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United States District Court

District of Oregon

Portland Division

AHM, by and through
her Guardian *ad litem* and father,
David Mark Morrison**,** and
David Mark Morrison, individually,

v.

Portland Public Schools,

Defendant.

Civil Action No. 3:11-cv-00739-MO

Declaration of
L. Lloyd Morgan
Addendum H – Poster
Incidence Rate Model

A Model to Predict Future Brain Tumor Incidence Increased Resulting from Mobile Phone Use

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Abstract

If cellphone radiation is causing brain cancer there are two possibilities: DNA repair is reduced, or DNA damage is increased. Using *age-adjusted* brain cancer incidence, model parameters are adjusted to obtain a best fit to the data. If these parameters reasonably fit, *age-specific* brain cancer data, then the model can be used to predict brain cancer incidence increase. For Nordic and USA countries we predict a 2-fold incidence increase, if DNA repair is decreased, or more than a 6-fold incidence increase , if DNA damage is increased.

Background

Case-control studies have found for users with the highest cumulative hours of use and/or for >10 years of cellphone use, risks for brain cancer (OR range: 1.6-2.6) [1-3]. We examined brain cancer incidence rates in Nordic countries and the USA. Sparsely populated regions in Sweden have shown higher incidence increase over time compared with more densely populated areas [4]. Since the beginning, age-adjusted brain cancer incidence rates have been increasing with two exceptions: the USA, and Sweden. "Rates" are expressed in brain cancer cases per 100,000 person-years.

Objectives

The first objective was to model brain cancer trends by extracting characteristic risk functions from data reported since data collection began and to use these for projecting future trends resulting from mobile phone use. We model two possibilities: decreased DNA repair and increased DNA damage. A second objective was to investigate recent incidence trends in rural areas.

Methods

Normally occurring DNA brain cell damage during one year was assumed to be associated with a cancer risk function that is increasing over time but normally balanced by a DNA repair function reducing the amount of potential carcinogenic damage over time. By adjustment of model parameters to a brain cancer risk function to obtain a best fit to age-adjusted brain cancer data, we can calculate age-adjusted brain cancer incidence rates into the future, as has been done previously for melanoma [5].

The model can account for increasing levels of initial DNA brain damage or decreasing DNA repair efficiency, caused by mobile phone use. If the model parameters result in an accurate comparison of age-specific brain cancer incidence to published data, then the model can be assumed to be a reasonable predictor of future age-adjusted brain cancer incidence. The incidence change over time in all 21 Swedish counties was analyzed vs. population density to see if the higher output power from mobile phones in rural areas still correlates with brain cancer incidence as it earlier has been reported [4].

Results

DNA brain cell damage has a long latency time requiring around 20+ years before the first 10 % of latent brain cancer cases would be detected. If mobile phones can *reduce the DNA repair efficiency* it may result in about a *2-fold increase in brain cancer* incidence by 2042. If mobile phone use also can generate *increased DNA brain damage*, there would be about a *6-fold increase in brain cancer* incidence, which could eventually result in a *9-fold increase in the 65-69 age cohort* by 2042. Brain cancer rates have been decreasing since 1990 in the USA, and since 1984 in Sweden. Since 1985 Finland's increasing rate has slowed. Norway's rate increased further since 1993. Denmark's rate has been steadily increasing since 1955.

Figure 1 shows age-adjusted brain cancer incidence rates for Denmark, Norway, Finland, Sweden and the USA. With each country, linear trend lines are shown for two ranges of years. **Table 1** summarizes what is seen in Figure 1. It shows the percentage change in these incidence rates, the associated R^2 correlation factor and the resultant p-value for each linear trend line.

We chose Norway's data to illustrate the general picture of what an increase in the age-adjusted brain cancer incidence rate after mobile phone use became common may portend.

