54, 67, 75, 84, 86–93]. Their origin is largely obscure except for a statistical association between limb/joint pain and (i) regular analgesic use, (ii) JHM, (iii) dislocations, and (iv) previous surgery [84]. Multiple studies demonstrated that chronic fatigue is a major contributor to disability in JHS/EDS-HT [85, 94, 95]. Associated complaints include muscle weakness [96], sleep disturbance [97], and other features of chronic fatigue syndrome [95].

Table 6: Forms of pain in EDSs also comprising JHS/EDS-HT.

Pain subtype	Manifestations	Key reference(s)
Nociceptive pain	Soft-tissue injuries	[86]
	Dislocations	[84]
	Arthralgias	[87]
	Back pain	[19, 88]
	Myalgias/myofascial pain	[19, 67, 75]
Neuropathic pain	Compression neuropathy	[89]
	Peripheral neuropathy	[89]
Dysfunctional pain	Complex regional pain syndrome types I and II	[47]
	Fibromyalgia	[53, 54]
	(Some) headache disorders	[90]
	Functional abdominal pain	[19, 91]
	Dysmenorrhea	[92]
	Vulvodynia/dyspareunia	[93]

Headache is a highly disabling form of pain in EDS [87, 90]. In JHS/EDS-HT, migraine seems the most common form of headache [98]. However, JHM, especially in form of cervical spine instability, is a possible trigger for other headache disorders, including new daily persistent headache [60], cervicogenic headache [99], and neck-tongue syndrome [100]. Headache attributed to spontaneous (idiopathic) cerebrospinal fluid leakage is a further form of headache possibly facilitated by connective tissue laxity and then associated with JHM [57]. Recently, postsurgical recurrence of Chiari I malformation and associated symptoms, also comprising headache, emerged as a predictor for an underlying heritable connective tissue disorder [40]. Taken together, these evidences may explain the extreme clinical heterogeneity, treatment resistance, and high impact on quality of life of headache in JHS/EDS-HT.

Consolidated evidence indicates that JHS associates with impaired proprioception at various joints, such as proximal interphalangeal joints [101] and knee [102], with consequent poorer joint kinesthesia and position sense [103]. Such a lack of proprioception, that likely affects multiple body segments, impairs balance and posture [104–107]. Clumsiness, tendency to falls, and fear of falling are direct consequences of this phenomenon [108]. The likely congenital nature of such a proprioception impairment may contribute to delayed autonomous walking, tip-toe walking, lack of crawling, clumsiness, and, possibly, dyspraxia, which are frequently reported in infancy and childhood by JHS/EDS-HT patients [109]. For this reason, a noncasual association between JHS/EDS-HT and developmental coordination disorder has been recently proposed [49].

5.5. Psychiatric Features. While emotional/behavioral distress is quite common in various EDSs and significantly contribute to disability, the relevance of psychologic/psychiatric features and their likely relationships with the underlying pathophysiology are generally overlooked in the management of these patients. By studying 48 EDS patients (including eleven with a clinical diagnosis of EDS-HT and five with JHS), Lumley et al. detected a high rate of anxiety, depression, anger, and interpersonal concerns [110]. Interestingly, access to psychiatric services was registered in ~2/3 patients. A considerable excess of emotional symptoms [111] and psychological distress and somatosensory amplification [38] are noted in JHS/EDS-HT patients. More specifically, JHS/EDS-HT is more common among patients suffering from anxiety and panic disorders and, in turn, these complaints are frequently reported in JHS/EDS-HT [112, 113]. Although psychological difficulties may be secondary to chronic pain and disability, ostracism, and avoidance of relationships, a primary (i.e., pleiotropic) and/or organic contributor may coexist. Accordingly, Eccles et al. described greater amygdale volumes in reportedly hypermobile compared with nonhypermobile subjects [114]. Additional findings included decreased volume of anterior cingulate and parietal lobe. Volumetrically abnormal regions are implicated in cognitive control of pain and negative emotions [115]. It is well known that behavior is influenced by the environment, via neural afferents, as an adaptive reply to the homeostatic need. Reactive behavior changes induce, in turn, autonomic arousal states which translate in action such a reply. Therefore, in JHS/EDS-HT, it is possible that, in the future, some behavioral/psychological characteristics could be unexpectedly linked to specific functional features, such as dysautonomia and lack of proprioception.

5.6. Cardiovascular and Pulmonary Features. Cardiovascular involvement is a feature of many HCTDs, including JHS/EDS-HT. Mild mitral, tricuspid, and aortic valve regurgitation is observed in ~25% patients with classic EDS or EDS-HT [119]. However, true mitral valve prolapse occurs in ~6% patients only [120, 121], and this incidence does not seem significantly higher than controls [120]. Early investigations pointed out a high rate of aortic root dilatation in EDS-HT with risk of possible life-threatening complications [122]. A subsequent study on 252 patients with classic EDS and EDS-HT fixed to 10.8% the overall incidence of aortic root dilatation in these conditions, with the latter showing the highest risk (12%) [121]. Of note, at variance with other HCTDs with reduced life span, in all but one patient aortic dilatation did not show any progression in adulthood.

Besides such minor structural heart anomalies, dysautonomia is, by far, the most clinically relevant cardiovascular feature in JHS/EDS-HT. Rowe et al. described 12 EDS patients (six with classic EDS, six with JHS/EDS-HT) with orthostatic intolerance demonstrated by orthostatic stress test [123]. Subsequent clinical and experimental studies drew attention to dysautonomia as a likely underlying mechanism for various visceral complaints in JHS/EDS-HT [91, 124]. More recently, postural tachycardia syndrome has been defined as the most specific form of cardiovascular autonomic dysfunction in JHS/EDS-HT [62].

Morgan et al. found an increased rate of asthmatic symptoms and atopy associated with increased lung volumes,

(a) (b) (c)

FIGURE 4: Visceroptosis of the gut in a 40-year-old woman with severely debilitating gastrointestinal functional complaints. Note marked gastroptosis (a) and pelvic localization of the small bowel (b) and transverse colon (c).

impaired gas exchange, and an increased tendency of both the lower and upper airways to collapse in JHS/EDS-HT [125]. Soyucen and Esen postulated that JHS/EDS-HT may predispose to asthma [126]. In fact, JHS/EDS-HT may lead to persistent childhood wheezing by causing airway collapse through a connective tissue defect affecting airways structure [126]. Further studies are needed to confirm this hypothesis.

5.7. Ocular Features. As previously described, blepharochalasis, antimongoloid palpebral slant, and blue sclerae are relatively common findings in JHS/EDS-HT [65, 127]. Further, though less common features include myopia, unilateral ptosis, and tilted optic disc [127]. A recent survey on 22 patients defined the JHS/EDS-HT phenotype as mostly consisting in xerophthalmia (i.e., positive BUT and Schirmer I tests), steeper corneas, pathologic myopia, and minor lens opacities and vitreal abnormalities [66]. Overall, ocular complaints are usually mild to moderate in JHS/EDS-HT and only a minority of them (i.e., xerophthalmia, and pathologic myopia) need treatment and monitoring, which, at the moment, can be carried out following standard procedures. As some of these features are quantitative and less influenced by age compared to JHM, it is expected that a more accurate ophthalmologic assessment will be included in a revised version of JHS/EDS-HT diagnostic criteria.

5.8. Gastrointestinal Features. Although not included in the Villefranche and Brighton criteria, gastrointestinal involvement is common in JHS/EDS-HT. Indirect evidence comes from several studies demonstrating a high incidence of JHS or (generalized) JHM among patients suffering from chronic (slow transit) constipation [41–43], hiatus hernia [58], Crohn's disease [48], faecal incontinence [50], rectal evacuatory dysfunction [63], and functional gastrointestinal disorder [56]. Typical gastrointestinal features include gastroesophageal reflux (74%) with or without hiatus hernia, chronic/recurrent gastritis (48%), symptoms of delayed gastric emptying, recurrent abdominal pain (68%), and constipation/diarrhea (72%) [19]. However, the range of bowel involvement may extend much beyond to include a wide variety of functional gastrointestinal disorders according to the Rome III classification [128].

The mechanisms underlying such a severe visceral involvement are obscure. Possible contributing factors may comprise (i) reduced fixation to adjacent structures causing visceroptosis and hernias, (ii) gut hypotonia/hypomotility, and (iii) structural anomalies (e.g., dolichocolon). A recent study demonstrating an increased rate of celiac disease in JHS/EDS-HT [129] adds complexity to the study of connections between connective tissue and bowel function, which appear also mediated by an abnormally functioning immune system. The apparent underdiagnosis of visceroptosis in JHS/EDS-HT has been recently pointed out [130]. Accordingly, while literature data concerning such a disease manifestation is scarce, clinical practice often reveals abnormal downward displacement of the gastrointestinal tract and kidneys (Figure 4). The impact of such anatomic features, as well as gut motility and length, in symptom development needs further clarification.

5.9. Gynecologic Features. Gynecologic aspects of JHS/EDS-HT have been largely ignored in the past. However, it is now clear that women with JHS/EDS-HT commonly suffer from irregular menses, meno/metrorrhagias, and severe dysmenorrhea [92]. The latter may only occasionally be related to an underlying gynecologic disorder, such as endometriosis, and, therefore, displays a (dys)functional origin in most cases. Fertility and pregnancy are usually unaffected by JHS/EDS-HT, although more attention should be posed on obstetric and anesthetic interventions in order to prevent some potentially life-threatening or disabling complications. Among them there are (i) the risk of anesthesia-induced hypotension facilitated by dysautonomia, (ii) meningeal fragility complicating in cerebrospinal fluid hypotension in case of epi/peridural anesthesia, (iii) proneness to pelvic prolapse after episiotomy, and (iv) an apparently increased rate of suture dehiscence and minor hemorrhages after surgery. Although, in the past, previous case reports or case series gathering data from different EDS forms suggested prudence in counseling pregnancy in this condition [131–140], recent data are more reassuring. Associated symptoms are influenced by pregnancy. But, while symptoms worsen in many cases, in other women they remain unchanged or improve during pregnancy [92].

Pelvic prolapse is the most debilitating gynecologic feature of JHS/EDS-HT, and, accordingly, it was comprised in the revised Brighton criteria [9]. Clinical manifestations mainly include urinary stress incontinence [64], uterine prolapse, and faecal incontinence [50]. Although prolapses may occur in the nullipara [141], they are most often facilitated by episiotomy and vaginal tears [92].

6. Disease Evolution

As recently outlined, JHS/EDS-HT displays marked phenotypic metatropism with extreme variability at various ages [19, 141]. A series of mechanisms may explain such a phenomenon. Firstly, excessive joint motion is not always detrimental and it often precedes by some years or decades of musculoskeletal pain. At the same time, JHM naturally decreases with age also in the prospectively symptomatic patient. This implies that many patients refer to the general practitioner or specialists when JHM is no more visible, at least, at Beighton score calculation. Secondly, as introduced by the Brighton criteria [9] and the modified Villefranche criteria [17], JHS/EDS-HT manifestations extend much beyond the musculoskeletal system and many disabling features progress uncoupled with joint motion. Thirdly, many patients develop a series of avoiding strategies, such as kinesiophobia, with the false hope of reducing disability. The consequence of such maladaptive cognitions is a progressive limitation of daily activity with aggravation of muscle deconditioning, and, eventually, musculoskeletal pain and fatigue [142]. The understanding of these processes allows to identify at least three distinct disease phases, whose knowledge may help physicians in suspecting JHS/EDS-HT at various ages. For details on the disease progress and phases delineation, see [19].

In addition to the postnatal progression of the disease, connective tissue fragility may also affect resilience of the membranes and cervix, as well as late fetal development. The neonatal presentation of JHS/EDS-HT may comprise slightly preterm birth, precipitous delivery, congenital dislocations at shoulders and clavicles, congenital hip dislocation (usually, unilateral), clubfoot, and, possibly, positional plagiocephaly. Presence of multiple features at birth should lead to investigate JHS/EDS-HT or apparently asymptomatic JHM (either visible or historical) in one of the parents, who may have transmitted the trait. Conversely, neonatal hypotonia, congenital scoliosis, dislocations at unusual sites (e.g., knee or occipitoatlantoaxial junction), fractures, and skin lacerations are never been reported in JHS/EDS-HT and are more typical of other HCTDs.

7. Diagnosis

To date, the diagnosis of JHS/EDS-HT is clinical in essence and based on the agreement of largely accepted diagnostic criteria together with the exclusion of other, partially overlapping HCTDs. Assessment of JHS/EDS-HT lays exclusively on accurate clinical history taking and extensive physical examination, including dermatologic, oral cavity, orthopedic, and neurologic evaluations (see Section 5).

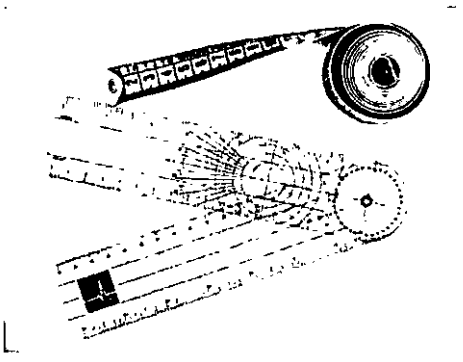

FIGURE 5: Illustrative examples of universal goniometer and flexible tape as essential tools for assessing joint mobility.

However, skin and joint motion assessment deserves annotations.

7.1. Skin Extensibility. Skin extensibility is difficult to assess due to the lack of standardized methodologies. Over the years, a series of tools have been proposed [143–145] but none of them is used in the clinical practice. More recently, skin extensibility measurement by using a suction cup has been proposed as a reproducible method [146]. However, a the moment, skin extensibility evaluation is largely left to the practitioner's experience. As a rule of thumb, skin is considered "hyperextensible" in an adult if it can be stretched by ≥ 1.5–2 cm at the dorsum of the hand (fourth metacarpal), and/or volar aspect of the forearm. Testing in areas of natural skin redundancy (i.e., extensor surfaces) should be avoided. Similar parameters are not available for measuring velvety/smooth skin. Assessing skin consistency and texture is harder in toddlers and infants due to their inherent softness of tissues.

7.2. Joint Mobility. In most joints, evaluation of range of motion is obtained by manipulation and is more accurate if supported by an anatomic (universal) goniometer, a ruler and/or a flexible tape (Figure 5). Various systems are available for identifying subjects with generalized JHM, and the Beighton score is the most widely accepted one (Table 7) [10]. Application of this score is not ideal in all situations. Firstly, specific subpopulations, such as females and prepubertal individuals, are inherently more "lax" than others. Secondly, many joints and groups of joints are not considered in the computation. Thirdly, JHM naturally decreases with age also in the affected/symptomatic individuals. Therefore, specific and/or disabling symptoms may set up or worsen when JHM is no further appreciable. In order to investigate JHM in joints other than those included in the Beighton score, standards are available for adult (i.e., ≥ 16 years of age) subjects (Table 8) [147]. Similarly, a set of specific questions has been outlined for detecting historical JHM in subjects who lost their double jointedness (Table 9) [117]. Similarly to skin extensibility, objective evaluation of variability in joint motion is difficult in toddlers and infants,

Table 7: The Beighton score for assessing generalized JHM.

Sign	Yes	No
Passive apposition of the right thumb to the flexor aspect of the forearm	1	0
Passive apposition of the left thumb to the flexor aspect of the forearm	1	0
Passive dorsiflexion of the right V finger beyond 90 degrees	1	0
Passive dorsiflexion of the left V finger beyond 90 degrees	1	0
Hyperextension of the right elbow beyond 190 degrees	1	0
Hyperextension of the left elbow beyond 190 degrees	1	0
Hyperextension of the right knee beyond 190 degrees	1	0
Hyperextension of the left knee beyond 190 degrees	1	0
Forward flexion of the trunk with the knees extended and the palms resting flat on the floor	1	0

Adapted from [10].
Note: the Beighton score ranges from 0 to 9. Generalized joint hypermobility is fixed for a total score of 5/9 or above for the Villefranche criteria and 4/9 or above for the Brighton criteria. Unstandardized modifications for specific population subgroups, such as children (*increasing* by 1 point these limits) and males (*reducing* by 1 point these limits), are reasonable. For noncollaborative subjects, a modified Beighton score lacking the spinal bending maneuver and a maximum score of 8 may be applied.

as the Beighton score is nearly useless in these subjects and alternative screening tools are not yet available.

7.3. Ultrastructural and Molecular Findings. At the moment, ultrastructural and molecular abnormalities are only occasionally identified in JHS/EDS-HT and no finding is pathognomonic of this condition. Therefore, skin biopsy and molecular testing do not take part to the standard diagnostic schedule in JHS/EDS-HT. In the past, the ultrastructural features of EDS-HT have been studied and their knowledge may be relevant, as such investigations can be performed for the differential in doubtful cases. In particular, early studies showed that single collagen fibers of the reticular dermis may present an enlarged and irregular section, but usually without coalescing in the typical "cauliflower-like" fibers observed in classic EDS [148]. Molecular analysis of *TNXB* and *COL3A1* is not confirmatory for JHS/EDS-HT, although it may be considered for differential diagnosis in case of partial overlap with the vascular and Tenascin X-deficient forms of EDS.

