Cell Phones and Brain Cancer

Gedaliah Farber[1], Kevin Leung[2], James Gomes[3],

[1]*Undergraduate Research Opportunities Program, Faculty of Health Science, University of Ottawa, Ottawa, ON, Canada,* [2]*Undergraduate Faculty of Health Science, University of Ottawa , Ottawa, ON, Canada,* [3]*Faculty of Health Science, University of Ottawa, Ottawa, ON, Canada*

Abstract

To date there are over 7 billion cell phone subscriptions worldwide and society's overall, collective, usage of these devices is increasing on a daily basis. Cell phones, communication towers and other communication devices emit electromagnetic fields (EMF). It is believed that prolonged exposure to EMFs may play a role in adverse health effects including tumour formation in the brain. The goal of this design is to gain a better understanding of the tumorigenesis in the brain from exposure to EMF in a dose-dependent manner. A review of the most current epidemiological literature appears to indicate that the most affected regions of the brain are those located ipsilaterally to the cell phones placement against the head. To study the effects of EMF and their role in tumorigenesis, rat astrocytes will be cultured *in vitro* and exposed to EMF in a dose dependent manner. RNA will be extracted from the exposed cells and expression of key genes associated with brain tumorigenesis will be assessed by RT-PCR techniques. The genes of interest in this study are MGMT, IDH1, Glyceraldehyde-3-phosphate dehydrogenase (GAPDH), BAX, HSP27, and B-actin and their role in apoptotic inhibitory pathways.

Introduction

- Cell phones are now being introduced to children at exceedingly younger ages and the long term effects of cell phone EMF exposure is not well understood.
- This research is of particular interest since the IARC, WHO has identified cell phones as potential carcinogens as they emit electromagnetic fields.

Magnetic and electric waves produced by current flowing through a conductor combine to create an electromagnetic field (EMF).

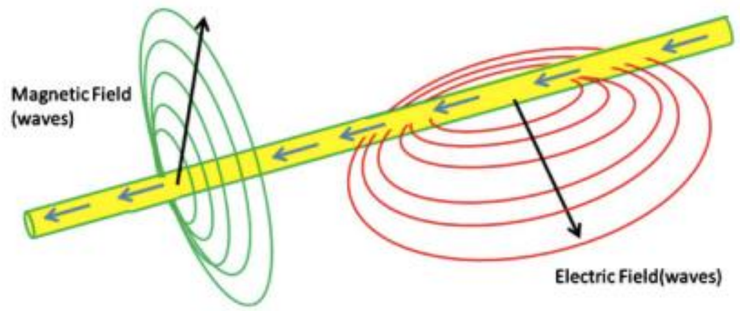

Figure 1:This diagram shows the formation of the Electric current flowing through a conductor, by either an antenna or a circuit inside the cell phone to generate both magnetic and electric fields. These fields consist of oscillating magnetic and electric waves which combine to form the EMF that is then released by the cell phone.

Aims and Objectives

- The purpose of this research is to determine all adverse health effects of exposure to EMF from cell phones and specifically the development of brain tumours.
- The objectives of this study was to identify the genes that are either silenced or expressed when exposed to EMF and to comprehend the mechanisms of tumourigenesis.
- The genes that play a role in the carcinogenesis of the brain will be assessed for expression and methylation status of the promoter region in DNA obtained from cells exposed in-vitro to EMF from cell phones.
- The cells (astrocytes) can then be exposed to radiation via a cell phone in a dose dependent manner, for a specific period of time and then be analyzed for tumour formation.

Methodology

- The most recent epidemiological studies were identified from databases such as PubMed, Scopus, and Ovid. The search criteria included terms such as electromagnetic fields (EMF) and electromagnetic radiation.
- The search was refined by adding brain terms such as astrocytes and glial cells and tumour related search terms such as glioblastoma. This search criteria resulted in a manageable amount of research literature that was reviewed for relevance by reading the title and the abstract.
- After excluding the non-relevant studies a total of xx studies relevant to this research were selected for data extraction.
- In the data extraction process information on the purpose of the study, population studied, methods used, samples collected and measurements made and results obtained was collected. Statistical details on risk, hazard and other factors was also collected. Information on the conclusions reported and inferences made was also recorded.
- Information on all genes of interest was collected and genes MGMT, GAPDH, BAX, B-Actin, IDH1, and JNK were identified as of interest for their role in the tumourigenesis of brain cells.

