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Long-Term Health Impacts of Cell Phone-Driven Radiofrequency Radiation Exposure in Humans

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This is to certify that the doctoral dissertation by

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has been found to be complete and satisfactory in all respects,
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Abstract

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Dissertation Submitted in Partial Fulfillment

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Abstract

Uncertainties still exist about the safety of cell phone use and the level of cell phone-driven radiation. The purpose of the current inquiry was to determine the long-term health impacts of cell phone-driven radiation via the use of cell phones. In this cross-sectional study, which was based on socio-ecological theory, secondary data from the 2012 National Health Interview Survey were analyzed to assess the difference in the prevalence of thyroid cancer, mouth/tongue/lip cancer, and heart disease between exposed and non-exposed/less exposed groups in the United States. Logistic regression was used to address three research questions. Findings showed that cell phone use was associated with cancer outcome. However, there was no statistically significant relationship between individuals who were heavy users or sometimes users of cell phones and thyroid or mouth/tongue/lip cancer when compared to individuals who rarely or do not use cell phones. There was a relationship between heavy/sometimes users and heart disease when compared to individuals who rarely/do not use cell phones. Yet, when all the confounders/covariates were included in the model, there was no statistically significant difference between the groups compared. Even when all the covariates were accounted for, age and sex were added in the model for thyroid cancer for both phoneuse 1 and 2. Findings reiterate the need for more rigorous attention to industrial quality control measures for cell phone use and also highlight the need for social awareness of the possible health implications of such use. Using study findings, policy makers may wish to explore the implementation of comprehensive regulatory measures to address cell phone safety.

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Dedication

I was driven by many factors including my own personal and professional life-course perspectives to undertake a PhD program. My decision to engage in the assessment of cell-phone driven radiation comes from both the previous evidence-based studies and the cultivated academic instinct instilled in me that allowed me to question the possible and plausible public health impacts associated with repeated use of cell phones. Ultimately, my gratitude goes out to researchers who came before me who have conducted inquiries into the public health impacts of cell phone use. Their work paved the way for my research study.

Acknowledgments

I advance my deepest gratitude to my chair, Dr. Robare; my committee members, Dr. Ferraro, Dr. McDoniel, Dr. Kachgal, the rest of URR team; the program director team; the academic advising team; the entire Walden staff; and my family and friends for their invaluable compromise throughout my academic pursuit. I appreciated these individuals' contributions towards my academic achievements throughout the entire dissertation process. Without these amazing individuals or groups' support, the completion of the dissertation process may not have been possible or as informative, memorable, and productive as it turned out to be. With their steadfast support and expert suggestions, the process enabled me to grow in terms of my research thinking processes and was a meaningful and amazing academic research experience, to say the least.

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Chapter 1: Introduction to the Study

Introduction

My dissertation topic is on the impacts of cell phone-driven radiofrequency radiation (RFR) on human health outcomes. In this study, I investigated the possible health impacts associated with RFR emitted or transmitted via cell phones. Regarding the study's operational constructs, the term "health impacts," "health outcomes," and "quality of life" refer to any of the chronic conditions such as thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease. I assessed prevalence rate differences in chronic conditions or health outcomes between groups regularly exposed to long-term cell phone driven RFR via the use of cell phone communication and a non-exposed or low exposed group. Therefore, the primary independent/predictor variable explored in this study was cell phone RFR exposure via cell phone use.

The cell phone connection systems in question can be modulated by any of the radio access technology features such as the Nordisk MobilTelefoni or Nordiska MobilTelefoni-Gruppen (NMT), Global System for Mobile Communication (GSM), Code Division Multiple Access (CDMA), and Long-Term Evolution (LTE) communication systems (Mun, Estrin, Burke, & Hansen, 2008: Wireless Intelligence, 2007. The NMT, GSM, CDMA, and LTE radio communication systems are common access features used in cell phone transmission depending on the providers' wireless network connection or the country's preferred network system(s) (Mun, Estrin, Burke, & Hansen, 2008: Wireless Intelligence, 2007). Regardless of the access features, all current

wireless systems emit or transmit RFR or radio waves (Mun, Estrin, Burke, & Hansen, 2008: Wireless Intelligence, 2007).

The NMT was the first fully automatic cellular phone system introduced in the market in 1981 and directed by the Nordic Telecommunications Administrations (PTTs) (Mun, Estrin, Burke, & Hansen, 2008: Wireless Intelligence, 2007). The GSM system was introduced in 1991 in Finland but currently captures more than 80% of the global market and operates in over 212 countries (Mun, Estrin, Burke, & Hansen, 2008: Wireless Intelligence, 2007). The CDMA was introduced in 1995 and currently captures about 17% of the global market, while the LTE was introduced in 2009 (Wireless Intelligence, 2007). Both the NMT and LTE services capture the smaller portion of the global market (Wireless Intelligence, 2007). The U.K., most European nations, and all other countries except Japan and South Korea operate on a GSM network system, while the United States and other parts of North America as well as some parts of Asia operate mostly on a CDMA network (Wireless Intelligence, 2007). In contrast, the Nordics and several other European countries operate on the NMT network (Wireless Intelligence, 2007).

Assessing the safety, toxicology, and epidemiological impacts of cell phone-driven RFR on human health outcomes is essential for advancing meaningful safety policies and standards for cellular phone use and operation (Balmori, 2016; Fehske et al., 2011; Kesari, Siddiqui, Meena, Verma1, & Kumar, 2013). An increase in unsafe RFR levels via cell phone use in the local or global context may substantially increase the incidence and prevalence of adverse health outcomes within the target population

(Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013). In this chapter, the background information, problem statement, purpose of the study etc., about the health impacts of RFR through the use of cell phone was explored and discussed.

Background

My rationale for evaluating the health impacts of cell phones on humans was based on evidence suggesting that biological effects are produced from cell phone RFR exposure. Cell phone use or cell phone-driven RFR exposure during calls is a risk factor for the onset of chronic conditions (Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013). Such biological effects due to prolonged use of cell phones during calls, present unprecedented public health concerns within societies that have adopted mobile phone communication systems as the primary means of communication (Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013). Also, scientists have suggested that cell phone-driven RFR emission or exposure alters not only the biological mechanisms of the exposed targets but affect plants and the environment (Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013; NTP, 2016; Tkalec et al., 2008). Based on these findings, experts have concluded that there is adequate evidence to suggest that cell phone use may be associated with serious adverse health impacts on human, animal, and environmental health (Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013; NTP, 2016; Tkalec et al., 2008). Figure 1 shows the basic diagrammatic structure of a GSM network and how it is linked to communication devices and sources.

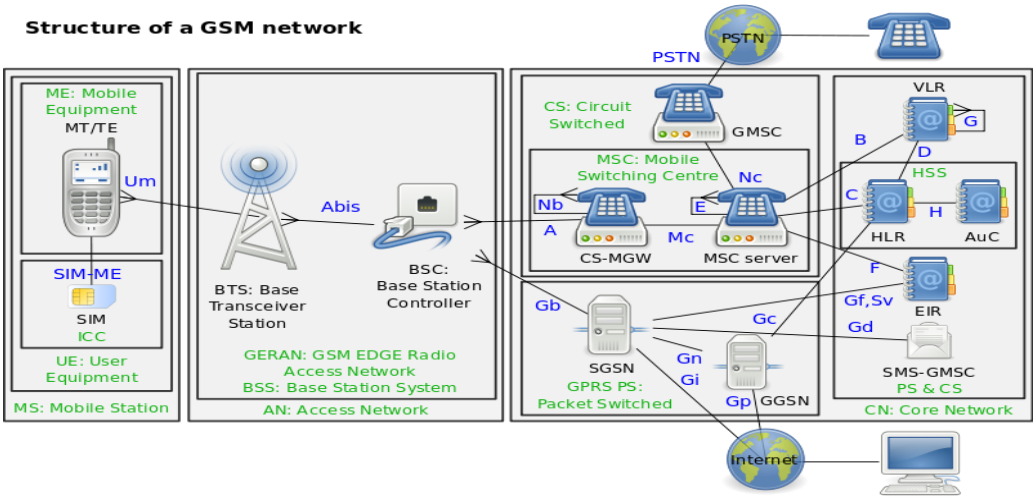


Figure 1. Structure of a GSM network. (Wikipedia.org, 2016).

In 2011, the International Agency for Research on Cancer (IARC), which is a part of the World Health Organization (WHO) responsible for conducting research on cancer and classifications of potential carcinogens, classified RFR as a possible and probable carcinogen to humans due to the increased risk for glioma (malignant brain cancer) observed with cell phone use (IARC, 2011). The inherent biosafety concerns on cell phone use promotes the need further epidemiological studies. One such need is this dissertation study which focused on the evaluation of the health impacts of cell phone-driven RFR exposure. The pathological diseases observed in rats via an experimental study design and in humans through observational or longitudinal study design following exposure to RFR are crucial evidence that supports this study rationale (Balmori, 2016; Kundi, 2009; Fehske et al., 2011; Kesari et al., 2013; NTP, 2016; Tkalec et al., 2008). Subsequently, unintentional biological effects and environmental changes could create public health and environmental health threats. Hence, long-term studies appear to be necessary and warranted.

Problem Statement

Researchers have shown in observational or experimental studies using plant species, animal species, and human cohorts which were exposed to certain levels of cell phone-driven RFR that there is evidence suggesting that the current RFR transmitted or absorbed by the body via cell phone use is carcinogenic or adversely affects health outcomes (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec, Malaric, Pavlica, Pevalek-Kozlina, & Vidakovic-Cifrek, 2008). Using an experimental research design to assess the toxicity levels of cell phone driven RFR in animals, National Toxicology Program (NTP) researchers concluded, for instance, that there are serious adverse health impacts of cell phone-driven RFR in an exposed group of animals (rats and mice) compared to the unexposed or control group (NCI, 2011; NTP, 2016). In the NTP study, the health impacts of RFR exposure on rats were not only linked to malignant glioma and glial cell hyperplasia but were also linked to Schwannoma and Schwann cell hyperplasia (NTP, 2016). Moreover, there is a differential health effect of the types of RFR exposure between GSM- and CDMA-driven cell phone RFR exposures in rats (NTP, 2016). There are also apparent variation effects based on sex in male and female rats (NTP, 2016).

The unintentional interference properties of cell phone during use is also another concern. It is known that cell phone interferes with medical devices such as pacemakers. It appears the challenges in quantifying the cumulative effect of cell phone driven RFR that produces biological effects or etiological onset of chronic conditions or clinical manifestation of disease is a serious concern in predicting the effects of the long-term exposure. It may take years or decades for certain types of chronic conditions to become

clinically observed or manifested (Gordis, 2009). Based on this, it seems to be more meaningful to conduct long-term studies than short-term inquiries on cell phone use on its impacts on chronic conditions (Balmori, 2016; Kundi, 2009).

Globally, cell phone use is increasing substantially; thus, there are growing concerns about public health efforts related to RFR exposures (Kundi, 2009). These concerns deal with both the monetary and non-monetary costs of long-term health impacts of cell phone driven-RFR (Balmori, 2016; Kundi, 2009). As discussed, suggestions advanced through short-term studies about RFR impacts on animals indicated severe adverse chronic conditions (Kesari et al., 2013). Perhaps, extensive and continuous RFR exposure in humans via cell phone use could lead to serious public health burdens (Kesari et al., 2013).

Cell phones were first introduced in the global marketplace for public use in the 1980s (Gow & Smith, 2006). In the United States alone, over 90% of adults are cell phone owners and users (Rainie, 2013). About 78% of youths aged 12-17 owns cell phones, and 37% of all teens have smartphones (Rainie, 2013). Globally, the number of cell phones is over 6 billion (97%) users based on the current population size (Rainie, 2013). The connections per 100 persons in the United States alone ranged from 103.1-118% (The World Bank, 2016). In the United Kingdom, cell phone connections is between 126-129.6% (The World Bank, 2016). The trend of global cell phone users grew from 4.01 billion users in 2013 to 4.61 billion users in 2016 (Statista, n.d.). According to the Statista (n.d.) report, the total global number of mobile phone users is estimated to reach 5.07 billion users by 2019.

As it currently stands in the 21st century, cell phone use is a common means of communication in both the social and business environments. The social or business environment necessitating the ubiquitous nature of cell phone ownership or usage is intricately linked to the sociocultural and technological dynamics of the modern society (Rainie, 2013). There are reported severe health consequences associated with cell phone-driven RFR emission (NTP, 2016).

Nevertheless, other epidemiologic investigators have indicated that there are limited risks associated with cell phone use due to lack of strong evidence-based information on the exposures (Auvinen, Hietanen, Luukkonen, &Koskela, 2002). Shrestha (2015) emphasized that uncertainties on duration and long-term use remained because a minuscule proportion of participants in the study reported cell phone use beyond 10 years. Thus, it is necessary and warranted to investigate the possible long-term impacts of cell phone RFR on the individuals' quality of life and health outcomes (Shrestha, 2015). Understanding the long-term health effects, if any, could allow health practitioners to conduct meaningful epidemiologic assessments to assess the public health significance of the effects.

Some investigators even suggested that there was no link between cell phone use and health outcomes. For instance, Kundi (2009) concluded that the increased risks of cell phone radiation to health outcomes were not met based on three epidemiologic criteria: The first criterion was that there is no available evidence-based exposure metric. Secondly, the observed duration of cell phone use was too low (Kundi, 2009). Thirdly, there was no evidence-based selection of end points for the different types of neoplasias,

and risk-effect association could not be possible due to the lack of etiologic hypotheses (Kundi, 2009). Kundi (2009) also indicated that selection bias, misclassification bias, and effects of the RFR to the proposed health outcomes probably and possibly reduced the risk estimates.

For many of the cross-sectional driven research designs conducted on RFR exposure and its link to health outcomes, recall bias could have led erroneously to increased risks (Kundi, 2009; Shrestha, 2015). In the cases where the findings may not have been spurious, and the evidence suggested an increased risk, the magnitude of such cannot be meaningfully evaluated due to insufficient information on duration and long-term use (Kundi, 2009; Shrestha, 2015). Shrestha (2015) suggested that excess risk was not observed with self-reported short or medium-term cell phone use. As a result, Shrestha (2015) emphasized that uncertainties on the duration and long-term use remained because a minuscule proportion of participants in the study reported cell phone use beyond 10 years. In a sharp contrast, additional findings in some retrospective cohort studies suggested increased risk of glioma and acoustic neuroma among cell phone users (Havas, 2009). However, from the cross-sectional studies conducted in the US, the researchers concluded that increased incidence of glioma or meningioma or non-central nervous system (CNS) cancer was not associated with cell phone use (Benson, Pirie, Schüz, Reeves, Beral, & Green, 2013; Havas, 2009).

Figures 2 and 3 show the positions of the salivary glands to the ear area. The diagram illustrates one of the body areas that could be directly affected by cell phone-

driven RFR due to use of a handheld phone for talking. The Figure 2 was retrieved from Therabreath.com.

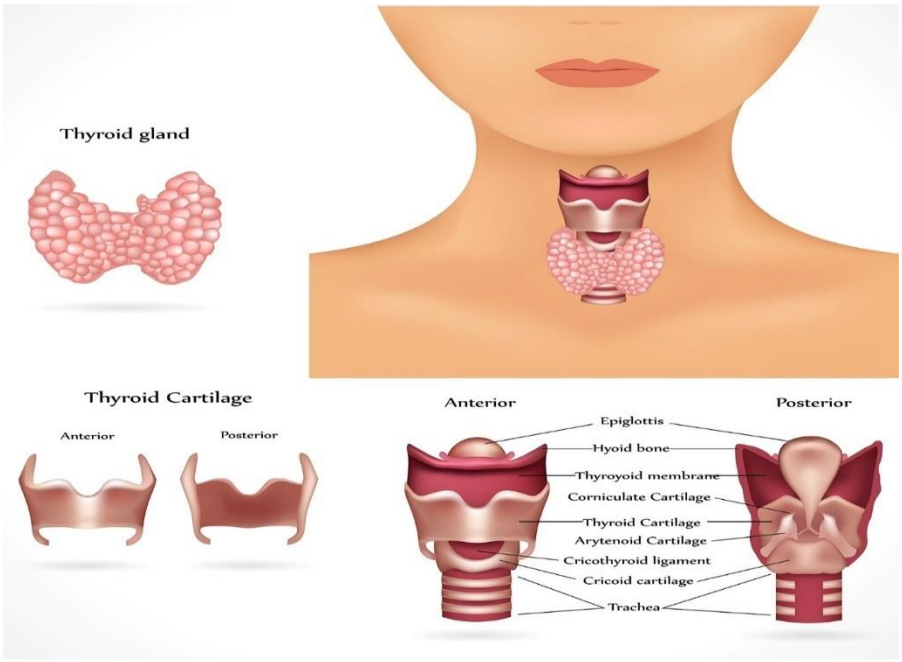


Figure 2. Thyroid Gland Diagram and Location in Human. (Stockfreeimages.com, 2017).

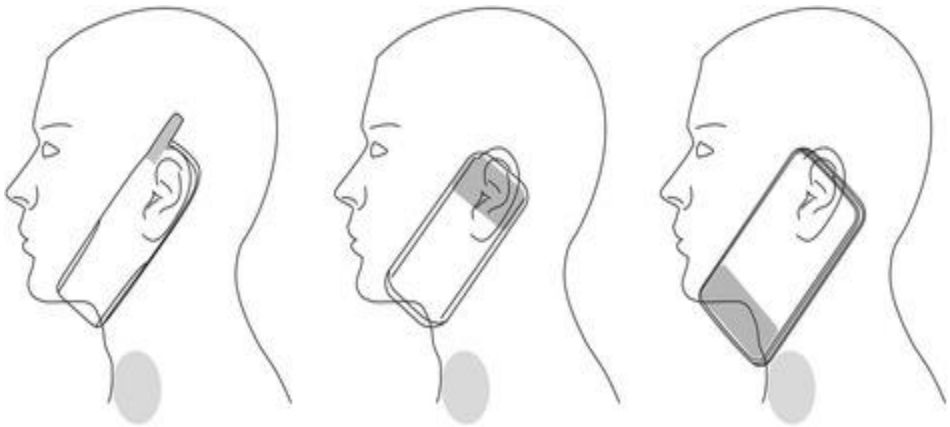


Figure 3. Handheld Cell Phone in Position to the Head Area. (Carlberg et al., 2015).

Figure 4 showed the possible locations of antenna in a typical cell phone. It is possible that the position of the antenna in a cell phone may differ that what is shown here. The antenna location or positioning is dependent on the manufacturers' design and utility model. The important thing here is that there could be more than one antenna in a cell phone design.

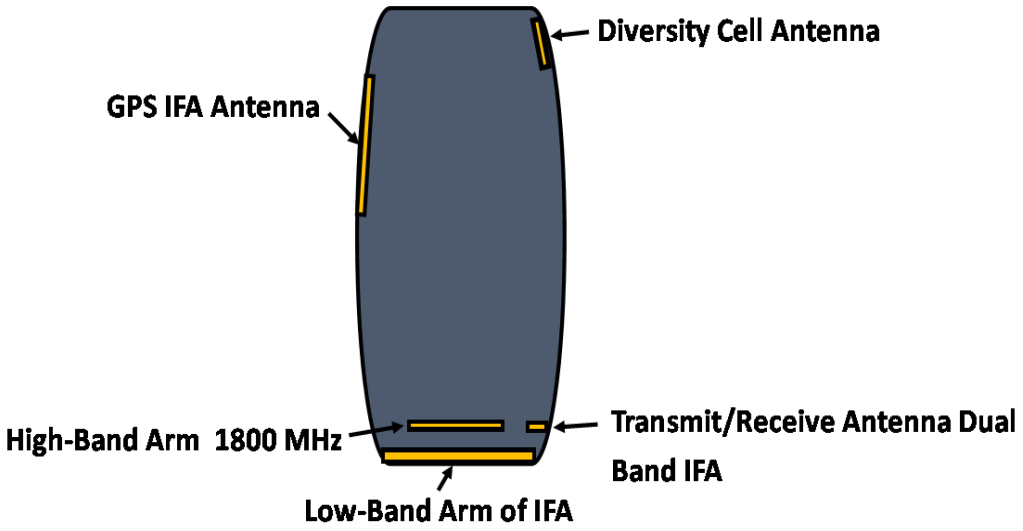


Figure 4. Cell Phone Antenna Positions. (antennatheory.com).

The primary health outcomes under study and gap in the literature for this inquiry (the health effects of a long-term exposure of cell phone use) are thyroid cancer, mouth/tongue/lip cancer, and heart conditions. For this inquiry, it is important as well to emphasize other reported health outcomes that are linked to cell phone-driven RFR exposures. By establishing the rationale for this study with reported health outcomes linked to cell phone RFR exposures, it garnered evidence-based support on the ideation of this research inquiry precept. Perhaps, also supported a deductive theoretical thinking process for the observed phenomenon. Glioma, meningioma, neurotic cancer, cardiac

schwannomas or heart tumors, acoustic neuroma, and other non-CNS conditions has been reported in several studies (Finkenthal et al., 1996; NTP, 2016; Taylor, Wargo, Alderman, Bradley, & Addiss, 2011; O'Neill, Teo, Davis, Henshaw, Lamburn, Maisch, Morgan, & Ahonen, 2011). The parotid gland tumor (PGT) and salivary gland tumor (SGT) are potential health outcomes associated with cell phone-driven RFR exposure (O'Neill, et al., 2011). Parotid and salivary organs are sets of glands adjacent to the ear (O'Neill, et al., 2011). These glands are located in the ear position where a handheld cell phone is placed during use (talk mode). Increased risks of PGT or SGT among heavy cell phone users has been reported by several researchers (O'Neill, et al., 2011). On average, the survival rate of PGT or SGT is 2.7 years while a 10-year survival rate is in the range of 14-26% (Havas, 2009). For a slow growth and painless symptomatic form, the tumor is likely benign 80% of the cases, but for a painful and nerve paralysis symptomatic form, the tumor is likely malignant 20% of the cases (Havas, 2009). The common concern highlighted with other health outcomes and especially PGT or SGT also applied to thyroid glands which are located in the neck area not far away from the parotid gland (PG) and Salivary gland (SG) area or the head area. In other words, if the PG and SG could be adversely affected by cell phone-driven RFR exposure or heavy cell phone use, it seems likely that the thyroid glands could also be affected.

PGT or SGT is a rare form of cancer and has not received public attention. SGT prevalence is approximately 70-75 benign and 8-14 malignant neoplasms yearly/million population in the UK, and about 1% of cancers in the US (Bradley & McGurk, 2013; Havas, 2009). In the western part of the world, the historical yearly incidence rate of

PGT or SGT is 1-3 per 100,000 people (Havas, 2009). It is present in people of any age but is common among older individuals (American Cancer Society, 2015). The estimated average age of patients at the time of diagnosis is 64 years old for all types and stages of SGT (American Cancer Society, 2015). At least from 5 years after diagnosis, roughly 72% of those diagnosed with PGT or SGT are alive (American Cancer Society, 2015). It appears, based on the available evidence, cell phone driven radiation is a possible predictive factor for thyroid cancer. Hence, in this research setting, the link between cell phone use and thyroid cancer, mouth/tongue/lip cancer, and health condition were evaluated.

The prevalence and incidence assessment conducted in 2014 by the Cancer Research UK showed that thyroid cancer is the 19th most common cancer cases in the UK (Cancer Research UK, 2014). Thyroid cancer cases are approximately 1% of all new cancer cases in the UK (Cancer Research UK, 2014). Among women in the UK, thyroid cancer is the 16th most common cancer (1% of the female), while in men, it is the 19th most common cancer (<1% of the male) (Cancer Research UK, 2014). Similarly, in 2017, the American Cancer Society (ACS) most recent estimates for thyroid cancer indentified approximately 56,870 new cases (42,470 women, and 14,400 men) of thyroid cancer in the US (American Cancer Society, 2017). About 2,010 deaths (1,090 women and 920 men) from thyroid cancer was reported (American Cancer Society, 2017). The lifetime risk of thyroid cancer varies; however, its diagnosis is common at younger age than most adult cancers (American Cancer Society, 2017). For instance, the proportion of thyroid cancer in children and teens in the US is 2% (American Cancer Society, 2017).

About 3 in 4 of thyroid cancer cases are women (American Cancer Society, 2017). The risk of being diagnosed with thyroid cancer has tripled in the past three decades and has become the most rapidly increasing cancer in the US (American Cancer Society, 2017).

The data on the incidence, prevalence, and mortality rate of thyroid cancer per 100,000 individuals in the US was reported by the National Institute of Health (NIH). The NIH risk estimate for the 2009-2013 cases on thyroid cancer showed that the number of new cases of thyroid cancer in the US was 13.9 per 100,000 annually among men and women (NIH, n.d.). It was also estimated that the number of deaths was 0.5 per 100,000 annually for both men and women (NIH, n.d.). Overall, in 2013, the number of persons living with thyroid cancer in the US was 637,115 individuals (NIH, n.d.).

Even though thyroid cancer occurs among both genders, it is one of the few cancers that are more common in females than males (Cancer Research UK, 2014). Perhaps, this observation is in part due to gender differences in exposure to the risk factors (Cancer Research UK, 2014). For instance, in the European age-standardized (AS) incidence rates, the AS rate are significantly higher for females than in males in England, Wales, Scotland, and Northern Ireland (Cancer Research UK, 2014). However, among females, there are geographical differences in the incidence rate (Cancer Research UK, 2014). Between women in different UK nations, the AS rate was significantly higher for females in England in comparison to Wales, Scotland, and Northern Ireland (Cancer Research UK, 2014). Also, the age-standardized incidence rates are significantly lower in Wales in comparison to England and Scotland (Cancer Research UK, 2014.

The researchers also found that there are no statistically significant differences between the other constituent countries of the UK (Cancer Research UK, 2014). Overall, in 2014, there were 3,404 new cases of thyroid cancer in the UK, which constituted 966 (28%) male cases and 2,438 (72%) in female cases (Cancer Research UK, 2014). Based on this information, the male to female ratio estimate was 4 to 10 or 1 to 2.5 respectively (Cancer Research UK, 2014). Furthermore, the crude incidence rate was estimated to about 3 new thyroid cancer cases per 100,000 males in the UK, while 7 new cases occur for every 100,000 females (Cancer Research UK, 2014

Overall, thyroid cancer incidence has increased for both genders in Great Britain by over 149% since the late 1970s (Cancer Research UK, 2013). Based on the European AS rate of thyroid cancer incidence estimate, males in Great Britain remained stable until 1994-1996 and had increased since then by 121% (Cancer Research UK, 2013). On the other hands, the female AS thyroid cancer incidence rates also remained stable until 1991-1993, but have since increased by 144% (Cancer Research UK, 2013). In the UK for the last decade, thyroid cancer AS incidence rates increased by 71% for both genders combined, and when stratified by sex, the increase was 70% for males and 73% for females (Cancer Research UK, 2013). Overall, the thyroid cancer incidence rates in both males and females by age groups in Great Britain increased (Cancer Research UK, 2013). Furthermore, in 2012, the estimated lifetime risk of developing thyroid cancer for men was roughly 1 in 480, and approximately 1 in 180 for women in the UK (Cancer Research UK, 2013).

According to Vanderpump (2011), thyroid disorders are prevalent, and its manifestation is determined by the availability of dietary iodine. However, Vanderpump (2011) did not identify all the possible factors that could directly or indirectly affect the dietary iodine availability. Though, from the data obtained from screening large population samples in the USA and Europe, the consensus was that the most common cause of thyroid disorders globally is iodine deficiency (Vanderpump, 2011). Such deficiency could lead to the onset of goiter and hypothyroidism (Vanderpump, 2011). Vanderpump (2011) also emphasized that most individuals with thyroid disorders have an autoimmune disease. They indicated that operational indicators of thyroid disorders include age, sex, environmental factors, and thyroid screening techniques; while emphasizing that there is increasing incidence of well-differentiated thyroid cancer currently being observed (Vanderpump, 2011). Multiple investigators have identified potential short-term health impacts of cell phone use or cell phone-driven RFR. However, the potential long-term risk for chronic conditions such as thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease have not been demonstrated.

Purpose of the Study

This study is an exploratory epidemiological research meant to draw attention to meaningful and evidence-based findings. The comparative epidemiological research inquiry about the health impacts involving long-term cell phone use on a variety of health outcomes has never been conducted or addressed. It appears that in the absence of an evidence-based long-term biosafety analysis of cell phone RFR impacts on human health, the sociocultural dynamics, industrial-based proliferations, and individual-based adoption

of unsafe cell phone technology could lead to high rate of health-related risks and outcomes. Currently, cell phones appear to be the common means of communication, and its adoption has increased over the years, globally, (see Figure 8) (CTIA, 2011; The World Bank, 2016). To address these concerns, in this study, secondary data containing information about participants' cell phone use behavior and ownership status was used to explore the correlational association between long-term cell phone-driven RFR exposure and health outcomes.

Accurate determination of prolonged (long-term) exposure and duration of cell phone use among eligible participants minimizes erroneous conclusions regarding the inferred associations between the cell phone exposure and the health outcomes investigated. The secondary data-driven questionnaire instrumentation approach employed in this study for the assessment of the effects of cell phone-driven RFR exposures on thyroid cancer, mouth/tongue/lip cancer, and heart disease did capture the exposure dosage in terms of the actual RFR measurements during cell phone use. There was also no measurement on the specific absorption rate (SAR) during cell phone use among the participants. This study lacks ideal conditions necessary to established spatio-temporal validation on the exposure and its link to the health outcome under investigation. Hence, plausible alternative explanation on the observed outcomes including unknown or unaccounted confounders and covariates effects are possible influences that distort the findings. In this study, the applied study design and method approaches specified in in the chapter 3, was the best available alternative design for a long-term study on chronic health outcomes among human subjects. It appears that The

RFR exposures could lead to serious adverse health outcomes or even death (NTP, 2016). For this reason, a cross-sectional design tolerates acceptable ethical standards for scientific studies involving human subjects. Therefore, the use of secondary data-driven cross-sectional (observational) study design for this epidemiologic study settings was meaningful, ethical, and rational in addressing the posed research questions. Overall, the study purpose was initiated as an exploratory epidemiologic study about the long-term effects of cell phone RFR exposure on human health outcomes. Also, the establishment of the proposed research inquiry's theoretical framework was based on the prior experimental studies conducted on animals, and other human-based observational studies (Havas, 2009; National Cancer Institute, 2011; National Toxicology Program, 2016). In many cases, researchers had reported or observed severe negative health impacts and manifestations of chronic conditions on the unit of analysis upon cell phone RFR exposure (Havas, 2009; National Cancer Institute, 2011; National Toxicology Program, 2016).

Research Questions and Hypotheses

In this study, I addressed the following research questions and hypotheses:

RQ1. What is the difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_o1: There is no difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_a1: There is a difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

RQ2. What is the difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_o2: There is no difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_a2: There is a difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

RQ3. What is the difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_o3: There is no difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_a3: There is a difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

Theoretical Foundation

The social-ecological theory is of great interest in this study because its operational contents and constructs are function of the sociocultural or sociopolitical or psychosocial or psycho-behavioral perspectives that provides an in-depth understanding of the interactive links between an exposure or effector and a response or an outcome variable in regards to public health promotion measures among individuals either in the micro-, meso- or exo- or macro-systems or all of the specified levels of individual or social interaction constructs (Bronfenbrenner, 1979, 1986, 1994, 1995). Therefore, the incorporation of the social ecological theory in this study as the functional theoretical foundation elevated our understanding of the interaction processes or links on health promotion measures. It provided the platform, which allowed the exploration of the extrinsic and intrinsic interactions between the health outcomes of interest (thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease) and exposure (cell phone use) among the specified target population investigated. It appeared by understanding the mode-of-action and biological effects of cell phone-driven RFR exposure on health outcomes through the literature review processes, the biological plausibility on thyroid, mouth/tongue/lip and heart is possible. Therefore, the social ecological theory was meaningfully applied in the exploration of the interactive links between cell phone-driven RFR exposures and thyroid cancer, mouth/tongue/lip cancer, and heart conditions. An in-depth explanation of the social-ecological theory was explored in the Chapter 2 section of the dissertation.