7.4. Diagnostic Criteria. JHS/EDS-HT is an exclusion diagnosis based on published diagnostic criteria. Actually, two distinct sets of diagnostic criteria exist: the Villefranche criteria for EDS-HT [4] and the Brighton criteria for JHS [9]. The Villefranche criteria emerged from the activities of an international group of experts mainly comprising pediatricians and clinical/medical geneticists and are typically used to evaluate children and young adults. Independently, JHS, alternatively termed hypermobility syndrome, generalized joint hypermobility syndrome, or benign joint hypermobility syndrome (the latter is now in disuse as JHS should be no longer considered a "benign" condition), has been studied by the rheumatologic community. The Brighton criteria consider the natural progressive loss of joint mobility by age and, therefore, are more adequate to assess adults. Consequently, in order to be considered affected by JHS/EDS-HT, a patient must meet either the Villefranche or the Brighton criteria. A further set of diagnostic criteria was proposed by Levy and was derived from the Villefranche criteria for EDS-HT [17]. It includes additional common features not previously considered, such as functional bowel disorder and cardiovascular dysautonomia, and, then, seems

Table 8: Standards for evaluating range of motion of adults' joints.

Movement	Maximal ROM
Shoulder elevation through flexion	180°
Elbow extension	190°–195°[1]
Elbow pronation-supination	170°[2]
Wrist flexion	80°
Wrist extension	70°
Wrist ulnar deviation	30°
Wrist radial deviation	20°
2nd finger MCP joint extension	45°
PIP and DIP joint extension	0°
Hip abduction with leg extended	45°
Hip adduction with leg extended	30°
Knee extension	180°–190°[1]
Ankle dorsiflexion	20°
Ankle plantar flexion	50°
1st toe MTP joint extension	70°
Mandible depression	35–50 mm
Mandible protrusion	3–7 mm
Mandible lateral deviation	10–15 mm
Neck rotation	11 cm[3]
Neck flexion	45°
Neck extension	45°
Neck lateral flexion	45°
Thoracolumbar spine lateral flexion	35°

[1]The lower and the upper end fits better for men and women, respectively.
[2]80° in supination and 90° in pronation from mid-position.
[3]From the tip of the chin to the lateral aspect of the acromion process.
DIP: distal interphalangeal, MCP: metacarpophalangeal, MTP: metatarsophalangeal, PIP: proximal interphalangeal, ROM: range of motion.

Table 9: A proposed questionnaire for investigating JHM by history.

(1)	Can you now (or could you ever) place your hands flat on the floor without bending your knees?
(2)	Can you now (or could you ever) bend your thumb to touch your forearm?
(3)	As a child did you amuse your friends by contorting your body into strange shapes or could you do the splits?
(4)	As a child or teenager did your shoulder or kneecap dislocate on more than one occasion?
(5)	Do you consider yourself double jointed?

Adapted from [117].

more inclusive. However, it has not yet been validated by the scientific community. In light of the recent discoveries concerning the protean manifestations of JHS/EDS-HT, the need of extensively revising and unifying available diagnostic criteria for this condition is urgent [149].

7.5. Differential Diagnosis. The extreme clinical variability of JHS/EDS-HT identifies a great number of partially overlapping (acquired and genetic) disorders showing the variable association of mucocutaneous fragility, JHM, chronic musculoskeletal pain and fatigue. Among them, there are other HCTDs with JHM, the "battered child" syndrome, bleeding disorders, and various rheumatologic conditions with chronic musculoskeletal pain, such as ankylosing spondylitis, rheumatoid arthritis, and fibromyalgia. The association of apparently unexplained features (see "clinical features") and severe physical disability observed in JHS/EDS-HT broadens the spectrum of the differential to include some neurologic disorders, including multiple sclerosis, amyotrophic lateral sclerosis, hereditary and acquired sensory-motor and/or autonomic polyneuropathies, and chronic fatigue syndrome, as well as myopathies featuring JHM [30]. Accordingly, the number of possibly useful investigations is myriads. Their use should be wisely evaluated case by case, as no standardized guidelines are available up to date. In the clinical practice, many JHS/EDS-HT patients reach the correct diagnosis after dozens of consultations, as well as costly and invasive/ineffective diagnostic procedures. Doubtful clinical pictures could be comprehensively investigated by screening for peripheral polyarthralgias (i.e., hands/feet X-rays, HLA-B27 testing, erythrocyte sedimentation rate, and rheumatoid factor, and C-reactive protein dosage) [150], chronic fatigue syndrome panel screening [151], serum creatine kinase and lactate dehydrogenase dosage, and electroneurography/electromyography.

Among the various HCTDs sharing some musculoskeletal features with JHS/EDS-HT, there are other EDSs, as well as the Loeys-Dietz and arterial tortuosity syndromes. Historical and physical clues for suspecting such conditions include papyraceous/hemosiderotic scars, molluscoid pseudotumors, subcutaneous spheroids, markedly visible subcutaneous vessels, triangular face with sunken eyes, early-onset cutis laxa and premature ageing, bifid uvula/cleft soft palate, hypertelorism, dolichocephaly, vascular complications, history of osteochondritis dissecans, and sudden death in close relatives. Persistence of the suspect of vascular EDS, Loeys-Dietz, or arterial tortuosity syndromes should be further investigated by extensive vascular imaging (i.e., whole-body angio-MRI; or brain angio-MRI plus thoracic and abdominal angio-TC; or heart, abdominal aorta, epiaortic and limb vessels Doppler ultrasound) followed by molecular testing (*COL3A1*, *TGFBR1*, *TGFBR2*, *SMAD3*, *TGFB2*, and *SLC2A10*) in specialized laboratories. Rapid detection of such rarer HCTDs is crucial for prognosis establishment due to their high risk of vascular accidents, a complication never reported in JHS/EDS-HT. Occasionally, differential diagnosis with classic EDS may need *COL5A1* and *COL5A2* molecular testing.

8. Principles of Management

Guidelines for managing JHS/EDS-HT are still lacking. General indications for the broader group of EDSs have been recently revised and proposed [152], but no program is available for JHS/EDS-HT up to date. Levy presented some suggestions specifically addressed to JHS/EDS-HT [17]. Here, previously published and author's personal experiences are summarized in order to present a structure for approaching treatment and prevention strategies in JHS/EDS-HT. It has not been emphasized enough that, at the moment, the long-term treatment of JHS/EDS-HT is largely unsuccessful in terms of amelioration of symptoms. In fact, after years of treatment cycles and follow-up evaluations, many patients still refer the complaints reported at first evaluation. This anticipates that, actually, the best result of all practitioners' efforts is to stabilize symptoms with short periods of complete/partial relief. It should be taken in mind that most of the following indications are not yet been confirmed by evidence.

8.1. Treatment of Pain: An Overview. Musculoskeletal pain is a major determinant for deterioration of quality of life in JHS/EDS-HT. Although it usually starts as occasional/recurrent joint pain facilitated/triggered by joint instability (e.g., dislocations and sprains), subsequently it becomes pathogenically heterogeneous usually manifesting in form of widespread myalgias and arthralgias and often with neuropathic features. Pain chronicization and resistance to treatment are the most relevant features influencing prognosis. The best management program should include drugs, physical therapy [88, 153, 154], cognitive-behavioral therapy [142], and adherence to a series of lifestyle recommendations [118]. For this reason, while occasional and low-to-moderate recurrent pain may be treated in an outpatient setting by the reference specialist (e.g., clinical geneticist, rheumatologist, physiatrist, or general practitioner), management of chronic or highly disabling recurrent musculoskeletal pain in JHS/EDS-HT usually needs a multidisciplinary approach.

8.2. Treatment of Pain: Medications. In my experience, drugs monotherapy is successful for treating occasional/recurrent pain of low-moderate intensity. The following alternatives are of potential use in the otherwise healthy adult with JHS/EDS-HT (adjustments are needed for children and nonhealthy subjects) and can be well managed by the general practitioner:

(1) ibuprofen 200–1,800 mg/day (mean: 1,200 mg/day) in one to three divided doses with a maximum of single dose of 600 mg;

(2) naproxen 1,000 mg/day in two divided doses;

(3) paracetamol 1,000–3,000 mg/day in up to three divided doses of 500–1,000 mg each; paracetamol can be administered in association with codeine phosphate (with a ratio of 30 mg codeine phosphate per 500 mg paracetamol) for a maximum daily dose of 3,000 mg for the former and 180 mg for the latter.

In case of inefficacy of monotherapy, other drugs may be used in alternative of or association with the above-mentioned medications:

(1) tramadol up to 400 mg/day in one of two divided doses with a maximum single dose of 200 mg; association of codeine phosphate and tramadol must be avoided;

(2) Cox-2 inhibitor (celecoxib) 200–400 mg in one or two divided doses in presence of documented osteoarthritis;

(3) pain modulator drugs, including tricyclic antidepressants and serotonin/norepinephrine receptor inhibitors, in presence of documented or presumed neuropathic pain; among them, amytriptyline is actually considered the best choice [142] with an initial daily dose of 10 mg with an increase of 10 mg/week (up to 100/day) after careful monitoring of pain relief and side effects (the preferred dose is usually 30–50 mg/day); duloxetine is a further promising drug; benefits from these drugs also include treatment of other satellite symptoms, such as depression, sleep disturbances, and irritable bowel disease;

(4) opioids (other than tramadol and codeine) may be effective for treating chronic musculoskeletal pain of moderate-to-high intensity, and their management usually needs specialist consultation; although the use of such drugs is usually successful in the short terms, *many chronic JHS/EDS-HT users of opioids* still suffer from intense and debilitating pain.

8.3. Treatment of Pain: Nonpharmacologic Resources. In addition to drugs, alternative interventions could be considered in isolation or combined with medications and lifestyle modifications (see "Section 8.6"). They include the following.

(1) Referral to a physical therapy specialist is expected for all JHS-EDS-HT patients in order to identify the need for specialized intervention, choose the best suited sport/fitness activity, and educate the patient to "joint" health; extensive information for the physical therapist is summarized in Hakim et al. [155].

(2) Cognitive-behavioral therapy is beneficial in all forms of chronic pain, including musculoskeletal and visceral pain as well as headache; therefore, cognitive-behavioral therapy is indicated in patients with debilitating pain not adequately treated by standard care (i.e., probably most painful sensations other than those related to dislocations and sprains).

(3) Application of braces for short periods may improve joint stability in case of recurrent sprains and their use needs specialistic consultation;

(4) Soft neck collars, waterbeds, adjustable air mattresses, and viscoelastic foam mattresses and/or pillows may improve quality of life related to headache and sleep quality.

(5) Crutches, canes, walkers, wheelchairs, and scooters may improve residual mobility in the most disable patients; while their use allows the patient to perform some activities easier, it is not free of side effects, such as joint traumas to the upper limbs and increased deconditioning of the lower limbs.

(6) Occupational therapist consultation is usually indicated for pain conditions influencing daily activity and, then, constricting life of the affected individual; it is needed in presence of loss of autonomy and disability at home, work, and school.

(7) JHS/EDS-HT pelvic pain is best managed with a multispecialist approach but a specific schedule is lacking; general recommendations are well summarized in Nelson et al. [156].

(8) Pain related to gastrointestinal functional disorder can be managed following available standards [157, 158].

(9) Topical lidocaine (cream or patch), local injections of anesthetic and steroids, and anesthetic nerve blocks are possible non- or mini-invasive procedures that prevent systemic assumption of drugs and, consequently, the risk of side effects; no systematic study is available, but their use seems of very limited success (see Section 8.7).

(10) Recently, prolotherapy with 10% dextrose was demonstrated successful in reducing pressure-induced *pain at the TMJ in the hypermobile subject* [159]; with caution, its use may be considered in other joints also.

(11) Although considered at risk of causing dislocation, chiropractic with application of low-force adjusting techniques may be successful in JHS/EDS-HT [160].

(12) Heat and hydrotherapy, acupuncture, and transcutaneous electrical nerve stimulation (i.e., TENS) are possible alternatives without overt contraindications in JHS/EDS-HT; their effectiveness remains untested.

8.4. Treatment of Pain: Points of Concern. Some therapeutic options display documented/presumed severe side effects or hopeless inefficacy and, then, should be considered most cautiously. Among them there are the following.

(1) Most of the orthopedic surgical interventions aimed at stabilizing joints, such as arthroscopic debridement, tendon relocations, capsulorrhaphy and arthroplasty, and reducing annulus hernias (e.g., high risk for recurrence, abnormal wound healing, adhesion formation, and pain amplification); surgery should be always postponed to more conservative approaches; when it is firmly requested, meticulous planning and communication to the patient of the low rate of success of this approach are mandatory.

(2) Generous prescription of periods of inactivity and abstention from regular sport activity (i.e., muscle deconditioning of rapid onset).

(3) Use of myorelaxants (i.e., amplification of joint instability with multiple dislocations with consequent exacerbation of pain and fatigue).

(4) Chronic local and systemic use of steroids (i.e., steroid-induced connective tissue damage on soft-tissues and bone).

(5) Use of antiplatelet drugs, for example, as acetylsalicylic acid (i.e., increased tendency to mucosal hemorrhages and ecchymoses).

(6) Use of antiepileptic drugs (i.e., exacerbation of dysautonomic symptoms).

8.5. Treatment of Fatigue. In JHS/EDS-HT chronic fatigue is likely multifactorial and pathogenic heterogeneous [118]. Although various contributors, such as muscle weakness, cardiovascular dysautonomia, sleep disturbance, malabsorption, respiratory dysfunction, and analgesic overuse, may be clinically identified and, possibly, managed, no study has been performed to systematically investigate these factors weighting their role isolatedly and possible treatments. The above-mentioned factors may influence the general wellness of affected individuals and all or most of them should be properly investigated in any JHS/EDS-HT patient displaying a disabling fatigue. Distinguishing between physiologic fatigue after physical activity or due to unhealthy lifestyle and pathologic fatigue may be difficult. However, the coexistence of persistent exertional dyspnea, unrefreshing sleep, postexertional malaise, and reduced stamina, combined with major limitations of the daily activities, likely indicates pathologic fatigue. Common comorbidities, most of which concurring by chance, should be properly investigated and treated [151]. Sleep hygiene, gastroenterologist and pneumologist consultation, and drug therapy adjustments are strategies which may alleviate fatigue-related disability. Nevertheless, cardiovascular dysautonomia seems the most relevant contributor to fatigue in JHS/EDS-HT [62].

Management of chronic fatigue and cardiovascular dysautonomia is firstly nonpharmacological, and life-style recommendations are summarized here, in part (see Section 8.6), and in Mathias et al. [62]. In patients in whom these procedures are ineffective, drug use could be considered. Fludrocortisone in a daily dose of no more than 300 μg is the first-line drug. Vasoconstrictors, mainly midodrine (at an initial dose of 2.5 mg/die which may be weekly raised up to 30 mg/day), are second-line alternatives and could represent a preferred choice in JHS/EDS-HT in consideration of the increased risk of osteopenia/porosis. Both fludrocortisone and vasoconstrictors are contraindicated in patients with systemic hypertension. In this case, β-blockers or clonidine may improve both the blood pressure and heart rate. β-blockers should be avoided in patients with asthma, a feature with a possibly increased rate in JHS/EDS-HT [126]. In patients with marked postprandial tiredness, octreotide at low dose (25–50 μg in three administrations before the principal meals) is a therapeutic option. Recently, Kanjwal et al. [161] identified modafinil as a possible therapeutic resource for managing chronic fatigue in orthostatic intolerance.

Table 10: Lifestyle recommendations for JHS/EDS-HT.

Recommendation
Promote regular, aerobic fitness
Promote fitness support with strengthening, gentle stretching, and proprioception exercises
Promote postural and ergonomic hygiene especially during sleep, at school, and at workplace
Promote weight control (BMI < 25)
Promote daily relaxation activities
Promote lubrication during sexual intercourse (women)
Promote early treatment of malocclusion
Avoid high impact sports/activities
Avoid low environmental temperatures
Avoid prolonged sitting positions and prolonged recumbency
Avoid sudden head-up postural change
Avoid excessive weight lifting/carrying
Avoid large meals (especially of refined carbohydrates)
Avoid hard foods intake and excessive jaw movements (ice, gums, etc.)
Avoid bladder irritant foods (e.g., coffee and citrus products)
Avoid nicotine and alcohol intake

Adapted from [118].
Note: these recommendations must be intended as flexible indications for ameliorating quality of life and do not represent lifesaving solutions.

For more details on management of pain and fatigue in JHS/EDS-HT, refer to [118].

8.6. Lifestyle and Nutritional Recommendations. In consideration of the chronic and progressive nature of JHS/EDS-HT and the nonexistence of decisive treatments, adherence to specific behavioral guidelines for preventing symptom onset and/or deterioration appears, at the moment, the most cost- and time-effective solution. Major limits still exist in appropriately selecting and testing reasonable recommendations. This lays down the still too fragmented knowledge on the pathophysiology of JHS/EDS-HT. Nevertheless, a list of likely harmless and potentially effective lifestyle recommendations can be identified (Table 10) [118]. Such suggestions are drawn based on general recommendations for preventing complications related to some common phenotype components of JHS/EDS-HT, such as proneness to joint damage and cardiovascular dysautonomia.