Results

Table 2 Summary of high grade glioma risk in epidemiological studies to date

Variable	Histology	Cell phone type	Hours of exposure	Latency (years)	Number of cases/ controls	OR	95% CI
Hardell (2006)	Astrocytoma	Digital	≤64	1–5	34/139	1.7	0.96–2.9
	Astrocytoma	Digital	>64	1–5	22/75	**2.1**	**1.05–4.1**
	Astrocytoma	Digital	≤64	5–10	18/44	**2.4**	**1.2–4.8**
	Astrocytoma	Digital	>64	5–10	40/67	**3.3**	**1.7–6.4**
	Astrocytoma	Analog	≤80	5–10	6/24	1.4	0.5–4.0
	Astrocytoma	Analog	>80	5–10	8/12	**3.9**	**1.3–12**
	Astrocytoma	Digital	≤64	≥10	0/0	–	–
	Astrocytoma	Digital	>64	≥10	15/18	**4.5**	**2.0–10**
	Astrocytoma	Analog	≤80	≥10	6/13	**3.2**	**1.05–9.6**
	Astrocytoma	Analog	>80	≥10	32/27	**7.4**	**3.4–16**
Hardell (2006b)	Astrocytoma	Digital	≤64	1–5	90/349	1.4	1.01–1.9
	Astrocytoma	Digital	>64	1–5	53/235	1.2	0.8–1.7
	Astrocytoma	Analog	≤85	1–5	13/67	1.0	0.5–1.9
	Astrocytoma	Analog	>85	1–5	8/19	1.9	0.8–4.7
	Astrocytoma	Digital	≤64	5–10	22/70	1.6	0.9–2.8
	Astrocytoma	Digital	>64	5–10	64/107	**2.9**	**1.9–4.4**
	Astrocytoma	Analog	≤85	5–10	22/63	1.6	0.96–2.8
	Astrocytoma	Analog	>85	5–10	13/64	1.0	0.5–1.9
	Astrocytoma	Digital	≤64	≥10	0/0	–	–
	Astrocytoma	Digital	>64	≥10	15/18	**3.8**	**1.8–8.1**
	Astrocytoma	Analog	≤85	≥10	8/26	1.4	0.6–3.3
	Astrocytoma	Analog	>85	≥10	51/58	**3.7**	**2.3–5.9**
Shuz (2006)	Gliomas (males)	Both	Regular use[a]	–	76/170	0.78	0.53–1.14
	Gliomas (females)	Both	Regular use	–	30/38	**1.96**	**1.10–3.50**
Lonn (2005)	Glioma III–IV	Both	Regular use	<5	83/213	0.9	0.7–1.4
	Glioma III–IV	Both	Regular use	5–9	55/139	0.8	0.5–1.2
	Glioma III–IV	Both	Regular use	≥10	16/38	0.8	0.4–1.5
	Glioblastoma	Both	Regular use	<5	50/213	0.9	0.6–1.3
	Glioblastoma	Both	Regular use	5–9	35/139	0.8	0.5–1.2
	Glioblastoma	Both	Regular use	≥10	9/38	0.7	0.3–1.6
Lakhola (2007)	Glioblastoma	Both	Regular use	<10	304/1633	0.75	0.61–0.92
	Glioblastoma	Both	≤75	≥10	25/111	0.66	0.41–1.07
	Glioblastoma	Both	>75	≥10	32/105	0.93	0.34–1.01
Christensen (2005)	Gliomas	Both	–	1–4	24/66	0.59	0.43–1.75
	Gliomas	Both	–	≥5	34/88	**0.55**	**0.32–0.96**
	Gliomas	Both	–	5–9	26/66	0.57	0.32–1.02
	Gliomas	Both		≥10	8/22	0.48	0.19–1.26

[a] Regular use is defined as at least one incoming or outgoing call per week for at least 6 months

(–) Dash denotes value not indicated in original report

Numbers in *bold* are statistically significant

Figure 2: This table is a summary of hish grade glioma cases presented in epidemiological research up to 2012. It includes; the histology, type of cell phone, exposure time (in hours), latency (in years), number of controls, and the statistical significance (as ndicated by the bold text).