Nature of the Study

The research design applied in this research inquiry was a cross-sectional approach. A cross-sectional design is a form of an observational study. The data used in the study to address the research inquiry was a secondary data generated by the 'National Health Interview Survey, 2012' (NHIS) (NCHS, 2013, 2015). The original data was collected via a survey or questionnaire-based approach (NCHS, 2013, 2015). The survey was administered by the United States Department of Health and Human Services (USDHHS) to the participants through the purview of the Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS) (NCHS, 2013, 2015). Based on the research questions' constructs, which involved quantifiable measures, the appropriate research method that aligned with the research inquiry is a quantitative research method. In other words, to produce meaningful findings, all the relevant variables, the independent variables (IVs), which included cell phone use and no cell phone use or few cell phone use must be quantifiable or objectively identifiable. Also, the dependent variables (DVs) (thyroid cancer, mouth/tongue/lip cancer, and heart condition) must be quantifiable as well. Known confounders or extraneous variables must be quantifiable to help draw meaningful inferential assessments from the study findings. In this study, the IVs and DVs' levels of measurements are both nominal or categorical, see chapter 3 for more detail.

Definitions

Blue Tooth: A wireless cell phone accessory designed to be used as a hands-free device with a handheld cellular or mobile phone. A blue tooth uses radio wave energy to

transmit communication signals. Its use also exposes a user to RFR or RF-EMF radiation.

Cell phone or mobile phone or cellular phone: A telecommunication device. All cell phones emit or transmit electromagnetic field (EMF) or radiofrequency (RF) (a radio or microwave energy) (Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011). The emitted or transmitted EMF is made up of waves of electric and magnetic energy transmitted via space (Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011). Electromagnetic energy is categorized based on the wavelengths and frequencies they emit or transmit in the electromagnetic "spectrum" (Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011).

Cell phone use: In this research setting, it includes the operation of any type and model of cell phone or mobile phone that could be used to initiate telephone calls (NHTSA, 2011a, NHTSA, 2011b). The initiated calls will involve the ability to talk via the cell phone either through a handheld or hands-free mode (NHTSA, 2011a, NHTSA, 2011b).

Cell phone-driven RFR or Cell phone-driven RF-EMF: An exposure to RF-EMF radiation through any cell or mobile phone source that emits or transmit RFR or RF-EMF wave when in use either during the initiation of dialing or receiving an incoming call or outgoing call or talking or texting. The cell phone-driven RFR or cell phone-driven RF-EMF could be modulated via the GSM or CDMA or any other types of the telecommunication modulation systems or features.

Cohort effect: The variations in the characteristic profiles among individuals who are defined by some shared temporal experiences or events, common life experiences, and biological factors, etc. (Gordis, 2009; Szklo & Nieto, 2014).

Electromagnetic energy: Electromagnetic energy (EME) or electromagnetic radiation (EMR) or electromagnetic field (EMF) is a radiant energy emitted from electromagnetic processes that transmit or emit specific range of wavelength, frequency, and energy (Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011). Radio waves or microwave energy or visible light is an example of electromagnetic energy (Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011).

Generational effect: Refers to how individuals or cohorts born at a certain time or age differs in behavior, social, and biological impulses from another sets of cohorts who are born at a different time (Gordis, 2009; Szklo & Nieto, 2014). Comparison of millennial social environment or life experience to the baby boomers' generation is an example of generational effect (Gordis, 2009; Szklo & Nieto, 2014).

Handheld cellular phone: A cell phone or mobile phone communication device that requires the use of the hand to talk or listen to conversations or perform other communication transmission functions (NHTSA, 2011a; NHTSA, 2011b). A handheld cell phone is typically held with a hand and placed close to the ear or head area adjacent to the ear or face cheek area or jaw area to talk. A handheld cell phone can be held with hands to send text messages or initiate a dial or call.

Hands-free cell phone: A cell phone or mobile phone design that is integrated with features that do not require individuals to use their hands to operate, talk, send a

text, or initiate a dial. A handheld cell phone can be operated with a phone accessory such as a blue-tooth, headset, and equipped with a speaker phone mode or feature (NHTSA, 2011a, NHTSA, 2011b).

Headset: A headset is a cell phone accessory designed for use as a hands-free device with a handheld cell phone. A headset uses wire connections to transmit communication signals from a connected phone to the listening source. Its use (to talk) reduces radio wave or electromagnetic radiation to the head/cheek/neck areas.

Health outcomes: Refers to chronic health conditions or adverse health conditions. An example of a health outcome or chronic health condition considered or explored in this study is thyroid cancer.

Heart conditions: Refers to a range of heart conditions that affect the heart. Heart conditions or diseases includes blood vessel diseases, coronary artery disease, heart rhythm problems (arrhythmias), and congenital heart defects (Mayo Clinic, n.d.). Heart disease or heart condition is often interchangeably used for the term "cardiovascular disease" (Mayo Clinic, n.d.). A cardiovascular disease (CVD) is health conditions that involved the heart and vascular systems. Narrowed or blocked blood vessels, is a condition that may lead to heart attack, chest pain (angina), and stroke (Mayo Clinic, n.d.).

High blood cholesterol: Cholesterol is a waxy substance found in the fats (lipids) in the blood. Having high cholesterol than the body needs could increase the risk of heart disease and coronary artery disease (Mayo Clinic, n.d.).

Hypertension: An increase in the blood pressure above normal. Hypertension can also be referred to as high blood pressure. It is a health condition where a long-term force of the blood against the artery walls is to high enough for a prolonged period that my lead to health problems, such as heart disease (Mayo Clinic, n.d.).

Period effect: Refers to the variation in a study setting caused by the influence of the year or period in which the study observations were made (Gordis, 2009; Szklo & Nieto, 2014).

Radiofrequency radiations or radio wave or microwave dnergy: A type of non-ionizing electromagnetic energy (Finkenthal et al., 1996; NTP, 2016).

Social determinant of health (SDH): Refers to the socio-structural, infrastructural, infostructural determinants and conditions in which people are born, grow, live, work and age (Cohen, Chavez, & Chehimi, 2012; Krieger, 2011; Schneiderman, Speers, Silva, Tomes, & Gentry, 2010; Wilkinson, & Pickett, 2010). Some of the SDH factors are socioeconomic status, education, the physical environment, employment, access to health care, social support and social networks (Cohen et al., 2012; Krieger, 2011; Schneiderman et al., 2010; Wilkinson & Pickett, 2010).

Speaker phone mode: An inbuilt speaker system feature in a handheld cell or mobile phone that allows the users to speak and listen through the speaker without placing the phone close to the head/cheek/neck areas.

Thyroid problems: Sets of known thyroid functions or lack of functions that may produce adverse health issues. Examples of thyroid problems are hypothyroxinemia, hyperthyroxinemia (hyperthyroidism), thyroid tumor or cancer, goitre, etc.

Unit of analysis: Refers to the entity that is being analyzed or observed in a study. It is the 'what' or 'who' that is being studied (Gordis, 2009; Szklo & Nieto, 2014).

Assumptions

In plant, animal, and human studies, researchers has shown either through an experimental or observational or longitudinal study design the association between RFR or RF-EMF exposure with several health outcomes including but are not limited to schwannomas, glioma, glial cell lesions, parathyroid tumor, salivary gland tumor, and heart conditions (Havas, 2009; Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011; Vanderpump, 2011). The findings from the experimental or observational research from other studies is indicative of the possibility of the link between cell phone-driven RFR exposure and other health outcomes. Thus, one of the key challenges encountered in this study was the extent to which the scope of the individualistic or ecological fallacy was demonstrated upon a long-term exposure of cell phone use.

In other words, the individualistic or ecological fallacy assumption explored in the previous body of literature in connection to cell phone-driven RFR exposure and the subsequent health outcomes was re-evaluated in this study, but specifically on thyroid cancer, mouth/tongue/lip cancer and heart conditions. The health effects of cell phone use (cell phone-driven RFR exposure) has been previously demonstrated with individuals or sets of the target population or within an ecological or a geographical or demographical location. However, it was not simple to show such health effects in all cases and how cell phone-driven RFR-EMF exposure or cell phone use could inherently and always produce the same outcomes (adverse health outcomes). There are many

reasons why researchers should not assume that cell phone use will always lead to adverse health outcomes. One of the key reasons is that the correlational association between and exposure and a given health outcome is heavily dependent on the link between the extrinsic and intrinsic factors within and between the unit of analysis under investigation (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014).

Another factor that could affect the accuracy of a study is the type of the research design employed for the research inquiry. For instance, the application of a cross-sectional or survey or questionnaire data collection approach, may not accurately establish the temporality sequence of the exposure as it relates to the health outcome in question. A cross-sectional approach could not be used definitively to ensure the integrity of the spatiotemporal exposure-health outcome sequence or define the scope of the exposure-outcome temporality sequence (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The multifactorial nature of the link between an exposure and a health outcome is another confounding factor that is always present. Multiple factors are intricately connected to health outcomes (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011). Some of these multifactorial intricacies are genetic variability of the subjects, known or unknown familial history of the subjects, duration of exposures, exposure to covariates, confounders such as age, demographic, and other social determinants of health (SDH) factors. More so, the responses provided by the participant on the level of phone use and duration of cell phone use could be subject to recall bias or rumination bias on the level

of exposure and duration of exposure (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). Also, the reliability and validity of the response provided by the participants were subjective and were not verified objectively. Also, the secondary data employed was not collected for the sole purpose of this study, thus, may not be the best measures of the concept and intent of the study.

Scope and Delimitations

The scope and delimitations of this study are characterized by time, sample size, and geographic area of the NHIS study. Therefore, the inherent characteristics of the NHIS dataset implicated individuals living in the United States in 2012. Also, the independent variable was delimited to cell phone-driven RFR-EMF exposures but not radio or microwave exposures from any other sources such as internet or TV, etc. On the other hand, the dependent variables are delimited to thyroid cancer, mouth/tongue/lip cancer, and heart condition or disease because these are the relevant health outcomes under investigation.

In terms of the scope of generalization, the findings were not extended beyond the target population or subject participants or unit of analysis used in the study. In fact, the conclusion derived from the study was not generalized to the entire population. As the study design used to address the posed research inquiries or research questions or hypotheses was a cross-sectional design, the only meaningful inferential conclusion drawn was a correlational association not a causal relationship.

An attempt to draw a causal relationship in this study based on the cross-sectional research design in the absence of an experimental or quasi-experimental study design was not made. A cross-sectional design employed to address the posed research questions and purpose of the study was appropriate. As the purpose of the study was to assess some specified health risk of long-term exposure to cell phone use. The causal links between cell phone use and health outcomes or the mechanisms or mode of action through which long-term cell phone uses or cell phone-driven RFR-EMF exposures lead to the specified adverse health outcomes was not explored. The risk explored in this study was based on establishing the difference in the prevalence of the specified adverse health outcomes (thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease) among long-term cell phone users and non-users.

Limitations

Establishing accurate measurement for the study measures is crucial to reduce spurious errors. Regarding the RFR-EMF exposure around the head or thyroid or heart area while using cell phone to talk, the data source' researchers did not record and stratify participants who used hand-held set or blue-tooth or those who used hands-free device such as a headset or the speaker to talk. The use of a blue-tooth or handheld cell phone to talk may direct RFR-EMF exposure to the head, thyroid, and chest areas than could be possible when the phone is in a speaker mode or hands-free device such as a wired-headset is used. By not recording the use of the specified phone accessories among the target population, the link between the exposure level and the specified health outcomes could be distorted towards or away from the null hypothesis.

The spatiotemporal sequence integrity cannot be definitively determined due the use of a cross-sectional design. In other words, there was no definitive evidence suggesting that the subjects were exposed to cell phone driven-RFR before the onset of thyroid cancer or mouth/tongue/lip cancer or heart condition and vice versa. Based on this limitation inherent to cross-sectional designs, the exposure-outcome sequence validity integrity may have been compromised or misclassified. Also, generalization of the findings on the effect of long-term exposure of cell phone use to the entire United State population outside the target population was not possible in this study.

Generational effect such as limitation in the participants' age among individuals who participated in the study could confound the results (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). Older individuals are predisposed to age-related chronic conditions. As people age, they are likely to develop chronic conditions such as thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease. As well, period effect such as the 'time' of the study or exposure time may not have been long enough to adequately and sufficiently reflect the health-related outcome onset exhibited by the genetic or metabolic or biological damage associated with repeated and long-term exposures of cell phone-driven RF-EMF or cell phone use among the target population (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). Furthermore, thyroid cancer, mouth/tongue/lip cancer, and heart condition or disease reported by the survey participants may not have been clinically confirmed or retrospectively verified by the interviewers.

Significance of the Study

Cell phone ownership and usership have increased substantially not just in the United States but globally, (see Figure 8) (Pettinger, 2012; The World Bank, 2016). There seem to be a plethora of concerns or evidence-based findings suggesting or implicated cell phones-driven RFR to many chronic health outcomes in animals or humans or even the environment (Havas, 2009; Finkenthal et al., 1996; NTP, 2016; Taylor et al., 2011; Vanderpump, 2011). As a result, health practitioners or epidemiologists must employ appropriate health promotion practices to identify, verify, and implement the best health promotion measures to minimize the risk of exposures in order to avoid global chronic health epidemics or pandemics. At best, there should be systematic cell phone RFR exposure/health outcome biosurveillance systems. In contrast, unaddressed increase of cell phone-driven RFR exposures to increase via cell phone ownership and use, especially when evidence of possible and probable life-threatening health risks are reported or shown, is an act of public health negligence. The adverse effect of RFR was demonstrated in plants. In several studies, RFR was linked to the inhibition of plant development pathways, an effect which consequently prevents the normal functioning of the whole photosynthetic systems or plant growth, and induced dwarfism in plants (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec, Malaric, Pavlica, Pevalek-Kozlina, & Vidakovic-Cifrek, 2008).

Adverse health outcomes resulting from exposure to long-term RFR facilitates monetary and non-monetary burdens and perhaps, could promote serious unanticipated ecological or environmental and health consequences. After over 30 years of cell phone

introduction in the marketplace, scientists applied mostly short-term studies to demonstrate the health impacts, environmental implications, and public health issues of cell phone driven RFR emission (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec et al., 2008). With plant seedlings, it was also shown that RFR could inhibit metabolic pathways, cause genetic/chromosomal aberration and could cause mitotic abnormalities by inducing lagging chromosomes, vagrants, disturbed anaphases and chromosome stickiness, and mitotic spindle impairment remarkably depending on the field frequencies, strength, and modulation (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec et al., 2008).

According to Kesari, Siddiqui, Meena, Verma, and Kumar (2013), the inconsistency observed on the biological effects of cell phone-driven RFR or EMF's exposures occurred in part due to the difficulty in controlling the predictor parameters. The biological effects are not only dependent on the proximity and magnitude of the affected unit(s) but involve the environmental parameters as well. Even with such conflicts and inconsistencies in conclusions, some of the health outcomes shown in previous studies were linked to cell phone use (Kesari et al., 2013). These health outcomes are not limited to genotoxic effects, childhood leukemia, neurological effects, cardiovascular effects, neurodegenerative conditions, infertility, immune system deregulation, brain tumors, inflammatory responses and allergic reactions (Kesari et al., 2013). Most if not all the conclusions drawn about cell phone-driven RFR risks on biological effects emphasized that prolonged exposures could lead to harsher health impacts (Kesari et al., 2013). Besides cell phones, regular and long-term use of

microwave devices such as microwave ovens could be associated with adverse effects on biological systems (Kesari et al., 2013; Fehske, Technische Universitat Dresden, Fettweis, Malmodin, & Biczok, 2011). Microwave radiations or RFR exposure increases the level of reactive oxygen species (ROS) and perhaps lead to neurodegenerative conditions (Kesari et al., 2013).

Electromagnetic radiation or RFR is an environmental pollutant, which has serious adverse effects on wildlife and the environment (Fehske et al., 2011). Cell phone-driven RFR is continuously irradiating environmental habitat, and many species could develop long-term adverse effects (Balmori, 2016). Some of the observed effects to species include interference in the natural defense systems, adverse health impacts, reproduction problems, and reduction species population within its natural habitat (Balmori, 2016). Behavioral cues of many animal species; birds, bats, and rats are influenced by the RFR or electromagnetic radiation (Fehske et al., 2011). Increasingly, RFR-driven pollutants or masts have been linked to the decline of animal populations and an increase in the deterioration of plants and animals' cohabitation (Balmori, 2016).

The identified gap in the literature inspired the exploratory intent of this study, in hope that the findings will arouse further objective and long-term epidemiologic investigations of RFR exposure on other health outcomes in humans. The primary justification for conducting the long-term study in human subjects was based on the knowledge that most etiologic risks of or exposure to chronic conditions, its prevalence and incidence, and the disease development are cumulative and long-term (Gordis, 2009). Cell phones have been in the global market for over 30 years. Therefore, the exposure

time for many people has been over three decades. With this study, human subjects who own and use cell phones and meets the specifics of the inclusion criteria defined in this study that fit the long-term exposure parameters were selected. Possible effects of confounders or covariates such as gender, age, race, marital status, and job status were evaluated in the model. Other exposures to ionizing radiation and other known carcinogen were not accounted for because these factors were not recorded in the secondary data.

The findings from this dissertation could be useful in facilitating meaningful industrial-based safety changes and informed knowledge transfer to users. Perhaps, the findings may foster positive social change by encouraging public policy advocacy and health promotion measures on this issue. Ultimately, informed research drives the inclination to change that would be needed to make the necessary changes needed to improve the quality of life of cell phone users. Also, the expected social change given the findings derived from this dissertation is a continuous process that requires further investigation on the topic..

Summary

Even with the extensive literature review on the short-term health effects of cell phone use or cell phone-driven RF-EMF exposure, uncertainties still exist on the published findings. Also, the lack of long-term epidemiological studies on mobile phone use justifiably supports the shared concerns about the need to bridge the uncertainty gaps through the application of long-term epidemiologic studies. With sufficient long-term studies on the effects of cell phone use on chronic diseases, it would be insightful how

the rapid increase in cell phone subscription and adoption increase over the years in the US and rest of the world could affect the population health in the near future.

The purpose of the study was tailored to the assessment of the long-term risk effects of cell phone use the prevalence of thyroid cancer, mouth/tongue/lip cancer and heart disease. Though, the secondary data employed in this analysis was generated via a survey data collection approach through a cross-sectional design. Therefore, it was difficult to advance conclusive suggestions on the temporality of the exposure-outcome sequence. With the application of a cross-sectional design, accurate prediction of the prevalence of thyroid cancer, mouth/tongue/lip cancer and heart disease was made. Detailed information on the literature reviewed and methodology employed are discussed in Chapters 2 and 3 respectively. Also, the data analyses and the conclusion drawn are presented in Chapter 4 and 5 respectively.

Chapter 2: Literature Review

Introduction

The evaluation of the effects of cell phone use on health outcomes was supported by evidence-based research conducted by many researchers. Based on the review of the literature, links between cell phone use and some chronic health conditions has been demonstrated but uncertainty still exists about the long-term implications of cell phone-driven RFR-EMF exposures on thyroid cancer, mouth/tongue/lip cancer, heart condition/disease, and many other chronic conditions (Balmori, 2016; Bolen, 1994; Fehske et al., 2011; Haggerty, 2010; Kesari et al., 2013; Racuciu, 2009; Tkalec et al., 2008). In this chapter, I explored published literature on cell phone health implications. My review includes the theoretical framework, key variables, sampling approaches, research methodology, research design, and relevant research findings of each research inquiry. Also, in this chapter, the literature search strategy and theoretical foundation were discussed. The chapter concludes with a summary of key points.

Literature Search Strategy

Literature for this research inquiry was accessed via the PubMed/NCBI, MEDLINE, Bioelectromagnetics site, epidemiology journals, Walden Library, EBSCO, Google Scholar, and other biomedical journals databases. I identified studies related to cell phone use or cell phone-driven RFR exposure and health outcomes. Very few citations from a non-peer reviewed articles or blogs or websites such as the NIH or CDC were used. The majority of the literature included in this review was published between 2001 to 2016 (See Table 1 for more detail.). A few references that were published

before 2000 were included. One is the original publication by Bronfenbrenner (1979), who pioneered the social-ecological model. Others are foundational research on the biological impacts of RFR in living cells.

Table 1

Distribution of Reviewed Literature, Books, and Websites/Blogs by Year

Literature year	No. of literature items	No. of websites/blogs	No. of books	% of peer-reviewed sources	% of total reference sources
1979	1			1.33	0.88
1985	1			1.33	0.88
1986	1			1.33	0.88
1994	2			2.67	1.77
1995	1			1.33	0.88
1996	1			1.33	0.88
1999	1			1.33	0.88
2001	1			1.33	0.88
2002	5			6.67	4.42
2003			1	0.00	0.00
2004	2			2.67	1.77
2005	4	1	1	5.33	3.54
2006	3		1	4.00	2.65
2007	3	1	1	4.00	2.65
2008	8		1	10.67	7.08
2009	10		2	13.33	8.85
2010	4	1	2	5.33	3.54
2011	8	5	2	10.67	7.08
2012	2	1	1	2.67	1.77
2013	8	2	2	10.67	7.08
2014	2	2	2	2.67	1.77
2015	3	3	1	4.00	2.65
2016	4	5		5.33	3.54
Total	75	21	17		

Grand Total	113
Total % of Peer-Reviewed Lit. list within 5-years	25.33
Total % of Reference List within 5-years	16.81
Total % of Peer-Reviewed Lit. list within 10-years	69.33
Total % of Reference list within 10-years	46.02
Total % of Peer-Reviewed Lit. list within 15-years	88.00

Total % of Reference list within 15-years	58.41
Total % of Peer-Reviewed Lit. list within 20-years	92.00
Total % of Reference list within 20-years	61.06
Total % of Peer-Reviewed Lit. list outside 20-years	6.67
Total % of Reference list outside 20-years	4.42

The query terms used for the literature search were, as follows: *Health outcomes and mobile phone radiofrequency radiation* [MeSH term/research phrase], *Thyroid cancer and mobile phone use* [MeSH term/research phrase], *thyroid problems or thyroid functions and mobile phone use* [MeSH term/research phrase], *thyroid cancer and mobile phone use* [MeSH term/research phrase], OR *thyroid cancer* [TX All Text] AND *mobile phone* [TX All Text]) AND *radiofrequency radiation* [TX All Text]. A search in the MEDLINE EBSCO database using search key terms such as *thyroid cancer* [TX All Text] AND *mobile phone* [TX All Text]) AND *radiofrequency radiation* [TX All Text] generated 1,748 peer-reviewed scholarly articles on the topic.

When the same search criteria were restricted to articles published between January 2006 to December 2016 (within 10 years of my anticipated graduation date), 315 published peer-reviewed articles were generated on the topic. When restricted to January 2011 to December 2016 (within a 5-year period of my anticipated graduation date), 178 published peer-reviewed articles were generated. Similar search techniques were used to find relevant literature in other databases.

Within the search criteria, when *mobile phone* term was changed to *cell phone* and under the same search engine (MEDLINE EBSCO database) as previously specified for literature search within a 5-year period, 77,786 peer-reviewed journals were generated instead of 178 journals. The reason this happened is that MEDLINE EBSCO database

included journals that are linked to *biological cell lines* as the part of the key term instead of limiting the literature search to only *cell phones* publications. Also, when the term *cell phone* was changed to *cellular phone*, a search within a 5-year period populated 39,135 peer-reviewed journals. A simple search with the keywords *cancer* [TX All Text] AND *mobile phone* [TX All Text]) AND *radiofrequency radiation* [TX All Text] generated 145 articles (within a 5-year period of my anticipated graduation date) on the topic. With an advanced search option, when the term *cancer* was used in one search field AND *mobile phone* in another search field AND *radiofrequency radiation* in another, the advanced search populated 63 peer-reviewed articles on the topic. When the search was limited to only humans, it produced 50 articles. However, in many cases, the literature search was not limited to humans. It was necessary, I concluded, to identify experimental design studies that were conducted in animals that could not otherwise be ethically conducted in humans.

Even a search with the terms *thyroid problems* [TX All Text] AND *mobile phone* [TX All Text]) AND *radiofrequency radiation* [TX All Text] within the last 5-years of my anticipated graduation, the keywords generated 2,135 peer-reviewed literature. Similarly, when the field options were not selected or were left blank, a search with *thyroid problems* [blank] AND "mobile phone" [blank]) AND *radiofrequency radiation* [blank] keywords generated 11 peer-reviewed articles on the topic. When limited to humans, the search was narrowed to 7 articles. All searches were restricted to publications written in the English language. The following search syntax is an example of one of the search details from the MEDLINE database: (*thyroid gland* [MeSH Terms]

OR (*thyroid* [All Fields] AND *gland* [All Fields]) OR *thyroid gland* [All Fields] OR *thyroid* [All Fields] OR *thyroid (usp)* [MeSH Terms] OR (*thyroid* [All Fields] AND (*usp*) [All Fields]) OR *thyroid (usp)* [All Fields]) AND functions[All Fields] AND (*cell phones* [MeSH Terms] OR (*cell* [All Fields] AND *phones* [All Fields]) OR *cell phones* [All Fields] OR (*mobile* [All Fields] AND *phone* [All Fields]) OR *mobile phone* [All Fields]).

Theoretical Foundation

Social-ecological theory (SET) is a concept that included sociocultural, sociopolitical, psychosocial, and psychobehavioral operational constructs to provide in-depth intrinsic and extrinsic interaction viewpoints about public settings, organizations, genetic subgroups and individuals in micro-, meso-, exo-, macro, and chrono-systems (Bronfenbrenner, 1979, 1986, 1994, 1995). SET was applied here to advance health promotion measures by addressing the extrinsic (cell phone use/cell phone-driven radiation) interactive links to the intrinsic outcomes such as thyroid cancer, mouth/tongue/lip cancer, and heart conditions within the US population exposed to cell phone driven RFR. It is evident from research findings that cell phone use has both social and biological connections (Balmori, 2016; Bolen, 1994; Fehske et al., 2011; Haggerty, 2010; Kesari et al., 2013; Racuciu, 2009; Tkalec et al., 2008). The interactive relationship observed in this study included the micro-, meso-, exo-, and macro-systems of the SET framework. In this epidemiologic investigation of health promotion measures, the application of the ecological model was an invaluable theoretical concept or conceptual framework in addressing the plausible and possible association between cell phone use and the specified health outcomes.

The application of SET required an in-depth understanding of the biological plausibility of the RFR risks, sources of RFR exposure, environmental interactions associated with cell phone use, effects on genetics and genetic predisposition, human biochemistry, and interaction patterns of radio wave technologies with the unit of analysis. The relevance of preventive or primary practices or approaches for optimizing health, delaying or controlling the onset of chronic diseases and reducing its severity within a target population is linked to the quality and levels of the adverse extrinsic exposures or the SAR absorbed by the body during usage or the rate of exposure (Fenech et al., 2011). Perhaps, incorporating the SET framework in the assessment of cell phone-driven RFR (an adverse extrinsic factor) to health effects provided insightful information on how the advancing knowledge and research in this area could be effectively applied to minimize adverse health outcomes, prevent, delay, and control health conditions associated with cell phone use. With such information, scientists and health practitioners could expand their understanding on how to explain cell phone-driven RFR effects on health outcomes using the SET framework, and how to apply the informed knowledge in transforming risks in modern radio wave-driven technologies, sociocultural behaviors, technological practices, and exploration of EMF and RFR implications to the advantage of the overall population quality of life and environmental integrity.

The interactive relationship between multifactorial variables or units could be explained using the ecological model. The attributes of the interactions play crucial roles in determining the target population quality of life or in the promotion or delay or prevention of adverse health outcomes within the target population (Aschengrau &

Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The model is sectioned into three parts or subgroups, and any of the three sections contained the extrinsic and intrinsic characteristic profiles (Satariano, 2005). An extrinsic factor such as the environment (physical or built or social environment) could be altered or modified, while an intrinsic factor such as race or age or genetics is non-modifiable and inherent or congenital or at a natural state (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Szklo & Nieto, 2014).

The first subgroup of the ecological model included the sociocultural/sociopolitical and psychosocial/psycho-behavioral constructs. It includes demographic (age, gender, race, and ethnicity), environmental (physical and built), social (social capital, living arrangements, social support, and social networks), psychosocial (self-efficacy, social control, and sense of coherence), socioeconomic, and physiological factors (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The second subgroup of the ecological model construct represents quality of life, which involves health and functional outcomes (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The extrinsic or intrinsic factors affects and is affected by other elements in the ecological model mix (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Szklo & Nieto, 2014). Within the ecological model mix, there are intra-interactions within each subgroup sections and inter-interactions between subgroup sections, interactive effects which could lead to health and functional outcomes (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano,

2005; Szklo & Nieto, 2014). The third subgroup section represents the vital status (alive or dead status) (Satariano, 2005).

Often, quality of life (health and functional outcomes) is a good indicator of the vital status. It is imperative noting, the ecological model was not in any way intended to predict specific causal relationships between an independent and dependent variable or across independent, intermediary/covariates/confounders, and dependent variables (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). In contrast, the ecological model represents a simple heuristic approach through which the epidemiologic processes and interplay or roles of intrinsic and extrinsic factors on quality of life and vital status could be explained (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The epidemiologic processes or the triad factors include the period or time, place or location, and persons or subjects which is often referred to as the 3 p's (period, place, and persons) (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). Factors which are critical in the distribution of a disease process or an adverse health condition (chronic conditions or infectious diseases or communicable diseases or even genetic conditions) (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Szklo & Nieto, 2014). The following figures from Satariano (2005) publication illustrated the interactive interplay of the interrelated (intra-/inter-) links of the ecological model (see Figure 5 for more details). On the other hand, Figure 6 is a reconstructed ecological model was tailored to represent the interrelated (intra-/inter-) links between mobile phone use or cell phone-

driven RF-EMF exposures and health outcomes (thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease) using the ecological model constructs.

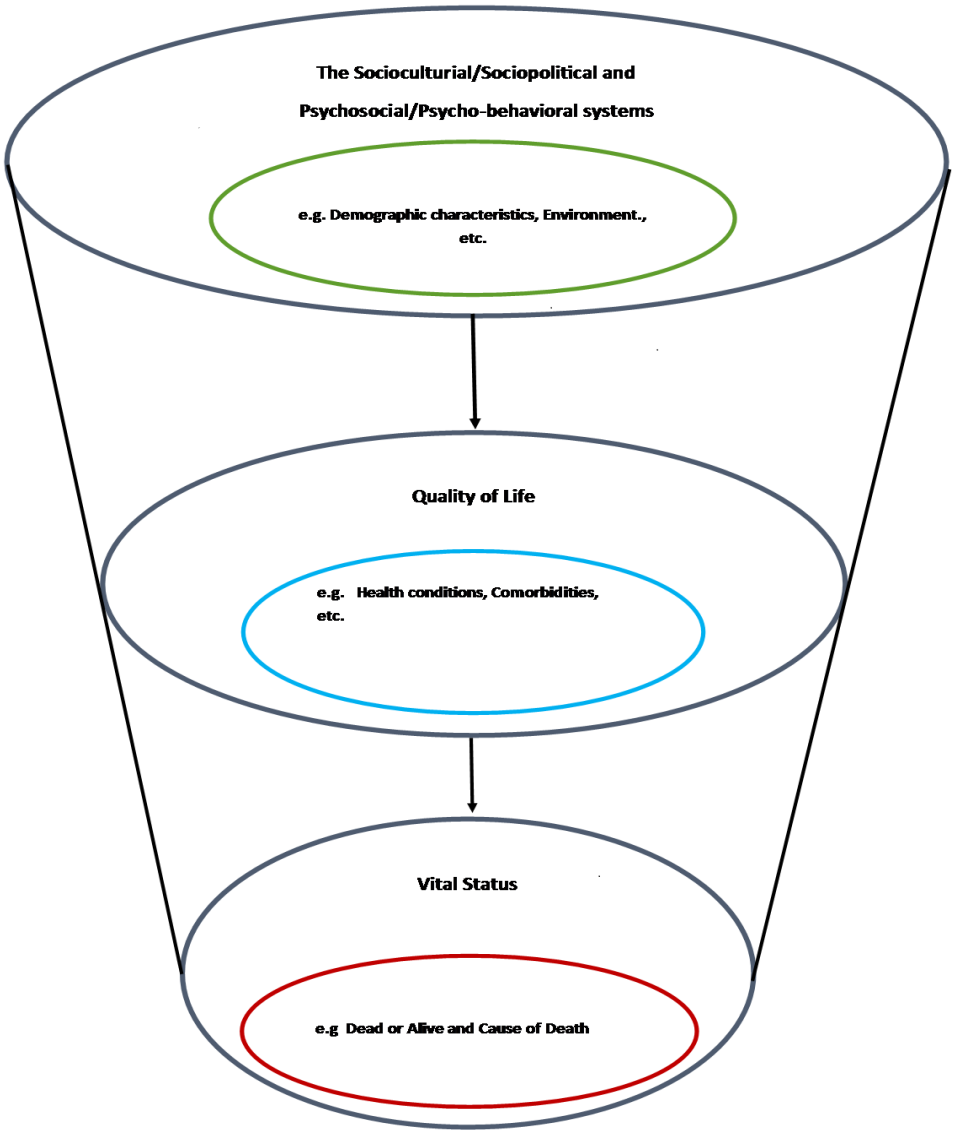


Figure 5. Ecological Model.