In addition to behavior modifications, adequate nutritional supplementations may be of some help in preventing/treating some features of JHS/EDS-HT. Although specific studies are still lacking, suggestions have been recently proposed [62, 162, 163]. In particular, dysautonomia-related fatigue may be partly managed by (i) generous daily water/liquid intake (2–2.5 lts) preferring isotonic solutions, (ii) high salt intake (to avoid in case of arterial hypertension), and (iii) daily assumption of carnitine (250 mg) and/or coenzyme Q10 (100 mg). Capillary/small vessels fragility may be improved by daily assumption of ascorbic acid, a

cofactor of prolyl and lysyl hydroxylases, which are enzymes involved in the biogenesis of collagens. Approximately 8–50 times the 60 mg recommended daily intake for adults is indicated as the dose for maximal improvement of such biological functions. In case of osteopenia, daily intake of therapeutic doses of vitamin D (usually 880 IU/day in adults) and calcium (usually 1,000 mg/day in adults) is indicated for lowering the risk of fracture. Vitamin D is present in a few foods, and many people, especially in USA and Europe, may not get enough sunlight, which is essential for endogenous production of vitamin D from cholesterol. Therefore, a daily supplementation of 200 mg or 400 mg vitamin D, for adults and children, respectively, may be recommended also in the nonosteopenic individual. A 1–5 mg daily intake of melatonin is considered a resource for improving sleepiness in various functional somatic syndromes, such as fibromyalgia. Similarly, melatonin may be effective in JHS/EDS-HT. Other nutraceuticals which have been thought beneficial, though still without evidence, in JHS/EDS-HT comprise vitamin E, vitamin B complex, vitamin K, glucosamine, chondroitin, γ-linolenic acid, pycnogenol, magnesium, zinc, methyl sulfonyl methane and silica.

8.7. Surgical and Anesthetic Issues. Major surgical complications, such as organ or vascular rupture, are typical of other EDS subtypes and have never been reported in JHS-HT. Nevertheless, there are some minor weaknesses in JHS/EDS-HT, such as cervical spine and TMJ instability/laxity, delayed wound repair, cardiovascular dysautonomia, and slight vascular fragility, that may be of concern in case of minor or major surgery. Accordingly, some red flags are identified for healthcare professionals approaching surgery in JHS/EDS-HT.

(1) Although surgery is not contraindicated in JHS/EDS-HT, the increased time requested for soft-tissue repair and the related risk of possibly unsatisfactory results and muscle deconditioning due to postsurgical recovery entail to pay more attention in planning invasive interventions.

(2) The mild soft-tissue fragility and delayed wound healing may be counteracted by doubling the waiting time before suture stitches removal.

(3) In case of local/minor surgery, consider the frequently reported resistance to intradermal lidocaine infiltrations and topical EMLA cream in JHS/EDS-HT [164, 165]; a double dose of anesthetic by intradermal injection as the first choice may be effective.

(4) Although evidence is lacking, local anesthetic resistance could manifest also in case of epidural anesthesia.

(5) Intubation should be performed with care due to TMJ and cervical spine instability and minor mucosal fragility; in adult patients with severe TMJ dysfunction, limited mouth opening may request the use of pediatric devices.

(6) Peridural anesthesia administration may request extra time due to premature spondylosis; meningeal fragility may associate with an increased risk of intracranial hypotension due to cerebrospinal fluid leakage.

(7) In case of total anesthesia, the coexistence of cardiovascular dysautonomia may increase the risk of hemodynamic changes; prophylactic early fluid loading and phenylephrine infusion should be considered [139].

(8) Although postsurgical hemorrhages are usually mild, their occurrence, especially in older subjects and toddlers as well as in case of concurrent chronic diseases, may expose the patient to unreasonable risks; prophylactic use of desmopressin (DDAVP) may be considered to reduce the chance of excessive bleeding.

8.8. Obstetric Issues. Besides the risk of disease transmission as previously discussed (see Section 4), the diagnosis of JHS/EDS-HT also has obstetric implications. While fertility does not appear affected by JHS/EDS-HT, particular attention should be posed on delivery planning for both operative and anesthetic implications (see Section 8.7). In addition, in order to minimize the risk of pelvic prolapses, Cesarean section should be considered the first choice when vaginal delivery without episiotomy cannot be anticipated [92]. Slightly preterm delivery due to premature rupture of the membranes or cervical insufficiency and precipitous delivery occur in ~10% and ~30% of cases, respectively, and should be carefully considered.

9. Conclusion: Ehlers-Danlos Syndrome, Hypermobility Type as a Model for Studying Functional Somatic Syndromes

Since its early definition as an HCTD with predominant rheumatologic manifestations, JHS/EDS-HT is emerging as a widespread disorder with reverberations in practically all organs and systems. Although most complications are not life-threatening and many patients have a nearly intact lifespan, the pervasive nature of the disorder often makes their life poor and restricted by worsening disability [166]. The spectrum of clinical implications of lax joints even outside rare and well-defined HCTDs seems to be wider than previously expected, in contrast to the quaint adage of considering JHM a benign, asymptomatic trait. Accordingly, Table 4 well illustrates the range of complaints/disorders linked to JHM. Although most studies are based on statistical analysis testing the occurrence of JHM among patients suffering from specific "common" afflictions, these preliminary observations may hide under a common milieau. The existence of such a correlation is foreshadowed by the convergence of most of these complaints in JHS/EDS-HT patients, who often "migrate" from one specialist to another referring every time a different complaint. Accordingly, an underlying disorder of the connective tissue may represent the missing link between JHM and the extra-articular dysfunctions capturing the practitioner's attention also in patients not satisfying the different sets of diagnostic criteria for JHS/EDS-HT.

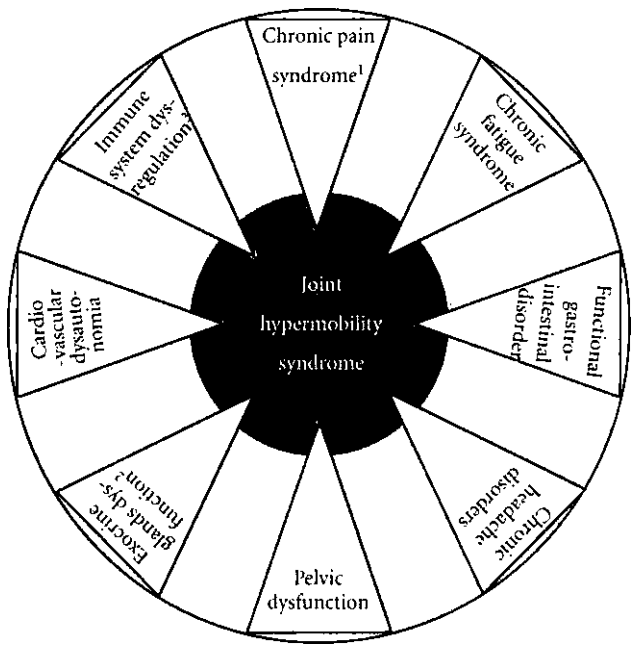

FIGURE 6: Schematic representation of extra-articular manifestations of Ehlers-Danlos syndrome, hypermobility type (alternatively termed joint hypermobility syndrome). The dark grey circle symbolizes the phenotypic spectrum of this condition, which includes a series of functional somatic syndromes and tissue/organ-specific dysfunctions (i.e., the white triangles, whose tips are indeed comprised within the dark circle). Outside the clinical spectrum of Ehlers-Danlos syndrome, hypermobility type, the single phenotypic components may be observed in isolation or, perhaps, in incomplete associations within the general population (the larger and light grey circle). It is expected that, in the future, the study of heritable dysfunctions of the connective tissue will move from the dark gray circle to the light gray one, as a prominent field of interest. [1]Mostly including fibromyalgia, myofascial pain and complex regional pain syndromes. [2]Comprising xerophthalmia, xerostomia, vaginal dryness, and abnormal sweating. [3]Asthma, atopy, gluten sensitivity, inflammatory bowel disease, and recurrent cystitis are all possible manifestations of an underlying immune system dysregulation.

Such an interpretation extends the horizons of the study of heritable anomalies of the connective tissues to a series of bridging phenotypes filling the gap between true HCTDs and (apparently isolated) functional somatic syndromes, such as fibromyalgia, chronic fatigue syndrome, and functional gastrointestinal disorder(s). In this perspective, JHS/EDS-HT may represent the prototype for testing the complex pathogenic correlations between a primary defect of the connective tissue and disorders of tissues other than skin, joints, and vessels. In fact, clinical practice anticipates a continuum among patients with the full-blown JHS/EDS-HT characteristics and others showing incomplete systemic manifestations (the so-called "overlap" or "borderline" phenotypes) or single organ dysfunctions constituting the JHS/EDS-HT extended phenotype (Figure 6). In this context, the unveiling of JHS/EDS-HT molecular basis and the related pathophysiology could have unexpected effects in understanding and, hopefully, better treating a wide variety of common functional disorders, which actually represent a great challenge for the healthcare system of most industrial countries.

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Use of the Gait Profile Score for the evaluation of patients with joint hypermobility syndrome/Ehlers–Danlos syndrome hypermobility type

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ABSTRACT

Gait analysis (GA) is widely used for clinical evaluations in various pathological states, both in children and in adults, such as in patients with joint hypermobility syndrome/Ehlers–Danlos syndrome hypermobility type (JHS/EDS-HT). Otherwise, GA produces a large volume of data and there is the clinical need to provide also a quantitative measure of the patient's overall gait. Starting from this aim some global indexes were proposed by literature as a summary measure of the patient's gait, such as the Gait Profile Score (GPS). While validity of the GPS was demonstrated for the evaluation of the functional limitation of children with Cerebral Palsy, no studies have been conducted in patients JHS/EDS-HT. The aim of our study was therefore to investigate the effectiveness of the GPS in the quantification of functional limitation of patients with JHS/EDS-HT. Twenty-one adult (age: 36.1 ± 12.7 years) individuals with JHS/EDS-HT were evaluated using GA and from GA data the GPS was computed. The results evidenced that the GPS value of patients was 8.9 ± 2.6, statistically different from 4.6 ± 0.9 displayed by the control group. In particular, all values of Gait Variable Scores (GVS) which compose the GPS were higher if compared to controls, with the exception of Pelvic Tilt and Foot Progression. The correlations between GPS/GVS and Lower Extremity Functional Scale (LEFS) showed significant relationship between GPS and the item 11 ("Walking 2 blocks") ($\rho = -0.56$; $p < 0.05$) and 12 ("Walking a mile") of LEFS ($\rho = -0.76$; $p < 0.05$). Our results showed that GPS and GVS seem to be appropriate outcome measures for the evaluation of the functional limitation during gait of patients with JHS/EDS-HT.

1. Background

Joint hypermobility syndrome (JHS), also termed Ehlers–Danlos syndrome hypermobility type (EDS-HT) (Tinkle et al., 2009), is an uncommon rheumatologic condition characterized by generalized joint hypermobility (JHM) and a wide variety of musculoskeletal and non-musculoskeletal findings related to congenital laxity of the connective tissue. JHS/EDS-HT is

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likely the most common inherited connective tissue disorder and diagnosis is based on specific diagnostic criteria (Beighton, De Paepe, Steinmann, Tsipouras, & Wenstrup, 1998; Grahame, Bird, & Child, 2000).

The JHS/EDS-HT clinical spectrum is wide and blurs with single organ functional disorders and, perhaps, normal variations. Musculoskeletal system is clearly one of the most commonly and severely affected anatomic structure with hyperextensibility of ligaments, tendons and capsules. In addition to capsuloligamentous laxity, various studies demonstrated lack of proprioception at knees, shoulders and proximal interphalangeal joints (Celletti, Castori, Grammatico, & Camerota, 2011; Hall, Ferrell, Sturrock, Hamblen, & Baxendale, 1995; Helliwell, 1994; Mallik, Ferrell, McDonald, & Sturrock, 1994; Rombaut, De Paepe, Malfait, Cools, & Calders, 2010). The relationship between reduced skeletal muscle proprioception and ligamentous laxity is obscure. However, previous studies (Castori, Camerota, Celletti, Grammatico, & Padua, 2010; Castori, Celletti, Camerota, & Grammatico, 2011; Celletti, Castori, Galli, et al., 2011; Ferrell et al., 2004) demonstrated that enhancement of proprioception by exercise and focal muscle vibration can ameliorate pain intensity and, consequently, quality of life.

In JHS/EDS-HT, lack of proprioception influences complex functions, including posture and gait. Accordingly, it can be hypothesized that, in JHS/EDS-HT, many musculoskeletal dysfunctions and their relationships with disability and quality of life are caused by a quite specific impairment of balance and movement. Postural studies showed postural instability with frequent falls and amplification of the fear of fall (Galli, Cimolin, et al., 2011; Rombaut et al., 2011). Surface electromyographic studies during postural exercises demonstrated lower *gluteus medius* activity and higher *gastrocnemius lateralis* activity in JHS/EDS-HT patients compared to controls during more difficult tasks, while *rectus femuris* was constantly more activated (Greenwood, Duffell, Alexander, & McGregor, 2011).

Also gait patterns are perturbed in JHS/EDS-HT. Rombaut et al. (2011) found that gait velocity, step length, and stride length were significantly smaller during walking in the JHS/EDS-HT subjects compared to the control group. Galli, Rigoldi, et al. (2011) showed decreased stiffness at ankles and hips with reduced dorsiflexion of the feet during stance and swing phase. Furthermore, there is a decrement of gait velocity, step and stride length, and cadence (Galli, Rigoldi, et al., 2011). In patients with JHS, knees showed reduced peak flexion and increased extension at mid stance during walk. All these findings imply functional relationships with lower limb impairment.

3D-Gait Analysis (GA) is an objective tool to measure and quantify the gait pattern of a subject and it produces a large volume of highly informative data; despite its objectivity this makes it an instrument that is sometimes complicated to use and difficult to interpret in clinical setting. Among the measures proposed by literature to quantify instantaneously the degree of functional limitation during gait, the Gait Profile Score (GPS) is a recent parameter which summarizes the overall deviation of kinematic gait data relative to normative data. This index was used in children with different neurological and orthopedic limitation (Beynon, McGinley, Dobson, & Baker, 2010) and in adults (Kark, Vickers, McIntosh, & Simmons, 2012) demonstrating to be useful in clinical practice with strong and significant correlations with clinicians' ratings of kinematic gait deviation (Beynon et al., 2010). However, the studies conducted using this index are scanty and to our knowledge the usability of the GPS in individuals with JHS/EDS-HT has not been investigated, yet. Since GA is recognized as a fundamental tool in the evaluation of gait patterns for patients with general movement disorders such as EDS, there is the clinical need to have a single measure of the quality of a particular gait pattern in JHS/EDS-HT.

The aims of this study are the following: 1) to quantify the gait alterations in patients in JHS/EDS-HT using a new summary index (GPS and GVS); 2) to search for a correlation between GPS (and GVS) and clinical measure to determine whether the GPS could be feasible for the characterization of gait in patients in JHS/EDS-HT.

2. Methods

2.1. Participants

Twenty-one adult (age: 36.1 ± 12.7 years; weight: 64.0 ± 16.4 Kg; height: 163.9 ± 5.9 cm) individuals with JHS/EDS-HT were enrolled in this study (JHS/EDS-HT group). All patients studied have attended a multidisciplinary service dedicated to HCTDs and were followed into the "joint hypermobility" outpatient clinic in the Division of Physical Medicine and Rehabilitation of the Umberto I University Hospital (Rome, Italy) and into the clinical genetics outpatient clinic at the Medical Genetics of the San Camillo-Forlanini Hospital (Rome, Italy). The patients referred to these services from the beginning of November 2010 through March 2011. The diagnosis of JHS/EDS-HT was established using published criteria (Beighton et al., 1998; Grahame et al., 2000). As JHS/EDS-HT is a diagnosis of exclusion, the absence of features suggestive of other heritable connective tissue disorders (like marfanoid body shape and brittle bones that are respectively the cardinal features of Marfan Syndrome and Osteogenesis Imperfectan) was assessed in a clinical genetics outpatient clinic. The patients with endurance sufficient to stand at least 20 minutes, assisted or unassisted, were recruited and screened at a baseline visit, which included a physical and neurological exam and a gait analysis.

Twenty adult participants, age matched, were recruited as healthy controls (Control Group: CG; mean age: 37.23 ± 8.91 years; weight: 66.9 ± 8.5 Kg; height: 171.3 ± 8.0 cm). Selection criteria for this group included no prior history of cardiovascular, neurological or musculoskeletal disorders. These participants showed negative Beighton score, normal muscle strength and no obvious gait abnormalities. The study was approved by the Ethics Research Committee of the Institute and written informed consent was obtained by the patients.

2.2. Instruments

The complete evaluation consisted of clinical examination and GA.

As concerns the clinical examination all patients were assessed using the Villefranche (Beighton et al., 1998) and Brighton criteria (Grahame, 2000; Grahame et al., 2000) and participants were considered with JHS/EDS-HT if meeting at least one of the two sets of diagnostic criteria. Additional extra-articular features were also investigated and registered. Hypermobility was assessed using the Beighton score that is a 9-point evaluation with attribution of one point in the presence of any of the following features: (a) passive apposition of the thumb to the flexor aspect of the forearm (one point for each hand), (b) passive dorsiflexion of the V finger of the hand beyond 90° (one point for each hand), (c) hyperextension of the elbow beyond 10° (one point for each arm), (d) hyperextension of the knee beyond 10° (one point for each leg), and (e) forward flexion of the trunk with the knees extended and the palms resting flat on the floor. Skin/superficial connective tissue features were assessed qualitatively on the basis of accumulated experience by palpation and gentle stretching of the skin at the volar aspect of the palm (at the IV metacarpal) and/or of the forearm. In addition patients were evaluated using the Lower Extremity Functional Scale (LEFS), which is a self-report questionnaire containing 20 questions in regards to different activities with a score from 0 (Extreme Difficulty or Unable to Perform Activity) to 4 (No Difficulty). The maximum score is 80 points, indicating very high function and the minimum is 0 points, indicating very low function (Binkley, Stratford, Lott, & Riddle, 1999).