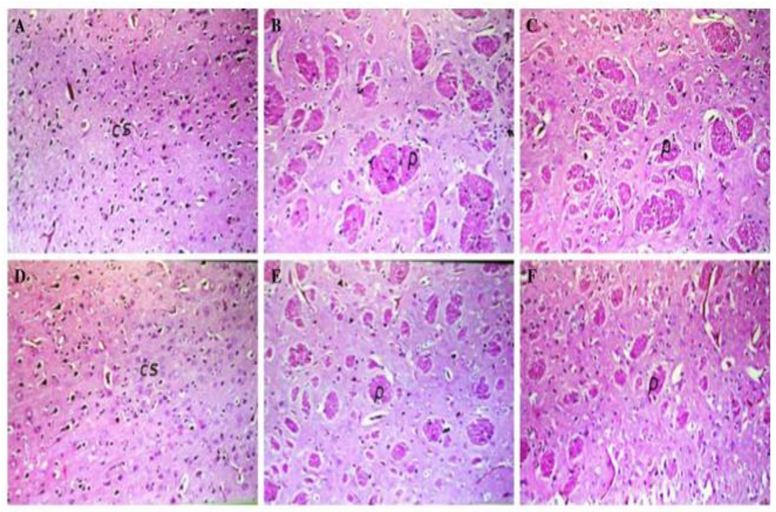

Figure 3: Microscopic representation of brain cells in young adult rats after samples were satined with hematoxylin and eosin 940. Samples a) and d) represent the "Young Control" and "Adult Control" groups respectively and show normal cerebral histological structure. Samples b) and c) represent the "Young Exposed" and "Activated Exposed" groups respectively and a number of plaque (P) formations are visible. Sample e), "Adult Exposed" and f) "Adult Activated" groups show multiple number of plaques and focal plaque formation respectively.

Tissue Culture

For this study astrocytes were obtained from rat brains and cultured in-vitro. Once cells were roughly (80%) confluent (approximately 1 million cells) they were separated into two separate samples and allowed to continuously grow is an incubation chamber set to 37°C. Cells were grown in the same fashion until a sufficient number was achieved to carry out the rest of the procedure and expose cell cultures to the cell phone radiation. These cells, when confluent, were exposed to EMF from cell-phones in a dose-dependent manner.
DNA and RNA was extracted from the exposed cells to assess gene expression and DNA methylation.

RT-PCR

Primers were designed for expression and DNA methylation for the genes of interest for use in RT-PCR analyses for expression and DNA methylation.
DNA primers for the selected genes in this study were designed using software from http://www.ncbi.nlm.nih.gov/ a sequence was selected to design and order a primer to replicate the genes of interest. Selection of the primer was conducted using another protocol which limited the number of secondary structures, CG%, and limit the number/type of nucleotides in hairpin structure which would form during RT-PCR and yield the most success. After the primer was selected, the CpG island was located as to identify the region of methylation for the particular gene of study.

Rattus norvegicus O-6-methylguanine-DNA methyltransferase (Mgmt), mRNA

NCBI Reference Sequence: NM_012861.1

GenBank Graphics

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>gi|6981201|ref|NM_012861.1| Rattus norvegicus O-6-methylguanine-DNA
methyltransferase (Mgmt), mRNA
CTCAGTTCCAGAGCCTGTGTACCGTTCCAGGTTACGGAGTTTCTGCACAGTTTGCAAACTGGAACTTGGC
AGAATGGCTGAGATCTGCAAAATGAAATACACGGTGTTGGACAGCCCTTTGGGGAAGATAGAGCTGTCCG
GCTGTGAGCGAGGCCTGCATGGGATACGATTTCTCAGTGGGAAGACCCCAAACACTGACCCCACAGAGGC
TCCAGCCTGTCCTGAGGTGCTCGGTGGGCCAGAGGGAGTGCCAGAGCCCCTGGTGCAGTGCACAGCCTGG
CTGGAAGCCTATTTCCACGAACCTGCAGCCACAGAGGGGCTTCCCTTGCCTGCTCTCCATCACCCTGTGT
TCCAGCAAGATTCATTCACCAGACAGGTGTTATGGAAGCTGCTGAAGGTTGTGAAATTCGGAGAAATGGT
TTCTTACCAGCAATTAGCAGCCCTGGCAGGCAACCCCAAAGCGGCTCGTGCAGTAGGAGGAGCGATGAGG
AGCAATCCAGTCCCCATCCTCATCCCCTGCCACAGGGTGATTCGCAGTGACGGTGCCATTGGCAATTACT
CTGGAGGAGGACAGACTGTGAAAGAGTGGCTTCTGGCCCATGAGGGCATCCCAACTGGACAGCCGGCCTC
CAAAGGCTTGGGTCTGATTGGGAGCTGGCTCAAGCCATCCTTCGAGTCTTCCAGCCCAAAGCCGTCTGGC
TGAAATTGAGTAACCGTTTGAATGACACATAGATGTAATGCGGTGTTGGAAGCGGATGTGTGGTGGGTAC
CACTATATTAAAAGAGCTGCATGTGTCCTGGGAAAAAAAAAA
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Figure 4: FASTA sequence for MGMT gene in *Rattus norvegicus* obtained from http://www.ncbi.nlm.nih.gov/ . From this sequence the CpG island can be identified and a favourable primer can be selected to run RT-PCR.

Conclusion

- It is still not possible to infer that there is a direct relationship between the formation of brain cell gliomas and exposure to EMF from cell phones and base station which emit them.
- Review of the literature expresses the need for further research to be conducted as most studies in the field yielded results that were either inconclusive or not statistically significant.
- Future plans for this research project include the identification of other target genes and the need for more experiments in order to gather an appropriate amount of data for analysis.

References

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