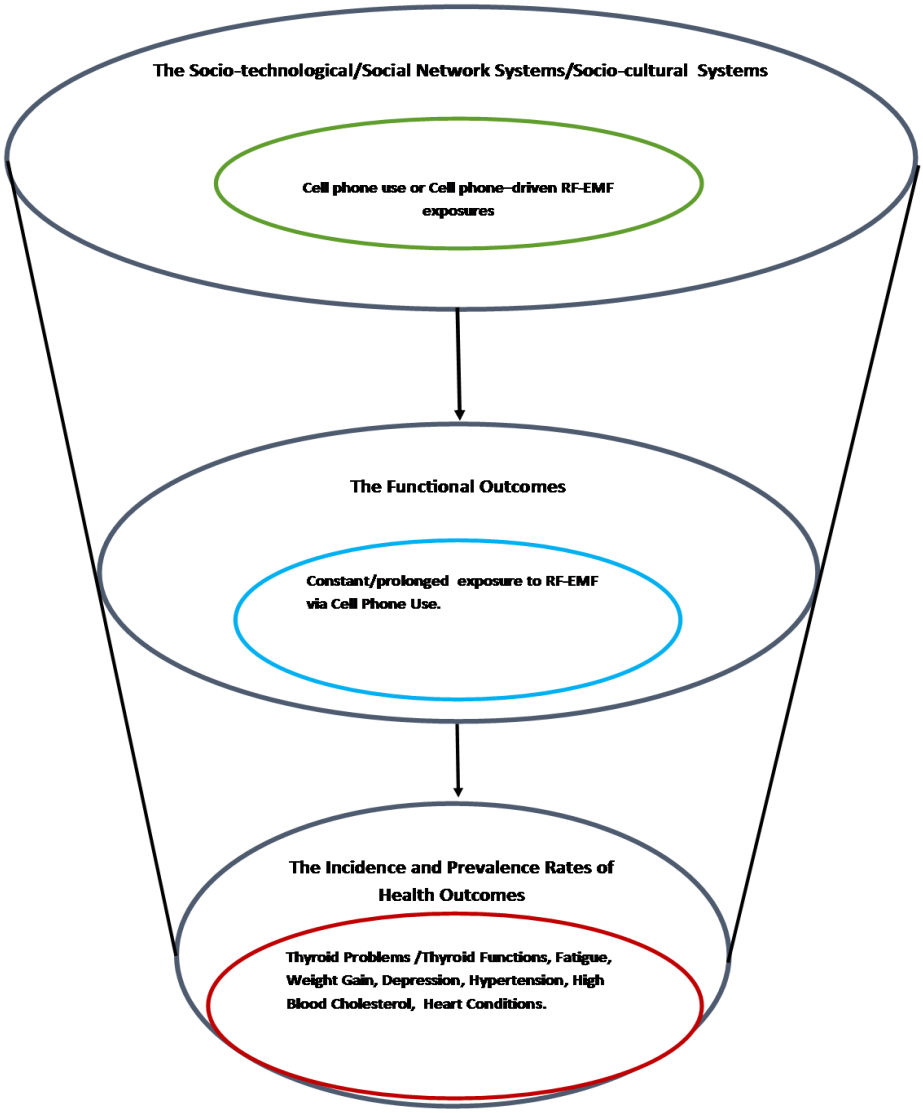


Figure 6. Ecological Model in relation to Mobile Phone use and Health Outcomes.

The application of the ecological model by Campbell, Dworkin, and Cabral (2009) on the evaluation of the impact of sexual assault on women's mental health is an example of how an ecological model could be applied in a variety of public health or population health or epidemiologic investigations. The epidemiologic factors that facilitate target population health assessment involve an in-depth understanding of the

persons, place, and period of the event. The integration of these three key factors in epidemiologic evaluations, especially in health outcomes, is a major part of the investigative scope of work within the ecological model constructs. Using the ecological model, Campbell, Dworkin, and Cabral (2009) represented the individuals or persons level (e.g., sociodemographics, biological/genetic factors), assault characteristics (e.g., victim-offender relationship, injury, alcohol use), microsystem factors (e.g., informal support from family and friends), meso/exosystem factors (e.g., contact with the legal, medical, and mental health systems, and rape crisis centers), macrosystem factors (e.g., societal rape myth acceptance), and chronosystem factors' (e.g., sexual revictimization and history of other victimizations) effects on mental health outcomes (e.g., post-traumatic stress disorder, depression, suicidality, and substance use) among sexual assault survivors.

Similarly, the ecological model representation of the interactions on how cell phone use affects quality of life (health outcomes-thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease) of persons who own/use cell phone or accustomed to heavy use of mobile phone were presented in this study as follows: The individuals or persons level factors represented the sociodemographics e.g., gender, age. Cell phone use characteristics or behavior included cell phone-driven RFR exposure among users, headset use or blue tooth use or speaker phone use or hands-free use. The microsystem factors included the use of other alternative forms of communication such as home landline telephone and its accessibility and availability. The meso and exosystem factors involve the evaluation of the attitude and ideology about cell phone culture. The

macrosystem factors included the assessment of the societal concept on cell phone or smartphone technology trend or usability. The chronosystem factors on the other hand, dealt with the life course perspective and historical account of the individual or societal quality of life as it related to cell phone use.

Literature Review Related to Key Variables and Concepts

Cell Phone Use Prevalence

The fact that uncertainties on the safety of cell phone use or cell phone-driven RFR exits, is indicative of the need to pursue further epidemiologic research on the topic. The ubiquitous and constitutive proliferation of cell phone ownership and usership have increased substantially not just in the US or UK but globally, (see Figure 7 and 8) (Pettinger, 2012; Statista, n.d.; The World Bank, 2016). Cell phone subscriptions in South Korea, Sweden, and United States increased substantially over the years, (see Figure 9) (Carlberg, Hedendahl, Ahonen, Koppel, & Hardell, 2015; CTIA, 2011; The World Bank, 2016). The global cell phone subscription is now over 7 billion, (Figure 8) (The World Bank, 2016; GSMA Intelligence, 2016). The incidence of thyroid cancer in the US, Canada, Israel, and many other countries are rapidly increasing in recent years (Safer EMR, 2016). A similar increase was observed in the Republic of Korea as well, and from the observation, Ahn, Kim, and Welch (2014) indicated that even in 2011 when the thyroid-cancer mortality remained relatively stable, the incidence rate of thyroid-cancer diagnoses in the Republic of Korea was 15 times greater that the incidence rate observed in 1993. They concluded that the rise in the incidence rate is attributed to over-diagnosis as a result of the widespread thyroid-cancer screening (Ahn, Kim, & Welch,

2014). In the mist of the potential uncertainties and gaps followed by the high prevalence of cell phone use/ownership, it is undoubtedly necessary and of public health interest to understand how the increase in the prevalence of cell phone use and ownership under the current transmittable radiofrequency radiation exposure impacted the global health outcomes, environmental health, and public health promotion measures.

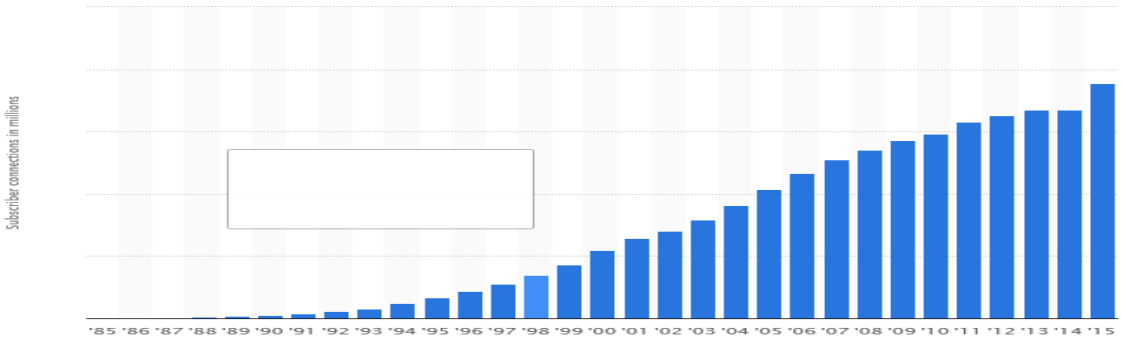


Figure 7. Mobile phone subscription in the US. (CTIA, 2011).

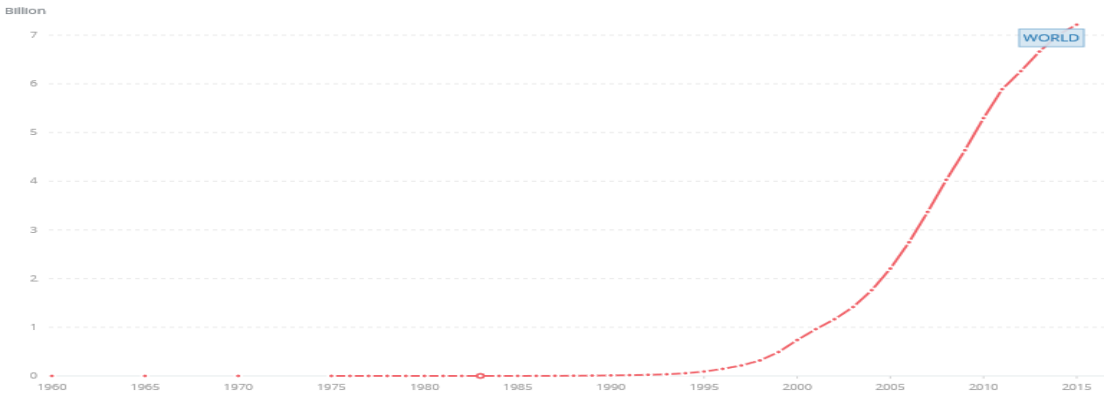


Figure 8. The global phone subscription (The World Bank, 2016; GSMA Intelligence, 2016)

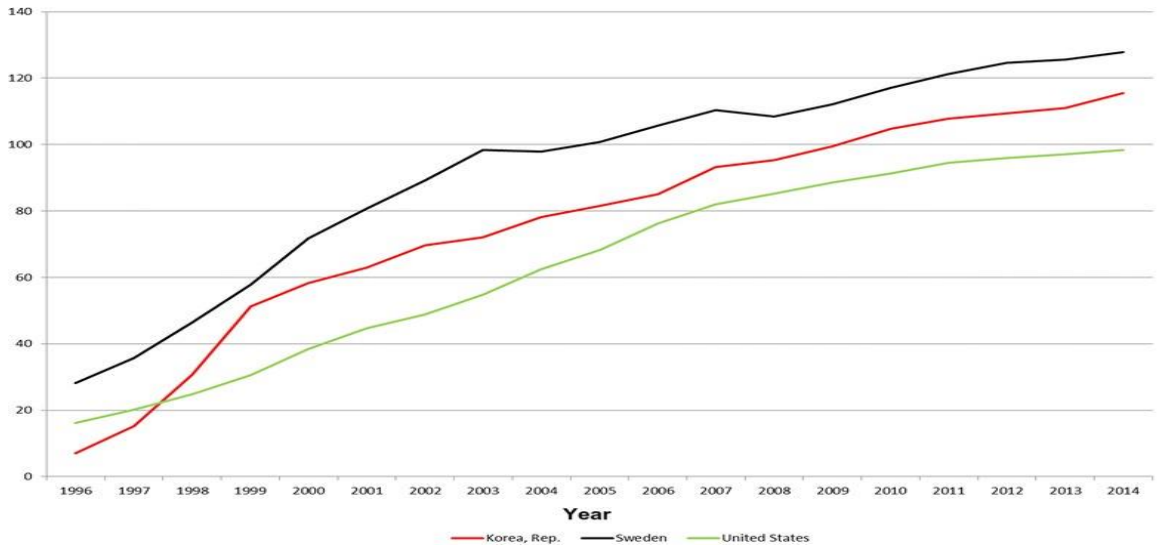


Figure 9. Mobile phone subscriptions per 100 persons in South Korea, Sweden and United States. (Carlberg, Hedendahl, Ahonen, Koppel, & Hardell, 2015).

Cell Phone-Driven RFR Transmission and Frequency Range

Over the years, a substantial increase of cell phone ownership and usership has been established. The primary and common electronic source of exposure to RFR among the specified target population in this current study was via cell phone use. In other words, the current mobile phone technology used by every subscriber emits RFR during the signal transmission cycle (Finkenthal, Greco, Halsey, Pena, Rodecker, Simms, …. & Schissel, 1996; NASA, 2010; NTP, 2016; Zamanian & Hardiman, 2005). Radiofrequency radiation or radio wave or microwave energy is a type of non-ionizing electromagnetic energy (Finkenthal et al., 1996; NTP, 2016). Within the electromagnetic frequency spectrum particularly the non-ionizing energy, mobile phone emits or transmits RFR between the range of 108 to 1012 radiofrequency (European Commission, 2005; NASA, 2010; Finkenthal et al., 1996; Zamanian & Hardiman, 2005).

The mobile phone radio frequency emission is within the ‘thermal high induced current’ or ‘heat generated’ energy frequency (European Commission, 2005; Finkenthal et al., 1996; Zamanian & Hardiman, 2005). Globally, the current cell phone technology is either modulated through technology features such as the Nordisk MobilTelefoni or Nordiska MobilTelefoni-Gruppen (NMT) or Global System for Mobile Communication (GSM) or Code Division Multiple Access (CDMA) or Long-Term Evolution (LTE) communication systems. Any of these radio access features could be used in cell phone transmission depending on the providers’ wireless network connection or the country’s preferred network system(s). Regardless, all the current wireless systems emit RFR or radio waves. See Figure 10 and 11 for the more information on the emission frequency of RFR.

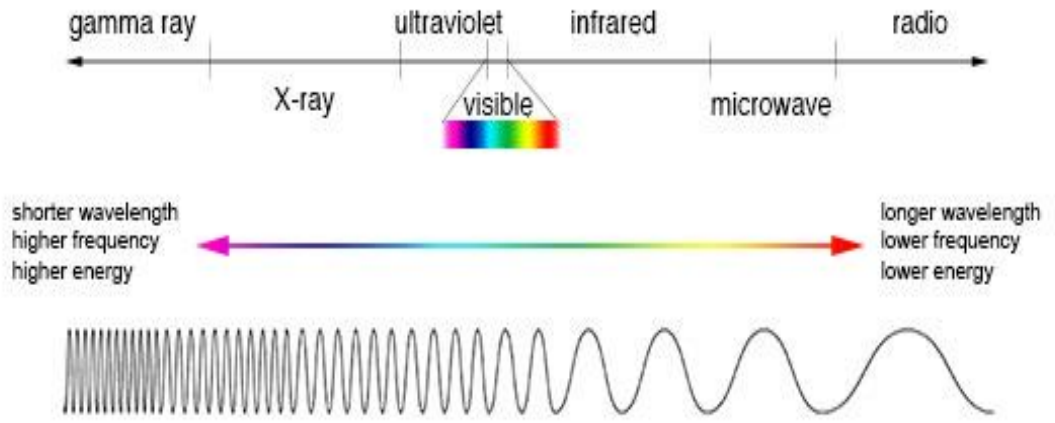


Figure 10. Electromagnetic Spectrum. (Source: https://imagine.gsfc.nasa.gov/science/toolbox/emspectrum1.html).

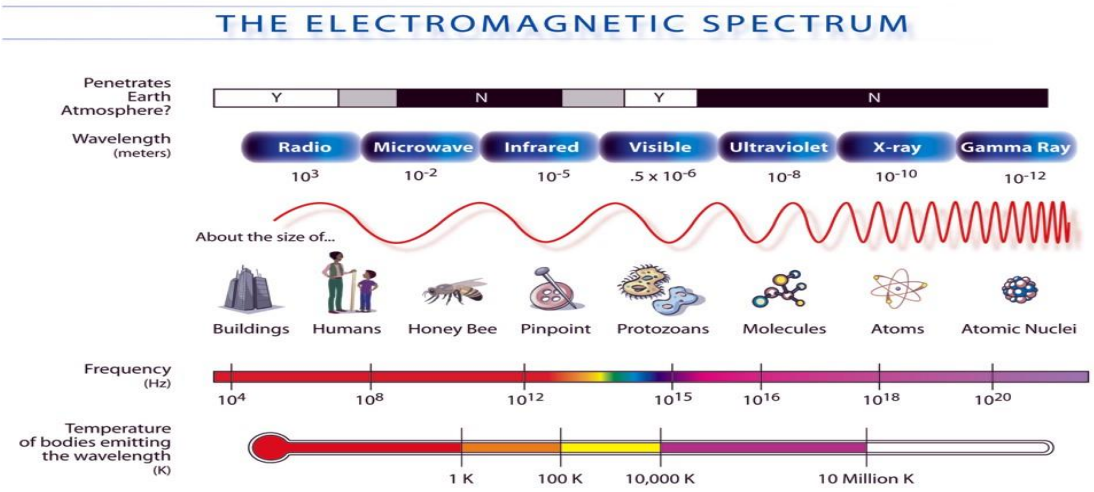


Figure 11. Electromagnetic Spectrum, Frequency Wavelength, and Emitted Temperature Range. (https://goo.gl/images/uilE5W).

According to Aghav et al. (2013), the power emitted or transmitted by a cell phone is in the range of 1 to 2 watts. The frequency ranged from 824 to 849 MHz for the CDMA modulation system, 890 to 915 MHz for the GSM-900 modulation, and 1710 to 1780 MHz for GSM-1800 modulation (Aghav et al., 2013). Tsung-Chieh and Hung-Wen (2010) reported that the current cell phones radio frequency operates in the range of 900-1800 MHz. Saini and Pandey (2013) indicated that during the cell phone calling mode a significant change to high RFR exposure occurs because a cell phone would emit the highest power during the calling mode transmission. According to Taylor, Wargo, Alderman, Bradley, and Addiss (2011), radiation exposures emitted via cell phones vary based on the antenna, phone model, configuration, and signal strength. They emphasized that a phone with weak signal strength produces higher levels of RFR or microwave exposure (Taylor et al., 2011).

Based on the frequency range of radio waves emitted or transmitted by cell phone communication systems, cell phone RFR is considered a high frequency (HF) or very high frequency (VHF) or ultra-high frequency (UHF) emitter or radio wave (European Commission, 2005; Finkenthal et al., 1996; Herter, 1985; NASA, 2010; Mignone & Barnes, 2011; Zamanian & Hardiman, 2005). Unlike other radio or microwave emitters or transmitters such as the TV and radio systems, cell phones emit or transmit radiofrequency radiation to the user or body within a proximal distance because of its utility and design application compared to the TV or other radio systems. As a result, many scholars and public health practitioners are concerned about the effect. Based on the plausible uncertainty and health outcomes associated with cell phone use, Sivani and Sudarsanam (2012) explored the possible effects of radiofrequency electromagnetic field (RF-EMF) on the biosphere. They indicated that at high or lower intensities, RF-EMF radiation exposure within the biological or molecular levels influences the neurotransmitter functions, calcium efflux, electrophysiology, cellular metabolism, blood-brain barrier, cellular and tissue morphology, gene functions, and protein expression in certain types of cells (Sivani & Sudarsanam, 2012).

They also noted that there are scarce short-term epidemiologic evaluations performed on the impacts of RF-EMF radiation in animals and insects such as frogs, bats, house sparrows, humans, honey bees in India. And at its worse, there are no long-term epidemiologic studies on the RF-EMF radiation impacts in animals while cell phone users and subscribers are substantially increasing in India (Sivani & Sudarsanam, 2012). As a result, the overall macro-biological or population-based health consequences of

constant and long-term cell phone-driven RFR exposure is unclear and could present serious health threats in public health measures if not appropriately addressed (Sivani & Sudarsanam, 2012). Therefore, there is urgent need to identify the frequency, intensity, and duration of non-ionizing electromagnetic fields associated with biosystem and ecosystem degradation (Sivani & Sudarsanam, 2012). Such measures could produce informed decision strategies in mitigating the possible health impacts associated with cell phone-driven RF-EMF radiations (Sivani & Sudarsanam, 2012). Also, users and technology innovators could integrate the information into product quality improvement as an opportunity to reduce RFR emission and re-educate the public on the proper use of wireless technologies (Sivani & Sudarsanam, 2012). Consequently, the informed awareness could help to ensure and maintain or improve the population health and environmental health integrity (Sivani & Sudarsanam, 2012). Also, long-term studies should be conducted to assess the effect of RF-EMF exposure on early-life and prenatal health outcomes (Sivani & Sudarsanam, 2012). The future epidemiologic study focus should be on children and young adults' behavioral modification to understand the link between neurological disorders and cancers, and its associations to cell phone-driven RF-EMF exposures (Sivani & Sudarsanam, 2012).

Plausibility of RFR and Biological Effects

Within the current body of literature, there is a lack of sufficient long-term human epidemiological studies on the association between cell phone-driven RFR and thyroid cancer, mouth/tongue/lip cancer, and heart conditions. Uncertainties and absence of conclusive evidence are concerns that facilitated the need for a meaningful long-term

epidemiologic study on the safety or impact of RFR among cell phone users. Based on these indicators, the need for long-term epidemiological studies is urgently warranted. However, through short-term studies, the link between cell phone use or cell phone-driven RFR exposure and many chronic conditions such as malignant glioma, glial cell hyperplasia, schwannoma, Schwann cell hyperplasia, other types of cancers and tumors, damage to fertility and reproduction damage to biological process, genotoxic effects, etc. has been demonstrated (Finkenthal et al., 1996; NTP, 2016; O'Neill, Teo, Davis, Henshaw, Lamburn, Maisch…& Ahonen, 2011; Zamanian & Hardiman, 2005).

The findings described by O'Neill, Teo, Davis, Henshaw, Lamburn, Maisch…and Ahonen (2011) suggested an increase in cancers and other tumors, damage to fertility and reproduction cells, genotoxic effects, and damage to the biological process. After a rigorous review of the evidence provided in the prior literature or past studies, they concluded that children and young individuals are prone to the negative long-term impacts of cell phone use(O'Neill et al., 2011). As a result, the risk of harm to cell-phone driven RFR or cell phone use among users has increased (O'Neill et al., 2011). Also, among individuals who used cell phones for 10 years or more, and for the duration of 30 minutes or an hour per day, a doubling of the incidence rate or risk of some brain tumors have been reported (O'Neill et al., 2011). There is also a relative association between cell phone use and the increase in the prevalence of parotid or salivary gland tumors (O'Neill et al., 2011).

In addition, the International Agency for Research on Cancer (IARC), an agency of the World Health Organization (WHO) in charge of research on cancer, classified RF-

EMF range 30 kHz-300 GHz as a Group 2B risk factor (IARC, 2011; O'Neill et al., 2011). In other words, that RFR is, 'possibly' carcinogenic factor to humans, which served as evidence to the potential health threat associated with cell phone use or cell phone-driven radiation (IARC, 2011; O'Neill et al., 2011). In furtherance, the association between RFR and sperm damage, impairment of female fertility, and damage to the unborn fetus were demonstrated in previous studies (O'Neill et al., 2011). The genotoxic impacts reported in several publications showed substantial impairment of DNA repair mechanism, damage to the DNA strands, and effects on gene expression (IARC, 2011; O'Neill et al., 2011).

According to O'Neill et al. (2011), the effects of cell phone use on the blood-brain barrier and reduction of the melatonin levels in humans after about 30 minutes of cell phone use per day were demonstrated. Cell phone use was also shown to affect the heat shock proteins to induce a stress response effect (O'Neill et al., 2011). Oxidative stress, cell apoptosis and damage to cell membrane were found to be associated with cell phone use or cell phone-driven RFR (O'Neill et al., 2011). It was shown that children's brain tissue was sensitive and highly conductive to cell phone-driven RFR, which suggested that RFR penetrates more readily in children's brain than in an adult and a bigger head size (O'Neill et al., 2011). As a result, prolonged and constant exposure of children to cell phone driven RFR, doubled the absorption rate of RFR to the head compared to a larger head (O'Neill et al., 2011). In furtherance, cell phone energy absorbed by children heads are concentrated in certain areas of the child's brain, and such localization was estimated at 3 times the absorption rate of RFR in that area of the brain (O'Neill et al.,

2011). Overall, it was concluded that the risk of brain cancer or tumor after prolonged mobile phone use or cell phone-driven RFR exposures was statistically significant in younger users when compared to adult users (O'Neill et al., 2011).

In support of plausible negative effects of prolonged cell phone use on biological systems, Kesari et al. (2013) emphasized the conflict and inconsistency or uncertainty in the previous conclusions regarding the biological effects of cell phone-driven RFR or EMF's exposures. As much, highlighted that the inconsistency or uncertainty was profoundly affected by the difficulty in maintaining a tightly controlled predictor parameter in a study (Kesari et al., 2013). Even in the presence of such conflicts and inconsistencies or uncertainties, several findings showed links between biological effects and cell phone-driven RFR exposures (Kesari et al., 2013). Effects which are not limited to childhood leukemia, genotoxicity, neurologic disease , cardiovascular condition, neurodegenerative, infertility, immune system deregulation, brain tumors, inflammatory responses, and allergenicity (Kesari et al., 2013). As such, Kesari et al. (2013), emphasized that biological effects are not only dependent on the proximity and magnitude of the affected unit(s), but involved the intricate relationship between the extrinsic or environmental parameters and intrinsic or biological systems. Hence, cell phone-driven RFR effects on bio-systems is heavily dependent on prolonged RFR exposures (Kesari et al., 2013). Therefore, regular and long-term uses of microwave devices including microwave ovens and cell phones are possible predictors of adverse effects on biological systems (Kesari et al., 2013). In addition, increased level of ROS

enhanced the effect of microwave radiations or RFR, which could lead to neurodegenerative conditions (Kesari et al., 2013).

Other biological effects reported on cell phone-driven RFR emission or exposure implicated not only the altering effects on biological mechanisms of animals but plants and environment health as well (Balmori, 2016; Fehske, Technische Universitat Dresden, Fettweis, Malmodin, & Biczok, 2011; Kesari et al., 2013; NTP, 2016; Tkalec et al., 2008). As a result, electromagnetic radiation or RFR is an environmental pollutant with the ability to adversely affect wildlife and the environmental habitats (Fehske, et al., 2011). According to Balmori (2016), cell phone-driven RFR through cell phone use continuously irradiates environmental habitat to induce adverse effects. As a result, many species could be affected by the long-term biological or environmental stress due to the RFR exposures (Balmori, 2016). These biological or environmental effects are not limited to the natural defense system reduction, reproduction problems, and species population reduction within its natural habitat (Balmori, 2016). The RFR-driven pollutants (cell phone masts or constant cell phone-driven RFR emissions) have been linked to the decline of animal populations and health deterioration of plants and animals cohabitation (Balmori, 2016). In addition, Balmori (2016) emphasized the potential and possible behavioral response associated with RFR or EMR in many animal species; birds, bats, and rats.

Even with plants, the adverse biological effects of RFR demonstrated by researchers were troubling. In plant, RFR exposure inhibited development pathways and consequently inhibited the whole photosynthetic pathways, growth pattern, and even

induced dwarfism (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec, Malaric, Pavlica, Pevalek-Kozlina, & Vidakovic-Cifrek, 2008). Upon repeated and constant plant seedlings exposure to RFR, it inhibited the metabolic pathways (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec et al., 2008). The exposure caused genetic or chromosomal aberration and mitotic abnormalities by inducing lagging chromosomes, disturbed anaphases and chromosome stickiness, and lead to mitotic spindle impairment (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec et al., 2008). The level of the damage heavily depended on the field frequencies, strength, and modulation of the RFR (Bolen, 1994; Haggerty, 2010; Racuciu, 2009; Tkalec et al., 2008). These biological findings presented sufficient evidence to support further studies on cell phone use and possible link to serious adverse health effects in humans, animal, plants and the environment health issues (Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013; NTP, 2016; Tkalec et al., 2008).

An '*animal-based experimental design*' is inherently the best research model employed by scientists to demonstrate possible public health issues that otherwise would produce negative ethical qualms or concerns if conducted in human subjects. Bas, Odaci, Kaplan, Acer, Ucok, and Colakoglu (2009a) explored the impact of 900 MHz RF-EMF exposures on the qualitative and quantitative functions of hippocampal pyramidal cells in the adult female rats. In the study, an animal model-based experimental design was used to investigate the impact of 900 MHz RF-EMF radiation exposure on pyramidal cells development in the cornu ammonis (CA) using 16-week-old female rats. After birth (postnatal), the 16-week-old rats' hippocampus were exposed to 900 MHz RF-EMF

radiation (Bas et al., 2009a). Three study conditions were established; the control group (Cont), the sham group (Sham), and the experimental group (EMF exposed) (Bas et al., 2009a). Throughout the duration of the study, the rats in the control group were not placed in the exposure tube and never exposed to the RF-EMF radiation (Bas et al., 2009a). The rats in the Sham group were in the exposure tube for 28 days, 1 hour per day, but did not receive any RF-EMF radiation exposure (Bas et al., 2009a). The rats in the experimental group were exposed to 900 MHz RF-EMF radiation for 28 days, 1hour per day within the exposure tube chamber (Bas et al., 2009a).

The SAR for the rats in the experimental group (EMF exposed) ranged from 0.016 (whole body) and 2 W/kg (in the head) (Bas et al., 2009a). From the necropsy samples of the rats in all the three groups (Control, Sham, and Exposed), the number of pyramidal cells in the CA were calculated, and the histopathological evaluation of the CA regions of the hippocampus were assessed (Bas et al., 2009a). From the comparative assessment of the quantitative number of pyramidal cells in the CA regions for all the three groups, a 900 MHz RF-EMF exposure on postnatal rats significantly decreased the number of pyramidal cells in the CA region compared to the numbers observed in rats in the other groups (the Control and Sham) ($p<0.05$) (Bas et al., 2009a). For the qualitative analysis, cell loss was observed in the CA region for the rats in the exposed (EMF group) compared to the other groups (the Control and Sham) (Bas et al., 2009a).

In a follow-up study, Bas, Odaci, Mollaoglu, Ucok, and Kaplan (2009b) using an experimental research design investigated whether chronic prenatal exposure to the 900 MHz EMF induced pyramidal cell loss in the hippocampus of newborn rats. The lack of

investigation on prenatal exposure to EMF or its effects on the development of the pyramidal cells of the cornu ammonis in postnatal prompted the need for the follow-up study (Bas, Odaci, Mollaoglu, Ucok, and Kaplan, 2009b). In the study, pregnant rats in the control group were not exposed to EMF while the pregnant rats in the experimental group were exposed to 900 MHz EMF radiation (Bas et al., 2009b). The exposure occurred during the 1st to 19th gestation days (Bas et al., 2009b). The offspring rats were delivered for both the control and experimental groups (Bas et al., 2009b). After delivery, a necropsy procedure was performed on the offspring rats at the end of the 4th week (Bas et al., 2009b). From the optical fractionator assessment of the rats' cornu ammonis (both the control and experimental groups), the exposure to 900MHz EMF radiation significantly reduced the total number of the pyramidal cells in the cornu ammonis in the exposed rats (experimental group) in comparison to the control group ($p < 0.001$) (Bas et al., 2009b).

Beason and Semm (2002) expanded the discussion of the RFR effects by examining the responses of neurons to the amplitude modulated microwave stimuli. In the study, they evaluated possible effects of pulsed RFR signals on neurons of the avian brain (Beason & Semm, 2002). The pulsated microwave signal stimuli used in the study were at similar frequency and magnitude (900 MHz, modulated at 217 Hz) as those produced by the current cell phone communication network systems (Beason & Semm, 2002). Based on the observation, the microwave or RFR-EMF stimulation induced neural activity changes in more than half of the brain cells (Beason & Semm, 2002). About 76% of the cells responded to the microwave or RF-EMF-induced stimulus, and

on average, increased their rates of firing by 3.5-fold (Beason & Semm, 2002). Other cells that did not increase their rate of firing showed a decrease in the rates of spontaneous activities, which suggested that cell phone-driven RFR exposure poses potential adverse biological effects (Beason & Semm, 2002).