As concerns the instrumental evaluation, all patients were evaluated instrumentally using an optoelectronic system with passive markers (ELITE2002, BTS, Milan, Italy) with a sampling rate of 100 Hz, two force platforms (Kistler, CH) and 2 TV camera Video system (BTS, Italy) synchronized with the system and the platforms for videorecording. In particular, the analogic signal acquisition is conducted using an A/D converter and all the signals are managed with a common clock and integrated inside an acquisition workstation.

2.3. Procedures

After the collection of some anthropometric measures (height, weight, tibial length, distance between the femoral condyles or diameter of the knee, distance between the malleoli or diameter of the ankle, distance between the anterior iliac spines and thickness of the pelvis), passive markers were placed at special points of reference, directly on the individual's skin, as described by Davis, Ounpuu, Tyburski, and Gage (1991), to evaluate the kinematics of each body segment. In particular they were placed at C7, sacrum and bilaterally at the ASIS, greater trochanter, femoral epicondyle, femoral wand, tibial head, tibial wand, lateral malleolus, lateral aspect of the foot at the fifth metatarsal head and at the heel (only for static offset measurements). The Davis marker-set was chosen as the protocol of choice to acquire the movement of lower limbs and trunk based on Ferrari et al. (2008).

2.4. Experimental protocol

After placement of the markers participants completed two or more practice trials across the plate walkway to ensure that the patients were comfortable with the experimental procedure. After familiarization, at least 6 trials were acquired asking the participants to walk at their self-selected velocity and barefoot along the walkway (10 m long). Average values of three consistent trials from each side foot were analyzed.

All the acquisitions were acquired by the same operator with experience, so to assure reproducibility of the acquisition technique and to avoid the introduction of errors due to different operators.

2.5. Signal processing

In this analysis, only kinematic data were considered and while ground reaction forces were also acquired during this study, they are not included in the present analysis and are not discussed in this paper.

In this study all kinematic graphs obtained from GA were normalized as a percentage of gait cycle producing sagittal kinematic plots of the pelvis, hip, knee and ankle for each cycle. Using specific software (BTS EliteClinic, version 3.4.109) data were exported in .txt and .xls files. From these data format we computed the Gait Profile Score (GPS) which summarizes the overall deviation of kinematic gait data relative to normative data (Baker et al., 2009). The GPS and MAP method was implemented as described by its authors (Baker et al., 2009) using our control data. GA data were then processed to obtain GPS and MAP according to the published method (Baker et al., 2009).

The GPS represents the root mean square (RMS) difference between particular gait trial and averaged data from people with no gait pathology. It has an advantage over the other indices as it is comprised of a number of gait variable scores (GVSs) representing an equivalent RMS difference for different kinematic variables. These can be displayed as a bar chart known as the Movement Analysis Profile (MAP).

The GPS is based upon a number of gait variable scores (GVS) each of which is the root mean square difference between a specific time normalized gait variable and the mean data from some reference population calculated across the gait cycle. Thus if $x_{i,t}$ is the value of gait variable i calculated at a specific point in the gait cycle t, and $\bar{x}_{i,T}^{ref}$ is the mean

value of that variable at the same point in the gait cycle for the reference population then the *i*th gait variable score is given by:

$$GVS_i = \frac{1}{T}\sum_{t=1}^{T}(x_{i,t} - \bar{x}_{i,T}^{ref} -)^2$$

where T is the number of instants into which the gait cycle has been divided. The GPS is then the RMS average of the GVS variables:

$$GPS = \frac{1}{N}\sum_{i=1}^{N} GVS_i^2$$

The overall GPS is based upon 15 clinically important kinematic variables (Pelvic Ant/Pst, Pelvic Up/Dn Obliquity and rotation of the left side and hip flexion, abduction, internal rotation, knee flexion, dorsiflexion and Foot Progression for left and right sides). In this analysis a GPS score for each side was used based on all nine GVS for that side.

As the GPS represents the difference between the patient's data and the average from the reference dataset, the higher the GPS value is, the less physiological gait pattern is.

2.6. Statistical analysis

GPS and GVS scores were computed bilaterally for each participant and the median and quartile range values of all indexes were calculated for each group (JHS/EDS-HT group and CG). Kolmogorov–Smirnov tests were used to verify if the parameters were normally distributed; the parameters were not normally distributed, so we used the Mann–Whitney U tests for comparing data of JHS/EDS-HT group and CG. With the proposed sample sizes the study will have a power of 86%. The Spearman coefficient (ρ) and the gamma coefficient (γ) were calculated to examine the relationship between LEFS total score, item 11 ("Walking 2 blocks") and 12 ("Walking a mile") of LEFS and GPS respectively. Among items of LEFS only item 11 and 12 were selected because they are the items related to walking ability. The correlation Statistical significance was set at $p < 0.05$.

3. Results

All the participants were able to complete both clinical and instrumented evaluations. An initial comparison between the GPS scores of the right and left limb was made for all patients. No statistical differences were found between the two limbs, indicating a symmetric gait pattern; subsequently, data from both sides were pooled.

The values of GPS and GVS for the pathological group and the CG were displayed in Fig. 1.

The GPS value of patients was 8.9 ± 2.6, statistically different from 4.6 ± 0.9 displayed by the control group ($p < 0.05$). In particular, all values of GVS were higher if compared to CG ($p < 0.05$), with the exception of Pelvic Tilt and Foot Progression, which are close to normality.

The GPS distribution with respect to LEFS was examined in order to investigate the presence of correlation among the GPS and clinical assessment: while no significant correlations were found between GPS and total score of LEFS ($\rho = -0.38$; $p > 0.05$), significant correlations were found with the item 11 ("Walking 2 blocks") ($\rho = -0.56$; $p < 0.05$) and 12 ("Walking a mile") of LEFS ($\rho = -0.76$; $p < 0.05$) (Fig. 2). The research of correlation was conducted also between "item 11" and "item 12" of LEFS and each GVS value, and results showed that existed a significant relationship of the "item 12" with the score related to Knee Flex-Extension, with $\rho = -0.49$ ($p < 0.05$), and to Ankle Dorsi-Plantarflexion, with $\rho = -0.46$ ($p < 0.05$).

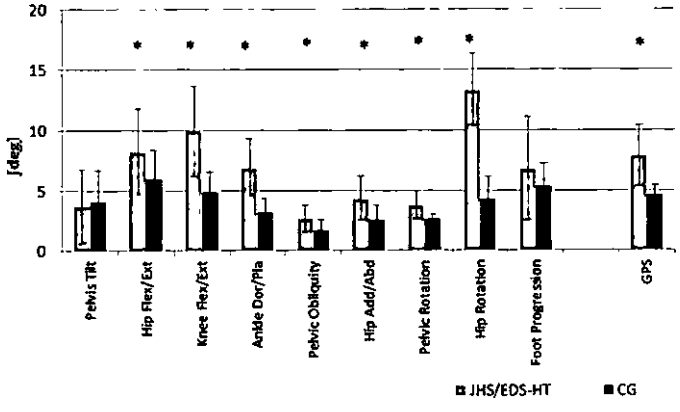

Fig. 1. Median and quartile range of GVSs and GPS values for the EDS-HT group and CG. *$p < 0.05$. EDS-HT group vs. CG.

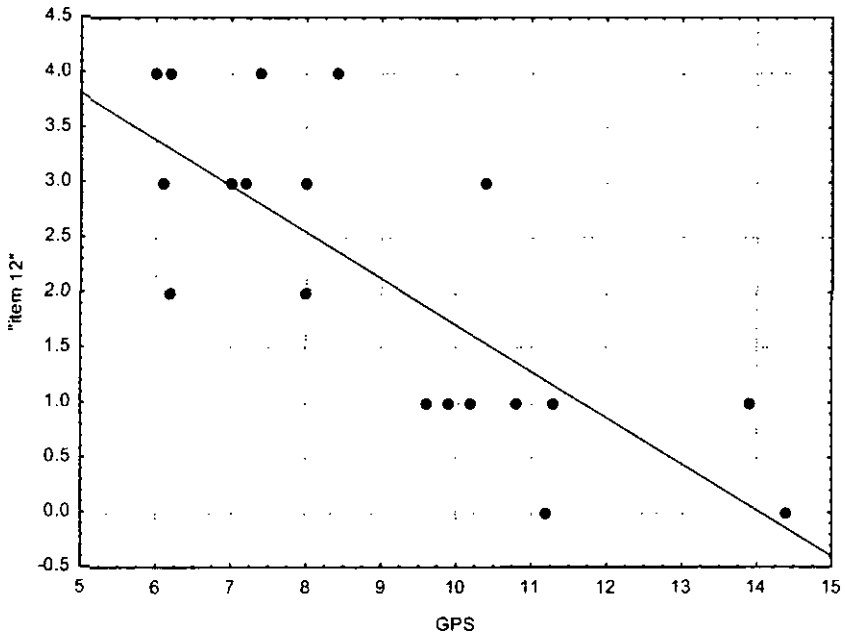

Fig. 2. Graph representing the correlation between GPS and "item 12" of LEFS for EDS-HT group ($\rho = -0.76$, $p < 0.05$).

4. Discussion

This study quantified the gait pattern in patients with JHS/EDS-HT using a summary measure recently proposed by literature, the GPS parameter. This study represents the first application of the GPS in this pathological state. We decided to use the GPS instead of the other summary indices proposed by the literature, such as Gillette Gait Index (Schutte et al., 2000), Hip Flexor Index (Schwartz, Novacheck, & Trost, 2000) or Gait Deviation Index (Schwartz & Rozumalski, 2008) because of the nature of GPS, which provides both a summary measure (GPS) and the single values (GVS) for each graph of gait analysis report. The selection of the parameters used for the Gillette Gait Index and Hip Flexor Index computations are, in fact, specific to gait characteristics of Cerebral Palsy and the Gait Deviation Index appears a measure of gait pattern independent from the specific pathology (Galli, Cimolin, De Pandis, Schwartz, & Albertini, 2012), but too global. On the contrary, the GPS is independent from the specific pathological state with the great advantage of the decomposition into GVS and MAP, which are not present in the other summary measures. In this way it provides useful insights into which variables (i.e. pelvis, hip, knee or ankle joint) are contributing to GPS (Baker et al., 2009).

As concerns the GPS value, our data evidenced that the gait pattern of JHS/EDS-HT participants was globally statistically different from CG. Since GPS can provide a global estimation of gait deviation, which may be related to pathology, it has the potential to be a value complement to a more detailed analysis of kinematics. In addition, thanks to GVS and MAP, it is possible to identify the level of actual deviations (hip, knee or ankle joints): from our data, the significant differences from the controls were related to all the lower limb joints, with the exclusion of pelvis on the sagittal plane (Pelvic Tilt) and foot on the transversal plane (Foot Rotation). These results are partially in agreement with previous literature (Galli, Cimolin, et al., 2011). Previous study showed significant differences mainly in terms of ankle joint, confirmed by our results, and not at knee and hip joints, not in agreement with our results. The reasons of these differences may be twofold. The first could be due to the different patients' number of the two studies, which is higher in the present study. Secondly, the reason could be connected to the different nature of the specific parameters used by Galli, Rigoldi, et al. (2011) and of the summary measures (GPS and GVS) used in this study. While specific parameters are computed in particular instant of the gait cycle, the GPS and GVS are computed point by point from the entire gait vector, including accordingly more information than specific indices.

In addition to previous results, we found also significant difference between JHS/EDS-HT and control group in terms of the hip internal-external rotation during walking, not evaluated in literature yet.

Interestingly, significant correlations were found with the item 11 ("Walking 2 blocks") and 12 ("Walking a mile") of LEFS, indicating that GPS seems to be sensible for measuring the function level in EDS patients. Not significant correlation was found with LEFS total score ($p > 0.05$); the reason could be connected to the different and wide motor aspects evaluated by the LEFS score compared to the gait-specific nature of the GPS. LEFS questionnaire, for example, assesses "putting on your shoes or socks", "squatting", "getting into or out of a car" and other activities different from walking and not included in the GPS, which is specific for gait. In addition, our results evidenced that a significant relationship of the "item 12" existed with the GVS and in particular with Knee Flex-Extension and Ankle Dorsi-Plantarflexion; it could indicated that the deterioration of function level may be directly connected to deterioration at distal (knee and ankle joints) level.

A potential weakness of this study may be the relatively small sample size, resulting in limited strength of our findings. However, JHS/EDS-HT is a rare genetic condition which affects no less than 1 in 10,000 in the general population and large experimental group is difficult to gather. In addition, from a statistical point of view with the proposed sample sizes, even if not large, the study has a power of 86%. Another bias of the study is that participants were only females and so not sex comparison has been possible. However it is reported that this pathological condition is dramatically more common in women (Castori et al., 2010). In addition the GPS methodology incorporates only kinematic patterns of the lower limb joints taking not in consideration spatio-temporal parameters and kinetics which have been demonstrated previously to differ from physiological values in these patients (Galli, Rigoldi, et al., 2011). Future work may focus on extension of the GPS to these elements in order to make it a more extensive measure of gait strategy.

Although some limitations are present, this work represents the first attempt to use a synthetic index in patients with JHS/EDS-HT. In this pathology, such as in the past for other pathologies like Cerebral Palsy, there is in fact the clinical need to develop specific and synthetic parameters to measure gait. From our results, the GPS seems to have validity as a summary measure of functional limitation during walking in JHS/EDS-HT patients and it could represent a useful tool in clinical settings to objectively quantify the degree of gait deviation from normality, stratify severity and to quantify the effects of rehabilitative treatments.

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APPENDIX C

Case Report

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Lipedema, a Rare Disease

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Lipedema is a chronic disease of lipid metabolism that results in the symmetrical impairment of fatty tissue distribution and storage combined with the hyperplasia of individual fat cells. Lipedema occurs almost exclusively in women and is usually associated with a family history and characteristic features. It can be diagnosed based on clinical history and physical examination. Lipedema is usually symmetrical, but spares the feet, is often painful to palpation, and is negative for Stemmer's sign. Additionally, lipedema patients can present with microangiopathies and lipomas. The well-known therapies for lipedema include complex decongestive therapy, pneumatic compression, and diet modifications. However, whether these treatments help reduce swelling is debatable. We encountered a case of lipedema that was initially misdiagnosed as lymphedema. The patient's clinical features and history were different from those typical of lymphedema, prompting a diagnosis of lipedema and she was treated with a complex decongestive therapy program.

Key Words Lipedema, Lymphedema, Complex decongestive therapy

INTRODUCTION

Lipedema is a chronic disease of lipid metabolism marked by a bilateral and symmetrical swelling of the lower extremities caused by impairment of symmetrical fatty tissue distribution and storage combined with hyperplasia of individual fat cells. It can be diagnosed using clinical features rather than diagnostic tests.[1] It almost exclusively affects women and 15% of patients have a family history of lipedema.[2] Lipedema occurs primarily in the lower extremities and is rarely accompanied by edema of the upper extremities.[1,3,4] Edema of the lower extremities is observed between the pelvic crest and the ankle, and occurs symmetrically on both sides.[3] Here, we report a case of lipedema with a review of the literature.

CASE REPORT

A 60-year-old female patient was admitted to the Department of Gastroenterology, Kosin University Gospel Hospital, for stomach evaluation because esophagogastroduodenoscopic findings at a regular health check-up were suspicious for stomach cancer. After admission, an esophagogastroduodenoscopy, colonoscopy, chest computed tomography scan, abdominal computed tomography scan, and bone scan were carried. No abnormal findings of stomach cancer were observed.

The patient reported a 3-year history of edema of both lower extremities, and was transferred to the

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Table 1. Circumference of Both Lower Limbs

		20 cm below the knee	10 cm below the knee	Knee	10 cm above the knee
At transfer	Rt.	38.0 cm	45.0 cm	49.0 cm	54.5 cm
	Lt.	40.0 cm	45.0 cm	48.0 cm	53.5 cm
At discharge	Rt.	36.5 cm	42.5 cm	44.3 cm	52.5 cm
	Lt.	36.5 cm	43.2 cm	46.0 cm	51.0 cm

Department of Physical Medicine and Rehabilitation for the evaluation and treatment of the lymphedema. The patient had undergone an operation for the fracture of the left tarsal bone 9 years previously and a hysterectomy with bilateral salpingo-oophorectomy due to uterine myoma 4 years previously. At presentation, the patient reported taking metformin (Dae Woong Pham, Seoul, Korea) and glimepirid (Han Mi Pham, Seoul, Korea) for diabetes, and valsartan (Novartis Korea, Seoul, Korea) and amlodipine (MSD Korea, Seoul, Korea) for hypertension. The patient's weight gain started 10 years earlier and the edema of both lower extremities, which showed intermittent improvement and deterioration, appeared 3 years previously. More recently, the edema had continued without any improvement, although the patient did not have any family history related to lower extremity edema.