Figure 2A shows the published age-adjusted brain cancer rate in Norway along with what the model predicts for 3 assumptions: 1) Mobile phone use has no effect on brain cancer rate; 2) mobile phone use decreases the DNA repair efficiency, and; 3) mobile phone use increases DNA damage. If there is no effect, then the rate would reach steady-state of 11 brain cancers per 100k person-years. Since the rate is already 27% higher, this model suggests there is an effect from mobile phone use. By 2042 the rate could reach 90 (a 6.4-fold increase), if increased DNA damage from mobile use is the mechanism. If the mechanism is reduced DNA repair, by 2042, the rate could reach 27 (a 1.9-fold increase).

Figure 2B shows 3 age-specific incidence rates in Norway (30-34 years, 45-49 years, and 65-69 years). It also shows the model data for two cases: mobile phone use increases DNA damage, and mobile phone use decreases the DNA repair efficiency. For the 30-34 age cohort, the rate was 11 in 2007. If mobile phone use increases the DNA damage the rate could increase to 17 by 2012 (a 1.5 fold increase), and could be stable thereafter. If mobile phone use decreases the DNA repair efficiency, the rate could increase to 9.1 and be stable thereafter. The reason the 30-34 age cohort's projected increase levels off is that this cohort has reached equilibrium because they would have been exposed similarly for all of the lives. A similar result would be true for all age cohorts.

For the 45-49 age cohort the rate was 18 in 2007. This could increase to 41 (a 2.3-fold increase) for a decreased DNA repair efficiency. For increased DNA damage it could increase to 125 (a 6.9 fold increase).

In 2007, the 65-69 year cohort's rate was 51. With increase DNA damage it could increase to 466 by 2042 and could still be rising (a 9.1-fold increase). With reduced DNA repair efficiency the rate could increase to 130 by 2042 (a 2.6-fold increase).

Discussion

The model used was based on the log-normal distribution that is determined by two parameters. One is the time to 50%, the median time, and the other is the spread of the distribution, here called dispersion. As we deal with a logarithmic time scale the dispersion is measured in time decades instead of linear time as used in the normal distribution. And by a mathematical transform we characterize the placement of the distribution in the time domain by the time to 0.1 % instead of time to 50 %. Further details on this method can be found in Hallberg [5].

The model is based on one distribution that is describing the risk of getting brain cancer over time due to cell damage acquired during one year of living. Another distribution is used to characterize the probability that such damages are repaired over time and no longer represent a risk of brain cancer.

The earliest age-adjusted brain cancer incidence rate comes from Denmark. From 1943-1955 the rate was increase a modest 0.3% per year. After 1955 the model assumes that the repair efficiency decreased somewhat from that point in time and onwards. By parameter variation a best fit was found for the combined risk function of increased DNA damage and decreased DNA repair efficiency to the reported age-adjusted brain cancer incidence rates up to somewhere between 1984 and 1993 for all countries examined, excepting Denmark.

For Norway it appears that the rate started to increase after 1993, while for Finland the rate of increase slowed from 1985. For Sweden, beginning in 1984, and for the United States beginning in 1990, the rate decreased (**Figure 1**).

In order to account for the increasing use of mobile phones we tested two hypotheses, which might explain the increasing rates in Norway. If the remaining repair capability since 1955 was further reduced this could explain the increasing rates since 1993 pointing to an approximate doubling in age-adjusted rates. If the increasing use of mobile phone increased DNA brain damage, the increase would require a few more years after 2017 to become obvious (**Figure 2B**). Our model, then predicts the age-adjusted brain cancer incidence would increase by factor of about 5.4 by 2042 (**Figure 2A**).

We can only speculate about the reasons behind the different trends noticed in different countries during the last decade. We can still see that rural areas in Sweden have rates that are increasing more than in urban areas, which might suggest that the average output power from the mobile handset is an important factor [4]. The fact that Sweden, and the USA now show decreasing rates is interesting, but we need to understand why?

Perhaps the immune systems is showing an "adaptive response" as reported by Sannino et al. [6] from exposure to low levels of mobile phone radiation. If so, there is the possibility that this immune response will eventually be overwhelmed by ever increasing exposures, resulting in a sharp increase in brain cancer similar to what is being seen in melanoma incidence. Today melanoma incidence is drastically increasing, especially melanoma on the skin of the head.