Through an experimental design approach, Belyaev, Hillert, Protopopova, Tamm, Malmgren, Persson… and Ringdahl (2005) exposed healthy (non-hypertensive) subjects and individuals reported as hypersensitive to EMF or microwaves. The source of the EMF or microwaves was from GSM modulated mobile phone (Belyaev et al., 2005). The characteristics of the EMF exposure were of 915 MHz emission frequency, 37 mW/kg SAR and 50 Hz magnitude field (power) and 15 muT peaks (Belyaev et al., 2005). The selected subjects or donors were stratified by gender and age (Belyaev et al., 2005). The sample analysis from the two groups (hypersensitive and healthy cohorts) was performed using a blind approach (Belyaev et al., 2005). The evaluation of the changes in chromatin conformation was measured using the anomalous viscosity time dependencies (AVTD) method (Belyaev et al., 2005). The 53BP1 protein surrounding the foci with DNA double-strand breaks (DSBs) was analyzed using the immunostaining in situ technique (Belyaev et al., 2005). The RFR exposure induced at room temperature by either 915 MHz or 50 Hz EMF or MW statistically predicted the condensation of chromatin conformation (Belyaev et al., 2005). The change response was similar to the effect of heat shock induced at 41 degrees centigrade (Belyaev et al., 2005).

The was a statistically significant difference in the response between healthy individuals and hypersensitive subjects (Belyaev et al., 2005). For exposed, there was a

distinct decrease in background level of 53BP1 signaling as well as after heat shock treatments (Belyaev et al., 2005). The decrease confirmed the AVTD analysis and perhaps indicative of the response in the decrease of 53BP1 and antibodies produced from a stress-induced chromatin condensation (Belyaev et al., 2005). However, the 915 MHz or 50 Hz MW exposure did not induce the 53BP1 foci (Belyaev et al., 2005).

With the pulsed-field gel electrophoresis (PFGE), apoptosis, morphological changes and apoptotic fragmentation of DNA were assessed (Belyaev et al., 2005). There was no apoptosis induced by exposure to 915 MHz and 50 Hz microwaves (Belyaev et al., 2005). A 915 MHz microwave and 50 Hz magnetic field induced comparable responses in lymphocytes in healthy and hypersensitive subject-donors (Belyaev et al., 2005). The induction was similar to the stress response induced by heat shock (Belyaev et al., 2005).

With the animal model, Belyaev et al. (2006) further explored the impacts of 915 MHz MW exposure transmitted or emitted via the GSM. The focus of the study was the MW impacts on rats' brain, gene expression, DNA breaks, and changes in chromatin conformation (Belyaev et al., 2006). The experimental rats were exposed to 915 MHz MW, and the control rats were exposed to the sham condition (2-hours) (Belyaev et al., 2006). The output power level was 2W, and the SAR for the MW radiation absorbed by the rats was 0.4 mW/g (Belyaev et al., 2006). The cell samples collected from the rats' brain, spleen, and thymus after the exposure were analyzed (Belyaev et al., 2006). The RNA extracted from the rats' cerebellum was analyzed for the gene expression and integrity. The assessment of the changes in chromatin conformation was used as the

basis for the evaluation of the stress response and genotoxic effects using the AVTD method (Belyaev et al., 2006). The gene expression characteristic profiles were measured using the Affymetrix U34 GeneChips consisting of 8800 rat genes and was analyzed with the compatible Affymetrix Microarray Suite (MAS) 5.0 software (Belyaev et al., 2006). For all the exposed rats, 11 genes in the cerebellum were upregulated in a range of 1.34-2.74-fold, and one gene was downregulated to about 0.48-fold ($p < .0025$) (Belyaev et al., 2006). The induced genes encode proteins with diverse regulatory functions including neurotransmitter regulation, blood-brain barrier (BBB), and melatonin production (Belyaev et al., 2006). The DNA double-strand breaks were analyzed using PFGE, and from the gel analysis, there were no detectable effects of the 2-hour 915 MHz GSM MW exposure on chromatin conformation and DNA DSBs (Belyaev et al., 2006).

Another biological plausibility on the impact of radiofrequency radiation emitted or transmitted via cell phones was demonstrated by Aghav, Tiwari, and Yande (2016). They evaluated the health impact of cell phone-driven RFR and RFR emitted from cell phone towers (Aghav, Tiwari, & Yande, 2016). In the study, 25 healthy human subjects (10 males and 15 females) aged 21-25 years old were exposed to cell phone RFR through a continuous calling mode for 35 minutes (Aghav et al., 2016). The heart rate (HR), mean arterial pressure (MAP), respiration rate (RR), the saturated percentage of oxygen (SPO2), and body temperature were the common health indicators assessed in the study. Similarly, the fixed exposure parameters or predictor variables explored were GSM SIM (AIRCEL), transmitter and receiver of cell phone handset (Moto G 3rd Gen-XT 1550),

the proximity between the caller and receiver, time of observations, and other relevant external parameters (Aghav et al., 2016). The observed RFR emission statistically predicted effects on pulse rate, MAP, and heart rate (Aghav et al., 2016).

Heart rate variability is one of the best representatives of the functionality of the autonomic nervous system (ANS) and a good indicator of pathological and physiological conditions. Saini and Pandey (2013) explored the effects of cell phone and base station transceiver (BTS) radiation on heart rate variability among 19 healthy male participants within the age group 23±4.3 years (Saini & Pandey, 2013). The heart rate variability was measured using the ECG during microwave radiation exposures (Saini & Pandey, 2013). The measurement exponent decreased when the cohorts experience higher radiation levels. The observed change demonstrated that cell phone radiation exposure influenced or caused the heart rate variability (Saini & Pandey, 2013). The change varied significantly with radiation level (Saini &Pandey, 2013).

Alhusseiny, Al-Nimer, and Majeed (2012) examined the effects of cell phone-driven RFR interference on cardiac conduction in patients with a history of ischemic heart disease. In the study, 356 participants (129 males and 227 females) were separated into three groups. Subjects without cardiac diseases (Group I), patients with ischemic heart conditions (Group II), and patients with a history of cardiac conditions not related to myocardial ischemia (Group III) (Alhusseiny, Al-Nimer, & Majeed, 2012). For the individuals in each group, a cell phone was placed at the belt level and over precordium (Alhusseiny et al., 2012). The cell phone was set in a turn-on ringing mode for 40 seconds (exposure) (Alhusseiny et al., 2012). The heart electrocardiogram readings

recorded in the turn-off mode were the baseline (Alhusseiny et al., 2012). Analyzed electrocardiogram readings among the exposed showed a statistically significant prolongation of the corrected QT (QTc) interval among the males in the Groups I and III ($p < 0.001$) (Alhusseiny et al., 2012). In the Group II, the QTc interval prolongation and change in the voltage criteria in male patients were statistically significant ($p = 0.01$ and $p = 0.001$ respectively) (Alhusseiny et al., 2012). The statistical changes observed among the male participants were not identified in female patients with ischemic heart conditions (Alhusseiny et al., 2012).

Djeridane, Touitou, and de Seze (2008) explored the influence of EMF/RFR emitted by GSM-900 mobile phone on the circadian patterns of gonadal steroids (cortisol and testosterone), adrenal, and pituitary (thyroid-stimulating hormone, growth hormone, prolactin and adrenocorticotropin) hormones in men. In the experimental setting, subjects were exposed to RF-EMFs via the use of a mobile phone for approximately 4 weeks (Djeridane, Touitou, & de Seze, 2008). The duration of the exposure was 2 hours per day and 5 days per week (Djeridane et al., 2008). The short-term exposure was performed to assess the biological plausibility effects of cell phone-driven RF-EMF on gonadal, adrenal, and pituitary glands (Djeridane et al., 2008). In 15-day intervals, four sampling sessions were performed as follows: A pre-exposure (before exposure) period, mid-exposure (in the middle of exposure period), post-exposure (end of or after the exposure) period, and 15 days later (post-test) samplings were collected (Djeridane et al., 2008).

Blood samples from the subjects were collected hourly at night time and every 3 hours during the daytime (Djeridane et al., 2008). The corresponding pre-exposure hormone concentration was the control baseline for each participant (Djeridane et al., 2008). The test parameters evaluated included maximum serum concentration, the time of the maximum, and hormone circadian patterns curve (area under the curve) (Djeridane et al., 2008). Based on the analyses, the circadian characteristic profiles of thyroid-stimulating hormone, adrenocorticotropin, prolactin, and testosterone were not disrupted by the mobile phone-driven RF-EMFs (Djeridane et al., 2008). However, there was a statistically significant decrease in the activity of the growth hormone and cortisol, 28% and 12% respectively (Djeridane et al., 2008). The significant decrease occurred at the maximum levels when comparing the 2-week (growth hormone and cortisol) and 4-week (growth hormone) exposure periods to the pre-exposure period (Djeridane et al., 2008). The difference observed did not persist during the post-exposure period (Djeridane et al., 2008). Based on the observations and findings, mobile phone-driven RF-EMF exposure did not influence the endocrine functions in men within the short-term exposure period (Djeridane et al., 2008).

The Specific RF Energy Absorption-Rate

The plausibility of cell phone-driven RFR effects on biological systems appears to be possible when RF energy interacts with a biosystem to induce changes in molecular or organismal level. Therefore, quantification of the SAR emitted through cell phone use is important. In other words, the approximation of SAR emitted and absorbed by the body during cell phone use should be established to support effective and meaningful

intervention approaches. Using series of meta-analysis evaluations on the cell phone use or cell phone-driven RFR, Baan et al. (2011) explored the carcinogenicity of radiofrequency electromagnetic fields. They indicated that close-proximity of cell phones to the ear while making a call could increase SAR values in the brain or ear area (Baan et al., 2011). They also emphasized that the SAR values absorbed by the body are heavily dependent on the design and position of the cell phone antenna (Baan et al., 2011).

The anatomy of the head, how the phone is held, and the quality of the connection or transmission between the tower or base station and cell phone, substantially contribute to the level of the SAR during cell phone use (Baan et al., 2011). Baan et al. (2011) also emphasized that the children' average SAR via cell phone use is about two times higher in the brain, and up to ten times higher in the bone marrow of the skull than in adults (Baan et al., 2011). They suggested that the use of a hands-free cell phone accessory reduces RFR exposure to the brain to about 10% (Baan et al., 2011). However, the use of a hands-free cell phone does not reduce the RFR exposure to other parts of the body (Baan et al., 2011). As such, RFR or EMF has been classified as 'possibly carcinogenic to humans' (Group 2B) (Baan et al., 2011).

Bakker, Paulides, Christ, Kuster, and van Rhoon (2010) assessed SAR among children exposed to electromagnetic waves. The exposure level of interest is between 10 MHz and 5.6 GHz (Bakker, Paulides, Christ, Kuster, & van Rhoon, 2010). The specified range is important for the evaluation the integrity of the International Commission on Non-Ionizing Radiation Protection (ICNIRP) (Bakker et al., 2010). The specified

measures were critical in the assessment of the ICNIRP EMF reference measures for the whole-body-averaged SAR(SARwb) and the peak 10 g spatial-averaged SAR (SAR10g)) (Bakker et al., 2010). The SAR assessments are the basis through which the exposure-response or dose-response interactions could be determined for health promotion measures (Bakker et al., 2010).

Bakker et al. (2010) evaluated whether SAR in children remained below the basic restriction reference during RFR exposure at the standard reference levels. In the study, they used a finite difference time domain (FDTD) modelling for the estimation of the SAR in the selected subjects at different 12 orthogonal EMF plane wave configurations (Bakker et al., 2010). From the modelling evaluations, they suggested that the sensitivity assessments showed an uncertainty of 53% for the SAR(wb) and 58% uncertainty for the SAR(10g) due to the variations in the simulation settings and tissue properties (Bakker et al., 2010). They concluded that the restriction of SAR (wb) in children was exceeded, accounting for about 45% increase in small children (Bakker et al., 2010). The maximum SAR of 10g found at body protrusions remained under the limit for all the conditions assessed (Bakker et al., 2010). The findings supported other results regarding RF SAR estimation; as a result, they recommended that the ICNIRP reference level limits should be re-evaluated (Bakker et al., 2010).

Biological Mechanisms

The effects of cell phone-driven RF-EMF appears to promote biological mechanisms that influence the normal function of the biology of a system. One of the mechanistic changes in the biological systems was demonstrated by Aly, Cheema,

Tambawala, Laterza, Zhou, Rathnabharathi, Barnes (2008). Aly et al. (2008) assessed the impact of 900-MHz radio frequencies on the chemotaxis of human neutrophils via an in-vitro study setting. In the study, participants' blood from healthy adult donors were exposed to different temperatures and 900-MHz RFR at 0.4 V/m (Aly et al., 2008). Based on the findings, cell phone-driven RFR effects on human neutrophils concentration gradients of Cyclic Adenosine 3', 5'-Monophosphate (C-AMP) was demonstrated (Aly et al., 2008). It shows that without RFR radiation exposure, the speed of the neutrophils increases as the temperature increase from 35 to 40 degrees (Aly et al., 2008). Also, without RFR exposure, the neutrophils' speed peaked at 40 degrees temperature and then decreased above 40 degrees (Aly et al., 2008). With the same increase in temperature from 35 to 40 degrees and in the presence of 900-MHz RFR exposure, the speed of neutrophils increased (Aly et al., 2008). The maximum speed observed with the 900-MHz exposure group exceeded the measured value at any temperature by about 50% (Aly et al., 2008). The estimated change in temperature due to the presence of 900-MHz RFR exposure was less than one micro-degree (Aly et al., 2008). Based on the findings, the mean response time of the neutrophils upon RFR radiation exposure was about 2.5 minutes (Aly et al., 2008).

Aalto, Haarala, Brück, Sipilä, Hämäläinen and Rinne (2006) demonstrated the effect of cell phones RFR on the human brain. Specifically, the focus was on RFR effects on regional cerebral blood flow (rCBF) among healthy participants (Aalto, Haarala, Brück, Sipilä, Hämäläinen, & Rinne, 2006). With a double-blind study approach, they evaluated human brain response to cell phone-driven RFR using a

photographic positron emission tomography (PET) imaging instrument (Aalto et al., 2006). A voxel-based statistical analysis was used (Aalto et al., 2006). An operational mobile phone predicted a local decrease in rCBF in the proximal inferior temporal cortex brain region (Aalto et al., 2006). Also, RFR exposure induced an increase the rCBF at the distant area of the brain around the prefrontal cortex region of the brain (Aalto et al., 2006). The findings provided the first evidence-based support about the response effects of cell phones induced RFR on the rCBF in humans (Aalto et al., 2006). However, the biological mechanisms associated the findings are not well known, but evidently, RFR induced changes in the brain neuronal activity (Aalto et al., 2006).

Andrzejak et al. (2008) examined the influence of cell phones use or cell phone-driven RFR on heart rate variability (HRV) parameters among young healthy individuals. The rationale for the study supports the ideation on the biological plausibility of adverse effects of RFR-EMF via the cell phones use, effects that could influence the functions of the autonomic nervous system (ANS) and regulates the circulatory system (Andrzejak et al., 2008). With 32 healthy students selected, the time and frequency domain of the HRV at rest were recorded using an electrocardiogram (ECG) and echocardiogram to assess the changes in the sympathovagal balance (Andrzejak et al., 2008). The frequency power measured included ultra-low frequency (ULF), very-low-frequency (VLF), low frequency (LF), high frequency (HF) and LF/HF ratio (Andrzejak et al., 2008). The ECG measurements were performed in the morning from 08:00 to 09:00 am and in a sitting position (Andrzejak et al., 2008). The participants' ECG was monitored as follows;

before making a cell phone call (period I), during the use cell phones for calls (period II), and after the cell phone calls (period III) (Andrzejak et al., 2008).

The maximal, mean, and minimal heart rate variation in both men and women participants were not statistically significant in the 20-minutes period before the mobile phone call (period I), during the 20-minutes mobile phone call (period II), and after the mobile phone call (period III). There were no arrhythmias episodes observed before, during, and after cell phone use or calls. However, the standard deviation of the normal sinus to normal sinus (SDNN) and standard deviation of the average of the normal sinus to normal sinus (SDANN) for the time domain HRV parameters for the period I, II, and III was estimated. The was a statistically significant difference during the 20-minutes calls or mobile phone use (period II) compared to the period I ($p<0.05$) and period III ($p<0.05$) (Andrzejak et al., 2008). The SDNN and SDANN among the male participants significantly increased during the use of cell phone or telephone calls when compared to the period (III) after the phone call was terminated ($p<0.05$) (Andrzejak et al., 2008). Similarly, the SDNN and SDANN parameters among the female counterpart increased significantly during the 20-minutes of a call made by a cell phone (period II) in comparison with the 20-minutes period (I) before the telephone call ($p<0.05$) (Andrzejak et al., 2008). The HRV parameters for the three frequency categories VLF, LF, and HF increased significantly over the 20-minute period of the telephone call (period II) in comparison to the 20-minutes period (period I-without cell RFR exposure) ($p<0.05$; $p<0.01$; $p<0.05$ respectively). Overall, the LF decreased significantly during the 20-minute period after the cell phone call (period III) in comparison to the period during the

telephone call (period II) ($p<0.05$) (Andrzejak et al., 2008). The LF/HF ratio was significantly lower during the telephone call (period II) in comparison to the period before and after the cell phone call (period II and III) ($p<0.05$) (Andrzejak et al., 2008).

Among women, the corresponding ECG parameters to VLF, LF, and HF increased significantly ($p<0.05$) (Andrzejak et al., 2008). There was a significant decrease in the LF/HF ratio ($p<0.01$) during the 20-minutes telephone call (among the period II group) compared to the ECG value before the telephone call (the period I group) (Andrzejak et al., 2008). In contrast, the LF/HF ratio significantly increased during the period after the cell phone call (period III) compared to the period during the telephone call (period II) ($p<0.01$) (Andrzejak et al., 2008). In men, the HF parameter significantly increased during the period of cell phone call (period II) compared to the period before the telephone call (period I) ($p<0.05$) (Andrzejak et al., 2008). Overall, the parasympathetic systematic tone assessed via the indirect evaluation of the heart rate variability increased (Andrzejak et al., 2008). In contrast, the sympathetic tone decreased during the cell phone call (Andrzejak et al., 2008). Based on the recorded observations, telephone calls with cell phones changed the autonomic balance in healthy individuals (Andrzejak et al., 2008). However, they cautioned that the changes observed in the HRV during the cell phone calls might not only have been affected by the electromagnetic field but perhaps, also influenced by speaking (Andrzejak et al., 2008).

Mitra, Milan, Koushik, and Subasish (2014) accounted and corrected the possible confounding effects of speaking, which Andrzejak et al. 2008 suggested could have influenced HRV changes during the cell phone calls. They accounted for the speaking

effects by eliminating talking, any sound, physical, and mental stress among the participants during the cell phone-driven RFR exposure, and at least allowed 2 hours before and after taking a meal before the exposure (Mitra et al., 2014). The exposure was performed in the evening in a fully rested setting and a sitting position (Mitra et al., 2014). No electronics devices or any other cell phones other than the one used by the participants were in the same room where during the study (Mitra et al., 2014). The health effects of mobile phone radiation in humans were evaluated based on age group and gender (Mitra et al., 2014).

In the study, the health effects of GSM and CDMA cell phone-driven radiation in humans were evaluated by quantifying changes in the blood pressure (BP), pulse rate (PR), heart rate (HR), respiration rate (RR), and body temperature (BT) among 20 healthy participants (10 males and 10 females) selected (Mitra et al., 2014). The participants' age ranges from 21 to 60 years old (Mitra et al., 2014). Observations were recorded after 30 minutes of exposure in both the silent and calling mode and when a cell phone handset was placed on the ear side. In 5% of the male participants within the age group of 21–40 years old, the observations made included rapid and arrhythmic heart rate, and rapid and irregular pulse rate (Mitra et al., 2014). Also, in 5% of males aged 41-60 years old, when a cell phone handset was placed on the participants' chest, some changes in the HR and PR were observed after 15 minutes of exposure (Mitra et al., 2014). About 5% of females and 10% of males aged 41-60 years old, showed changes in HR and PR after 30 minutes of exposure when cell phone handset was placed on the participants' chest (Mitra et al., 2014). Based on the findings, Mitra et al. (2014)

suggested and proposed further investigation in larger population set to verify their results, a study setting that could inform a better and stronger conclusion.

It appears that the totality of the mechanistic interaction of MW or RFR-EMF with biological systems is not well established. Belyaev, Markovà, Hillert, Malmgren, and Persson (2009) demonstrated the effects of MW energy using the universal mobile telecommunication systems (UMTS) and GSM mobile phones induced long-term inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes. The inhibition of the DNA DSBs repair mechanism and the misrepair of DNA in stem cells are part of the critical multistage process that leads to the onset of various types of chronic conditions such as leukaemias, tumors, gliomas, etc. (Belyaev, Markovà, Hillert, Malmgren, & Persson, 2009). The UMTS, unlike GSM, emit wide-band MW signals (Belyaev et al., 2009). They concluded that it was possible that UMTS microwaves could produce or induce more biological effects and possibly, adverse health risks than the GSM radiation emissions due to the unique wideband frequency characteristics (Belyaev et al., 2009). In the study, among hypersensitive and healthy participants, it was shown that the UMTS microwave exposures affected the chromatin (Belyaev et al., 2009). Also, among the hypersensitive and healthy subjects, the formation of the DNA-DSB co-localizing 53BP1/gamma-H2AX DNA repair foci in the lymphocytes was inhibited (Belyaev et al., 2009). The observed effects of MW energy on 53BP1/gamma-H2AX foci upon exposure persisted up to 72 hours (Belyaev et al., 2009). As a result, they suggested that the observed MW-induced response lasted longer than the stress-induced response to heat shock exposures (Belyaev et al., 2009). They emphasized that

the effects of GSM microwaves heavily depended on the mobile phone carrier transmission frequency (Belyaev et al., 2009). Overall, there were significant differences in the effects between healthy and hypersensitive subjects upon exposure to the UMTS microwaves and 915 MHz GSM microwave regarding the formation of the DNA repair foci (Belyaev et al., 2009). The observations were different for hypersensitive ($p <$ *0.02*[53BP1]//*0.01*[gamma-H2AX]), but there was no significant difference for the control participants ($p > 0.05$) (Belyaev et al., 2009). With the non-parametric analysis, the specificity of the differences between the GSM and UMTS microwave effects on hypersensitive subjects was not demonstrated (Belyaev et al., 2009).

Markovà, Malmgren, and Belyaev (2009) also demonstrated the inhibition effects of mobile phones-driven microwaves on 53BP1 focus formation in the human stem cells within differentiated cells. In the study, they examined whether cell phone-driven microwaves emitted or transmitted via the GSM and UMTS induced DSBs or affected DSB repair mechanisms in stem cells (Markovà, Malmgren, & Belyaev, 2009). Based on the tumor suppressor data analysis, the TP53 binding protein 1 (53BP1) foci formed DSB locations or sites (DNA repair foci), indicating that cell phone-driven MWs inhibited the formation of 53BP1 foci primarily fibroblasts and mesenchymal stem cells in humans (Markovà et al., 2009). In the study, GSM frequency of 915 MHz and UMTS frequency band of 1947.4 MHz inhibited all cell types, but microwave exposure level of 905 MHz did not inhibit 53BP1 foci in differentiated cells of the fibroblasts or lymphocytes (Markovà et al., 2009). In contrast, 905 MHz MWs had some inhibition effects in stem cells (Markovà et al., 2009). The strongest MW effects were observed in stem cells

(Markovà et al., 2009). Therefore, stem cells are most sensitive to cell phone-driven MW exposure and react to MW frequencies more readily than differentiated cells (Markovà et al., 2009). A phenomenon critical in cancer risk assessment and advancing knowledge on the mechanistic link between MW energy and cancer (Markovà et al., 2009). Based on the findings, Markovà et al. (2009) suggested that stem cells are the most relevant and meaningful cellular model for the validation of the safety and risk of cell phone-driven MW or mobile phone communication signals.

Carpenter and Sage (2008) conducted a review of prudent public health policy about EMF exposures. From the review, information about public health approach needed to advance health promotion measures regarding the effects of RF-EMF radiation exposures was identified (Carpenter & Sage, 2008). They concluded that there was an association between several health conditions including alteration of the autonomic control of the heart, leukemia, brain tumors, and neurodegenerative conditions, and various sources of RF-EMF radiation exposures (Carpenter & Sage, 2008). However, indicated that uncertainty remains on the biological mechanism(s) of the observed effects (Carpenter & Sage, 2008). Therefore, precautionary actions must be taken to minimize the RF-EMF radiation from all known sources (Carpenter & Sage, 2008). Failure to advance immediate preventive measures on MW or EMF or RFR exposure could lead to adverse health outcomes on many chronic diseases in the future (Carpenter & Sage, 2008).

Cell Phone-Driven RFR and Health Outcomes

There are several studies published on the impacts of cell phone use and health outcomes. Majority of the studies are short-term. Cumulatively, the body of literature on the topic provided informed and evidenced-based basis to conduct further studies on the impacts of cell phone use and health outcomes. For instance, Rajkovic, Matavulj, and Johnsson (2006) showed the effect of 50 Hz electromagnetic field (EMF) on thyroid gland using an experimental study design. The unit of analysis used for the two-month study was male rats (Rajkovic, Matavulj, & Johnsson, 2006). According to Rajkovic et al. (2006), the rats were exposed to an EMF (100-300 microT, 54-160 V m-1) for a 1-month period. The one-month exposure was induced on the scale of 5 days per week for 4 hours, daily (Rajkovic et al., 2006). Among the rats exposed to the EMF, predominance microfollicles with less colloid content and dilated blood capillaries were commonly observed and present (Rajkovic et al., 2006). The stereological analysis of the follicular epithelium, interfollicular tissue, blood capillaries, and thyroid activation index showed a statistically significant difference between the exposed rats and control group (Rajkovic et al., 2006). Among the EMF-exposed rats, the volume density of colloid was significantly lower in counts than the controls, and the ultrastructural analysis of thyroid follicular cells for the EMF-exposed group showed colloid droplets in the thyrocyte, but with very few large-diameter droplets (Rajkovic et al., 2006).

Unusual changes in the lysosomes, granular endoplasmic reticulum, and cell nuclei among the exposed rats were observed when compared to the control group (Rajkovic et al., 2006). Based on the findings, EMF or RFR exposure has a stimulative

effect on thyroid gland as shown in both the light microscope and ultrastructural level (Rajkovic et al., 2006). The findings supported the uncertainty concerns regarding the impacts of prolonged exposure to EMF or RFR (Rajkovic et al., 2006). It also demonstrated the effects of EMF radiation on chronic conditions and molecular-epidemiologic levels (Rajkovic et al., 2006).

Abramson, Benke, Dimitriadis, Inyang, Sim, Wolfe, and Croft (2009) evaluated the link between mobile phone use and changes in cognitive function in young adolescents. The study design was a cross-sectional epidemiologic assessment among secondary school students. In the study, the students were 7th graders composed of 317 individuals (144 boys and 173 girls) (Abramson, Benke, Dimitriadis, Inyang, Sim, Wolfe, and Croft, 2009). The average age of the students enrolled in the study was 13 years old (Abramson et al., 2009). The participants were selected from 20 secondary schools around Melbourne, Australia (Abramson et al., 2009). Based on the completed exposure questionnaire administered to the participants, cognitive test battery and the Stroop colour-word test were used to assess the link between cell phone exposure and cognitive function among the selected students (Abramson et al., 2009).

After adjusting the covariates age, gender, ethnicity, socio-economic status and handedness, it was concluded that the accuracy of working memory was poorer among students or children who reported more use of mobile phone voice calls (Abramson et al., 2009). Similarly, the reaction time for a simple learning task was shorter, associative learning response time was also shorter, and accuracy was poorer among students or children who reported more use of mobile phone voice calls (Abramson et al., 2009).

The completion time for Stroop word naming tasks was longer among students who reported more use of cell phone voice calls (Abramson et al., 2009). The effect of the total short text messages generated per week by the students was analyzed, and the cognitive assessment for the text messages was similar to those of the cell phone voice calls (Abramson et al., 2009). As a result, they suggested that the observed cognitive changes were unlikely due to cell phone-driven RFR exposure (Abramson et al., 2009). However, Abramson et al. (2009) concluded that cell phone use was directly associated with a faster response, but, showed a less accurate response to a higher level of cognitive tasks or functions. Therefore, the observed behaviors were probably acquired or learned due to the frequent use of cell phone (Abramson et al., 2009).

Agarwal, Deepinder, Sharma, Range, and Li (2008) investigated the effect of cell phone use on various markers of semen quality among men attending infertility clinic. Assessment of the study was conducted based on the participants' active cell phone usage behavior (Agarwal, Deepinder, Sharma, Range, & Li, 2008). In the study, 361 participants were enrolled (Agarwal et al., 2008). The participants were divided into four groups: Group A represented only the participants who do not use cell phones (Agarwal et al., 2008). The group B included only participants that use cell phones for a duration of less than 2hrs per day (Agarwal et al., 2008). The group C represented participants that use cell phones 2-4hrs per day (Agarwal et al., 2008). Lastly, group D included participants that use cell phones more than 4hrs per day (Agarwal et al., 2008). After the established sperm parameters (liquefaction time, volume, viscosity, pH, viability, sperm count, motility and morphology) were assessed, the mean sperm viability, count, motility,

and normal morphology among four different cell phone user groups were statistically significant (Agarwal et al., 2008). The sperm parameters for all the four groups decreased as the duration of daily exposure to cell phones use increased (Agarwal et al., 2008). Therefore, the use of cell phones decreased the semen quality among the men observed, a phenomenon linked to the decrease in the sperm viability, count, motility, and normal morphology (Agarwal et al., 2008). The decrease in the sperm parameters was heavily dependent on the exposure duration of cell phone use or cell phone-driven RFR and not the initial semen quality or biological integrity (Agarwal et al., 2008). Figure 12 below showed the sperm parameters mean score recorded by Agarwal et al. (2008).

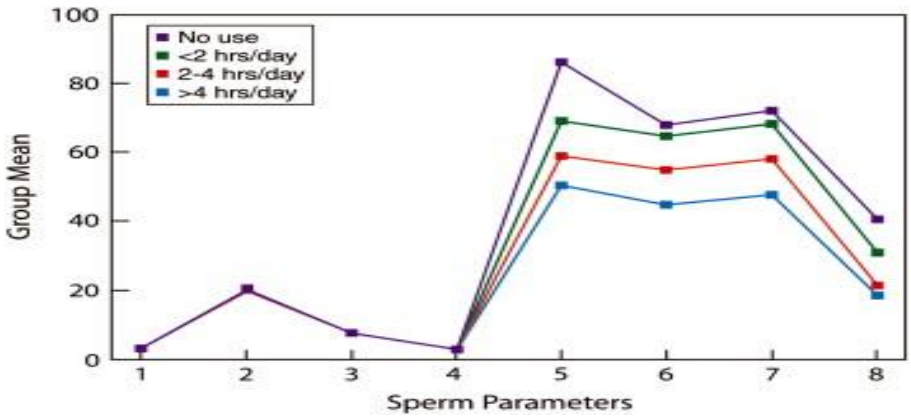


Figure 12. Sperm Parameters and Exposures. (Agarwal et al., 2008).

In the follow-up of the Agarwal et al. (2008) publication; Agarwal, Desai, Makker, Varghese, Mouradi, Sabanegh and Sharma (2009) demonstrated the effects of cell phone-driven radiofrequency electromagnetic waves on male semen. In this study, the source of the RFR exposure was via cell phone, which was either in a talk mode (experimental condition) or an off mode (control condition) (Agarwal, Desai, Makker,

Varghese, Mouradi, Sabanegh and Sharma, 2009). The semen samples were collected from 23 healthy donors and 9 infertile patients (Agarwal et al., 2009). The semen samples from the participants were divided into two groups, one experimental group and the control or unexposed group (Agarwal et al., 2009). The semen under the experimental condition was exposed to cell phone-driven RFR in talk mode for 1 hour (Agarwal et al., 2009). The semen under the control condition had identical parameters except that it was never exposed to cell phone-driven RFR (Agarwal et al., 2009). The primary outcome measures assessed in both conditions were sperm motility, viability, ROS, total antioxidant capacity (TAC), ROS-TAC score, and DNA damage (Agarwal et al., 2009). Agarwal et al. (2009) concluded that the semen samples exposed to the cell phone-driven RF-EMW had structural damage, DNA breakage, a decrease in sperm motility, and low viability, all of which were significant.

There was an increase in the ROS level, and a decrease in the ROS-TAC score (Agarwal et al., 2009). In the unexposed group, the levels of TAC and DNA damage had no significant difference compared to the unexposed semen. As a result, they advanced a cautionary warning to cell phone users emphasizing that cell phone-driven RF-EMW emitted during use could induce oxidative stress in sperm (Agarwal et al., 2009). Therefore, cell phone users should avoid keeping a talk-mode cell phone in their trouser pocket because the emitted RFR could pose an adverse risk to the spermatozoa and impair male fertility (Agarwal et al., 2009).