Muscle strength and sensory and muscle stretch reflexes of both the upper and lower extremities were normal on a physical examination that was carried out following transfer of the patient. The circumferences of both lower limbs were measured 10 cm proximal and distal to the lateral condyle of the femur to assess the degree of lower extremity edema (Table 1). Soft edema with negative Stemmer's sign was evident. Obvious differences in both lower extremities were not observed and edema was not evident in the ankles or feet. Upon examination, petechiae were noted in both lower extremities and a lipoma was observed under the right knee joint (Fig. 1, 2). The patient reported tenderness in lower extremities on palpation and, specifically, more severe pain on the outer surface of the left thigh. There were no abnormalities noted upon neurological examination.

Blood tests for diseases of the thyroid gland, heart, and kidney were all normal. Three-dimensional computed tomography angiography was performed to determine whether the edema was a result of vascular lesions.

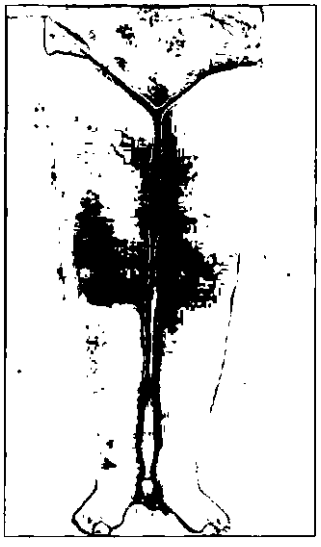

Fig. 1. Photograph of both lower limbs of the patient. Swelling is seen in both lower limbs except for the feet.

Fig. 2. Photograph of the patient's right lateral thigh. Multiple micropetechiae are seen on the right lateral thigh and calf.

Vascular lesions, including deep vein thrombosis, were not observed. Technetium-99m human serum albumin lymphangiography was also conducted to assess the presence of lymphedema, but there were no abnormalities (Fig. 3). In addition, motor nerve and sensory nerve conduction studies were performed to identify the cause of the lower extremity pain. The amplitude of the motor nerve action potential for both deep peroneal nerves, which were recorded in the short extensors of the toes, was reduced. There was no nerve action potential in the right superficial peroneal nerve. The amplitude of the sensory nerve action potential was reduced in the left superficial peroneal and both

sural nerves. However, it was difficult to diagnose this as clear polyneuropathy as there were no abnormalities in the onset latencies and nerve conduction velocities and there were no denervation potentials on needle electromyography.

We suspected that the absence of, and reduction in, potentials was due to serious edema and the increase in subcutaneous tissue rather than an abnormality of the nerve conduction velocity due to peripheral polyneuropathies because there was no motor or sensory response irregularities except for tenderness on palpation in the neurological exam (Table 2).

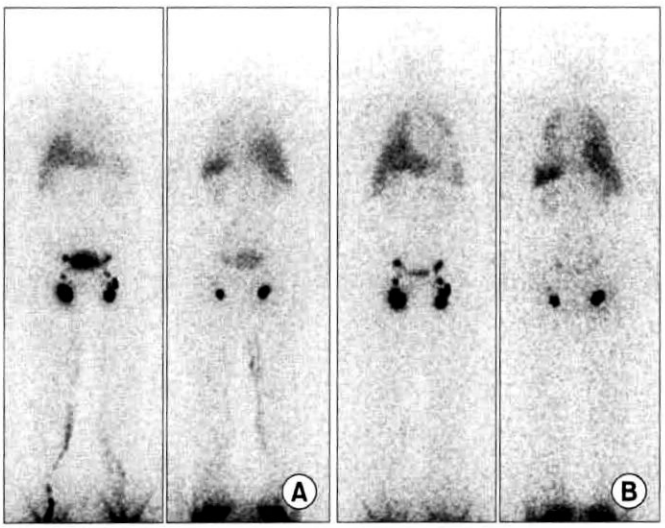

Fig. 3. Results of Technetium-99m human serum albumin lymphoscintigraphy. (A) Lower extremity Technetium-99m human serum albumin lymphoscintigraphy obtained 30 minutes after the injection of radionuclides revealing normal lymphatic drainage. (B) Lower extremity Technetium-99m human serum albumin lymphoscintigraphy obtained three hours after the injection of radionuclides revealing normal lymphatic drainage.

We diagnosed the patient with lipedema based on the edema with negative Stemmer's sign that occurred symmetrically in the legs, clinical history, physical exam findings, and other diagnostic test results. Complex decongestive therapy was administered to reduce the edema and pain. Bandaging was not applied due to the patient's pain. Manual lymphatic drainage with low intensity and remedial massage were performed during the first week of treatment. Thereafter, complex decongestive therapy including bandaging was actively performed after the patient's tenderness on pressure was reduced. Upon symptom improvement, dietary modifications were attempted in consultation with a nutritionist in conjunction with an exercise prescription that focused on aerobic exercise. The circumferences of both lower extremities were measured at one-week intervals using the same method beginning immediately after transfer to the Department of Physical Medicine and Rehabilitation. The treatment continued for a month. There was no significant change in total weight over the treatment period, but the reduction in edema resulted in decreased circumference of 2.75 cm and 2.45 cm in the right and left lower extremities, respectively. The circumferences of the lower extremities were reduced by 2.75 cm on average compared to measurements taken prior to treatment (Table 1).

Table 2. Nerve Conduction Data of Lower Limbs

	Amplitude (μV) Distal/Proximal	Conduction velocity (ms)	Latencies	
			Distal	Proximal
Motor				
Rt. tibial	9,800/5,000	44	3.2	12.2
Rt. peroneal	1,600/1,200	51	3.1	10.4
Lt. tibial	9,200/5,200	45	5.1	13.4
Lt. peroneal	1,400/1,100	49	3.2	11.1
Sensory				
Rt. superficial peroneal	Not evoked			
Lt. superficial peroneal	6	52	2.1	2.8
Rt. sural	8	66	1.6	2.6
Lt. sural	4	5	1.9	2.7

DISCUSSION

Lipedema is a condition that occurs bilaterally and symmetrically in the lower extremities and arises from the deposition of fat tissue starting at the hips and ending at the ankles, in a pattern that is visually similar to riding breeches.[4] A family history of lipedema is noted in many patients, as it is a disease that occurs due to an abnormal distribution of fat because of genetic or hormonal abnormalities. Edema is also observed in the hips and both legs due to abnormal fat distribution, but not in the ankles and feet.[1-7] The prevalence of lipedema in women has been reported as 11%. Other characteristics of lipedema include hematomas or petechiae that can easily arise from a minor shock or slight touch due to the increased fragility of the microvessels. In addition, tenderness can develop in response to small stimuli and palpations, and it is possible that the variably-sized nodules formed by the accumulation of subcutaneous fats are palpable.[1,6]

Lipedema tends to be generally misdiagnosed as lymphedema. In our patient, the edema in both lower extremities was misdiagnosed as lymphedema because of a medical history of hysterectomy. However, unlike the indicators for lymphedema, lymph node dissection was not performed as part of the hysterectomy for uterine myoma, but the presence of bilateral symmetrical edema led us to suspect lipedema. Additionally, drugs are often suspected as the cause of edema of the bilateral extremities; however, in this case, the lower extremity edema differed from drug-induced generalized edema because only bilateral lower extremity edema sparing the ankles and feet was observed. Drug-induced edema could also be excluded because the medical history was unremarkable for drugs such as diuretics or immunosuppressants, which are known to cause edema.[1,4]

Contrary to lymphedema, a family history of lipedema is common. Lipedema occurs bilaterally and symmetrically, and edema is not distinctively observed in the ankles and feet. Stemmer's sign is absent in lipedema because skin fibrosis is rare, and it can be accompanied by damaged skin microvessels due to accumulation of fat, microhematomas or petechiae due to circulatory disturbance.[1-6] Serious pain on palpation is relatively common in lipedema compared to lymphedema, and it is rare to find a medical history of cellulitis.[5,7] This patient was diagnosed with lipedema based on characteristics that were consistent with the clinical features of lipedema, including bilateral edema of the lower extremities, negative Stemmer's sign, and tenderness on applied pressure.

The progression of lipedema can be divided into three stages according to skin conditions and the sizes of the fat nodules. In the first, early stage of lipedema, soft skin and small, evenly distributed nodes can be observed in thick subcutaneous tissues. The skin appears orange peel-like in the second stage with larger, unevenly distributed nodes of subcutaneous tissue. In the third stage of lipedema, subcutaneous fat tissue projects outside the skin of the knees or thighs, which hinder mobility.[1,3] This patient was diagnosed with second-stage lipedema based on the presence of large fat nodes and orange peel-like skin.

Petechiae of this patient were observed in the pretibial portion of the lower extremities, and lipomas were distinctly observed on ultrasonography. After active complex decongestive therapy, the size of the lipomas decreased slightly, but remained. Lipomas are not generally found in patients with lipedema, but some cases of patients with lipedema accompanied by multicentric lipomas have been reported. The pathogenesis with respect to the accumulation of fat tissue is the same, so it is thought that there is a correlation between the two diseases.[5] Presently, histological examinations of the lipomas and micropetechiae were not performed because the patient's clinical symptoms and medical history were significant for the characteristics of lipedema. However, histological methods of diagnosis can be considered for this disease.

The symptoms of peripheral polyneuropathies due to diabetes were not observed in this patient, but the patient reported pain from weak pressure. The cause of this pain was not obvious, but it is thought that sensory nerves from each sympathetic nerve fiber are distributed in fat cells and impairment such inflammation of these autonomous nerves can cause tenderness and pain. This impairment may lead to a misinterpretation of protopathic sensory inputs such as sensations of pressure, temperature, or postures. When this impairment is accompanied by microangiopathies, the degree of pain can be increased. Presently, an improvement in pain was

be evident after low-intensity manual lymphatic drainage and remedial massage performed for one week.[4] In addition, the circulation of lymph is generally normal in patients with lipedema. However, when the disease is chronically progressed, circulatory disturbance occurs due to the pressure of fat cells on lymph collectors at the superficial layers. Moreover, microangiopathy occurs in the regions of edema, altering microcirculation and leading to increased permeability and protein-rich fluid extravasation, which further increases the amount of lymph. In prolonged courses of lipedema, the lymph vessels are unable to maintain their function, and altered microcirculation leads to impaired lymph transport capacity and the accumulation of lymph fluid. The high protein and fat contents of lymph fluid induce fibrosis with positive Stemmer's sign and subsequent progression to lipolymphedema.[3,8]

If adequate treatment is not administered even though the disease has progressed to chronic lipolymphedema, it has a similar prognosis and progresses to lymphedema. Thus, lipedema can be seriously damaging to the quality of life due to a reduction in the patients' mobility and aesthetic issues.

Active treatment for lipedema is required because early diagnosis and treatment can determine the patient's long-term prognosis. Therapy for lipedema can be largely divided into conservative treatments to reduce edema and surgical treatments such as liposuction. Conservative treatment can first be performed with complex decongestive therapy, which is usually used for patients with lymphedema. Although the long-term therapeutic effects for patients with lipolymphedema are good, the therapeutic effects for those with simple lipedema can be slow and weak. Complex decongestive therapy cannot affect fat tissue, but can contribute to treatment by reducing interstitial edema. No bandages should be used until the pain subsides because, unlike lymphedema patients, those with lipedema report pain and hypersensitivity in the edema areas when complex decongestive therapy is performed. The use of bandages after the pain disappears is helpful in reducing edema. In our case, manual lymphatic drainage was performed for this patient without bandages for one week because of reported tenderness upon the application of pressure to the legs during early treatment. There was an improvement in tenderness after one week of treatment. After the loss of tenderness, complex decongestive therapy including manual lymphatic drainage and bandaging were performed for a month and a reduction in leg circumference was observed. As in the present case, manual lymphatic drainage is typically performed every day during the edema reduction phase and twice weekly in the subsequent preservation phase. After edema reduction through complex decongestive therapy, compression stockings can be considered. According to several reports, a reduction in the excessive fatty tissue in lipedema is possible if the compression stockings are worn constantly and if compression bandages are applied at night.[3,4,6,9] However, continuous treatment is critical because the edema will recur or worsen if complex decongestive therapy is stopped.[1]

Presently, conservative treatment produced a reduction in pain and edema. However, surgical treatment can be considered for patients with lipedema who do not respond to conservative treatment.[1] Liposuction was performed under general anesthesia in the early 1990s, but this was associated with some complications such as excessive bleeding or permanent lymphedema due to damage to the lymphatic system. Now, the damage to important tissues such as nerves or blood vessels can be minimized by performing liposuction with thin (2-4 mm diameter) catheters under local anesthesia with advanced anesthesia and operation technologies. For this reason, liposuction is currently the standard surgical treatment method. Complex decongestive therapy for an early period after the operation is helpful in preventing the recurrence of edema because there is still the possibility of damage to the lymphatic system after the operation.[1,3-5,7]

Additionally, a better treatment effect for obese patients can be expected if their weight management is successful through proper exercise and diet modifications.[1,3] Presently, although loss of weight and body fat was expected with diet modifications and aerobic exercise, these effects were not significant.

According to the differing pathophysiologies of lipedema, which occurs due to the accumulation and impaired distribution of fat tissues, and lymphedema, which occurs due to the accumulation of lymph because of abnormalities in lymph circulation, their progression and prognoses differ. Thus, distinguishing between the two diseases at an early stage is important for both

clinicians and patients to understand the prognosis and determine the best treatment approach. It is extremely likely that lipedema can be improved if proper treatment is applied before 35-years-of-age, but delayed management makes the prognosis of lipedema similar to that of lymphedema as the disease progresses to lipolymphedema.[3]

If lipedema is not treated, complications that are detrimental to mental health and life-threatening can occur. These complications include mental problems such as eating disorders and generalized complications including hypertension, diabetes, and heart failure. Furthermore, patients may develop pseudo-Bartter's syndrome, which is characterized by hyperaldosteronism and hypokalemia due to the excessive use of diuretics, and joint problems in the spine or lower extremities due to excessive body weight.[3] This patient had continued daily life without special treatment after the occurrence of lipedema, and serious complications resulting from increased body weight (except for hypertension and diabetes) were not observed. Lipedema is a rare disease that has been scarcely reported in Korea. If it is diagnosed and treated early by taking into account its causes and clinical characteristics, complications can be reduced so that the patient can maintain better health and quality of life.

Lipedema is rare and is diagnosed based on a patient's medical history and physical examinations. To diagnose lipedema accurately, it is essential for it to be clearly distinguished from other diseases that can cause lower extremity edema. Lipedema can be easily confused with lymphedema, which is relatively common, because lipedema is a comparatively rare disease. Knowing the clinical features and differences in medical histories from the patients with lipedema will enable clinicians to achieve a good prognosis through quick diagnosis and treatment.

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APPENDIX D

English Translation

Liposuction of Lipedema to Prevent Later Joint Complications

Josef Stutz, Schwarzenbach am Wald

Josef Stutz

Summary

Lipedema is a symmetric fat disorder in women which affects their legs and arms.

Abnormal fat accumulations at the proximal inner thigh cause an abduction of the leg axis which leads to a change in gait, and to an unnatural physiological strain on the leg joints (knock knee).

Using liposuction, this abnormal fat can be reduced, and the leg axis and gait can be corrected.

Key words: lipedema, leg axis, knock-knee, arthrosis.

Lipedema is a symmetrical fat-distribution disease, usually presenting in the legs, less often in the arms. It primarily affects women. This subcutaneous fat tissue increases over time; however, the rate of progression occurs in an unpredictable manner. The physical change is usually noticed by affected patients at the end of puberty as a disproportion between the upper and lower halves of their bodies. Since the disease often runs in families, there is likely a genetic component; since it affects primarily women, there are likely hormonal causes, as well.

Lipedema typically causes a symmetrical growth of fat tissue in the legs, from the iliac crest to the ankles, and in the arms, from the shoulder to the wrist. The feet and hands, however, are always spared from increased fat tissue.

In later stages, the patients are prone to develop orthostatic edema, especially during hot weather and at the end of the day. Additionally, there is a noticeable tendency of affected areas to bruise even after only minor trauma, as well as have increased pain to the touch (1, 4, 13). There is no direct correlation between the extent of the increased abnormal fat tissue and the degree of discomfort.

Due to dynamic insufficiency of the lymphatic drainage in the extremities, lipedema can progress into a lymphostatic edema, referred to as lipo-lymphedema. The unbalanced state of afflux to and drainage from the limbs leads to a chronic failure of the lymphogenic transport capacity; after about 15-20 years, fibrosis of the cutis and lymphedema with liposclerosis develop as an expression of a chronic final condition, resistant to therapy (2, 11).

There is no reliable epidemiological data regarding the frequency of lipedema, although the literature refers to a prevalence of 10-15%. Lipedema is not a specific form of obesity, since in obese patients, the increase of fat tissue is distributed over the entire body.

Diagnosis

In addition to the physician taking a thorough medical and family history, the disease is diagnosed by clinical examination. Noting the locations of fat distribution is most important. A large difference in diameter between a slender upper body and bulky buttocks, legs and arms, with slender hands and feet is characteristic. By palpation, noting consistency and thickness, it is possible to establish the border between the disordered adipose tissue of lipedema and healthy, physiologically normal fat tissue. In addition, the surface of the skin of the affected areas feels cold to the examiner's touch. A significant paradox can be found in a comparative pinch test: the outside of the thigh is typically more painful to the touch than the inner thigh. Also, if the back of the upper arm and the fat at the edge of the stomach are

palpated at the same time, the patient reports increased pain in their arm.