Age-adjusted brain cancer rates have been increasing since data collection first began in the Nordic countries (Figure 1). The longest time trend began in 1943 in Denmark, and it indicated that the increasing incidence started to increase further from 1955 onwards. Sweden and the USA show decreased rates beginning about 1984 in Sweden and about 1990 in the USA. Finland's increased rate slowed somewhat from about 1985. Denmark's rate has increased steadily at 1.4% per year since 1955. Norway's rate had increased 1.4 % per year until about 1993. Afterwards its rate nearly doubled to 2.7% per year.

These results are summarized in **Table 1**.

For our model we have chosen to use brain tumor incidence in Norway. Model parameters were determined that resulted in a best fit to the Norwegian age-adjusted brain cancer incidence rate. **Figure 2A** shows the published data and the model data for the 3 possibilities, no effect at all, reduced DNA repair efficiencies and increased DNA damage. **Figure 2B** shows selected age-specific incidence rates (30-34 years, 45-49 and 65-69 years) and model data for the two latter possibilities.

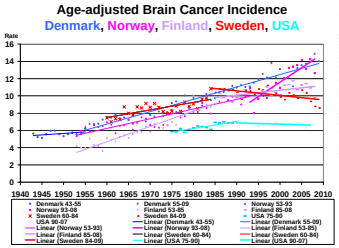

Figure 1. Incidence trends of brain and nerve tumors in the USA and the Nordic countries.

Conclusions

Our model suggests that a substantial increase in brain cancer may occur in the future. The true test of any model is: *does it make reasonably accurate predictions?* For our model this increase would occur around 2020-2030.

In summary, we conclude that no dramatic increase in age-adjusted brain cancer incidence rates have been seen *yet* in the USA or in the Nordic countries. The increasing rates of brain cancer in Norway might be seen as a warning sign. Monitoring brain cancer incidence rates is of great importance.

However, because the reporting of this data is delayed by several years, it cannot serve as an early warning system. In order to be an early warning system, cancer registries will need to improve the timeliness of their data reporting. Given the consequences this model predicts, public health contingency planning is reasonable. An obvious result of such contingency planning, given the implications of this model, would predict a dire shortage of neurosurgeons with a resultant increase in brain tumor mortality.

References

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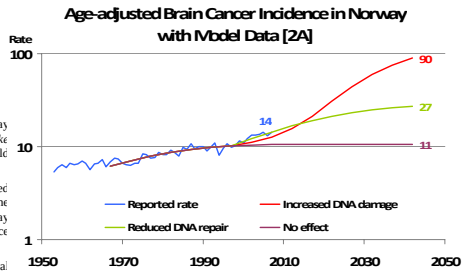
Country	Range of Years	Annual Rate Change	R^2	p-value
Denmark	1943-1955	0.3%	0.8162	0.070
	1955-2009	1.4%	0.9287	10^{-6}
Norway	1953-1993	1.4%	0.8119	10^{-6}
	1993-2008	2.7%	0.7445	10^{-6}
Finland	1953-1985	2.9%	0.8769	10^{-6}
	1985-2008	0.67%	0.3975	4.8×10^{-4}
Sweden	1960-1984	1.0%	0.5693	10^{-6}
	1984-2009	-0.53%	0.3839	3.7×10^{-4}
USA	1975-1990	1.3%	0.8124	10^{-6}
	1990-2007	-0.2%	0.2418	0.019

Table 1. Age-adjusted brain cancer incidence rate changes by country from Figure 1



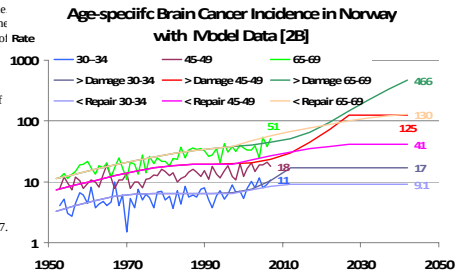


Figure 2A. Age-adjusted brain cancer incidence rates in Norway and calculated predictions based on increased DNA damage and reduced DNA repair Efficiency.

Figure 2B. Reported and calculated rates of brain tumors in Norway for the age groups 30-34 years, 45-49, and 65-69 years.