Before the Agarwal et al. (2009) study, the biological and genetic epidemiologic relevance for evidence-based investigative studies on the possible impacts of cell phone

use or cell phone-driven RFR on DNA integrity in the male germline was demonstrated by Aitken, Bennetts, Sawyer, Wiklendt, and King (2005). It appears that if biological or genetic epidemiologic effects are linked to cell phone use, the impacts would not be restricted to only DNA damage in male germ cells but could span to population-based epidemiologic or public health outcomes. Aitken et al. (2005) used an animal-based experimental design to evaluate the health-related effects associated with cell phone radiation. In the study, mice were exposed to 900 MHz MW energy, which is the level of RF transmitted by commercial cell phones (Aitken, Bennetts, Sawyer, Wiklendt, & King, 2005). The exposure duration was 12 hours per day for 7 days (Aitken et al., 2005). The specific absorption rate of the RFR exposure was estimated at 90 mW/kg (Aitken et al., 2005). The short-term cell-phone RFR did not induce significant changes in the sperm number, morphology and vitality among the exposed mice (Aitken et al., 2005). However, among the exposed mice, the real-time polymerase chain reaction (qPCR) analysis showed that both the mitochondrial genome and nuclear beta-globin locus were damaged (Aitken et al., 2005). The DNA damage was statistically significant $p < 0.05$ and $p < 0.01$ respectively (Aitken et al., 2005). As a result, it was concluded that cell phone-driven RF-EMR might not have a substantial effect on male germ cell development, but it has a significant genotoxic effect on the epididymal spermatozoa (Aitken et al., 2005). Aitken et al. (2005) emphasized that the findings deserved further investigations.

Al-Khlaiwi and Meo (2004) conducted an epidemiologic study using a cross-sectional survey approach to evaluate the association of cell phone-driven RFR with

fatigue, dizziness, tension, headache, and sleep disturbance within a target population in Saudi Arabia. The findings provided invaluable and evidence-based information on health outcomes and social awareness on the use of cell phones (Al-Khlaiwi & Meo, 2004). A total of 437 human participants (55.1% male and 39.9% female) were recruited (Al-Khlaiwi & Meo, 2004). The selected participants owned and used cell phones (Al-Khlaiwi & Meo, 2004). Surveys or questionnaires administered to the participants assessed the historical behavior of cell phone use and based on the information gathered, the association between cell phones use, and health outcomes or hazards was evaluated (Al-Khlaiwi & Meo, 2004). The reported descriptive analysis or percentage of the health outcomes observed in the study were headaches (21.6%), sleep disturbances (4.0%), tensions (3.9%), fatigues (3%) and dizziness (2.4%) (Al-Khlaiwi & Meo, 2004). Based on the participants' response to the survey or questionnaire's questions, there was an association between the use of cell phones and health outcomes or hazards (Al-Khlaiwi & Meo, 2004). It was concluded that cell phone or cell phone-driven RFR is a health risk factor (Al-Khlaiwi & Meo, 2004). Therefore, excessive use of cell phones or long-term cell phone use should be avoided (Al-Khlaiwi & Meo, 2004). Perhaps, health practitioners should facilitate health promotion measures about cell-phone use health indicators via group discussions, public presentations, advocacy, and electronic and conventional media sources (Al-Khlaiwi & Meo, 2004).

Lönn, Ahlbom, Hall, Feychting and the Swedish Interphone Study Group (2005) conducted a study on the assessment of long-term mobile phone use and brain tumor risk. The study was a case-control design (Lönn, Ahlbom, Hall, Feychting, & the Swedish

Interphone Study Group, 2005). The cases were individuals aged 20–69 years old and who were diagnosed with glioma or meningioma between 2000–2002 in Sweden (Lönn et al., 2005). The control group were randomly selected and were stratified based on age, gender, and residential locations (Lönn et al., 2005). Based on the information collected from individuals with cases of brain tumor, 371 (74%) and 273 (85%) participants had glioma and meningioma respectively (Lönn et al., 2005). Meanwhile, 674 (71%) participants were the control group. The estimated odds ratio for regular cell phone use was 0.8 for individuals with glioma and 0.7 for those with meningioma (Lönn et al., 2005). The evaluation of cell phone use accounted more than 10-years duration of use (Lönn et al., 2005). However, there was no significant increase in risk for ipsilateral phone use for tumors located in the temporal and parietal lobes (Lönn et al., 2005). Regardless of the type of phone, amount of use, and tumor histology, the odds ratio did not increase (Lönn et al., 2005). Therefore, based on the findings, cell phone use did not predict an increased risk of meningioma or glioma within the target population (Lönn et al., 2005).

Lahkola et al. (2007) conducted an international collaborative case-control study involving 1209 meningioma cases and 3299 population-based controls on mobile phones use and risk of meningioma. The study was conducted in five North European countries (Lahkola et al., 2007). The historical account of mobile phone use (regular cell phone use once a week for 6 months, duration of use, cumulative number and hours of use) were generated via personal interviews (Lahkola et al., 2007). Other indicators of cell phone use were evaluated for the assessment of meningioma risk, and the variables were

stratified by age, sex, country, and region (Lahkola et al., 2007). Among regular cell phone users, the risk of meningioma was lower than participants who never use a cell phone or non-regular users (*OR = 0.76*) (Lahkola et al., 2007). The risk of cell phone use never increased based on lifetime years of use, years of first use, cumulative hours of use or number of calls (dose-exposure) (Lahkola et al., 2007). The observations were similar for analogue and digital phone networks, age, and sex (Lahkola et al., 2007). In the study setting, the findings did not provide support for any link between mobile phone use and risk of meningioma (Lahkola et al., 2007).

Arnetz, Akerstedt, Hillert, Lowden, Kuster and Wiholm, (2007) examined the effects of 884 MHz GSM wireless communication signals on the self-reported symptom, cognitive function, and electroencephalographically (EEG) recorded sleep. Possible medical conditions and biochemical factors that could interfere with the study variables were evaluated (Arnetz, Akerstedt, Hillert, Lowden, Kuster & Wiholm, 2007). Accordingly, the participants were first habituated (participated in the habituation sessions) (Arnetz et al., 2007). Subsequently, were followed up with two sessions, which involved exposure to either the sham treatment or 884 MHz GSM wireless communication signals for 3 hours (Arnetz et al., 2007). The exposure average was 1.4 W/kg including periods of discontinuous transmission (DTX) and Non-DTX (Arnetz et al., 2007). The RFR exposure was directed to the left hemisphere (Arnetz et al., 2007). The total number of the sample size used in the study was 71 subjects (36 women and 35 men) (Arnetz et al., 2007). Overall, 38 participants (22 women and 16 men) reported

symptoms linked to the use of mobile phone (SG) (Arnetz et al., 2007). The remaining participants reported no cell phone-related symptoms (NG) (Arnetz et al., 2007).

The data on health indicators collected before, during, and following the exposure or sham sessions included self-reported symptoms of a headache, cognitive function, mood, and electroencephalographic recordings (Arnetz et al., 2007). During 884 MHz sessions in comparison with the sham exposure, sleep was initiated 1-hour after (Arnetz et al., 2007). As a result, the latency period to reach the first cycle of deep sleep (stage 3 sleep) was prolonged and the amount of stage 4 sleep was decreased among the RFR exposed subjects (Arnetz et al., 2007). Based on the analysis, during the laboratory exposure (884 MHz cell phone-driven RFR session), elements of sleep process were adversely affected (Arnetz et al., 2007). Participants who reported no symptoms indicated more headaches during the RFR exposure than in the sham exposure (Arnetz et al., 2007). The participants from either group (SG or NG) could not detect or sense the real exposure (884 MHz RFR) status more frequently than would be possible by mere chance alone (Arnetz et al., 2007).

Brain tumors and salivary gland cancer have been outcomes of interest in the public debate regarding the effects of cell phone use or cell phone-driven RFR exposures on health status. As a result, Auvinen, Hietanen, Luukkonen, and Koskela (2002) conducted a study in Finland using a registry-based case-control design. The total number of brain tumor and salivary gland cancer cases enrolled in the study were 398 and 34 samples respectively (Auvinen, Hietanen, Luukkonen, & Koskela, 2002). The subjects were diagnosed in Finland in 1996 (Auvinen et al., 2002). Based on the data

analysis, cell phone use was not associated with brain tumors or salivary gland cancers (Auvinen et al., 2002). However, there was a weak association between gliomas and the analog cell phone use (Auvinen et al., 2002). Auvinen et al. (2002) emphasized the implication of an inherent limitation such as a registry-based approach as a barrier in the risk assessment because of lack of information or verification of the accuracy of the information presented about the exposure (Auvinen et al., 2002).

In furtherance, Goldwein and Aframian (2009) explored the effects of handheld cell phone on parotid gland secretion based on the criteria that a correlational relationship between cell phone and salivary gland tumors exists. In the study, parotid saliva was collected from 50 healthy participants from both the dominant side and non-dominant side of the head based on the level of handheld cell phone use (Goldwein & Aframian, 2009). Among the right-sided dominant cell phone users, lower total protein concentration was observed in the dominant side compared to the non-dominant area (Goldwein & Aframian, 2009). The difference in the protein concentration was statistically significant (Goldwein & Aframian, 2009). In other words, cell phone use on the dominant side predicted higher saliva secretion rate in that area compared to the non-dominant side (Goldwein & Aframian, 2009). Therefore, the parotid glands adjacent to the dominant side had elevated salivary rates and decreased protein secretion, which is an indication of the continuous impacts of cell phone-driven RFR exposure (Goldwein & Aframian, 2009). Based on the findings, further investigation and large-scale longitudinal studies on the topic was encouraged (Goldwein & Aframian, 2009).

Sangün, Dündar, Çömlekçi, and Büyükgebiz (2015) explored the effects of cell phone-driven RF-EMF on the endocrine system in children and adolescents. As the level of sensitivity to the effects of the RF-EMF exposure increases, the SAR value increases as well, this assumption was the basis of the study (Sangün, Dündar, Çömlekçi, & Büyükgebiz, 2015). As a result, children with increased SAR are at a greater lifetime cumulative risk over the course of their lifetime due to early age exposure factor (Sangün et al., 2015). The inconsistencies and uncertainties about evidence-based causality studies on the association between cell phone-driven RF-EMF exposures and endocrine system are the key barriers to this research topic (Sangün et al., 2015). However, there are unignorable amounts of investigative findings suggesting an increased risk of cancer, hematologic effects, metabolism problems, endocrine functions, and cognitive impairment links to mobile phone-driven RF-EMF exposures (Sangün et al., 2015). Also, according to Sangün et al. (2015), cellular phone-driven RF-EMF exposure on the reproductive system and growth are challenging. Growing concerns on the adverse or serious adverse effects of cell phone-driven RF-EMFs exposure on thyroid functions, glucose homeostasis, adrenal hormones, and melatonin levels were observed (Sangün et al., 2015). Therefore, the health threats posed by mobile phone-driven RF-EMF exposure in children should be taken seriously and perhaps, classified as a public health hazard (Sangün et al., 2015).

Carlberg, Hedendahl, Ahonen, Koppel, and Hardell (2015) examined the increasing incidence of thyroid cancer in the Nordic countries using the Swedish Cancer Registry data. The time-period investigated was from 1970 to 2013 (Carlberg,

Hedendahl, Ahonen, Koppel, & Hardell, 2015). The communication devices included in the study are mobile and cordless phones (Carlberg et al., 2015). The communication devices used by the participants emit RF-EMF to the brain and thyroid gland during use (Carlberg et al., 2015). The increase in the incidence of thyroid cancer of the papillary type was noticeable due to its sensitivity to radio waves radiation (Carlberg et al., 2015).

The age-adjusted incidence rate of thyroid cancer in women significantly increased during the study period, Figure 13 (Carlberg et al., 2015). Also, while two joinpoints were identified (1979 and 2001), there was a significant increase in the age-adjusted incidence rate during the last period of 2001-2013 (Figure 13) (Carlberg et al., 2015). In men, they also found that there was an increase in thyroid cancer incidence during 1970-2013 (Figure 14) (Carlberg et al., 2015). In men, there was a joinpoint from 2005-2013, and the age-adjusted increase was statistically significant in men (Figure 14) (Carlberg et al., 2015). Based on the NORDCAN data, other Nordic country's thyroid cancer incidence increased significantly during the same time-period (Carlberg et al., 2015). Among the Nordic countries, there was a joinpoint observed in 2006 for both men and women; which showed a statistically significant increase of thyroid cancer incidence during 2006-2013 in women (APC +6.16 %) and men (APC +6.84 %) (Carlberg et al., 2015). Thus, reinforcing the findings observed with the Swedish Cancer Register data (Carlberg et al., 2015). The increasing trend in the incidence rate in Sweden was primarily due to thyroid cancer of the papillary (Carlberg et al., 2015). They also suggested that the thyroid cancer increase over time is not attributed to improved diagnostic procedures, but rather that an increase in the exposure to RF-EMF (non-

ionizing radiation) ionizing radiation and (e.g. medical computed tomography (CT) scans) should be explored further for this outcome (thyroid cancer) (Carlberg et al., 2015). In parallel to the increase in thyroid cancer, the out-going mobile phone minutes and mobile phone subscription also increased during the period (Figure 15) (Carlberg et al., 2015).

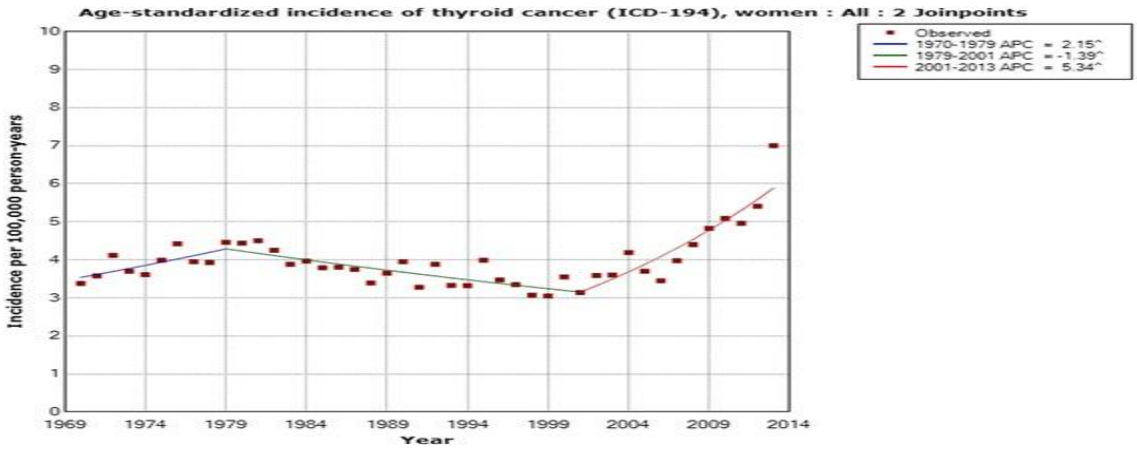


Figure 13. Thyroid Cancer Incidence Rate in Women. (Carlberg et al., 2015).

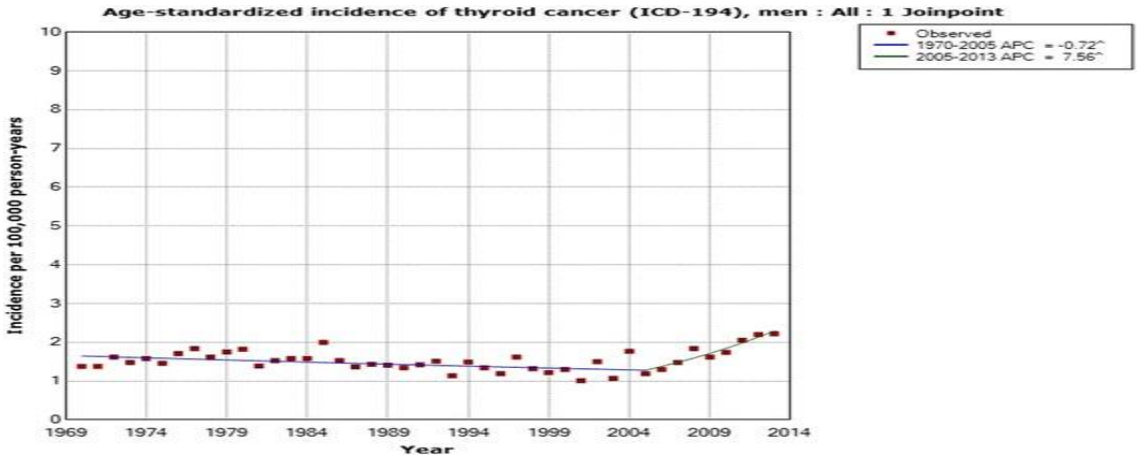


Figure 14. Thyroid Cancer Incidence Rate in Men. (Carlberg et al., 2015).

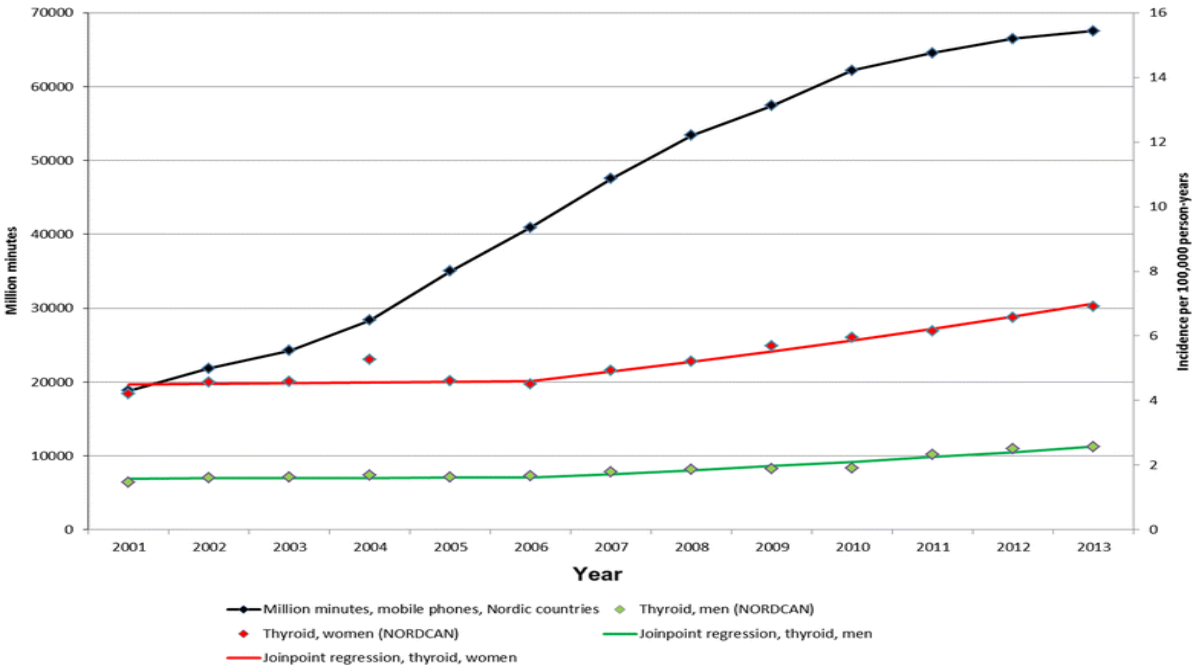


Figure 15. Out-Going Mobile Phone Minutes. (Carlberg et al., 2015).

Bhargavi, Balachandrudu, and Nageswar (2013) explored the effect of EMF radiation of two cell phone technologies with different frequencies and power levels on health outcomes (Bhargavi, Balachandrudu, & Nageswar, 2013). The experimental study was conducted using 10 human subjects (Bhargavi et al., 2013). The exposure duration for cell phone use was 10 minutes of talking time (Bhargavi et al., 2013). Electroencephalogram was used to monitor the brain signals during the10 minutes of exposure (Bhargavi et al., 2013). Cell phones modulated via the GSM communication feature showed a larger effect on the brain signals compared to cell phones modulated through the CDMA transmission system (Bhargavi et al., 2013). Despite this finding, they suggested that further high-quality research is necessary to advance meaningful health promotion measures (Bhargavi et al., 2013).

RFR Exposure Sensitivity to Generational or Age and Cohort Effect

The assessment of the association between cell phone use and brain tumors in children and adolescents, a multicenter case-control study performed by Aydin et al. (2011) provided additional information to clarify the uncertainty concerns regarding the influence of cell phone use on brain tumor outcomes among cell phone users. The primary hypothesis proposed by Aydin et al. (2011) was that children and adolescents are more vulnerable to health outcomes associated with cell phone use or cell phone-driven RFR exposure than adults. The multicenter sites included in the study are Denmark, Sweden, Norway, and Switzerland (Aydin et al., 2011). Individuals (children and adolescents) with the cases of brian tumor aged from 7–19 years old and were all diagnosed between 2004 and 2008 (Aydin et al., 2011). There was a total of 998 individuals included in the study (352 cases and 646 controls) (Aydin et al., 2011). Subjects in the control group were randomly selected and matched by age, sex, and geographical region (Aydin et al., 2011). Both the case and control group were subjected to interview process regarding their cell phone use (Aydin et al., 2011). Cell phone use or ownership was verified by the cell phone operator records when and if available (Aydin et al., 2011). Based on the risk analysis, brain tumor diagnosis among regular cell phone users was not statistically significant compared to the nonusers (Aydin et al., 2011). Children who were exposed to cell phone use or cell phone-driven RFR exposure at least in the past 5 years from the time of the study in comparison to children who never regularly used cell phones were not at increased risk of brain tumor (Aydin et al., 2011). In contrast, among the subset of the participants whose operator recorded data was

available showed the temporality sequence of cell phone use or subscription before the tumor onset (Aydin et al., 2011). Among those groups of individuals, the brain tumor risk was directly associated with the time elapsed (Aydin et al., 2011). However, there was no clear association between the dose-outcome response because there was no increase in the risk of brain tumors in the areas of the brain exposed to highest amounts of RFR (Aydin et al., 2011).

The spatial SAR differences among cell phone users were evaluated between adults and children using a standardized specific anthropometric mannequin head phantom (Christ, Gosselin, Christopoulou, Kühn, and Kuster, 2010). Different cortex areas were evaluated using the imaging-based head phantoms (for adults and children brain) exposed to various models of cell phones (Christ et al., 2010). The evaluation analysis implicated an age-dependent tissue-specific exposure of cell phone users and there were uncertainties about the effects of age-dependent dielectric tissue characteristics and age-dependent proportions of the face, ear, and skull on the global and local RFR absorption of the brain tissues (Christ et al., 2010). Due to the closer proximity of the cell phone to the brain (cortex, hippocampus, and hypothalamus) and eye tissues during use, the locally induced fields were significantly higher (*>3 dB*) (Christ et al., 2010). The effects to the bone marrow were even larger (*>10 dB*) due to its high conductivity properties (Christ et al., 2010). On the other hand, the pineal gland tissues did not exhibit an increase in the magnetic field perhaps due to its distance from the phone and not as a function of ageing (Christ et al., 2010). As a result, the study

findings did not support the hypothesis of age-dependent changes of the spatial peak SAR in the head via cell phone use (Christ et al., 2010).

Rezk, Abdulqawi, Mustafa, Abo El-Azm, and Al-Inany (2008) also demonstrated the effect of EMF on fetal and neonatal heart rate and cardiac output (COP) following acute maternal exposure to cell phone-driven EMF radiation. The study was conducted in Egypt with 90 women (Rezk, Abdulqawi, Mustafa, Abo El-Azm, & Al-Inany, 2008). The women had uncomplicated or uncompromised pregnancies including 30 full-term healthy newborn infants (Rezk et al., 2008). Their ages ranged from 18-33 years old (Rezk et al., 2008). In the study, pregnant women were exposed to cell phone-driven RF-EMF via phone-dialing mode for 10 minutes on a daily basis during the pregnancy term and after birth (Rezk et al., 2008). Rezk et al. (2008) observed a statistically significant increase in fetal and neonatal HR. They found that the decrease in the stroke volume and COP before and after cell phone exposure was statistically significant (Rezk et al., 2008). Also, the changes observed were attenuated with the increase in gestational age (Rezk et al., 2008).

Summary and Conclusions

The major themes observed through the literature review process about the effects of cell phone use or cell phone-driven RF-EMF exposure are uncertainty and reproducibility. To maintain a sustainable validity, the findings must be reproducible in different target population and settings through either an experimental design settings (causality studies) or longitudinal or observational design settings (correlational studies) in either an animal or human model. Lack of consistency and reproducibility of the

impacts of the predictor variable (cell phone use) on any of the published health outcomes (cancers and other suspected chronic conditions) makes it a challenging task to advance causality ideation or even correlational inference conclusively on the subject matter. Based on the literature review, it was and will always be easier to control confounders or covariates such as other non-ionizing radiation, dose-response, cohort effects, generational effects, and period effects, etc. in an experimental or quasi-experiment design than was or would be possible with observational or longitudinal study design. The longitudinal or observational-driven study design employed by researchers in some of the human epidemiological studies were prone to misclassification bias, investigators' bias, recall bias, participants' bias, selection bias, etc., and inherently lacks precise dose-response exposure integrity.

Besides, in many cases, if not all, there were no data on the SAR for the longitudinal or observational study design. With a longitudinal or observational design such as a cross-sectional study or case-control study, a clear spatiotemporality cannot be established between the exposure and health outcome (s) or biological effects. However, what is known or have been consistently observed in many the studies was that the health outcome(s) or biological effects were observed or estimated were statistically significant (not by mere chance alone). In few cases, the health outcomes or biological effects were not statistically significant. In cases or studies where the test statistics in a study was statistically significant, the biological mechanism for such observation or effect was not known.

Either through experimental or observational or longitudinal study design, much of the overall studies conducted for any given health outcome(s) or biological effects are short-term studies. Thus, lack of 'long-term' epidemiologic study in the US and most of the global community is the shared concern or gap in the literature for this research inquiry or topic. As a result of lack of long-term studies on the impact of cell phone use or cell phone-driven RF-EMF exposure among the target population in the US, fulfilling this gap with the specified health outcomes could advance in-depth insights on the prolonged and cumulative impacts of cell phone driven-RF-EMF exposure. Perhaps, could promote clarity on the biological links between the dose or exposure-response. The methodology used in this current study to address the research inquiry was discussed in chapter 3 of the dissertation.

Chapter 3: Research Method

Introduction

The purpose of this study was to evaluate differences in the prevalence rate of specified health outcomes (thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease) between cell phone users and non-cell phone users. Estimating the prevalence rate of these outcomes is essential in determining the health and social implications associated with handheld cell phone use and, consequently, to help public health practitioners advance appropriate health promotion measures and precautionary steps on safe cell phone use (Rajkovic et al., 2006; NTP, 2016). Findings from this study may promote evidence-based policy and regulatory guidelines on cell phone design and use.

To support these potential social awareness and policy outcomes, I sought to use meaningful, reliable, and valid research methods. The method used in this study was consistent with epidemiologic study approaches. Chapter 3 contains a description of the research methods used in this study. Other methodological factors considered included sample size estimation, sampling procedures, secondary data sources, assessment of the relevant test variables, evaluation of the test variables' levels of measurements, the statistical approach, target population, gender, and ethical implications.

Research Design and Rationale

I performed a secondary data analysis using a cross-sectional design. A cross-sectional research design is appropriate for the evaluation of the prevalence and incidence of an outcome because it is meant to capture relevant overview information (a snap shot)

of a phenomenon in a sample population (Aschengrau & Seage, 2014; Creswell, 2009; Gordis, 2009; Krieger, 2011; Moeller, 2011; Rudestam & Newton, 2015; Szklo & Nieto, 2014). Also, a cross-sectional research design provides investigators with the opportunity to predict the risk of an outcome given exposure at a given point in time or over a long period (Aschengrau & Seage, 2014; Creswell, 2009; Gordis, 2009; Krieger, 2011; Moeller, 2011; Rudestam & Newton, 2015; Szklo & Nieto, 2014). In the absence of an experimental study design, which can be used to assess causal relationships, a cross-sectional design can be used to assess inferential correlational associations between variables (Aschengrau & Seage, 2014; Creswell, 2009; Gordis, 2009; Krieger, 2011; Moeller, 2011; Rudestam & Newton, 2015; Szklo & Nieto, 2014). In this study, a survey-driven data collection approach was used. This data collection approach was the approach used by the primary USDHHS/CDC/NCHS investigators, based on the primary intent of the data collection, which was to assess the prevalence and incidence of cell phone use and compare health outcomes among different user groups.

Data Source and Variables

Secondary dataset from the NHIS 2012 study conducted by the USDHHS, CDC, and NCHS) were used for the current study. The NHIS 2012 data were generated via a survey-driven cross-sectional approach directed towards the individuals and households' health status in the United States (Inter-University Consortium for Political and Social Research [ICPSR], 2016; NCHS, 2013, 2015). The purpose of the NHIS fosters and synthesizes valuable information from the US households to advance health outreach programs to improve population health and medical knowledge locally, nationally, and

globally (ICPSR, 2016). Initially, NCHS began gathering national health information in 1957 through the Health Interview Surveys (HIS), which was later called NHIS (ICPSR, 2016, NCHS, 2013, 2015). The questionnaire and target population used for this study were from the NHIS 2012. The ICPSR number assigned to this study is ICPSR-36146. The identifier for the data set that contain the cell phone questions is called the 'Family Level' while the identifier for the second dataset which included the thyroid cancer, mouth/tongue/lip cancer, and heart disease questions is called the 'Sample Adult Level' (ICPSR, 2016, NCHS, 2013, 2015).

The NHIS is an annual interview conducted by the NCHS and CDC (ICPSR, 2016). The primary purpose of the NHIS studies is to monitor the health status of the U.S. population through meaningful data collection and analysis on a broad range of health topics (ICPSR, 2016). The NHIS questions are similar every year, and the repeated items are identified as the "core questions" (ICPSR, 2016). Since 1997, the core questions in the NHIS have been divided into three parts: The Family, Sample Adult, and Sample Child levels (ICPSR, 2016). Also, the 2012 NHIS contains enhanced questions on health care access and utilization (ICPSR, 2016). The Family level contain the questions on cell phone use while the Sample Adult level contain questions about thyroid cancer, mouth/tongue/lip cancer, heart condition or disease, and other health outcomes.

The 2012 NHIS data is made up of six core data files: The three disability questions test files, a paradata file, a functioning and disability file, and two complementary, and alternative medicine files (ICPSR, 2016; NCHS, 2013, 2015). The NHIS questionnaire covers the following supplemental topics: the Sample Adult

questionnaire on subjects of immunization, complementary and alternative medicine, non-cigarette tobacco use, voice, speech, and language; the Family questionnaire on subjects of food security; and the Sample Child questionnaire on subjects of mental health, mental health services, immunization, complementary and alternative medicine, balance, voice, speech, and language (ICPSR, 2016; NCHS, 2013, 2015). The 2012 NHIS core data files also contain Disability Questions Tests which includes Person-level data collection through a field test of six disability questions (ICPSR, 2016; NCHS, 2013, 2015).

The 'Disability Questions Tests 2012 files' are in three separate files while the fourth disability supplement test file was released as part of the Sample Adult Core called the 'Adult Functioning and Disability Level' (ICPSR, 2016, NCHS, 2013, 2015). The 'Adult and Child Alternative Health Supplement' components of the data set were intended to advance knowledge on alternative medical services, and the questions posed therein focused on the frequency or regularity application or use of various types of alternative therapies, the reason of use, and the associated costs (ICPSR, 2016; NCHS, 2013, 2015). The information on the survey and data collection processes such as the response rate, interview times, number of contact attempts and keystrokes of the interview were recorded in the 'Paradata Level' file (ICPSR, 2016; NCHS, 2013).

Overall, the NHIS is meant to generate information on the amount and distribution of illness and health outcomes, including evaluation of the effects of disability and chronic impairments, and the types of health services individuals receive (ICPSR, 2016; NCHS, 2013). The NHIS provides a continuous sampling and interview

processes on the civilians and noninstitutionalized target population in the US through core surveys and supplemental datasets (ICPSR, 2016; NCHS, 2013). The health information provided through the supplemental NHIS data are not limited to child health care and immunization, substance abuse, AIDS knowledge and attitudes, preventive care, dental care, nursing care, self-care, prosthetic appliances, and hospitalization (ICPSR, 2016; NCHS, 2013). All the information is maintained as microdata files, which are retained permanently since 1963 (ICPSR, 2016; NCHS, 2013).

The 2012 NHIS interview process was similar to the NHIS interview conducted a year prior (NCHS, 2013; NCHS, 2015). All information collected for the sample adult was collected from adults unless the participant is physically or mentally unable to respond (ICPSR, 2016; NCHS, 2013; NCHS, 2015). In such situations, a knowledgeable proxy was allowed to answer the sample adult questions (ICPSR, 2016; NCHS, 2013; NCHS, 2015). Information about the sample child was collected from a knowledgeable adult or who may or may not be an adult (ICPSR, 2016; NCHS, 2013; NCHS, 2015).