Imaging examinations

Besides CT (12), an MRI can also help to determine the location as well as the extent of the lipedemic fat distribution. Sonography has proved successful to depict the subcutaneous fat tissue (5). The thickness layer and increased echogenicity of the lipedema fat tissue allow it to be differentiated from normal subcutaneous fat. In addition, compression sonography can help to evaluate the remaining compressibility to determine the stage of the disease, and can enable the clinician to judge the degree of pain. Furthermore, to clarify possible coexisting lymphedema, an indirect lymphangiography can be used, as well as a functional lymphoscintigraphy to evaluate the lymphatic drainage.

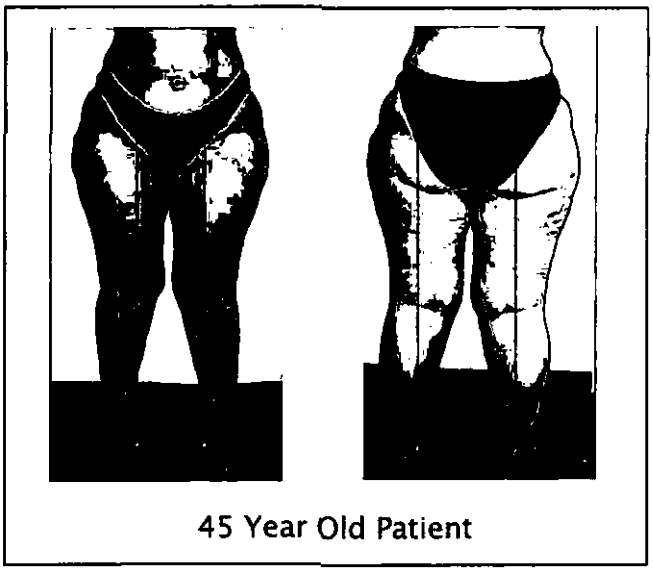

Fig. 1: Leg axes misalignment in a lipedema patient

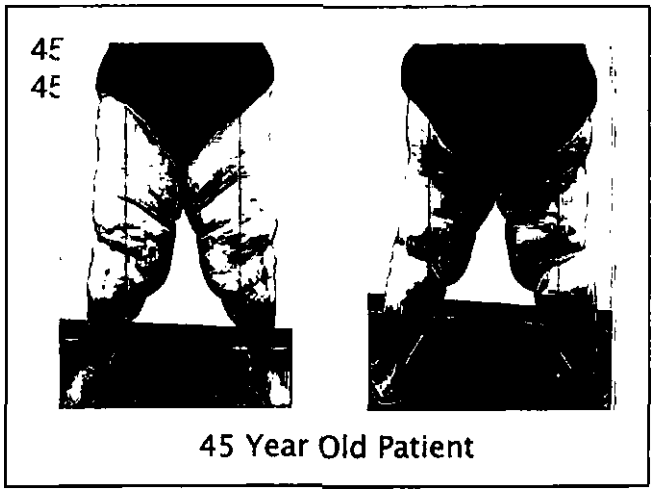

Fig. 2: Incorrect leg axis loading caused by inner thigh fat of lipedema patient.

Joint involvement

The abnormal accumulations of fat on the legs, especially those on the proximal inside of the thighs, cause affected patients develop a characteristic gait pattern (14). To avoid chafing of the skin, especially in the warmer months, patients tend to abduct their legs while walking, resulting in their being held in an upside-down V-position. These changes can manifest long before the diagnosis of lipedema is made. The patients only notice the increase in circumference of the legs, and believe that the joint pain they feel is due to to the accompanying weight gain. As this fat continues to accumulate, the abduction of the legs becomes wider, and the misaligned joint axes become clinically relevant. The improper stress in abduction causes a valgus deformity in the knee joints, and later a "skew-foot" position of the ankle joint and an apparent varus shift of the hip joint. This pseudo coxa-vara position, caused by the abduction of the legs, causes the typical "duck walk" of lipedema patients. This malposition of the joints occurs even in lipedema patients of normal weight. Knock-knees appear in obese patients as well, but in contrast to the malpositioning caused by lipedema, knock-knees due to obesity are accompanied by a valgus position of the hip joint.

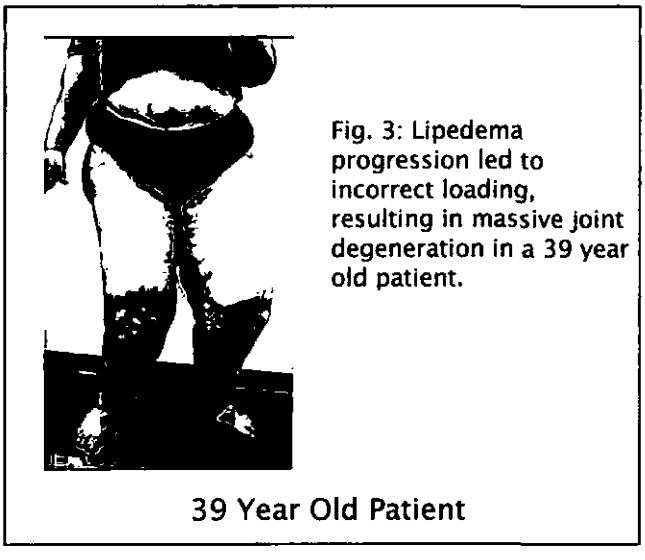

Fig. 3: Lipedema progression led to incorrect loading, resulting in massive joint degeneration in a 39 year old patient.

After the diagnosis and the start of conservative treatment, the situation for lipedema patients may be improved temporarily, since compression treatment prevents skin injuries on the insides of the thighs. The gait impediment, however, remains, since the abnormal lipedemic fat that causes the typical abduction of the legs remains, as well. Therefore, reestablishment of the normal physiological leg axis is impossible to achieve even with consistent conservative therapy, including lymphatic drainage and compression treatment. The early joint alteration

often leads to repeated mobility impairments and, in some cases, to early disablement (sometimes before age 30), despite conservative treatment. To make matters more complicated, many lipedema patients have developed eating disorders due to years of effort to reduce the size of their legs through exercise and diets. A metabolism that is accustomed to a low-calorie diet, complicated by the inability to exercise sufficiently because of joint pain, leads to a cycle of weight gain, further worsening the situation for the joints.

Confirmation of the misalignment of the joints

While taking a medical history, the clinician should inquire about manifested joint pain. Are the soles of the shoes worn off unevenly, especially on the medial side of the foot? Has the patient repeatedly been reprimanded in her youth to walk "correctly," or was her "unusual" gait alluded to in physical education class in school? Was she unable to participate in exercise due to leg pain?

In addition, a clinical examination is performed. Often, an abduction of the lower legs is displayed in a relaxed standing position as well as while walking. The distance between the inside of the ankles is demonstrably larger than the distance between the inside of the knees, and is much bigger than the distance between the proximal insides of the thighs which, in advanced lipedema patients, are always touching. The patient is asked to adduct the ankles; to achieve this, she must rotate her knees inward, since the accumulated fat on her inner thighs prevents a normal adduction of the ankles.

In the early stages, joints usually have a normal range of motion; however, tenderness or pain upon even gentle palpation can often be appreciated above the lateral knee joint space.

If the joint problems have been present for a longer period of time, patients often have previous orthopedic examinations and/or x-rays available. Usually, there are no radiographically visible changes, as generally, a knock-knee position is not depicted in an x-ray because it requires specific, long shots. These involve a high exposure to x-rays, and these views cannot be performed in many imaging centers.

Because of these limitations, the author (in collaboration with orthopedic colleagues) had been searching for an x-ray free and reproducible examination, and decided on a podometric 3D gait analysis. By way of a radio-controlled in-shoe recorder, using a special pressure-sensor sole, the pressure zones underneath the foot can be precisely measured. This video controlled gait analysis is much more exact than the traditional angle measurement, and can be reliably repeated and reproduced using standardized parameters.

These examinations show a pathologically increased pressure on the medial edge of the foot in the majority of lipedema patients. Therefore, there are detectable characteristic changes regarding the pressure zones of the foot in lipedema patients, despite the compensating "skew-foot" movements of the ankle.

Long-term studies were designed to ascertain if there was a normalization of the distribution of pressure on the feet of lipedema patients after the surgical removal of the inner thigh lipedema fat.

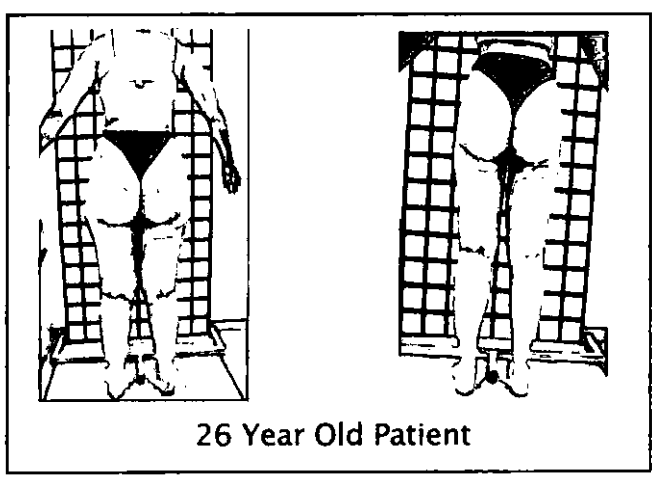

Fig. 4a: Inner thigh lipedema fat causes internal rotation of the leg axes.
Fig. 4b: Six months after WAL liposuction of medial lipedema fat, the leg axes are corrected.

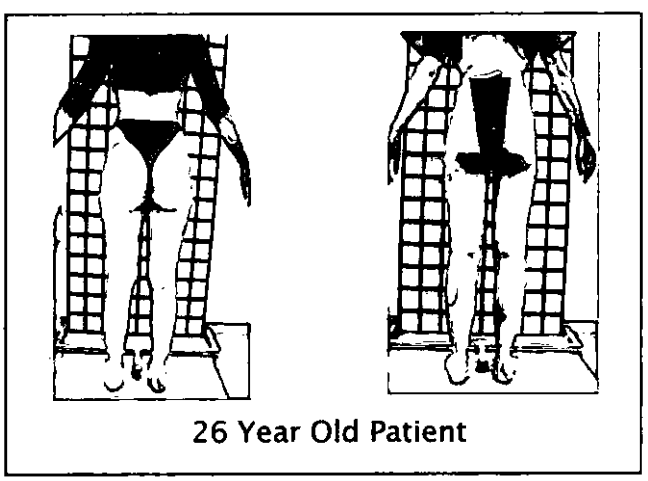

Fig. 5a: Before WAL liposuction
Fig. 5b: Six months after WAL liposuction

Treatment

Because of the pathophysiology of lipedema, exercise and diets do not reduce the fat mass in the legs. Conservative therapy, if the therapeutic regimen is consistently applied, can contribute to reducing the edema and the discomfort from strain and the pain of pressure. However, conservative therapy understandably has no effect on the localized increase in lipedema fatty tissue.

Since the late 1990s, lipedema has been treated surgically (7) – initially, against massive resistance of lymph physicians and therapists. This original resistance was legitimate, since lipedema was regarded solely as a cosmetic-aesthetic dysfunction, and the results of liposuction using the dry suction methods of that time ranged from disappointing to disastrous. The crisscross technique used in dry liposuction, which was reasonable in aesthetic surgery, ended up destroying numerous lymph vessels in the lipedema patient, which inevitably lead to a post-surgical lymphedema. Anatomical studies have concluded that lymph vessels are rather robust against sheering powers in a longitudinal direction, but can be easily damaged in a transversal axis (3). This led to the insight to use suction longitudinally, in the direction of the lymph axis only. Later, immune-histological examinations were performed to prove more precisely that, with appropriate technique, there was no damage to the lymphatic structures (8, 10). Only suction using tumescent local anesthesia creates the necessary environment in the fat tissue to enable a gentle removal of the diseased fat cells.

Since this early research, numerous successful liposuctions have been performed on lipedema patients.

These encouraging results have been backed up by studies demonstrating an improvement of the quality of life and a reduction of pain (even up to the point of no pain), and a significant reduction in the tendency to develop edema was proven, as well (9).

Conclusion

Even if the lipedema patient in the early stages of the disease only suffers a slight disproportion between her upper and lower body, lipedema is still very debilitating for her, due to daily insults and dirty looks from other people; the knowledge of potential progression to later complications such as bilateral lymphedema is even more devastating psychologically.

From the author's more than ten years' experience of treating lipedema surgically with liposuction, osteoarthritis of the large leg joints represents the most severe orthopedic complication of lipedema. This often requires one or more total joint replacements, which are performed without treating the actual cause of the malpositioning of the leg axis.

Liposuction of lipedema is the only treatment that can remove the mechanical impediment to the normal gait - namely, the abnormal lipedema fat accumulation on the proximal inner thigh - therefore, liposuction works to prevent early joint deterioration from osteoarthritis of the knee and ankle. In addition, it corrects the characteristic abnormal gait found in lipedema. The frequently used orthopedic measures are appropriate to relieve patients of pain for a certain time span; however, even joint replacement surgeries are not ultimately curative in the lipedema patient, since they neither remove the mechanical gait impediment, nor correct the resulting malpositioning of the leg axis.

This finding supports viewing lipedema as the cause of serious orthopedic disease, and consequently, necessitates explaining to lipedema patients early on that surgical removal of the abnormal fat should be viewed as necessary preventive therapy. Later complications that significantly affect the patients' mobility can be avoided only by surgically removing the abnormal lipedema fat on the legs.

Besides the significant increase in quality of life for the patients (6), removal of lipedema fat through liposuction will result in a considerable reduction in costs to the health care system. Not only will patients need less frequent lymphatic drainage after liposuction, but the costs of orthopedic intervention, up to and including joint replacement, can be avoided, as well.

Liposuction of lipedema has been demonstrated to correct of the malpositioning of the leg axis and the gait, and in addition, to improve the patients' quality of life.

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ORIGINAL ARTICLE

Water Jet-Assisted Liposuction for Patients with Lipoedema: Histologic and Immunohistologic Analysis of the Aspirates of 30 Lipoedema Patients

J. J. Stutz · D. Krahl

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Abstract Lipoedema is a fat distribution disorder causing massive, bilaterally symmetrical enlargement of the lower and in some cases the upper extremities in women. The atraumatic, anatomically appropriate procedure of water jet-assisted liposuction available today represents a promising treatment for these patients who generally suffer from severe subjective and objective impairment. Liposuction treatment can bring long-term improvement if the operative technique focuses on lymph vessel preservation. Immunohistologic analyses show minimal evidence of lymph vessel structures in lipoaspirates. The histologic analysis of the aspirates documents a relatively specific removal ("apheresis") of primarily intact lipocytes with low vascular amount.

Keywords Liposuction · Water jet-assisted liposuction · WAL · Body-jet · Lipoedema · Intact lipocytes · Atraumatic liposuction procedure · Intact lymph vessels

Introduction

Unlike (primary) lymphoedema, lipoedema is characterized by a bilaterally symmetrical, diffuse accumulation of adipose tissue [1]. This disease manifestation, which is mainly limited to the women, is localized primarily to the lower extremities, from the buttocks to the ankle joints, with the thighs and lower legs most affected. The often disfiguring enlargement and painful swelling subjectively impair the patient. Because the torso is not affected, the abnormal fat distribution results in an overall imbalance of body proportions [2–4]. The symptoms of this condition were first described in detail by Allen and Hines in 1940 [5]. It is characterized by orthostatic edema, tenderness, and increased risk of hematoma development. This disproportionate increase in leg circumference in relation to a slender torso cannot be reversed by physical exercise or diet. The course of the disease is progressive (Fig.1).

In the past ten years liposuction has become an established method for treating lipoedema, complementary to conservative treatment options. Liposuction is acknowledged as a possible therapeutic option in the guidelines of the German Society for Phlebology [6]. The aim of therapy is to reduce the circumference and volume of the extremities and remodel the leg contours. However, the first attempts at treating lipoedema with liposuction had adverse results. A worsening of the volume resulted from liposuction procedures in which operators had used a random combination of application directions along both the longitudinal and transverse axes. The unfavorable results occurred as consequences of surgical traumatization, especially to the lymph vessels, which can lead to lipolymphoedema [1]. Cornely [7] observed that "For years there has been a controversial discussion whether the liposuction of lipedema can be carried out without damaging the lymphatic system of the patient. Some authors keep claiming that the liposuction of lipedema is an obsolete method of treatment but this is not true. If the diagnosis of lipedema is undoubted, liposuction with tumescence anesthesia is carried out according to the method described by Klein" [7].

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Fig. 1 Lipoedema

Liposuction was reconsidered as a possible treatment for lipoedema after long-term successful results had been observed with anatomically appropriate liposuction methods in which the application was limited exclusively to the longitudinal axis of the extremity. A maximally atraumatic liposuction method was greatly supported by the development of tumescent local anesthesia and the use of thin cannulas [3, 8–10]. Liposuction has since established itself as a therapeutic option among the treatment possibilities for lipoedema [11, 12]. It has also been confirmed that damage to the local lymphatic vascular system must be avoided to the greatest possible extent to ensure long-term improvement of the postoperative results. Orienting analyses performed on avital tissue [13, 14] and the first intravital analyses [15] support a histomorphologic lack of damage to the lymphatic vascular system.

Today water jet-assisted liposuction (WAL) is a treatment method that avoids the disadvantages of the tumescence method, such as volume stress and osmotic effects, without increased traumatic effect.

Aim of the Study

The aim of this study was to conduct a histologic and immunohistologic evaluation of water jet-assisted liposuction (WAL) on a larger group of patients in order to evaluate its effect on lymph vessels, blood vessels, and lipocytes. For a series of 30 patients who underwent consecutive WALs on the inner knees, the entire aspirate for both legs underwent histologic and immunohistologic analyses to evaluate the structural integrity of the lymph vessels. This treatment site was chosen because the ventromedial lymphatic bundle, out of anatomic necessity, is denser in the knee region [16]. The lymphatic collectors run from the back of the foot to the inguinal lymph nodes and the ventromedial bundle extends dorsally behind the medial condyle of the femur. From an anatomical point of view, this area is associated with the highest risk to lymph vessels in liposuction.

Another aim of the study was to verify that WAL can be performed under local anesthesia with tumescent solution and that the normally required firm elastic consistency and high tissue pressure of the treated areas is no longer necessary, and also that the previously required preinfiltration period is obsolete.

Anatomical-Pathologic Considerations

The epifascial lymphatic system of the lower extremities extends as the ventromedial lymphatic bundle, with more than six main lymphatic collectors, from above the malleolus medialis to the inner knee on the medial condyle parallel to the longitudinal axis of the extremity. In the knee area the bundle of lymph collectors lies dorsomedially to the medial femur condyle [17]. Because of the dense bundling of the lymphatic collectors at the medial knee, a region especially exposed in liposuction, there is an increased risk of mechanical injury during treatment. This factor is associated with the risk of operative worsening from lipoedema to lipo-lymphoedema.

On the dorsa of the feet the lymphatic collectors lie above the venous system superficially and are in contact with the dermis-subcutis border here. In all other areas of the leg—including the knee region—the lymphatic collectors run below the venous system at various subcutaneous depths depending on the thickness of the subcutaneous fat. Some collectors have a close spatial relationship to perforating veins. In the thigh region the lymphatic collectors form three levels. In the area of the subinguinal confluence of superficial inguinal veins (Crosse), the superficial inguinal lymph nodes, which are responsible for the drainage of the lower extremities and outer genital region, are closely associated with the outlet of the saphenous vein and may be at risk here in varicose vein surgeries [18].

Lymphoscintigraphic studies of lipoedema patients have shown lymphatic insufficiency without morphologic changes in the lymph vessels as they are found in cases of lymphoedema. Lymphoscintigraphy is therefore a useful method for the clarification of differential diagnoses [19, 20].

The function of the lymph vessels can be understood histomorphologically, or ultrastructurally, in consideration of their mural content of smooth musculature. While lymph vessels with a muscle layer stimulate the flow of lymph using contractile activity, sections of vessels with fragmentary or inadequate mural muscle elements support resorptive activity of the intercellular fluid [21].

Multiple microlymphatic aneurysms of lymph capillaries, especially on the distal extremity, have been described as an anatomical-pathologic characteristic in patients with lipoedema [22].

Typical histologic findings of tissue sections from lipoedema patients show an increase of interstitial fluid with edema of the dermis and septa, an accumulation of mast cells, and a degeneration of adipocytes [23]. A patient who has suffered from lipoedema for years can spontaneously develop functional lymphoedema. This development can be forestalled through early diagnosis and therapy [1].

Diagnosis

The diagnosis of lipoedema is based on clinical findings: in addition, a high-resolution duplex ultrasound [24] is performed. If the clinician has reason to suspect that the patient's lymphatic system has already been affected, an indirect lymphangiography and a lymphoscintigraphy [25] are conducted.

Materials and Methods

Thirty female patients between 21 and 63 years of age with pre-existing pronounced lipoedema (for stages see Table 1) underwent water jet-assisted liposuction (WAL) (body-jet® system, human med AG, Schwerin, Germany) on both legs under standardized conditions with reduced quantities of Klein's [26] solution (1.0-1.5 L) and without bloating of the tissue. The entire aspirate of the inner knees (proximal lower leg and distal thigh) from both legs was histologically and immunohistologically analyzed. The operations were performed using a standardized prodecure. The infiltration was performed in all cases at Range 2 using a body-jet infiltration cannula (diameter = 3.5 mm) until sufficient anesthesia was attained with the infiltration solution. The aspiration procedure was then begun immediately without waiting for fluid infiltration.

In the WAL procedure a fan-shaped water jet is directed at the subcutaneous space in order to separate the adipose cells from the tissue, and at the same time the injected fluid, along with the detached fat cells, is suctioned off mechanically by means of a defined vacuum pressure. For all procedures the irrigation-aspiration cannula (3.5 mm) [16] was directed strictly along the axis of the lymph collectors. After the operation on the first leg, the second extremity underwent the same treatment. The vacuum was set at a constant 0.6-0.8 bar. The quantity of aspirated supernatant ranged from 250 to 2350 ml (Table 1).

Comparison to Tumescent Liposuction Techniques

In tumescent liposuction techniques local anesthesia [9] is frequently used. For this method large quantities of NaCl solution with small amounts of adrenalin and local anesthetic agents (Klein's solution [26] are introduced into the suprafascial space in preparation for the mechanical removal of the adipose tissue. The purpose of this procedure is to "tumesce" (swell) the aspirated area to achieve a tissue consistency comparable to the firm consistency of a watermelon. With this "supertumescence," according to Sattler [9], shearing forces and severe tissue traumatisztion can be avoided. In the tumescent procedure an "infiltration period" of 0.5-1.5 h on average is needed to give the fluid enough time to penetrate into the adipocytes through pressure and osmosis. At the beginning of the infiltration procedure the solution is introduced into the subcutaneous adipose tissue. This solution initially spreads along the connective tissue septa and separates the fat lobules in a process known as hydrodissection. Only then are the adipose cells mechanically removed from the aggregate by means of vacuum pressure. The adipose cells that are aspirated using the tumescent technique have been distended to many times their natural size. Therefore, the aspirate in the suction container is completely different in appearance to that obtained with WAL. In WAL the treated adipose tissue is not bloated and the infiltrating solution is aspirated simultaneously with the adipose tissue; consequently, there appears to be less "supranatant" fat in the aspirate. A comparison may be helpful: In the past, with the tumescent technique between 6 and 10 L [27] of fluid have been used for the infiltration of the front thighs of lipoedema patients in order to achieve the desired firm elastic consistency. When WAL is used, only 1-1.5 l of Klein's solution [26] are required. Therefore, it is not possible to directly compare the supernatants of the different aspirates. The actual quantity would have to be deduced using defined centrifugation.

Immunohistologic Analyses

The critical regions of the inner knee (proximal lower leg and distal thigh) were treated and aspirated separately; the lipoaspirate obtained from the knee area of both legs was also collected separately. The fat-containing operation product floating on the surface of the irrigation solution was skimmed off mechanically for further analysis. Standardized paraffin embedding and histologic

Table 1 Patient data, volume of aspirated fat, and procedure parameters

Patient No.	Age (years)	Size (cm)	Weight (kg)	Fat (%)	BMI	Stage of lipoedema	Waist-to-hip ratio	Aspirate (ml) (supranatant fat)
27196	29	180	65.2	18.8	20	III/1	0.75	250
27219	40	165	79.4	33	25	III/1	0.78	1400
27211	36	157	65	29.8	26	II/1	0.62	1350
25346	40	166	66	35	24	II/1	0.78	500
27390	34	170	71.2	25.5	25	III/1	0.73	600
27215	25	166	73.4	25.3	26.5	II/1	0.72	2000
27319	38	162	87	36.2	33.5	III/1	0.8	1250
27409	36	158	57.2	24.5	22.5	III/1	0.66	650
27463	41	170	70	27.3	24	III/1	0.71	200
27250	36	164	70.4	29.7	26	II/1	0.68	1000
19679	28	164	71.6	30.9	26.5	III/1	0.73	1600
21279	34	172	79	33	26.5	III/1	0.72	1150
27545	23	172	62.8	19.2	21	III/1	0.6	650
26165	63	168	87.4	39.7	31	III/2	0.8	650
26500	38	162	84.4	38.4	32.5	II/1	0.62	2350
27319	38	162	87.2	37.3	33.5	III/1	0.79	1100
10865	36	165	70	30	25.5	III/1	0.69	900
26951	24	161	96.6	30.4	37.5	III/1	0.72	750
27627	37	162	84.4	32.2	32	III/2	0.7	2650
27863	21	174	55.9	17.7	18	III/1	0.78	950
27928	38	174	75.4	28.7	25	III/1	0.63	1500
27925	21	164	80.2	35.6	30	III/1	0.68	1550
27810	23	170	73	25.4	25	III/1	0.68	800
27903	24	174	82.6	30.5	27	III/1	0.68	1300
27187	42	170	72.2	32.9	25	III/1	0.68	1100
10865	36	165	69.2	29	25	III/1	0.68	800
27010	40	163	98.2	39.5	37.5	III/1	0.85	1400
28054	22	175	87.8	34.1	29	III/1	0.68	1100
27600	33	168	63	21	22	III/1	0.69	550
28251	39	161	66.8	29.9	25.7	III/1	0.63	1400

preparation were performed in the laboratory following formalin fixation and centrifugation. The immunohistochemical markers CD31 (vascular endothelium) (DAKO) and D2-40 (selective marker for lymphatic endothelium) (Zytomed, Berlin) were used with the detection system K5005 DAKO alkaline phosphatase red rabbit/mouse (DAKO Cytomation, Hamburg) and chromogen Fast Red. Heat-induced antigen demasking was performed at pH 9.0.

For each specimen, analysis was performed on three step sections stained with conventional hematoxylin & eosin (H&E) as well as one immunohistochemically prepared slide for each. The area of the section examined per slide was 3.0 × 2.0 cm. The analysis was performed independently by two experienced histopathologic examiners. Skin tissue sections exhibiting clear results for both markers were used as positive controls (Figs. 2–4).

Results

The adipose tissue present in variously sized fragments in the aspirate consisted primarily of intact single cells and smaller aggregates of adipocytes which morphologically survived the operative removal from the connective tissue aggregate. Moderate quantities of blood capillaries were consistently detected in each field of study through the expression of CD31. Positive staining with D2-40 antibodies was detected for only two patients, with very few lymphatic lumina (maximum of 1/visual field).

Blood vessels (arterioles, venules, capillaries), with their endothelial lining, show up as oval or ring structures with anti-CD31 antibodies (Fig. 2). When stained with the D2-40 antibody, lymph vessels show up in the tissue section as an unrounded contour or as collapsed endothelial tissue (Fig. 3). In the level of the subcutaneous adipose tissue,

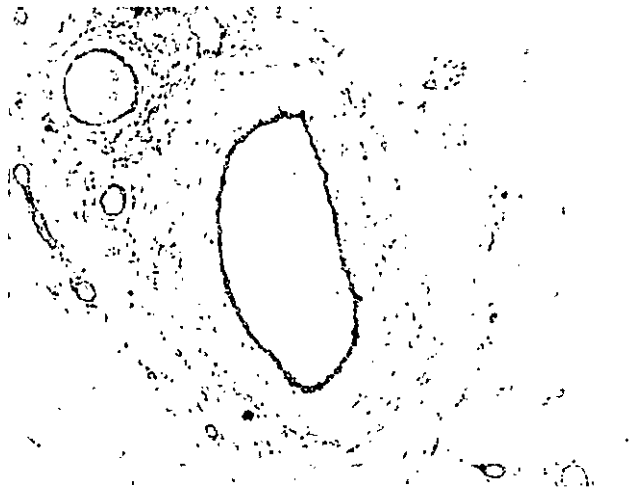

Fig. 2 Controls: CD31 antibody

Fig. 3 Controls: D2-40 antibody

Fig. 4 Controls: lipocyte complexes

blood vessel and lymph vessel tracts pass through the collagen-fibrous septal connective tissue.

The findings for the individual cases, including biographical data and information on disease stage, are presented in Table 2. The evaluated parameters were the histologic overview of the adipoaspirate with regard to the preservation of lipocyte morphology (no evidence of cell membrane rupture) and the immunohistologically imaged density of truncated blood capillaries and lymph vessels in the tissue preparations. The results were classified semiquantitatively as 0 = not detected, ((+)) = detected in very low levels, (+) = detected in low levels, + = detected, and ++ = detected in high levels.

Slides for evaluation were prepared for all patients. The lipocytes were contained in larger connected complexes (Figs. 4 and 5) or in dispersion to smaller aggregates and single cells (Fig. 6), some accompanied by strands of collagen-fibrous connective tissue (Fig. 5). Dispersion to single cells was generally correlated with a greater degree of single-cell damage in the form of membrane rupture (Figs. 6 and 7). Focal bleeding was found in one case (Fig. 8).

Lipocytes Predominantly Intact

In 28 of the 30 investigated lipoaspirates (patients), the lipocytes were found to be predominantly (> 70%) intact. Two of the 30 investigated adipoaspirates contained, for the most part, separately dissociated adipose cells with distinct signs of destruction and collapse of the cell membrane (Figs. 6 and 7). Immunohistologically, all specimens were shown to contain blood vessels in the images with anti-CD31 antibody (Figs. 7, 9–11). These vessels primarily had the smallest capillary caliber with an average diameter of 0.05-0.1 mm (Figs. 10 and 11). Pieces of venules were found in isolated cases. The number or density of the blood vessel structures ranged from 3 to 20 per microscopic field of vision at medium magnification.

In contrast with the CD31-immunostained vascular images, negative staining results were obtained for the lymphatic endothelial cell marker D2-40 (Figs. 12 and 13). In the sections stained with D2-40, antibody lymph vessels with collapsed walls were found in only two cases: in case 6 focally (Fig. 14) and in case 23, detectable in very low levels (Fig. 15). No intact lymph vessels were detected.

Discussion

The lipoaspirate obtained through liposuction consists of a mixture of subcutaneous tissue components. In the context of tumescent local anesthesia, after initial suprafascial

Table 2 Lipoedema: semiquantitative analysis

No.	Patient No.	Age (years)	Clinical stage of lipoedema	Histology: lipocyte fragments	Immunohistology	
					Blood vessel endothelium = CD31	Lymph vessel endothelium = D2-40
01	27235	40	III/1	+	+	0
02	27390	34	III/1	+	+	0
03	27215	25	II/1	+	+	0
04	27319	38	III/1	(+)	+	0
05	27409	36	III/1	+	+	0
06	27463	41	III/1	++	++	(+)
07	27250	36	II/1	+	+	0
08	19679	28	III/1	+	+	0
09	21279	34	III/1	(+)	(+)	0
10	27545	23	III/1	+	+	0
11	26165	63	III/2	(+)	+	0
12	26500	38	II/1	+	+	0
13	27319	38	III/1	+	(+)	0
14	10865	36	III/1	++	+	0
15	26951	24	III/1	+	+	0
16	27627	37	III/2	(+)	+	0
17	27863	21	III/1	+	+	0
18	27928	38	III/1	(+)	+	0
19	27925	21	III/1	+	+	0
20	27810	23	III/1	+	+	0
21	27903	24	III/1	+	+	0
22	27187	42	III/1	++	+	0
23	10865	36	III/1	+	+	((+))
24	27010	40	III/1	+	+	0
25	28054	22	III/1	(+)	+	0
26	27600	33	III/1	+	+	0
27	28251	39	III/1	+	+	0
28	27296	32	III	(+)	(+)	0
29	28137	36	II/1-2	+	+	0
30	27491	28	III/1	(+)	+	0

0 = not detected; ((+)) = detected in very low levels; (+) = detected in low levels; + = detected; ++ = detected in high levels

hydrodissection, a perilobular infiltration of the adipose tissue lobules is performed, followed by the desired intralobular infiltration. The three-dimensional expansion of the subcutaneous space allows the aspiration of adipose tissues with reduced shearing force, therefore minimizing injury to blood and lymph vessels [10, 12]. In vibration-assisted liposuction, the isolation of adipose cells from the tissue aggregate occurs as a result of the differences in the moments of inertia of the adipose and connective tissues. With the water jet-assisted liposuction method (WAL), adipose cells are mobilized in a comparable manner without causing injury to the vessels. Preservation of the collagen-fibrous septal connective tissue framework creates optimal conditions for postoperative recovery with fibrous tissue retraction [10]. The connective tissue framework also provides channels for both the blood and lymph capillaries. Previous histologic analyses of lipoaspirates were performed primarily to investigate adipose cell integrity [28].

Vessel parts show up as fragments in lipoaspirates, which complicates the task of identifying them reliably using histomorphologic methods. Lymph capillaries have much thinner walls than blood capillaries and are more difficult to identify in tissue sections. Therefore, the special problems presented by the identification of the fragile lymph vessel parts in lipoaspirates and their differentiation from blood capillaries could be anticipated. This challenge can be overcome with immunohistochemical techniques that allow an extremely specific (color) marking of special tissue components.