The selected primary questions that addressed the posed research questions for this dissertation are derived from the 'Sample Adult Level' and the 'Family Level' data. The core purpose of both datasets was intended to assess the health status of the recruited participants and consequently, address the subjects' lifestyle (ICPSR, 2016; NCHS, 2013; NCHS, 2015). For thorough data assessment in this current study, at least the participants' demographics such as age, socioeconomic status, race, and gender was

evaluated. Also, appropriate stratification adjustments were made whenever possible to minimize the covariate effects of known confounders.

The core foundation in the development of this dissertation's construct parameters for the independent variable (cell-phone ownership/cell phone-driven RFR exposure or no cell phone ownership/no RFR exposure/low RFR status) was based on the key questions about the duration of cell phone use and cell phone exposure or ownership. The key questions in the NCHS' 2012-NHIS questionnaire about the independent variable of interest that is relevant in this study are as follows (see Table 2 for more detail).

Table 2

The NHIS Questions and Response Options on Cell Phone Use

Question on Cell Phone	**Response Options**
Is there at least one telephone INSIDE your home that is currently working and is not a cell phone?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know
Not including cell phones, have you or your family been without telephone service for one week or more during the past 12 months? Do not include interruptions of phone service due to weather or natural disasters?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know
Not including cell phones, how long were you or your family without telephone service in the past 12 months?	000 = Less than 1 week 007-365 = 7-365 days 997 = Refused 998 = Not ascertained 999 = Don't know

Do you or anyone in your family have a working cell phone? (This the primary question that identified cell ownership for my study).	01-10 =1-10 phones 97 = Refused 98 = Not ascertained 99 = Don't know
Of all the telephone calls that you or your family receives, are all or almost all calls received on cell phones, some received on cell phones and some on regular phones, or very few or none received on cell phones? (This is the primary exposure question for my study).	1 = All or almost all calls received on cell phones 2 = Some received on cell phones and some on regular phones 3 = Very few or none on cell phones 7 = Refused 8 = Not ascertained 9 = Don't know

Source: 2012 NHIS Codebook (NCHS, 2013)

The determination of the health outcome(s) or dependent variable of interested was based on the assessment of thyroid cancer, mouth/tongue/lip cancer, hypertension or blood pressure, and heart condition health status of the participants, which were part of the questions stated in the 2012 NHIS questionnaire (NCHS, 2015). The questions about the primary health outcome of interest as stated in the 2012 NHIS questionnaire that is relevant in this study are as follows (see Table 3 for more detail).

Table 3

The NHIS Questions and Response Options on Health Outcomes

Question on Health Outcomes	**Response Options**
Have you EVER been told by a doctor or other health professional that you had...Cancer or a malignancy of any kind?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know

What kind of cancer was it?	1 = Mentioned 2 = Not mentioned 7 = Refused 8 = Not ascertained 9 = Don't know
What kind of cancer ... mouth/tongue/lip?	1 = Mentioned 2 = Not mentioned 7 = Refused 8 = Not ascertained 9 = Don't know
Have you EVER been told by a doctor or other health professional that you had ... Coronary heart disease?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know
DURING THE PAST 12 MONTHS have you had... Coronary heart disease?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know
Have you EVER been told by a doctor or other health professional that you had ...Any kind of heart condition or heart disease (other than the ones I just asked about)?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know
DURING THE PAST 12 MONTHS have you had...Any kind of heart condition or heart disease (other than the ones I just asked about)?	1 = Yes 2 = No 7 = Refused 8 = Not ascertained 9 = Don't know

Source: 2012 NHIS Codebook (NCHS, 2013)

The questions and response options stated in Table 3 were retrieved from the 2012 NHIS questionnaires. The 'Family level' questionnaire contains information about the cell phone use (NCHS, 2013; NCHS, 2015). The 'Sample Adult level' questionnaire contains information about thyroid cancer, mouth/tongue/lip cancer, and heart disease

status (NCHS, 2013; NCHS, 2015). The questions and response options in Table 3 (from the 2012 NHIS questionnaire codebook) were used to address the research questions posed in this current study as they contain the key elements of this dissertation inquiry. Even when the data collection process for this study involved secondary data source (the 2012 NHIS data sets), compliance to the Walden IRB standards, international, and local ethical standards were thorough in maintaining scientific integrity involving human subjects' personal information and wellbeing. The entire secondary dataset contents were evaluated to ensure that a complete de-identification of the participants' information was maintained in the original dataset, as well as in the final data set used in the statistical analysis for this current dissertation. In maintaining ethical standards, compliance to the 'right to use' request for the 2012 NHIS data access was submitted to the CDC-NCHS (NHIS@cdc.gov) via email. In response to the data access request, the NCHS indicated that the 2012 NHIS datasets were available in the NCHS public domain and the ICPSR sites. In addition, the Walden IRB review process was sought for the approval process for access to the 2012 NHIS data set before the data analysis was performed in Chapter 4 of this dissertation.

Methodology

The test variables in this study included the dependent variable, independent variables, mediating variables, moderating variables, covariates, and confounders. The primary dependent variables are thyroid cancer, mouth/tongue/lip cancer, and heart condition or disease. The primary independent variable is cell phone use/cell phone-driven RFR exposure. Individuals who do not use cell phone use or those that use cell

phone rarely are the 'control' or reference group. The number of cell phones owned was a mediating or moderating factor. Other mediating and moderating factors that could have influenced the outcome of the study are individuals with mental problems, speech problems, menopause problems. Substance abuse, interacting medications/vitamins, food choice lifestyle, the level of physical activities, alcohol use, smoking, and tobacco use habit could have also influenced the analysis.

In this study, all confounders are covariates. A covariate is a variable that is linked to both the risk factor or exposure and the outcome. A covariate may or may not interact with the exposure or risk factor. The exposure or risk factor is cell phone-driven RFR via the use of cell phone. The health outcomes under investigation are thyroid cancer, mouth/tongue/lip cancer, and heart condition or disease. The confounding variable of interest is age. Other covariates considered are race, gender, marital status, employment status, familial history, and menopausal status.

Diagrammatic illustration of the confounders and covariate of the study

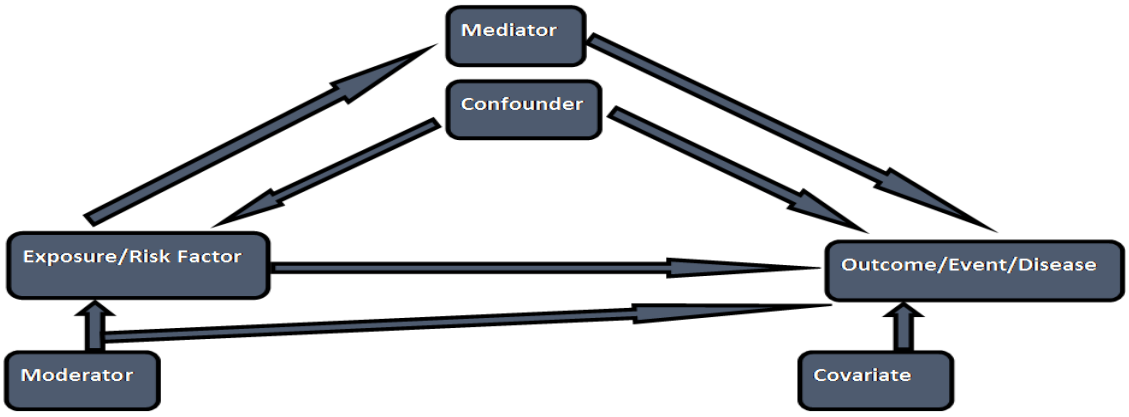


Figure 16. Confounder vs. Covariate and Mediator vs. Moderator.

Figure 16 is the diagrammatic relationship between several test variables or exposures or risk factors and the outcome variables. Figure 17 specifically shows the inter-relationship between test variables (independent variable, covariates, confounders) in this current study setting and the exposure or risk factors and the outcome variables. It is not possible to account for all the mediators, moderators, covariates and confounders in this study because the scope of the analysis for this study is limited to the information captured in the secondary data set, (see Figure 17 for more detail).

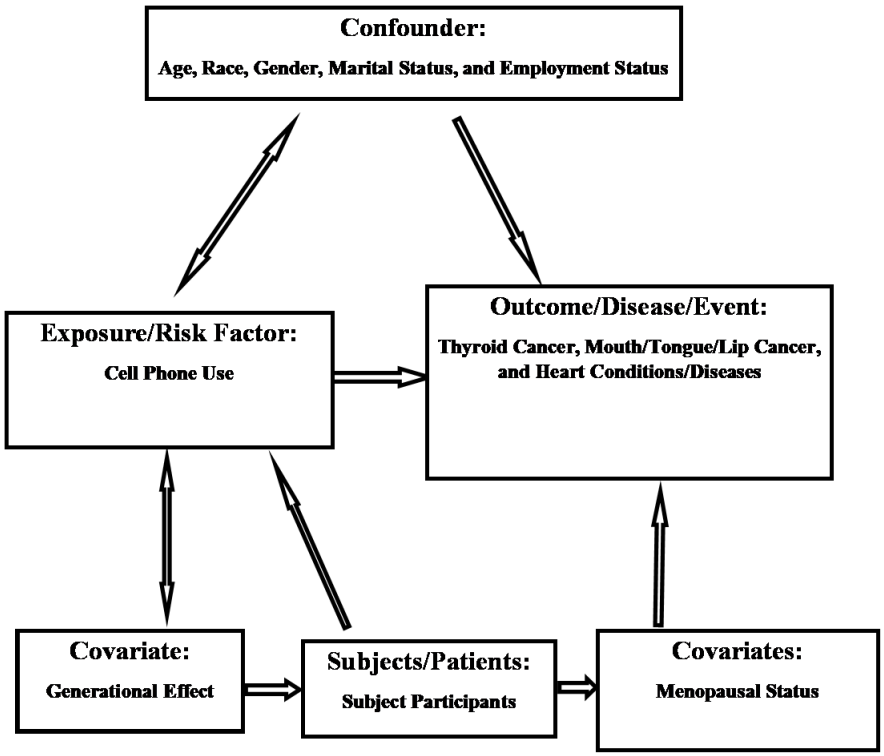


Figure 17. Confounder and Covariate and its Relationship to the Outcomes.

Confounders are covariates, but not all covariates are necessarily confounders (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011). In Figure 17, the generational effect is linked to the exposure or risk factor (cell phone use/cell phone-

driven RFR exposure) and indirectly to the health outcomes (thyroid cancer, mouth/tongue/lip cancer, and heart disease). Therefore, the generational effect is a mediator or moderator. Covariates such as age, race, gender, marital status, and employment status were both linked to thyroid cancer, mouth/tongue/lip cancer, and heart disease, and the cell phone use/cell phone-driven RFR exposure, which makes the variables a confounder. On the other hand, familial history and menopause status were represented as a covariate because they are directly linked to thyroid cancer, mouth/tongue/lip cancer, and heart disease but not cell phone use/cell phone-driven RFR exposure. Exploring this further, the following evidence-based descriptions were presented to support the concept. In the descriptions below, the use of the term 'health outcome' represents thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease.

Covariate Rationale: Familial history

Familial history is a covariate when:

- Familial history is associated with the health outcome risk (individual with familial history of the outcome from either parent or both parents could be prone to the health outcome than those that did not have any familial history regardless of the exposure to cell phone use).
- Familial history is not associated with the exposure to high use of cell phone calls/cell driven RFR exposure.
- Familial history is only linked to the health outcome risk but not considered a confounder.

- The cohort groups (exposure and non-exposure groups) were selected based on the exposure criteria of cell phone use, and the familial history is not associated with high cell phone use/cell phone driven RFR exposure. Hence, with a large randomized sample size for the cohort groups, the inter familial history profiles of the cohort groups should be similar. Therefore, under this operational sampling technique, the inter familial history variations may not influence the statistical analysis.

However, if there is a significant difference in the inter familial history composition of the cohorts, the statistical analysis could be influenced. Hence, the statistical analysis should be adjusted for or stratified by familial history status, or perhaps, subjects with familial history should be excluded from the study if and when possible.

Covariate Rationale: Menopause status

Menopause status is a covariate when:

- Menopause status (for female) is associated with the health outcome risk (individual at different menopause status may be exposed to different health outcome risk due to hormonal changes and hormonal replacement therapy (HRT) use if applicable regardless of the exposure to cell phone use).
- Menopause status is not associated with the exposure to high use of cell phone calls/cell driven RFR exposure (Jackson et al., 2008; Rainie & Zickuhr, 2015).
- Menopause status is only related to the health outcome risk, but not considered a confounder (Oxford University, n.d.).

- The cohort groups (exposure and non-exposure groups) were selected based on the exposure criteria of cell phone use, and the menopause status is not associated with high cell phone use/cell phone driven RFR exposure. With a large randomized sample size, the inter menopause status profiles of the cohort groups should be similar. Therefore, under this sampling technique, the inter menopause variations may not influence the statistical analysis.

In cases where there is a significant difference in the inter menopause composition of the cohorts, the statistical analysis could be influenced. In such a case, the statistical analysis should be adjusted or stratified by menopause status.

Confounder Rationale: Generational effect

Generational effect is a mediator/moderator or confounder when:

- Generational effect such as the 'number/duration' of cell phone owned/used (by frequency of cell phone use or time spent in handheld cell phone per call) is associated with the risk of the health outcome. Also, 'in time', people who could afford cell phone ownership and cell phone subscription costs may have different risk factors based on the how often they use cell phone for calls. Perhaps, these group of individuals may have high cell phone driven RFR exposure than those with limited access to cell phones.
- Generational effect is associated with the cultural and social norms of cell phone use and, influenced by the behavioral attributes linked to cell phone use within the context of the social construct of the target population (Rainie & Zickuhr, 2015). The high demand for cell phone use could be linked to 'social-pressure' or

'environmental-pressure-driven' lifestyle behavior (Rainie & Zickuhr, 2015). In such a social environment, individuals are more inclined to own or use cell phones for daily activities (Rainie & Zickuhr, 2015).

- Generational effect within this study operational construct is associated with both the health outcome risk and cell phone use. Generational effect is a mediator or moderator or confounder variable.
- The study cohorts were selected based on cell phone use/cell phone-driven RFR exposure criteria. The generational effect is associated with cell phone use. Therefore, cell phone users may have a profound attitude towards the technology utility and application based on the generational effect. In turn, the utility and application lifestyle could influence health outcome risks.

Cautiously, the health outcome risk may appear to be higher among cohorts that use cell phones often. In reality, high risk for the health outcome is associated to the generational effect. Therefore, for the statistical analysis purposes and accuracy, known generational effect factors should be adjusted or stratified accordingly.

Confounder Rationale: Age

Age is a confounder when:

- Age is an example of generational effect factor.
- Age could promote or diminish the inclination for cell phone ownership and usership. Also, people who could afford cell phone ownership and cell phone subscription costs may have different risk factors based on how often they use cell

phones for calls and perhaps, may have high cell phone-driven RFR exposure than individuals with limited access to cell phones

- Age is associated with cultural and social norms of cell phone use and influenced by the behavioral attributes linked to cell phone use within the context of the social construct of the target population (Rainie & Zickuhr, 2015). The high demand for cell phone use could be linked to 'peer-pressure' or 'age-driven-pressure' lifestyle behavior. Hence, individuals are more inclined to own or use cell phones for daily activities (Rainie & Zickuhr, 2015).
- Age is associated with both the health outcome risks and cell phone use, therefore, age is a confounder variable (Gesing, Lewiński, & Karbownik-Lewińska, 2012).
- The study cohorts were selected based on cell phone use/cell phone-driven RFR exposure criteria. Since age is associated with cell phone use, users may have profound age effect that is linked to the health outcome or its associated risks.

The health outcome risk may appear to be higher among cohorts that use cell phones often. In reality, high risk of the health outcome is associated with age. Therefore, the statistical analysis should be adjusted or stratified by age.

Confounder Rationale: Race

Race is a confounder when:

- Race is associated with the health outcome risk (individual in certain race groups could be at a higher risk for thyroid cancer, mouth/tongue/lip cancer, and heart disease than those in other race groups).

- Race is associated with exposure to the high use of cell phone calls/cell driven RFR exposure (Jackson, Zhao, Kolenic III, Fitzgerald, Harold, & Von Eye, 2008). In other words, individuals within certain race groups disproportionally use cell phones than those in other racial groups.
- Race is linked to both health outcome risk and cell phone use (Jackson et al., 2008).
- The cohort groups (exposure and non-exposure groups) were selected based on cell phone use exposure criteria. Race is not associated with cell phone use or cell phone-driven RFR exposure (see Figure 17). With a large randomized sample size for the cohort groups, the racial composition should be similar. With a large sampling size, race may not influence the statistical analysis.

Therefore, to assess the significant difference if any, in the racial composition of the cohorts, the statistical analysis should be adjusted or stratified by race.

Confounder Rationale: Gender

Gender is a covariate when:

- Gender is associated with the health outcome risk (Women could have a different risk to the health outcome than their Men counterparts).
- Gender is associated with the exposure to the high use of cell phone calls or cell driven RFR exposure (Jackson et al., 2008).
- Gender is related to both health outcome risk and heavy cell phone use. Hence, it is a confounder variable (Jackson et al., 2008).

- The cohort groups (exposure and non-exposure groups) were selected based on cell phone use criteria. Gender is associated with high cell phone use or cell phone-driven RFR exposure (see Figure 17). In a large randomized sample size study, the intra-gender genetics and social characteristic profiles of the cohort groups should be similar. Perhaps, when the intra-gender variations are similar, gender characteristics will not influence the statistical analysis. An inter-gender variability should be accounted by gender stratification.

Perhaps, if there is a difference in the intra and inter-gender composition of the cohorts, the statistical analysis could be influenced by such differences.

Confounder Rationale: Marital status

Marital status is a confounder when:

- Marital status is associated with the health outcome risk (marital status may expose individuals to different biological penetrance effects and socioeconomic status that could enhance or diminish the health outcome risk regardless of the exposure to cell phone use).
- Marital status is associated with the exposure to the high use of cell phone calls or cell driven RFR exposure (Rice & Katz, 2003).
- Marital status may be related to both cell phone use and a health outcome risk (Rice & Katz, 2003).
- The cohort groups (exposure and non-exposure groups) were selected based on the cell phone use criteria. Marital status is associated with high cell phone use or cell phone-driven RFR exposure. With a large randomized sample size for the

cohort groups, the inter-marital status profiles should be similar. As such, the inter-marital status variations may not influence the statistical analysis.

If there is a significant difference in the inter marital status composition of the cohorts, the statistical analysis could be influenced by such differences. Therefore, the statistical analysis should be adjusted or stratified by marital status.

Confounder Rationale: Employment status

Employment status is a confounder when:

- Employment status is associated with the health outcome risk (Employment status may expose individuals to different biological effects and socioeconomic status that could promote thyroid cancer, mouth/tongue/lip cancer, and heart disease risks regardless of the exposure to cell phone use or perhaps, the level of cell phone use and RFR exposure).
- Employment status is associated with the exposure to the high use of cell phone calls or cell phone-driven RFR exposure (McHugh, Marcum, & Bonauto, 2016).
- The cohort groups (exposure and non-exposure groups) were selected based on the cell phone use criteria, and the employment status is associated with high cell phone use or cell phone calls (Shin, 2014; Zhang, Amos, & McDowell, 2008). With a large randomized sample size of cohorts, the inter-employment status profiles of the cohort groups should be similar. Therefore, the inter-employment status variations may not influence the statistical analysis.

If there is a significant difference in the inter employment status composition of the cohorts, the statistical analysis could be influenced by such differences. As a result, the statistical analysis should be adjusted or stratified by the employment status.

Population

The target population for the 2012-NHIS study included adults living in the US national territories. The NHIS sample size varies each year, but the sample size may be augmented if necessary (NCHS, 2015). In 2011-2012, the NHIS sample size in 32 states including the District of Columbia was augmented (NCHS, 2015). The sample size was augmented by 13% and 21% in 2011 and 2012, respectively (NCHS, 2015). Based on the NCHS report, the purpose of the augmentation was to increase the number of states through which reliable state-level estimates could be generated (NCHS, 2015). The 2012 NHIS's 21% increase in the sample size made the 2012 NHIS study the largest sample size since the current sample design was introduced in 2006 (NCHS, 2015).

The 2012 NHIS sample size or the interviewed samples included 42,366 households, with about 108,131 subjects or persons in 43,345 families (Family level component) (NCHS, 2015). The adult participants interviewed for the 'Sample Adult' portion of the study, provided a self-reported response to all posed questions in the questionnaire unless the adult participant was mentally or physically unable to respond to the questions (NCHS, 2015). The total number of individuals interviewed was 34,525 persons or subjects (NCHS, 2015). Throughout the interview process, there were about 468 cases where a knowledgeable proxy responded to the question instead of an adult

(NCHS, 2015). The age criteria for the 'Sample Adult' component was 18 years of age and older (NCHS, 2015).

The total household response rate was 77.6% (NCHS, 2015). The non-interview rate was 22.4% (NCHS,2015). Out of the 22.4%, 14.6% of the non-interview rate were the result of respondents' refusal and unacceptable partial interviews (NCHS, 2015). While the remaining 7.8% from the non-interview rate was primarily the result of failure to locate an eligible respondent at home, after repeated contact attempts (NCHS, 2015).

The conditional response rate for the 'Family level' component was 99.0% (NCHS, 2015). The 99.0% response rate for this component was estimated by dividing the total number of completed family interviews (43,345) by the total number of eligible families, which was 43,785 families (NCHS, 2015). In contrast, the unconditional or final response rate for the 'Family level' component was 76.8% (NCHS, 2015). The unconditional response rate was calculated by multiplying the conditional rate (99.0%) by the household response rate of 77.6% (NCHS, 2015).

The Sample Adult component's conditional response rate was 79.7% (NCHS, 2015). Similarly, the response rate was estimated by dividing the total number of completed Sample Adult interviews (34,525) by the total number of eligible sample adults (43,323) (NCHS, 2015). For the Sample Adult component, the unconditional or final response rate was 61.2%, which was calculated by multiplying the conditional response rate (79.7%) by the final family response rate (76.8%) (NCHS, 2015).
The NHIS 'Sample Child' component of the secondary data was not directly relevant in this dissertation. However, the record shows that the interviewed sample for the 'Sample

Child' component (based on the response from a knowledgeable adult in the family) was 13,275 children under the age of 18 years (NCHS, 2015). Based on the information, the conditional response rate for the 'Sample Child' level was 90.7% (NCHS, 2015). An estimate derived by dividing the total number of completed Sample Child interviews (13,275) by the total number of eligible sample children (14,637) (NCHS, 2015). The unconditional or final response rate for the Sample Child level was 69.7% (NCHS, 2015). Similarly, the Sample Child level's final response rate was calculated by multiplying the conditional rate (90.7%) by the final family response rate (76.8%) (NCHS, 2015).

In summary, one adult per family was randomly selected to participate in the 'Sample Adult questionnaire' (NCHS, 2013; NCHS, 2015). A knowledgeable adult in the household provided information for the 'Sample Child questionnaire' if and when applicable (NCHS, 2013; NCHS, 2015). The selected adults responded for themselves to the questions posed unless they are physically or mentally challenged or incapable to do so (NCHS, 2013; NCHS, 2015). Also, a knowledgeable adult in the household or caretaker may answer the posed questions in place of the selected individuals if the selected adult is physically or mentally incapable of answering (NCHS, 2013; NCHS, 2015). Similarly, for the 'family questionnaire', all adults who are members of the household, and who are 17 years of age or older, and who are at home during the time of the interview were invited to participate and to respond to the questions voluntarily, but one was selected (NCHS, 2013; NCHS, 2015). In the absence of adults' presence at home at the time of the interview process, a responsible adult family member who is at

least 18 years of age or older residing in the household provided the reported information (NCHS, 2013; NCHS, 2015).

Procedures for Current Questionnaire

The 2012 NHIS study was a collaborative effort between the NCHS and US Census Bureau (NCHS, 2013; NCHS, 2015). The US Census Bureau's interviewers collect the survey data or information (NCHS, 2013; NCHS, 2015). Each interviewer has a personal badge that identified the individual as an employee of the US Census Bureau (NCHS, 2013; NCHS, 2015). All the interviewers and personnel involved with the data collection process were employees of the federal government (NCHS, 2013; NCHS, 2015). The interviewers and personnel were trained by the US Census Bureau based on the specified procedures produced by the NCHS (NCHS, 2013; NCHS, 2015).

For ensuring data integrity, a signed statement was issued to guarantee the confidentiality of the information collected (NCHS, 2013; NCHS, 2015). All the 2012 NHIS data collected through the personal household interviews were conducted by authorized and trained interviewers (NCHS, 2013; NCHS, 2015). Each interview process was performed according to the NCHS stipulated procedures and protocols (NCHS, 2013; NCHS, 2015). Depending on the number of individuals in the family and health status of family members, the average time to finish all the parts of the survey is about an hour (NCHS, 2013; NCHS, 2015). The NHIS composed of multi-core parts (NCHS, 2013; NCHS, 2015). The 'Family Core' questionnaire was designed to collect information on persons in the family (NCHS, 2013; NCHS, 2015). The measures included information on household composition; health insurance coverage; linkage to

administrative databases; basic indicators of health status and utilization of health care services; and basic demographic characteristic profiles—race, sex, ethnicity, age, and income (NCHS, 2013; NCHS, 2015).

In each family, one sample adult, and one sample child (if applicable) are randomly selected (NCHS, 2013; NCHS, 2015). Information about each family was collected using the 'Sample Adult Core' and the 'Sample Child Core' questionnaires (NCHS, 2013; NCHS, 2015). The 'Sample Adult Core' and the 'Sample Child Core' questionnaires differ inherently in some measures because health issues between adults and children are not usually the same (NCHS, 2013; NCHS, 2015). Both questionnaires were tailored to allow interviewers the ability to collect basic information on health-related behaviors, health status, and health care services (NCHS, 2013; NCHS, 2015). The NHIS questionnaire is a dynamic process because each year, supplemental questions are added to reflect new public health findings or needs, and to collect detailed information on core topics or to address the unmet needs (NCHS, 2013; NCHS, 2015).

Sampling and Sampling Procedures

The NHIS is one of the largest survey-driven studies in the United States aimed in recruiting thousands of families or households or persons in the US into the national cross-sectional study design in an attempt to address several health indicators (NCHS, 2013; NCHS, 2015). Therefore, the application of reliable sampling approaches is necessary to capture valid measures. The 2012 NHIS samples were selected through a multistage process (NCHS, 2013; NCHS, 2015). The sampling approach began with the selection of geographic areas, referred to as the primary sampling units (PSU) (NCHS,

2013; NCHS, 2015). The PSU was defined within the sampling strata (NCHS, 2013; NCHS, 2015). The NHIS files available to the public consisted of variance estimation strata and variance estimation PSUs (NCHS, 2015). To limit disclosure risks and other sensitive information the files were similar but not identical to the sampling PSUs and sampling strata (NCHS, 2013; NCHS, 2015). At least, two variance estimation PSUs were required to perform the variance calculations in each variance estimation stratum (NCHS, 2013; NCHS, 2015). Cases where only one variance estimation PSU is the variance estimation stratum, the PSU is referred to as a "singleton PSU" (NCHS, 2013; NCHS, 2015). The presence of a singleton PSU in a variance estimation stratum, the application of special techniques was required to generate the appropriate variance estimates (NCHS, 2013; NCHS, 2015).

Complex sample design software packages such as SUDAAN, Stata 10, R (plus the Survey add-on package) can compute appropriate variance estimates for a singleton PSU (NCHS, 2015). In contrast, software such as SPSS, Stata 9, SAS survey procedures could not be used to compute appropriate variance estimates for singleton PSUs (NCHS, 2015). Therefore, the NCHS teams generated supplemental files that allow users compute variance estimates appropriately (NCHS, 2015). For instance, NCHS emphasized that the use of Stata 9 for the statistical analysis with a non-supplemental file will generate missing values for standard error estimates (NCHS, 2015). Similarly, the use of SPSS and SAS analyses with non-supplemental files or data would produce standard error estimates that are slightly smaller (NCHS, 2015).

The sampling approach for the selected participants was performed in such ways that each subject in the target population had a known non-zero probability of selection, and the selection is random (NCHS, 2013; NCHS, 2015). The multistage sampling approach used for the 2012 NHIS was representative of noninstitutionalized individuals of the US population (NCHS, 2013; NCHS, 2015). Person's basic weight was recorded to enhance for proper analysis of person-record data, and each file's weights were based on the unit of analysis (NCHS, 2013; NCHS, 2015). The 'Weight-Final Annual' (WTFA) estimate was based on design and ratio adjustments (nonresponse and post-stratification included) (NCHS, 2015).

The participants were diversified with a broad range of backgrounds, health experiences, and lifestyle behaviors or perspectives (NCHS, 2015). With a diversified target population, the selection pool provides meaningful evidence when evaluating the effects of a particular exposure or risk factor and its relationship to health conditions. Such evaluation quality may not be possible with a smaller and non-diversified target population. Subjects who participated in the NHIS were not advised or asked to change their lifestyle behaviors or perspectives (NCHS, 2015). These selected subjects or families were only asked to provide their medical information, lifestyle, risk or exposure factors, and life-experience based on the posed questions contained in the 2012 NHIS research questionnaire (NCHS, 2015). Individuals or families who accepted the invitations to participate in the study and are within the acceptable parameters of the inclusion or exclusion criteria for recruitment into the study gave informed consent

(NCHS, 2015). No compensation or other incentives were advanced and provided for participation in the 2012 NHIS study (NCHS, 2015).

The 2012 NHIS questionnaire database contained the variables needed to address the research questions posed in this current study. An official secondary data access request via email was sent (by me) to the NCHS and ICPSR to the following email addresses listed; NHIS@CDC.gov and icpsr-user-support@umich.edu respectively. The NCHS and ICPSR teams responded to the email requests. Both organizations indicated that no special permission was needed for access to the 2012 NHIS dataset and any other publicly released questionnaires uploaded or published in the public domain (either in the NCHS or ICPSR website) for either the purpose of dissertation or future research publication. Furthermore, via an email correspondence the NCHS team indicated that all the de-identified public data and documentation about the 2012 NHIS study were available at the following link: (https://www.cdc.gov/nchs/nhis/data-questionnaires-documentation.htmhttp://www.cdc.gov/nchs/nhis.htm). In the ICPSR website, the data sets and de-identified publicly released questionnaire for the 2012 NHIS study are located at the following link: (https://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/36146 ?searchSource=find-analyze-home&sortBy=&q=NATIONAL+HEALTH+SURVEY %2C+2012). According to the information received from the NCHS team via email correspondence, the proper citation format for the data is located on page 6 of the following link (ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Dataset_Documentation/ NHIS/2015/ srvydesc.pdf). Page 6 provides documentation on how to cite the data (NCHS, 2015). The links listed do not contain the totality of the information due to

confidentiality reasons (NCHS, 2013; NCHS, 2015). Some variables and contents were restricted from the public domain (NCHS, 2013; NCHS, 2015). The list of the restricted variables is located at the following link (https://www.cdc.gov/rdc/b1datatype/dt1225 .htm) (NCHS, 2013; NCHS, 2015).

Researchers interested in the restricted variables may request access to the data set through the NCHS' Research Data Center (RDC) for special permission (NCHS, 2013; NCHS, 2015). By submitting a research proposal request for restricted data access, a review will be conducted by a committee to determine whether or not to grant or deny data access (NCHS, 2013; NCHS, 2015). In cases, where the RDC committee approves a request, the individual or organization requesting access to the restricted data will have permission to the variables and the dataset of interest requested (NCHS, 2013; NCHS, 2015). Access to restricted data may not be cost-free but may involve some data and administrative fees for accessing the internal data files (NCHS, 2015). Detailed information about the restricted data request form is located in the NCHS' RDC website (http://www.cdc.gov/rdc/), or an email request could be sent to rdca@cdc.gov for further inquiry (NCHS, 2013; NCHS, 2015). The approach used for sample selection in the current study (dissertation) using the 2012 NHIS secondary data to meet the minimum estimated sample size requirement of 95% or more statistical power and 95% confidence level was to use the whole data sample size of over 43,000 families. The sample was already randomly selected based on the NCHS sampling approach.

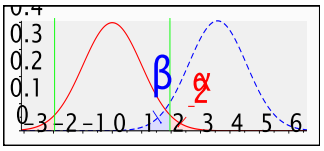
Power Calculation for Logistic Regression

For at least generating the effect size or odds ratio of 1.3, the estimated sample size required for this study using a logistic regression with an alpha value of 0.05 and 0.95 statistical power is 1188 samples (see Table 4, Figure 18 and 19).