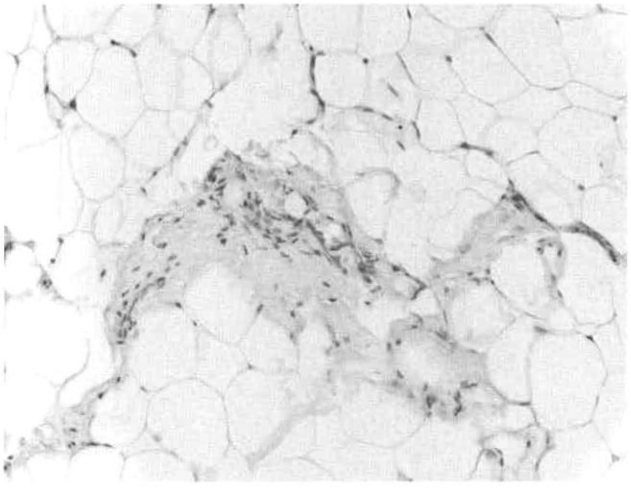

Fig. 5 Lipocyte complexes with strands of collagen-fibrous connective tissue

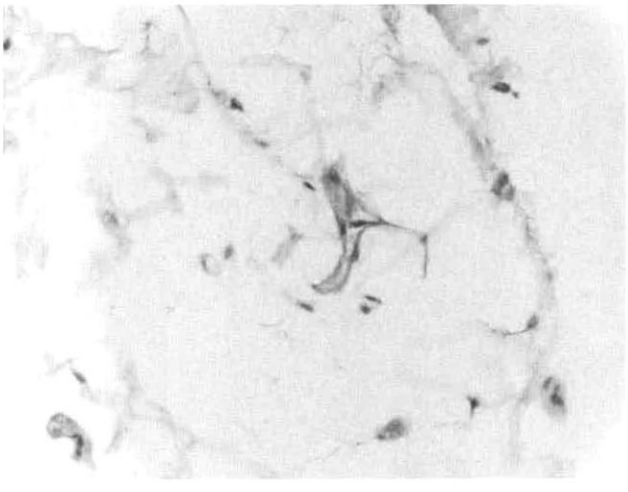

Fig. 6 Lipocytes, dispersion to smaller aggregates and single cells

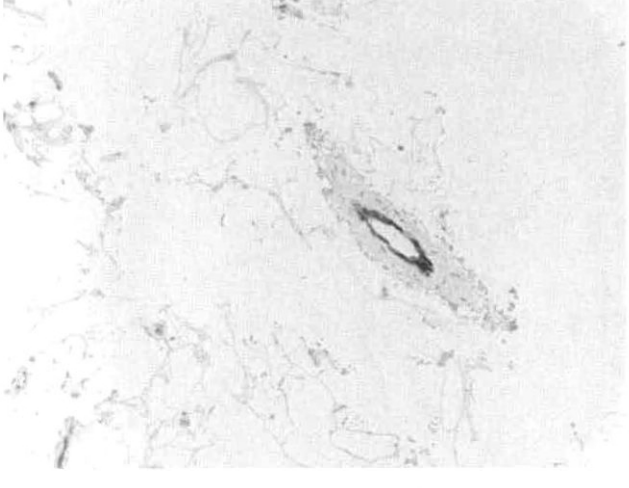

Fig. 7 Blood vessels with anti-CD31 antibody

Fig. 8 Focal bleeding in one case

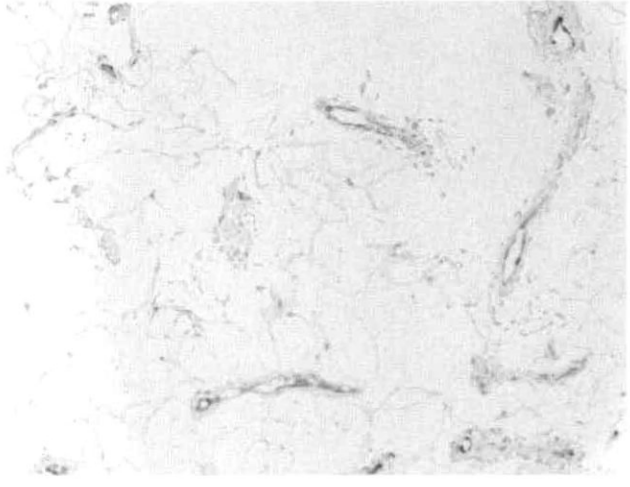

Fig. 9 Blood vessels with anti-CD31 antibody

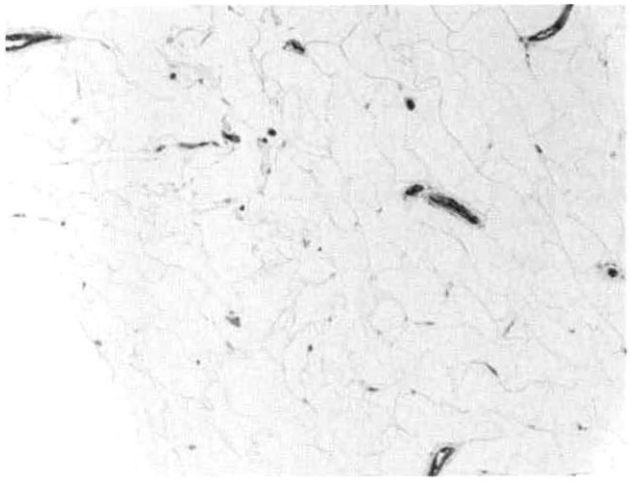

Fig. 10 Blood vessels with anti-CD31 antibody

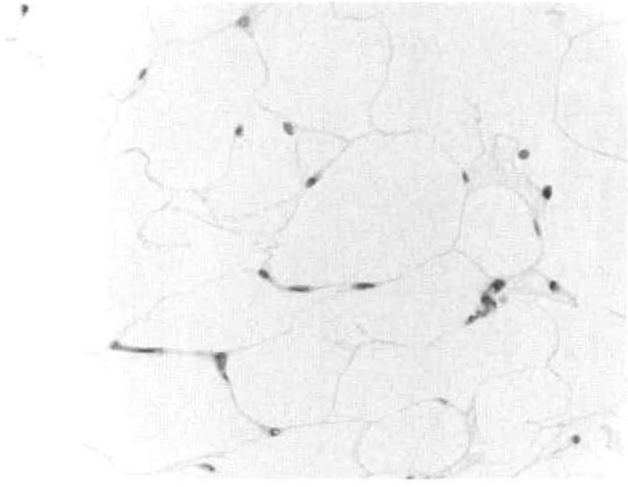

Fig. 11 Blood vessels with anti-CD31 antibody

Fig. 12 Negative staining results for the lymphatic endothelial cell marker D2-40

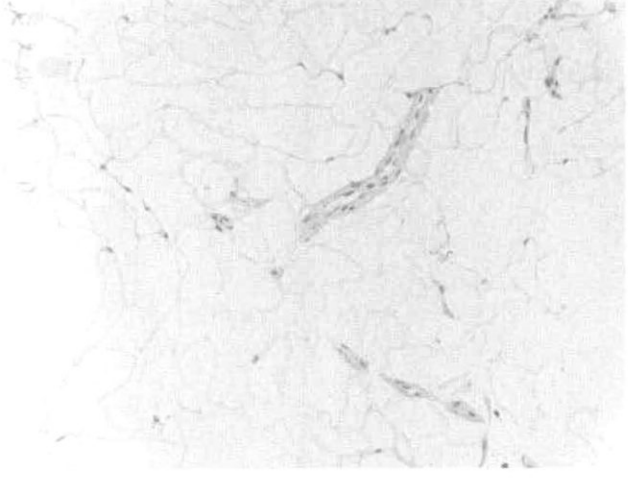

Fig. 13 Negative staining results for the lymphatic endothelial cell marker D2-40

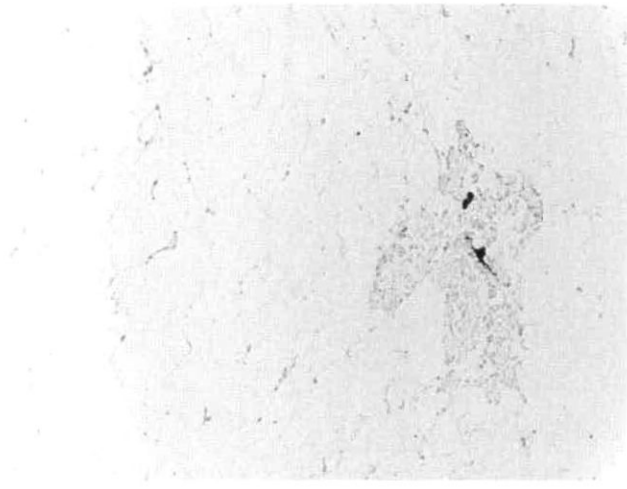

Fig. 14 Lymph vessels with collapsed walls (case 6 focally)

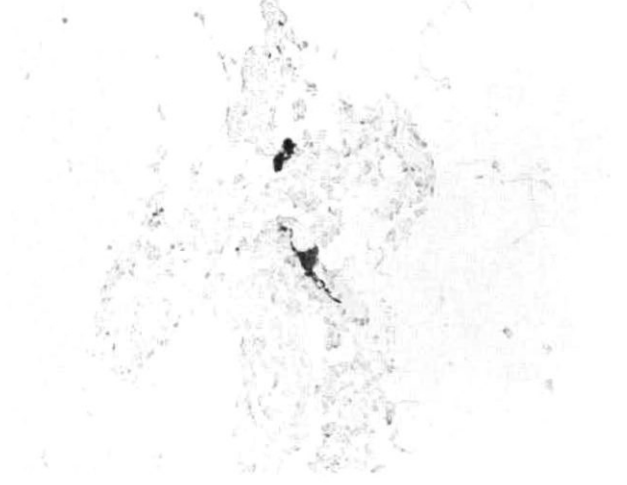

Fig. 15 Lymph vessels with collapsed walls (case 23, detectable in very low levels)

Antibodies to smooth muscle actin, among others, can be used as immunohistologic markers for blood vessels. CD31, on the other hand, is a marker for blood vessel endothelium which does not stain the endothelial lining of lymph capillaries.

The selective representation of lymph vessels has only recently become possible with the introduction of new markers such as the monoclonal antibody D2-40 [29]. Since then, additional, in part closely related antigens have been described as characteristic for lymph vessels and the corresponding antibodies (e.g., anti-podoplanin) introduced for histopathologic diagnostics. The use of the selective lymph vessel marker D2-40 makes it possible to assess whether and to what extent lymph vessels, in complete or fragmented form, are present in the adipoaspirates of patients with lipoedema. A parallel staining with the anti-CD31 antibody, on the other hand, identifies blood-carrying vessels. This method of immunohistologic investigation has already been

demonstrated using histologic preparations from five female patients who had been treated with anatomically appropriate vibration liposuction applied along the longitudinal axis of the extremity under tumescent local anesthesia [15]. According to these results, lymph vessels are practically undetectable in adipoaspirates, while blood capillaries are always present. From these findings it can be concluded that the operative trauma from liposuction causes no relevant damage through the destruction or mobilization of lymph capillaries. This evidence is crucial for the further methodology, practice, and development of liposuction because lymph vessels represent an especially vulnerable and exposed structure in the typical operation site of lipoedema patients.

Summary

Our research has confirmed on a larger number of patients that to a large extent damage to the lymph vessels can be avoided with the use of water jet-assisted liposuction and that this treatment method can produce results that are methodologically equivalent to the tumescence method [15]. The adipose tissue present in variously sized fragments consisted primarily of intact, single cells and smaller aggregates of adipocytes which, for the most part, had morphologically survived the mechanical operative stress. Blood capillaries were consistently detected in moderate quantities per visual field by means of CD31 expression. These vessels were equivalent to blood capillaries in routine staining with hematoxylin & eosin, in part with luminal erythrocytes. Lymph vessels stained with D2-40 were found in only 2 of the 30 cases in our study, and in very small amounts (Table 2). In summary, limited histomorphologic traces of the traumatization of adipose cells and blood capillaries were found in the histologic slides with almost no histologic correlate for lymph vessel injury.

The results suggest that WAL, when applied using anatomically appropriate techniques (working strictly along the longitudinal axis of the extremity), represents a method of treatment without substantially traumatizing the lymphatic vascular system. In comparison with the tumescence method, there are no side effects associated with the water jet method.

Conclusion

The atraumatic, anatomically appropriate procedure of water jet-assisted liposuction (WAL; body-jet®) available today represents a promising treatment for lipoedema patients who generally suffer from severe subjective and objective impairment. Liposuction treatment can bring long-term improvement if the operative technique focuses on lymph vessel preservation. Immunohistologic analyses show minimal evidence of lymph vessel structures in lipoaspirates. The histologic analysis of the aspirates documents a relatively specific removal ("apheresis") of primarily intact lipocytes with low vascular amount. The analysis of liposuction aspirates from 60 lower extremities obtained from the inner knee area, which represents an especially high-risk region for this type of operation, showed that only minimal or no injury was done to the lymph vessels, if the liposuction procedure was performed strictly parallel to the axis of the lymph collectors.

The immunohistochemical evaluation also confirmed the assumption that a state of tumescence is not required for the WAL procedure thus preserving the structural integrity of lymph vessels. It was also proven that when the WAL technique is used, the preinfiltration period for the tumescent fluid did not have to be observed.

A paradigm shift has thus occurred with the introduction of water jet-assisted liposuction. For this method no tumescence (firm-elastic infiltration condition with high tissue pressure) is necessary. Likewise, no preinfiltration period for the homogenization of the adipose tissue is required. The aspiration procedure is started immediately after the anesthesia has taken effect.

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APPENDIX E

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March 27, 2015

PECO
Brenda Eison, Supervisor
2301 Market St.
Philadelphia, PA 19103

Re: Acct # 234-690-1005
1191 Telegraph Rd., West Chester, PA 19382
Medical Necessity To Keep Electricity On

Dear Ms. Eison,

I understand that PECO will shut off electricity on April 3, 2015 unless a new smart meter is installed on the above mentioned property, to which Laura and John Murphy, the owners, are opposed. I have no interest in the outcome of this dispute but it is an unequivocal medical necessity for electric service to be maintained without interruption.

Both the owners are elderly and in fragile health for a number of reasons which is documented extensively by a number of health care providers. Their water comes from a well and is dependent on electricity. Any unecessary or prolonged interruption in electric service would seriously jeopardize their health.

Your attention, understanding and courtesy in resolving this important health issue is greatly appreciated.

Sincerely,

Peter J Prociuk MD

APPENDIX F

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-1: With regard to the AMR meter currently installed at the Murphy residence ("Murphy AMR Meter"),

a. When was the Murphy AMR Meter installed?
b. What is the make, model and specifications of the Murphy AMR Meter?
c. How many times per day does it transmit remotely?
d. How long is each transmission?
e. How far does it transmit remotely?
f. What are its peak emissions?
g. Does it transmit EMF to the electric wires inside the Murphy household?

PECO Answer to Murphy I-1:

a. May 8, 2002
b. Siemens/L+G/Duncan electro-mechanical meter with a Landis + Gyr AMR Communications Transmit Only Meter Module (TOMM)
c. 288 times per day for its scheduled transmissions (every 5 minutes)
d. 20 milliseconds. Total daily on-air time transmitting is 5.76 seconds
e. The expected useful distance is less than 1 mile
f. 1 watt or 33 dBM
g. No transmissions are made to communicate with facilities, devices, or wires inside the household

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-2: Does the Murphy AMR Meter transmit to each of the Murphy electrically powered devices, such as the HVAC system, computers, lamps, refrigerator, etc. through the airwaves or through the household wiring?

PECO Answer to Murphy I-2:

The Murphy AMR meter does not transmit to electrically powered devices within the residence.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-3: Are the transmissions from the Murphy AMR Meter sent wirelessly or via wire?

PECO Answer to Murphy I-3:

The transmissions from the Murphy AMR meter are sent wirelessly.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-4: How often does PECO collect data emitted from the Murphy AMR Meter?

PECO Answer to Murphy I-4:

Data is transmitted from the Murphy AMR meter every five minutes. The data is compiled at an intermediate facility and then delivered to PECO electronically once a day.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to
Interrogatories Complainant Laura
Sunstein Murphy, Set I

Murphy I-5: How is the Murphy AMR Meter usage data read by PECO

a. For billing purposes?
b. For outage purposes?
c. For any other purpose?
d. Name any and all other purposes.

PECO Answer to Murphy I-5:

a. Yes, the AMR meter usage data read by PECO is used for billing purposes
b. Yes, the AMR meter usage data read by PECO is used for outage purposes
c. Yes, the AMR meter usage data read by PECO is used for other purposes
d. Primarily theft detection and related analytics.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-6: When was the last time the Murphy AMR Meter was checked by PECO on site?

PECO Answer to Murphy I-6:

The Murphy AMR meter was tested for accuracy on April 15, 2002, prior to installation. PECO has not identified any onsite checks.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-7: For what purpose was the Murphy AMR Meter checked on site by PECO?

PECO Answer to Murphy I-7:

For accuracy. See Answer to Murphy I-6.

Responsible Witness: Glenn Pritchard

PECO Energy Company's Answers to Interrogatories Complainant Laura Sunstein Murphy, Set I

Murphy I-8: Is the Murphy AMR Meter able to be reprogrammed? If yes, what possible changes can be made to the Murphy AMR Meter programming as to its emissions?

PECO Answer to Murphy I-8:

No. It is not possible to reprogram the Murphy AMR meter to change the number of transmissions made by its communication module.

Responsible Witness: Glenn Pritchard