Table 4

Protocol of Power Analyses

z tests - Logistic regression			
Options:	Large sample z-Test, Demidenko (2007) with var corr		
Analysis:	A priori: Compute required sample size		
Input:	Tail(s)	=	Two
	Odds ratio	=	1.3
	Pr(Y=1|X=1) H0	=	0.2
	α err prob	=	0.05
	Power (1-β err prob)	=	0.95
	R^2 other X	=	0
	X distribution	=	Normal
	X parm μ	=	0
	X parm σ	=	1
Output:	Critical z	=	1.9599640
	Total sample size	=	1188
	Actual power	=	0.9501294



Figure

Figure 18: Central and Non-central Distributions.

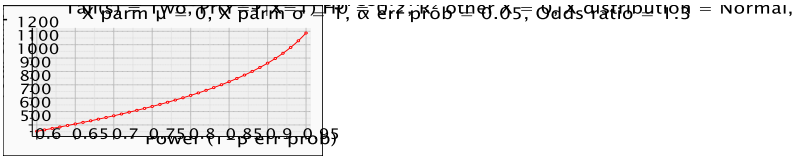


Figure 19: GPower Plot for sample size estimation

Data Analysis Plan

The type of statistical approaches used by the NHIS depended heavily on the construct of the research inquiry, levels of measurements of the DVs and IVs, and the purpose of the research inquiry. For instance, the statistical software used in the recent NHIS publications and data set descriptions included the statistical package for social sciences (SPSS), statistical analysis system (SAS), R, Stata, ASCII, and Excel/TSV (ICPSR, 2016; NCHS, 2015). In this current study, I used the SPSS for the data analysis. The types of the statistical tool for the data analysis used depended heavily on the construct integrity of the research questions, levels of measurements of the DVs and IVs, and the purpose of this research inquiry. In this study, the IV's (cell phone use) level of measurement was a categorical variable, and also quantitative. A categorical variable for cell phone use was produced by posing the following question; Of all the telephone calls that you or your family receives, are all or almost all calls received on cell phones? The response options for the question were coded as follows; 1) All or almost all calls

received on cell phones; 2) Some received on cell phones and some on regular phones; 3) Very few or none on cell phones; 7) Refused; 8) Not ascertained; and 9) Don't know. The second question related to the exposure or ownership of cell phone was a quantitative measure. The quantitative questionnaire question was; How many working cell phones do you or people in your family have? The response options were grouped by the number of cell phones owned or present in the household.

On the other hand, the DVs (thyroid cancer, mouth/tongue/lip cancer, and heart condition or disease) were measured as a nominal variable (cases or no cases). Based on the specified levels of measurements for the IV and DVs for this study research inquiry, a binary and multiple logistic regression is appropriate and was used for the statistical analyses (Aschengrau & Seage, 2014; Creswell, 2009; Frankfort-Nachmias & Nachmias, 2008). Similar statistical method criteria were used for the covariate or confounder interaction and modification effect evaluation within the statistical model. The application of appropriate statistical approaches helped in advancing meaningful explanation on whether there was a meaningful difference between the exposure groups, and if so, whether the difference was significant. Accordingly, the risk (odds ratio) was calculated based on the estimation of the health outcomes' differences or similarities between groups exposed to cell phone RFR those who were not exposed to cell phone RFR or those with minimal exposure.

The data manipulation process for the 2012 NHIS dataset included coding and recoding. For the cell phone use or exposure (IV), the responses reported in the 2012 NHIS dataset were consolidated to only three responses: ‘All or almost all calls received

on cell phones'; 'Some received on cell phones and some on regular phones'; and 'Very few or none on cell phones'. The three responses were coded in the data variable as 1, 2, and 3 respectively. The DV (yes/cases and no/no cases) were coded as 1 for the 'yes' or 'cases', and 2 for the 'no' or 'no-cases'. The gender variable was coded as well. Other confounders and covariates such as age group, marital status, race, and employment status were coded accordingly. The normality curve was plotted to identify the outliers. Before the data analysis, the codebook and data dictionary were reviewed for data integrity assessment, missing data counts, and computation of appropriate adjustments of the missing data. Subjects with a familial history of thyroid cancer, mouth/tongue/lip cancer, and heart conditions were not excluded from the sample statistical analyses because the number of cases observed was small.

The primary research questions and hypotheses that would be addressed in this study are stated as follows:

Research Questions and Hypotheses

RQ1. What is the difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_{o1}: There is no difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_{a1}: There is a difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

The IV addressed in this research question was cell phone use based on little (few) or no cell phone use or exposure and reception of all or almost all calls on cell phones. The DV cases evaluated was thyroid cancer prevalence difference between cell phone users and non-cell phone users or few users. The covariates evaluated were age, race, gender, marital status, and employment status. Gender, marital status, and employment status were all nominal or categorical variables. Therefore, for analysis involving these variables, multiple logistic regression was used to address the effects of these variables on thyroid cancer. Variables that showed an interaction effect or influenced thyroid cancer in the presence of the IV was added to the logistic regression model.

RQ2. What is the difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_{o2}: There is no difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_{a2}: There is a difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

RQ3. What is the difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_{o3}: There is no difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_{a3}: There is a difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

For the research question 2 and 3, the IV evaluated was cell phone use. The DV for research question #2 was mouth/tongue/lip cancer, and heart conditions for question #3. The assessment of the health outcome was based on the prevalence difference estimation between cell phone users and non-cell phone users or those that rarely use cell phones. Similarly, the covariates evaluated were age, race, gender, marital status, and employment status. Gender, race, age group, marital status, and employment status were either a nominal or categorical variable. Therefore, multiple logistic regression was used to estimate the interactive effects of these variables on mouth/tongue/lip cancer or heart conditions in the presence of cell phone use exposure.

Threats to Validity

In this study, the threats to validity considerations include the internal and external validity concerns. In the absence or lack of internal validity, external validity would not be achieved (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011;

Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The primary internal validity concern in this study was misclassification bias. Misclassification bias specific to this study may have been incurred due to the way the survey questionnaire questions were constructed. The questionnaire was not primarily constructed to evaluate the participants' cell phone use or exposure and their RFR exposure levels. For instance, the following questions were not specific to the direct user; 'Of all the telephone calls that you or your family receives, are all or almost all calls received on cell phones?' 'And How many working cell phones do you or people in your family have?' (NCHS, 2015). This question could apply or not apply to the individual with the health out evaluated. The two primary questions about cell phone exposure from the 2012 NHIS are the key questions that addressed the research questions posed in this dissertation. Based on the possibility of misclassification occurrence, the responses generated from the two questions about cell phone exposure or use were self-reported responses and perhaps subjective. The accuracy of cell phone use or exposure could not be confirmed. There were no quantifiable instruments used by NCHS to validate the responses and accurately measure the cell phone-driven RFR exposure levels and duration of the exposure, such as the participants' phone records. As a result, there could have been instances where the participants are prone to recall bias or respondents' bias. Such biases could have led to misclassification bias and subsequently led to either a Type I or Type II error.

Another concern that could have produced an internal validity threat was that the 2012 NHIS questionnaire did not include any question to assess whether a handheld mode or inbuilt cell phone speaker or wireless blue tooth or wired-headset accessory was

frequently used by the participant while answering a phone call or talking on a cell phone. The lack of differentiation and clarification of means of exposure is crucial because the use of a headset and inbuilt speaker reduces the cell phone-driven RFR exposure to the head, neck area, and chest. In contrast, individuals using a blue-tooth and handheld cell phone frequently to talk, have higher cell phone-driven RFR exposure. Therefore, the lack of verifiable classification of the level of exposure based on the individual cell phone use behavior could have led to gross misclassification bias. Such misinformation bias could have distorted the findings either towards or away from the null hypothesis. Therefore, these potential internal validity threats could have inadvertently influenced the external validity integrity. One way this could have been corrected is to verify the participants' cell phone ownership and usage information (phone record). Unfortunately, the participants' phone records were not captured in the 2012 NHIS secondary dataset or questionnaire administered by the NCHS.

Scientific data must be valid to be reliable. Reliability and validity increase the chances of internal and external validity (Aschengrau & Seage, 2014; Gordis, 2009; Krieger, 2011; Moeller, 2011; Satariano, 2005; Szklo & Nieto, 2014). The 2012 NHIS contained relevant variables that addressed the current research questions. The construct's parameter implicated with the independent variable (cell-phone ownership or cell phone-driven RFR status or no cell phone ownership or no RFR exposure or low RFR exposure) was based on the duration of use and cell phone ownership. The availability, consistency, and accuracy of responses to the cell phone use or exposure question may have reduced the chances of misclassification bias. The 2012 NHIS was a

national study involving many states in the US with approximately 43,345 families, 42, 366 household and 108,131 participants, which may have enhanced the external validity integrity of this study (NCHS, 2015). The sample size was large. Therefore, as all the samples were used for the statistical analysis, the findings were representative of the target population at risk for the health outcomes under investigation in the US. Unfortunately, the subjects' medical record or medical history and familial history were not evaluated, which posed an inherent threat to validity regarding the attributable effect of the exposure to the health outcomes evaluated. The descriptive statistics of the participants' age, socioeconomic status, race, employment status, and marital status were evaluated.

Ethical Procedures

The 2012 NHIS protocols and procedures contained informed consent information (NCHS, 2015). The 2012 NHIS study processes require consistent adherence to the informed consent, confidentiality, security, and ethical standards (NCHS, 2015). The recruitment process, data collection, and other areas of the study were monitored for the sole purpose of protecting and respecting the privacy and personal information of the subjects in the study (NCHS, 2015). The study activities conformed to current local, regional, federal, and international ethical and legal guidelines about the informed consent, confidentiality, personal health information, and the use of human biological outcomes specified in the NIH guidelines (NCHS, 2015; NIH, 2007). All information associated with the 2012 NHIS were handled appropriately based on standard ethical guidelines.

The processes of the 2012 NHIS adhered to the guidelines outlined in HIPAA (ICPSR, 2016; NCHS, 2015; NIH, 2007). The personal identifying information was secured and separated from the public databases (NCHS, 2015). The data information was de-identified, and the statistical analysis did not include any individual names. The personal or household's residential addresses were protected and removed from public access. The previous publications by the NCHS were reported in de-identified format (NCHS, 2015). This current publication is also de-identified. Enrollment and participation in the 2012 NHIS were entirely voluntary (NCHS, 2013; NCHS, 2015). All participants provided informed consent. The NCHS provided other relevant information about participation. The provided information includes the following:

- The participants' future health care will not be affected in any way, due to their decision on whether or not to participate in the study (NCHS, 2015).
- The participants are free to withdraw at any time during the interview (NCHS, 2015).
- All collected information are safeguarded, and sensitive information is highly confidential. The information and collected data should only be used for research purposes and in such cases, must not identify the individuals from which the data or information was collected (NCHS, 2015).
- Participants' information is collected into microdata files that are edited to remove all personal identifiers (NCHS, 2013; NCHS, 2015). The edited files are released to the public via the NHIS website or other collaborative partners' sites (NCHS, 2013; NCHS, 2015).

- The confidentiality of participants' responses and participation is assured under Section 308(d) of the 'Public Health Service Act' (NCHS, 2013; NCHS, 2015).
- The NHIS study is very important in furthering scientific research. The collected data are used solely for research and statistical purposes (NCHS, 2015). Therefore, additional analyses in the future cannot be specified or known at present (NCHS, 2015).
- When the NCHS staff prepare, and release analytical reports, each participants' response is combined with many other respondents' responses. Hence, no information that could identify any individual is publicly released (NCHS, 2013; NCHS, 2015).
- Statistically analyzed data are published in several types of reports, which may be released through the internet or in journal sites (NCHS, 2013; NCHS, 2015).
- The study was approved by the Institutional Review Board (IRB) (NCHS, 2015).
- For more information on how participants' privacy is respected and protected visit the following link: https://www.cdc.gov/nchs/about/policy/confidentiality.htm (NCHS, 2013; NCHS, 2015).
- For more information about the available microdata files and reports, visit the link below (https://www.cdc.gov/nchs/nhis/index.htm) (NCHS, 2013; NCHS, 2015).

Regardless of the health status, the 2012 NHIS participants were randomly selected and were representative of all types of households and families in the US (NCHS, 2013; NCHS, 2015). Even when participation is voluntary, another household or family or person cannot be selected to replace participants who were selected but was

unable to participate or perhaps refused participation (NCHS, 2013; NCHS, 2015). As a result, it is possible that such households could be underrepresented in the national estimates if such cases frequently occurred (NCHS, 2013; NCHS, 2015). Among the eligible households in the sample pool, the annual response rate of NHIS was approximately 90% (NCHS, 2013; NCHS, 2015).

The NCHS' data security procedures were tightly secured to prevent unauthorized invasion and disclosure of participants' data (NCHS, 2013; NCHS, 2015). For instance, the NCHS uses secure data networks, data encryption, and other security techniques that strictly adhere to the federal mandates and regulations on personal and sensitive information security (NCHS, 2013; NCHS, 2015). All responses collected by the US Census Bureau were securely transmitted to the NCHS (NCHS, 2013; NCHS, 2015). Once the data is received at the NCHS branch, authorized NCHS employees initiated data edits, and the removal of personal identifiers from the datasets before the data file is uploaded to the public use files and domain sites (NCHS, 2013; NCHS, 2015).

Participation required informed consent for the storage of the 2012 NHIS data or information obtained from the participants for any current or future research (NCHS, 2015). Participants were also encouraged to ask questions or send in comments or requests on any aspect of the study either through writing or by calling the study's toll-free number (NCHS, 2015). The approval process for the IRB was sought for the 2012 NHIS study by the NCHS team (NCHS, 2015). For the current dissertation, the Walden IRB approved permission for access to the 2012 NHIS dataset before the data analysis was performed.

Summary

The opportunity provided by the NCHS' 2012 NHIS dataset archivE substantially advanced informed knowledge that provided key information on health determinants among the US households. Such information further advanced our understanding about several health risk factors associated with handheld cell phone use or cell phone-driven RFR exposure and other health outcomes besides those under investigation in this study. Perhaps, it could in the future help researchers and policymakers propose early preventative measures for the exposures, outcomes, interventions, and practical corrective approaches. The findings from this study were presented in Chapter 4, while the conclusion drawn was presented in Chapter 5 of the dissertation.

Chapter 4: Results

Introduction

Chapter 4 contains the quantitative analyses and results generated from this research inquiry. In the chapter, I estimated the prevalence of thyroid cancer, mouth/tongue/lip cancer, and heart conditions. I calculated the prevalence estimates based on whether there was an association between the investigated health outcomes and the level of cell phone use or cell phone-driven RFR exposure. The NCHS-NHIS-2012 data, which included surveys from several states in the United States was used to estimate the prevalence and risk differences of thyroid cancer, mouth/tongue/lip cancer and heart disease between persons who received all calls/almost all calls and those who received some or no calls on cell phones. I performed stratification by race, age, gender, employment status, and marital status in the analyses when necessary and applicable. Mobile phone use and the specified health outcomes were recorded through a self-reported survey via an interview process by trained professionals. The measured predictor variables and the reported health outcomes were analyzed descriptively and inferentially. For the inferential analyses, I used binary and multiple logistic regressions to evaluate the following research questions and hypotheses:

RQ1. What is the difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_o1: There is no difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_a1: There is a difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

RQ2. What is the difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_o2: There is no difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_a2: There is a difference in the prevalence of mouth/tongue/lip cancer between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

RQ3. What is the difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones?

H_o3: There is no difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

H_a3: There is a difference in the prevalence of heart condition/disease between individuals who received all or almost all calls on cell phones compared to individuals who received very few or no calls on cell phones.

Using the 2012 NHIS dataset and SPSS software, I performed descriptive analyses of cell phone use or exposure, thyroid cancer, mouth/tongue/lip cancer, and heart conditions. The prevalence and risk analyses of thyroid cancer, mouth/tongue/lip cancer, and heart conditions were performed as well to address the research questions and hypotheses. The conclusion drawn from the analyses was presented in Chapter 5 of this dissertation, along with a discussion of the limitations of the research inquiry and suggestions for further studies.

Data Collection

The 2012 NHIS data were collected via a face-to-face interview conducted at the participants' homes when it is possible to do so (ICPSR, 2016; NCHS, 2015). A follow-up phone interview was conducted to complete the survey (ICPSR, 2016; NCHS, 2015). The US census bureau administered the 2012 NHIS interviews nationwide to include households that were representative of the U.S. population (ICPSR, 2016; NCHS, 2015). With a multistage area probability sampling approach, non-institutionalized samples of U.S. civilians were selected each month (ICPSR, 2016; NCHS, 2015). The information gathered from selected civilians was about the health and characteristic profiles of each member of the household (ICPSR, 2016; NCHS, 2015).

Collection of the NCHS 2012 NHIS Dataset

I obtained the 2012 NHIS dataset via the NCHS website (https://www .cdc.gov/nchs/nhis/index.htm). The 2012 NHIS dataset was also available on the ICPSR website (https://www.icpsr.umich.edu), which I also explored for relevant information about the study. The NCHS and ICPSR database are open source sites for de-identified part of the 2012 NHIS data (ICPSR, 2016; NCHS, 2013). From both websites, I downloaded the 2012 NHIS data as 'sav' files. The 'sav' file is the SPSS file format and does not require any additional compatibility conversion for use with the SPSS software. The downloaded zip files also contained the necessary codebook saved as a pdf file. For access to the 2012 NHIS dataset via the ICPSR website, an online membership registration with the ICPSR is required. As I was already a registered ICPSR member, obtaining the data from the ICPSR website did not require additional registration processes. The data set needed for this study was in two different files, the 'sample adult' and 'family level' files. I merged the two datasets and conducted appropriate data manipulations before using the dataset for the data analysis.

NHIS 2012 Structure and Sampling

The 2012-NHIS sample size augmentation started in 2011. The 2012 NHIS study samples included individuals, families, and households from 32 states and the District of Columbia in the United States (ICPSR, 2016; NCHS, 2013). The increase in the number of states for the 2012 NHIS sample recruitment was meant to ensure reliable estimates that were representative of the U.S. population (ICPSR, 2016; NCHS, 2013). Trained census interviewers from the U.S. Census Bureau agency collected the data for the 2012-

NHIS (ICPSR, 2016; NCHS, 2013). In the U.S. Census Bureau regional offices, a total of 750 trained Census interviewers or field representatives (FRs) were directed by the health survey supervisors for the surveys (ICPSR, 2016; NCHS, 2013).

The interviewers or FRs annually received thorough training in basic interviewing procedures, concepts, and procedures that are unique to the NHIS protocols (ICPSR, 2016; NCHS, 2013). The interviewers are periodically observed by supervisors (ICPSR, 2016; NCHS, 2013). The quality assurance and integrity of the interviewers' work were monitored by the PANDA system (ICPSR, 2016; NCHS, 2013). The PANDA system is a high performance and data analysis program used to provide periodic or routine monthly checks on response and completion rates (ICPSR, 2016; NCHS, 2013). All supervisors involved in the 2012 NHIS were career civil service employees selected via an examination and testing process (ICPSR, 2016; NCHS, 2013).

The 'household respondent' from each household was at least the legal age for a given state (ICPSR, 2016; NCHS, 2013). In many states in the US, the legal age without requiring informed accent from the parents for an interview is 18 years. In Alabama and Nebraska, the legal age is 19 years, and 21 years in Mississippi (ICPSR, 2016; NCHS, 2013). The household respondent provided basic demographic and relationship information about all the members of the household (ICPSR, 2016; NCHS, 2013). In a multi-family household, a single household respondent provided the household information for the family (ICPSR, 2016; NCHS, 2013). There were enhanced chances for selection of Blacks, Asians, and Hispanics aged 65 years or older (ICPSR, 2016; NCHS, 2013).

The first stage of the multistage area probability sampling plan involved selections of 428 primary sampling units (PSU's) (ICPSR, 2016; NCHS, 2013). The 428 PSUs were drawn from 1,900 PSUs (ICPSR, 2016; NCHS, 2013). A PSU is a county or small group of contiguous counties or metropolitan statistical area (ICPSR, 2016; NCHS, 2013). Geographically defined PSUs covered the 50 states and the District of Columbia within the US (ICPSR, 2016; NCHS, 2013). Two types of second-stage units were used within a PSU; the area segments and permit segments (ICPSR, 2016; NCHS, 2013). Area segments are geographically defined with an expected eight, twelve, or sixteen addresses (ICPSR, 2016; NCHS, 2013). Housing units built after the 2000 census are under the 'permit segment' criterion (ICPSR, 2016; NCHS, 2013).

Results

This section of chapter 4 contains the descriptive and inferential statistics for this study. The descriptive statistics for the independent variable (cell phone use), outcome variables (thyroid cancer, mouth/tongue/lip cancer, and heart conditions/disease), confounders (participants' age, race, marital status, employment status), and covariates (menopausal status) were performed. Following the descriptive analysis, I present the inferential statistics to address the research questions and hypotheses on the association between cell phone use and the prevalence of thyroid cancer, mouth/tongue/lip cancer, and heart disease. Tables 5-20 and figures 20-36 represent the distribution of the 2012-NHIS family and 'sample adult' level data files used in this study. Tables 21-68 represent the inferential statistics' assessments of the 2012-NHIS 'family' and 'sample adult' level data files used in this study.

Descriptive Statistics for the Family Level Data

In this section of the results, I provided the descriptive analysis of all the relevant predictor variables (cell phone use and duration of use). The predictor variables were presented in Table and Figure formats. Also, I provided a written explanation for each table and figure represented in this section of the results.

Phone and Cell Phone Use-Based Questions

In Table 5 below, a total of 43,345 participants were documented for the family level data file. Out of the 43,345 participants, 42,337 subjects had complete response information while 1,008 participants have at least one piece of a missing information. Table 5 and Figure 20 shows the distribution of families that have a working phone/landline inside the home. For Table 5 and Figure 20 descriptive analysis, the phone distribution described working phone in homes excluding cell phones.

Table 5

Working Phone in Home Excluding Cell Phone

		Frequency	*Percent*
Valid	Yes	24889	57.4
	No	17448	40.3
	Total	42337	97.7
Missing	Refused	80	.2
	Not ascertained	21	.0
	Don't know	11	.0
	System	896	2.1
	Total	1008	2.3
Total		43345	100.0

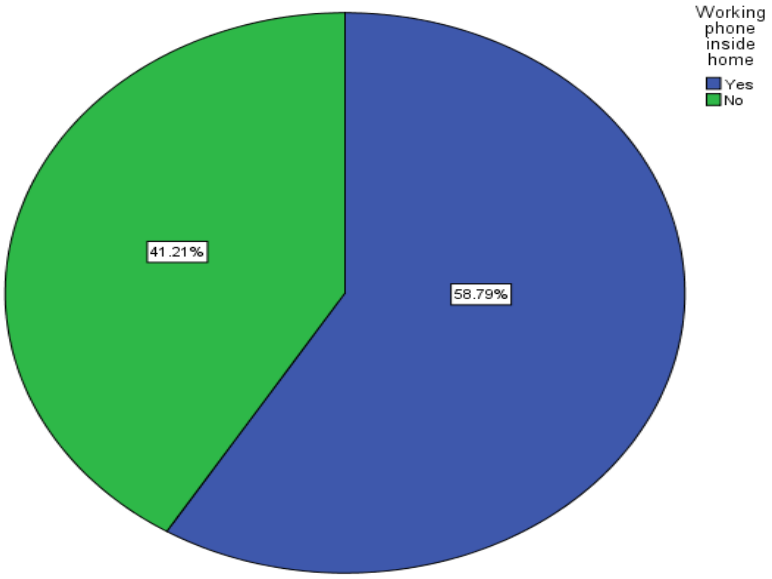


Figure 20. Working Phone inside Home.

Table 6 and Figure 21 show the proportion of participants that reported possession of at least a working cell phone in the family. Out of 43,345 participants, 38,136 (88%) reported that there is a working cell phone in the family. About 5,065 participants (11.7%) reported that there is 'no' working cell phone in the family. For the missing data on this question, 124 participants (0.3%) refused to answer the question, 19 participants (<0.1) do not know whether there is a working cell phone in the family, and 1 participant (<0.1) was recorded under 'system' as missing data.

Table 6

Working Cell Phone in Family

		Frequency	*Percent*
Valid	Yes	38136	88.0
	No	5065	11.7

	Total	43201	99.7
Missing	Refused	124	.3
	Don't know	19	.0
	System	1	.0
	Total	144	.3
Total		43345	100.0

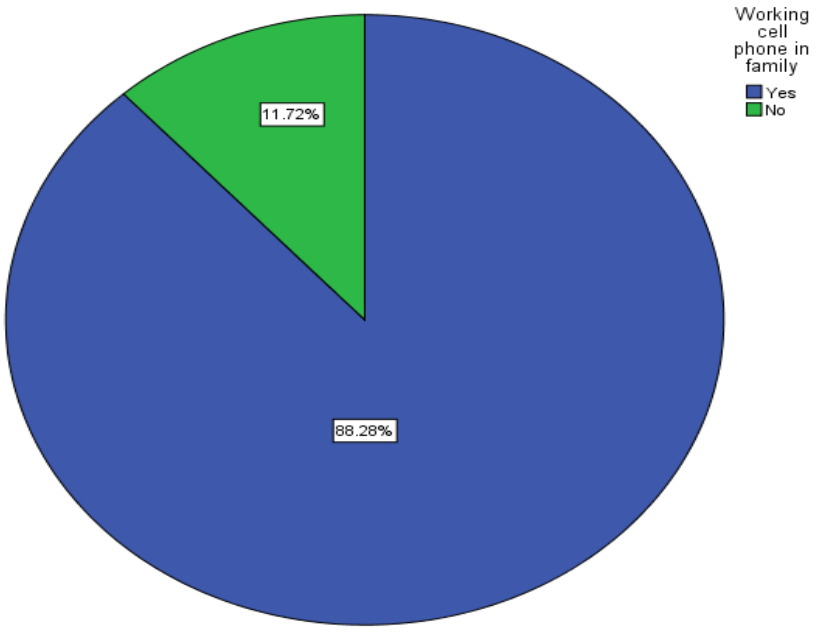


Figure 21. Working Cell Phone in Family.

In Table 7, a total of 43,345 participants were documented for the family level data file. Out of the 43,345 participants, 20,758 participants were valid or have complete information/response while 22,587 participants have some missing information/responses for this particular question. The valid category in Table 7 and Figure 21 contain the proportion of participants that had a complete response on the posed questions for the survey. For the response rate on the question, "*Of all the telephone calls that you or your*

family receives, are all or almost all calls received on cell phones, some received on cell phones, and some on regular phones or very few or none received on cell phones?", out of 20,758 participants that were not reported as missing data, 31.9% of the participants (6,617 participants) indicated that all or almost all calls were received on cell phones. About 41.6% participants (8,638 participants) indicated that some calls were received on cell phones and some calls on the regular phone. And 26.5% of the participants (5,503 participants) reported that very or no calls were received on cell phones. Accounting both the valid (20,758 participants) and missing sample data (22587 participants), which added up to 43,345 participants, the cell phone use proportion decreased. Using 43,345 as the total sample size as opposed to 20,758 participants (which excluded the missing data) for the cell phone calls received, and with this adjustment, 15.3% of the participants received all calls or almost all calls on cell phones, 19.9% received some calls on cell phones, and 12.7% of the participants received very few calls on cell phones.

For the missing data, there are three categories that were characterized as missing information/responses, these are 'Refused', 'Don't know', and 'System'. In Table 7, out of the 22, 587 participants with missing information/responses, 29 participants (0.1%) refused to answer the questions, 21 people (<0.1%) selected the "Don't know" response, and 22,537 participants (52%) were reported as 'System'. The 'system' missing values represented situations where the participants did not provide any response or never reported any response using the provided response options in the questionnaire. Figure 22 below shows the descriptive distribution of calls received by the participants on cell phones based on the three categories (*all or almost all calls received on cell phones, or*

some received on cell phones and some on regular phones, or very few or none received on cell phones).

Table 7

Received Calls on Cell Phones

Received calls cell

		Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*
Valid	All or almost all calls received on cell phones	6617	15.3	31.9	31.9
	Some received on cell phones and some on regular phones	8638	19.9	41.6	73.5
	Very few or none on cell phones	5503	12.7	26.5	100.0
	Total	20758	47.9	100.0	
Missing	Refused	29	.1		
	Don't know	21	.0		
	System	22537	52.0		
	Total	22587	52.1		
Total		43345	100.0		

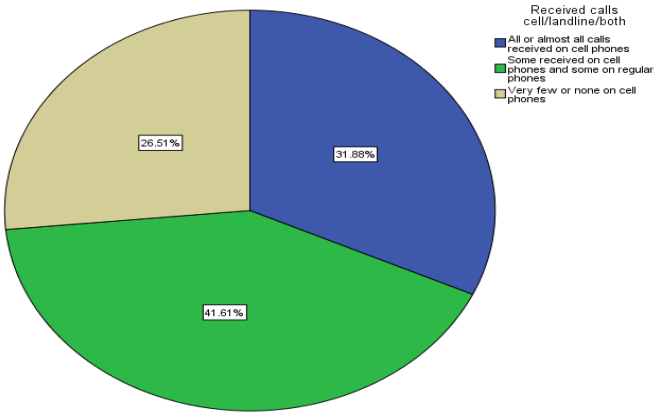


Figure 22. Received calls on Cell Phones.

Descriptive Statistics for the Sample Adult Level Data

In this section of the results, I provide the descriptive analysis of all the relevant health outcomes-thyroid cancer, mouth/tongue/lip cancer, and heart conditions/disease. In this section of the analysis, I descriptively show and explain the participants' age, race, marital status, employment status, and menopausal status for each of the health outcomes under investigation specified in each posed research question. The Tables and Figures representing the descriptive analysis for these variables are also summarized in this section of the proposal. The following health outcomes were analyzed.

Thyroid Cancer, Mouth/Tongue/Lip Cancer, and Heart Conditions/Disease

The respondent's distribution on the prevalence question, "*Have you EVER been told by a doctor or other health professional that you had...Cancer or a malignancy of any kind*?" was represented in Table 8. In Table 8 and Figure 23, the valid percent estimation included only individuals with a complete response and excluded the missing values. The percent column in Table 8 represent individuals with complete response and

those with missing values. There are 7.2 total percent and 9.0 valid percent among individuals who checked ‘Yes” to the question 'ever told by a doctor you had cancer?'. About 72.4 total percent and 91 valid percent of the participants indicated ‘No” to the same question. Out of the total sample size of 43,345 participants interviewed, 34,505 respondents were valid, while 8,840 participants refused or provided no response. Refusal or lack of response was considered as missing data.

Table 8

Proportion of Cancer Diagnosis by a Doctor

Ever told by a doctor you had cancer

		Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*
Valid	Yes	3118	7.2	9.0	9.0
	No	31387	72.4	91.0	100.0
	Total	34505	79.6	100.0	
Missing	Refused	10	.0		
	Don't know	10	.0		
	System	8820	20.3		
	Total	8840	20.4		
Total		43345	100.0		

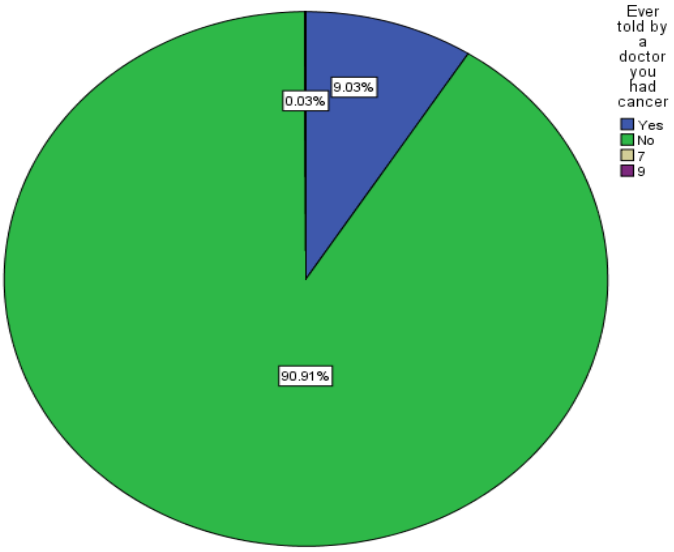


Figure 23. Cancer Diagnosis by a Doctor.

Table 9 and Figure 24 below represents the self-reported responses about thyroid cancer outcomes among the selected population. In Table 9, 79 participants (2.5 valid percent) reported or mentioned having thyroid cancer. In contrast, 3,027 participants (97.5 valid percent) did not report thyroid cancer outcomes. A total of 40,239 participants (92.8%) out of the total sample size (43,345) was recorded as missing cases for the thyroid cancer outcome.

Table 9

Proportion of Thyroid Cancer

What kind of cancer ... Thyroid

		Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*
Valid	Mentioned	79	.2	2.5	2.5
	Not mentioned	3027	7.0	97.5	100.0

	Total	3106	7.2	100.0
Missing	Refused	5	.0	
	Don't know	7	.0	
	System	40227	92.8	
	Total	40239	92.8	
Total		43345	100.0	

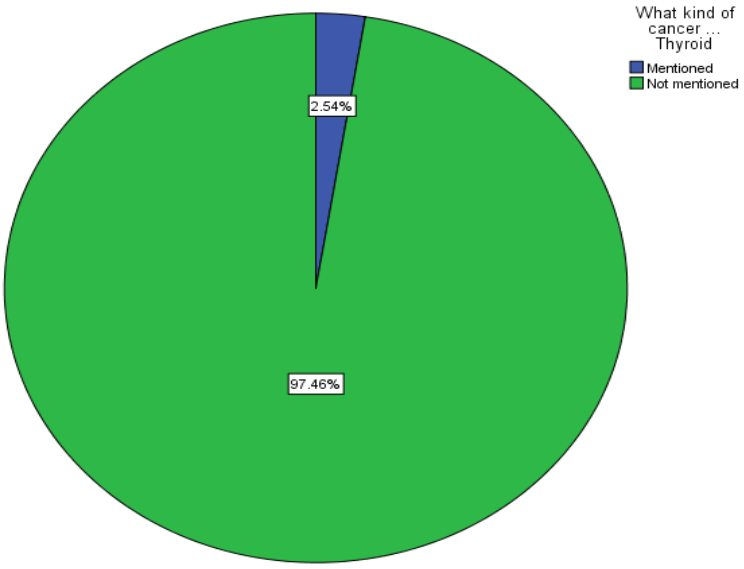


Figure 24. Thyroid Cancer.

Table 10 and Figure 25 below shows the self-reported distributions of mouth/tongue/lip cancer among the selected population. Here, 20 participants (0.6 valid percent) reported or mentioned having mouth/tongue/lip cancer. On the other hand, 3,086 participants (99.4 valid percent) did not report cases of mouth/tongue/lip cancer. A total of 40,239 participants (92.8%) of the total sample size (43,345) was recorded as missing cases for the mouth/tongue/lip cancer outcome.

Table 10

Proportion of Mouth/Tongue/Lip Cancer

What kind of cancer ... mouth/tongue/lip

		Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*
Valid	Mentioned	20	.0	.6	.6
	Not mentioned	3086	7.1	99.4	100.0
	Total	3106	7.2	100.0	
Missing	Refused	5	.0		
	Don't know	7	.0		
	System	40227	92.8		
	Total	40239	92.8		
Total		43345	100.0		

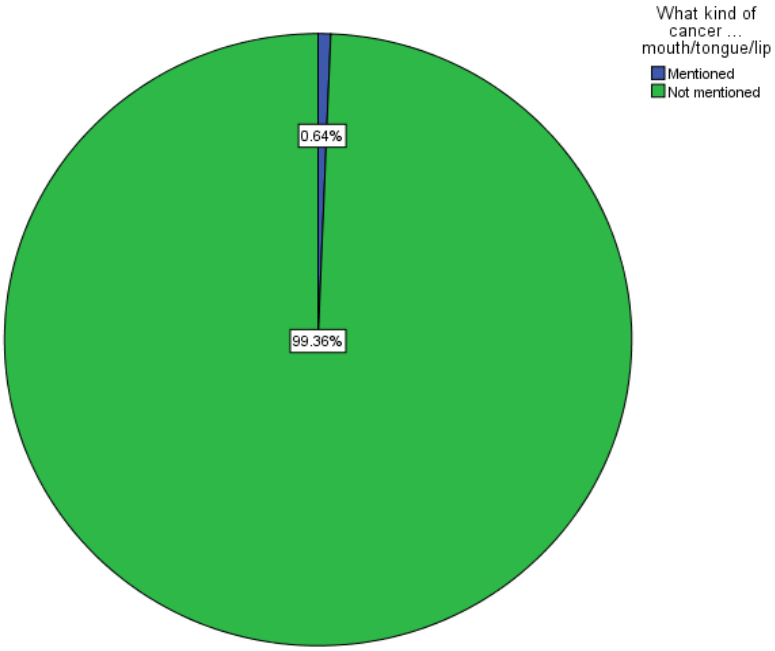


Figure 25. Mouth/Tongue/Lip Cancer.

Figure 26, 27, 28, and Table 11 shows the central tendency, dispersion, and distribution of the age of the selected sample population of when they were first diagnosed with mouth/tongue/lip cancer or thyroid cancer or both. Table 11 also shows

the descriptive statistics of the duration of heart conditions. The reported cases of mouth/tongue/lip cancer, thyroid cancer, and heart conditions (as a number of a unit) are 20, 77, and 732 respectively out of the overall total of 43,345 participants. However, 43,325; 43,268; and 42,613 participants' responses were reported as missing cases respectively. The average age at first diagnosis with mouth/tongue/lip cancer and thyroid cancer were 52.15 and 44.75 years respectively. The duration of the heart disease per number of units is 14.94. The other dispersion and distribution values such as median, mode, standard deviation, etc. are shown in Table 11.

Figure 26. Age First Diagnosed with Mouth/Tongue/Lip Cancer.

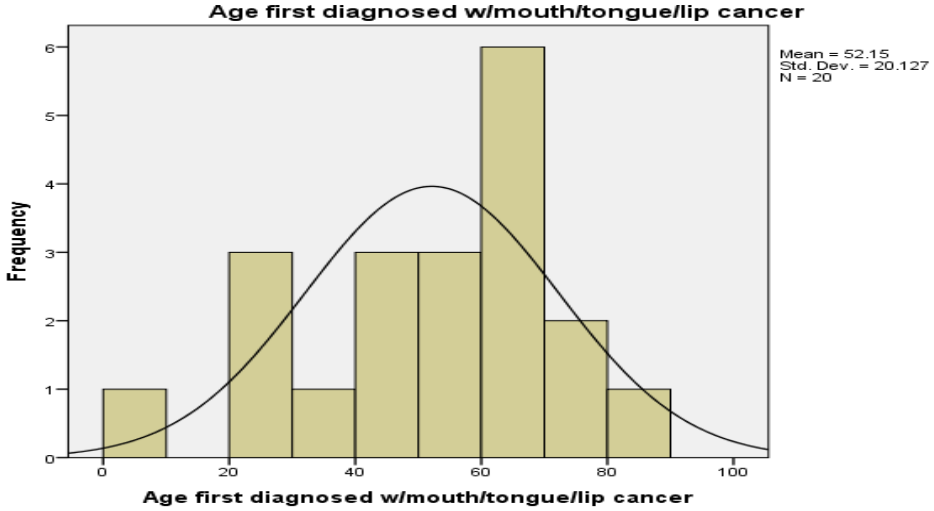


Figure 27. Age First Diagnosed with Thyroid Cancer.

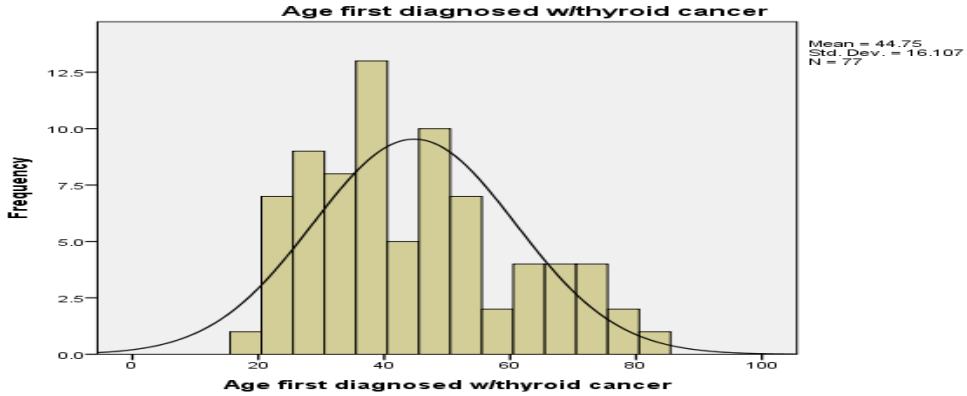


Figure 28. Duration of Heart Problems.

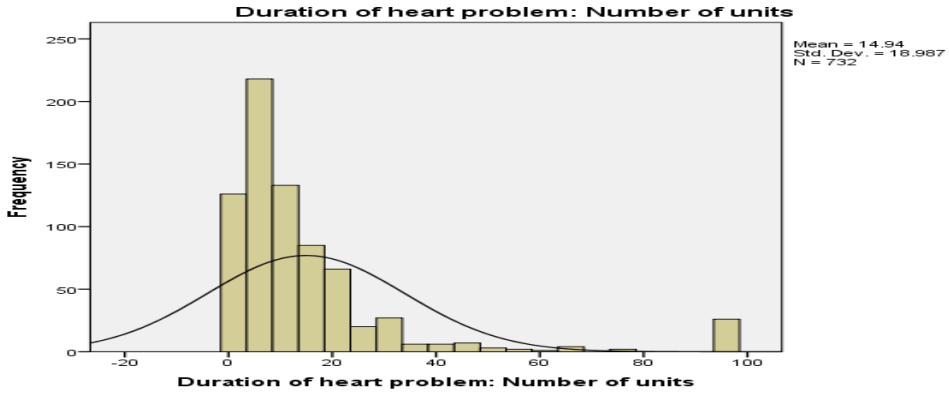


Table 11

Age at First Diagnosis

Statistics

		Age first diagnosed w/mouth/tongue/lip cancer	*Age first diagnosed w/thyroid cancer*	*Duration of heart problem: Number of units*
N	Valid	20	77	732
	Missing	43325	43268	42613
Mean		52.15	44.75	14.94
Median		57.00	42.00	10.00
Mode		49[a]	39	10
Std. Deviation		20.127	16.107	18.987
Variance		405.082	259.451	360.517
Skewness		-.774	.536	3.054
Std. Error of Skewness		.512	.274	.090
Kurtosis		.318	-.511	9.831
Std. Error of Kurtosis		.992	.541	.180
Range		80	67	95
Minimum		4	18	1
Maximum		84	85	96

a. Multiple modes exist. The smallest value is shown

Descriptive Statistics of the Confounders

The descriptive statistics for the selected confounders in this section of the results provided information on the central tendency or dispersion of distribution frequency of the confounders as well. The identified confounder variables are gender, race, age, marital status, and employment status. In Tables 12-19 and Figure 29-35, the descriptive analysis of the participants' gender, race, age, marital status, and employment status were discussed below. The descriptive analysis provided an overview of the dispersion and distribution frequency of the confounder variables.

Confounders (Gender, Race, Age, Marital Status, and Employment Status)

For each of the confounders described, Table 12 shows the total sample size (N), the valid cases or cases with complete self-reported responses, and the total missing values for each listed confounder-gender, race, age, marital status, and employment status. The number of participants who reported their gender, age, race, and marital status was 34,525. However, 25,880 participants reported their current and recent employment status. The missing value for participants who did not report their gender is 8,820. The missing value among individuals who did not report their race, age, and marital status was 8,820 and 17,465 participants for current employment status.

Table 12

Confounders' Distribution

Statistics

		Gender	*Race coded to single/multiple race group - MRACRPI2*	*Race coded to single/multiple race group - MRACBPI2*	*Age*	*Marital Status*	*Ever worked*	*Current/most recent job also longest held job*	*Number of years on the job*	*Age first diagnosed w/mouth/tongue/lip cancer*	*Age first diagnosed w/thyroid cancer*	*Menopausal problems, past 12 months*	*Duration of heart problem: Number of units*
N	Valid	34525	34525	34525	34525	34525	14469	25880	32128	20	77	4136	732
	Missing	8820	8820	8820	8820	8820	28876	17465	11217	43325	43268	39209	42613

Table 13 and Figure 29 shows the distribution and frequency of self-reported information about participants' gender. The total self-reported valid cases for gender were 15,273 (44.2%) for males, and 19,252 (55.8%) for females. The total missing values for gender--both male and female participants who did not report their gender or whose gender was not identified was 8,820.

Table 13

Gender Distribution

		Frequency	*Percent*	*Valid Percent*
Valid	Male	15273	35.2	44.2
	Female	19252	44.4	55.8
	Total	34525	79.7	100.0
Missing	System	8820	20.3	
Total		43345	100.0	

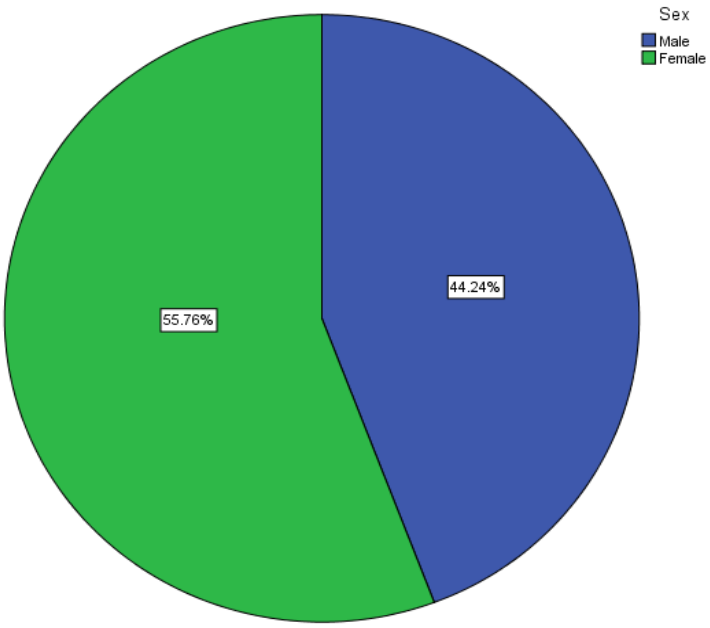


Figure 29. Gender/Sex.

Table 14 and Figure 30 shows the distribution and frequency of participants' race groups. The total valid cases of participants who self-identified as White was 26,214 (75.9%). There were 5,452 (15.8%) Black/African American, 413 (1.2%) Indian (American)/Alaska Native, 408 (1.2%) Asian Indians, 449 (1.3%) Chines, 518 (1.5%) Filipino, 849 (2.5%) other Asian groups, and 120 (0.3%) individuals as a multiple race group. There are 102 participants (0.2%) whose primary race was not releasable. Also, there are 8,820 participants in total who did not report their race.

Table 14

Race Distribution

		Frequency	*Percent*	*Valid Percent*
Valid	White	26214	60.5	75.9

	Black/African American	5452	12.6	15.8
	Indian (American), Alaska Native	413	1.0	1.2
	Asian Indian	408	.9	1.2
	Chinese	449	1.0	1.3
	Filipino	518	1.2	1.5
	Other Asian (See file layout)	849	2.0	2.5
	Primary race not releasable (See file layout)	102	.2	.3
	Multiple race, no primary race selected	120	.3	.3
	Total	34525	79.7	100.0
Missing	System	8820	20.3	
Total		43345	100.0	

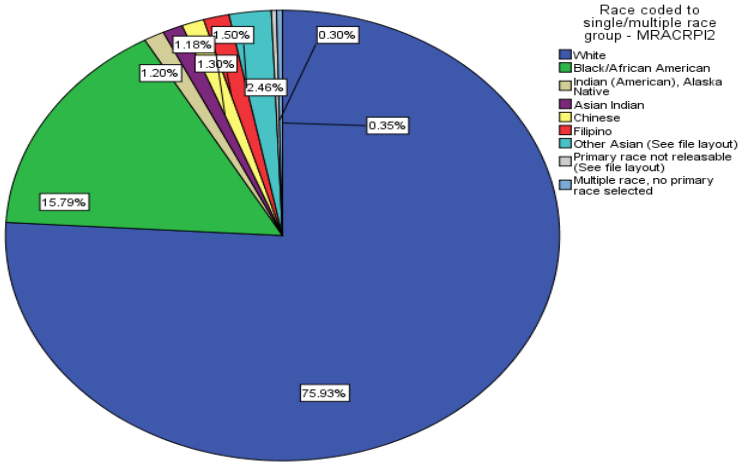


Figure 30. Race Proportion.

Table 15 and Figure 31 shows the central tendency, dispersion, and distribution frequency of the participants' age. The total valid number of participants who reported their age was 34,525 (79.7%), and 8,820 (20.3%) did not report their age. The participants' age had a normally distributed curve around the sample mean value of 48.53. The median age was 48 years old while the mode age (age mostly reported) in the sampling was 85 years old. The estimated participants' age standard deviation and variance estimate were 18.165 and 329.971 respectively. The estimated value for the skewness and Kurtosis values are 0.209 and -0.940 respectively. The skewness and kurtosis values (0.209 and -0.940 respectively) supported the normality distribution pattern of the participants' age. The estimated participants' age range was 67 years. The minimum and maximum age reported in the study were 18 and 85 years old respectively.

Table 15

Age Distribution

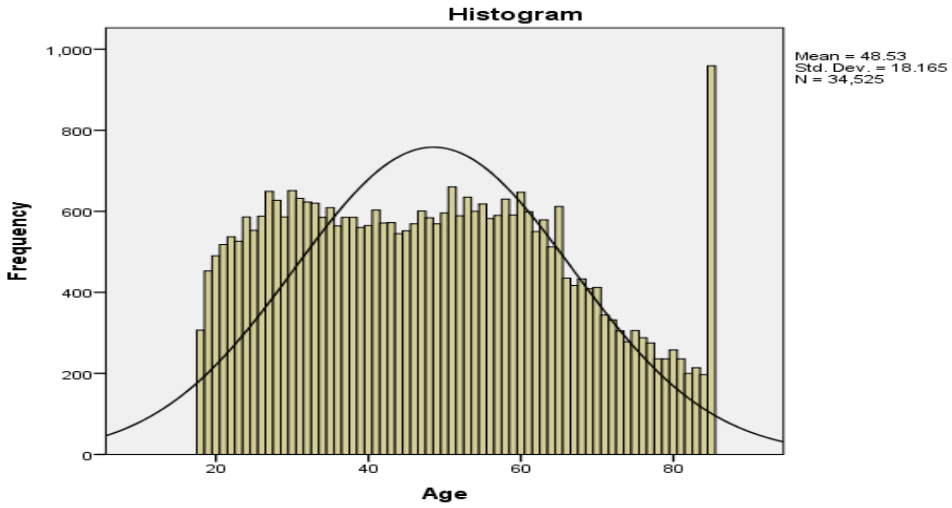
Statistics

Age

N	Valid	34525
	Missing	8820
Mean		48.53
Median		48.00
Mode		85
Std. Deviation		18.165
Variance		329.971
Skewness		.209
Std. Error of Skewness		.013
Kurtosis		-.940
Std. Error of Kurtosis		.026
Range		67
Minimum		18

Maximum	85

Figure 31. Age Distribution.



The marital status distribution in Figure 32, and Table 16 illustrates the participants marital status with the spouse in the households and no spouse in the homes. The marital status distribution also included information on individuals who are widows, divorced, separated, never married, living with the partner, and with unknown marital status. The total valid cases of participants who reported marital status was 34,525 (79.7%). A total of 8,820 (20.3%) did not report marital status. Also, 14,371 participants (41.6%) were identified as married with ‘spouse in household' while 559 participants (1.6%) identified themselves as married with spouse not living in the household (see Figure 32 and Table 16 for more detail). The valid percentage for participants who identified as widowed, divorced, separated, never married, living with a partner, and with unknown marital status were 9.5%, 13.9%, 3.0%, 24.0%, 6.1%, and 0.2% respectively.

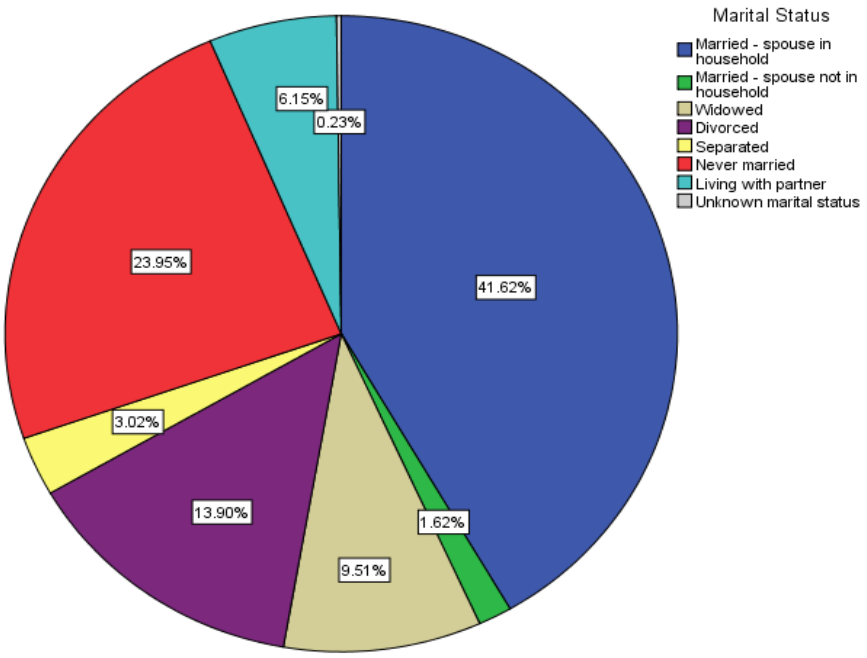


Figure 32. Marital Status Proportion.

Table 16

Marital Status Distribution

		Frequency	*Percent*	*Valid Percent*
Valid	Married - spouse in household	14371	33.2	41.6
	Married - spouse not in household	559	1.3	1.6
	Widowed	3285	7.6	9.5
	Divorced	4798	11.1	13.9
	Separated	1041	2.4	3.0
	Never married	8270	19.1	24.0
	Living with partner	2123	4.9	6.1
	Unknown marital status	78	.2	.2
	Total	34525	79.7	100.0
Missing	System	8820	20.3	
Total		43345	100.0	

The employment status of the participants based on the ‘number of years on the job’ is represented in Table 17 and Figure 33. The total number of participants who reported the number of years on the job is 32,128 (74.1%) cases. About 11,217 individuals (25.9%) did not report their number of years on the job (recorded as missing). Participants’ employment status based on the ‘number of years on the job’ had a normally distributed curve around the sample mean value of 9.80 years. The median ‘number of years on the job’ is 6 years while the estimated mode value is zero (0). The standard deviation and variance for the ‘number of years on the job’ are 10.202 and 104.075 respectively. The skewness and Kurtosis values are 1.078 and 0.064 respectively. Based on the two values (1.078 and 0.064), the distribution curve is normal for the ‘number of years on the job’. The range value based on the ‘number of years on the job’ is 35 years. The minimum and maximum values on the ‘number of the years on the job’ are 0 and 35 years respectively.

Table 17

Employment Status Distribution

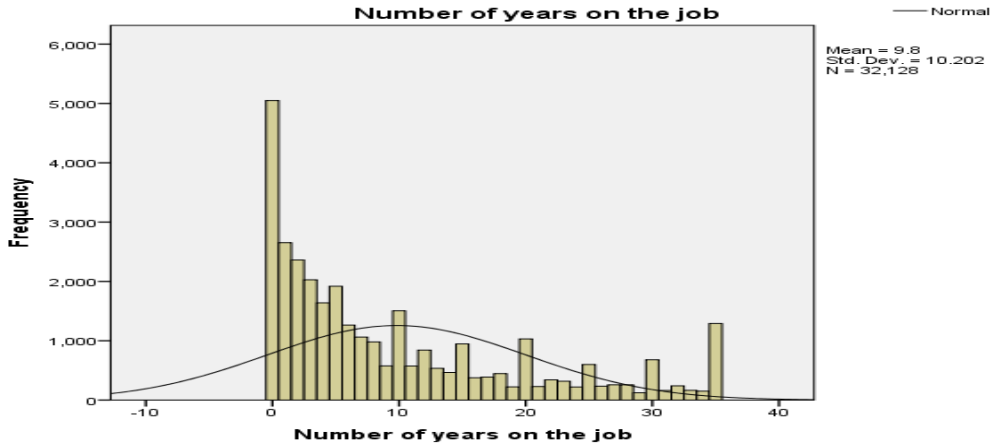
Statistics

Number of years on the job

N	Valid	32128
	Missing	11217
Mean		9.80
Median		6.00
Mode		0
Std. Deviation		10.202
Variance		104.075
Skewness		1.078
Std. Error of Skewness		.014
Kurtosis		.064

Std. Error of Kurtosis	.027
Range	35
Minimum	0
Maximum	35

Figure 33. Distribution of Years of Employment.



The employment status of the participants based on the question specification criterion 'ever worked?' is represented in Table 18 and Figure 34. Out of a total of 14,469 participants who responded to the 'ever worked' question, the total valid number of participants who reported 'yes' on the question is 12,529 (86.6%). Also, 1,940 individuals (13.4%) reported 'no' to the question while 13 participants refused to answer the question, and 5 people selected the 'Don't know' response. In total, 28,858 participants (66.6% of the total participants) identified as the 'system' missing value.

Table 18

Employment Proportion

Ever worked

	Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*

Valid	Yes	12529	28.9	86.6	86.6
	No	1940	4.5	13.4	100.0
	Total	14469	33.4	100.0	
Missing	Refused	13	.0		
	Don't know	5	.0		
	System	28858	66.6		
	Total	28876	66.6		
Total		43345	100.0		

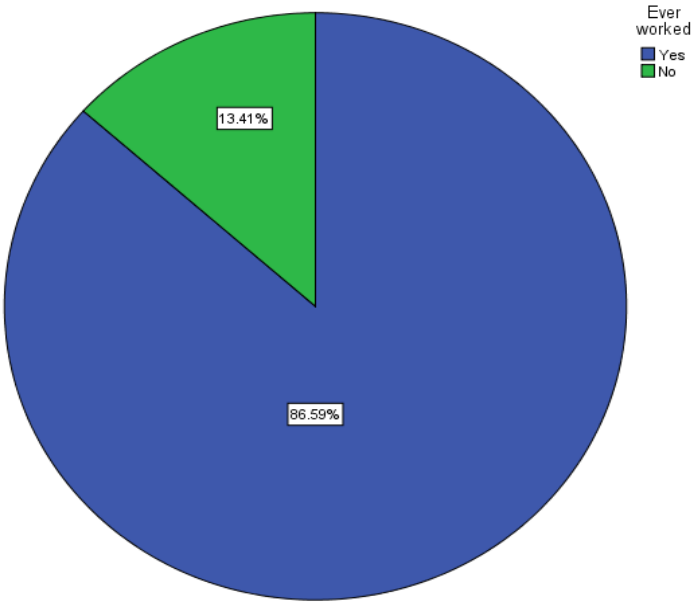


Figure 34. Employment Proportion.

The participants' employment status based on the criteria of the ‘current/most recent job/longer held job’ is represented in Table 19 and Figure 35. Out of a total of 25,880 participants who responded to the employment status question, a total valid count

of 15,452 (59.7%) had a job, while 10,428 individuals (24.1%) did not have a job. About 71 participants refused to answer the question, and 55 participants selected the 'Don't know' response. In total, 17,339 participants (40 % of the total sample size) were the system missing value.

Table 19

Current Employment Status and Years of Employment

Current/most recent job also longest held job

		Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*
Valid	Yes	15452	35.6	59.7	59.7
	No	10428	24.1	40.3	100.0
	Total	25880	59.7	100.0	
Missing	Refused	71	.2		
	Don't know	55	.1		
	System	17339	40.0		
	Total	17465	40.3		
Total		43345	100.0		

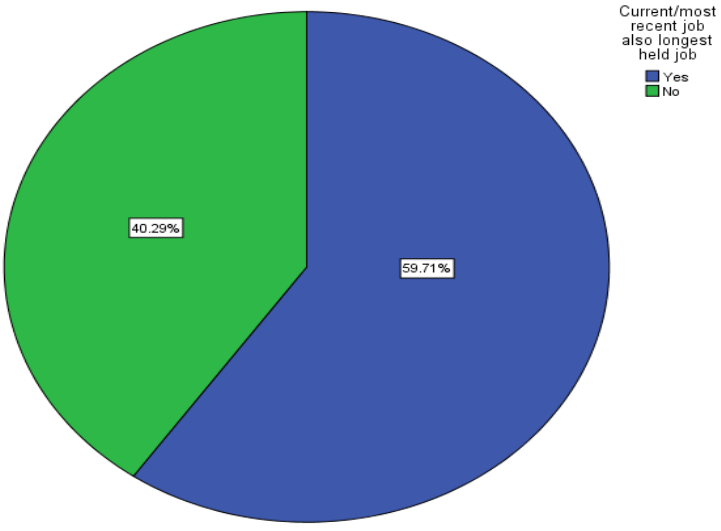


Figure 35. Distribution of Current/Most Recent Job also Longest Held Job

Descriptive Statistics of the Covariate:

This section of the study result focuses on the descriptive analysis of the covariate (menopausal status) (see Figure 36 and Table 29 for more detail). In this study setting, the menopausal status is a covariate. Its attributes in this research setting is a function of the level of the interactive effects on thyroid cancer, mouth/tongue/lip cancer, and heart disease outcomes..

Confounders—Menopausal Status

The descriptive analysis of the menopausal status only applied to women participants selected in this study. Women participants were assessed on whether they had 'menopausal problems in the past twelve months'. Out of 4,136 participants who responded to the question regarding 'menopausal problems in the past 12 months', 1,485 (35.9%) women indicated they had menopausal problems. Whereas, 2,651 individuals (64.1%) had no menopausal problems when posed with the same question. Six participants (< 0.001%) refused to answer the question. One person (< 0.001%) was 'not ascertained' while 10 participants (< 0.001%) selected 'Don't know'. In total, 39,192 participants (90.4%) is the 'system' missing value.

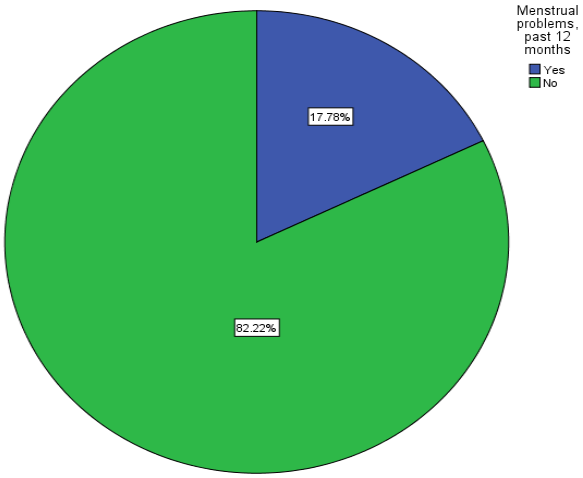


Figure 36. Proportion of Menopausal Problems.

Table 20

Distribution of Menopausal Problem

Menopausal problems, past 12 months

		Frequency	*Percent*	*Valid Percent*	*Cumulative Percent*
Valid	Yes	1485	3.4	35.9	35.9
	No	2651	6.1	64.1	100.0
	Total	4136	9.5	100.0	
Missing	Refused	6	.0		
	Not ascertained	1	.0		
	Don't know	10	.0		
	System	39192	90.4		
	Total	39209	90.5		
Total		43345	100.0		

Inferential Statistics:

In this section of the results, the inferential statistics are presented to address the three research questions and hypotheses. The research questions and hypotheses on

whether there is a difference in the prevalence of thyroid cancer or mouth/tongue/lip cancer or heart disease based on cell phone use behavior were evaluated. The differences evaluated were whether individuals who received all/almost all calls on cell phones (heavy users) or individuals who received some calls on cell phones had a difference in the prevalence rate compared to individuals who received very few calls/no calls on cell phones (rare/no users). The data was analyzed and assessed on whether there is a statistically significant difference between the exposed group and non-exposed group (heavy users vs. rare/non-users respectively). The effect size (odds ratio) was estimated to show the level of the magnitude of the effects if any. The confounders and covariates identified in the study were accounted for in the statistical model to help minimize the chances of distortion of the true attributable effect of the primary predictor variable (cell phone use or exposure). The following are the formulation of the prevalence calculation and inferential analyses for each of the research questions.

Prevalence Formula

Prevalence = Number of cases/Total population size.

Note:

1. *Prevalence* estimation could be measured in a closed cohort or in an open or general population.
2. *Prevalence* could be estimated in a cross-sectional design set up
3. For the *'prevalence'* estimation, the 'old' and "new" cases are summed up in the numerator.

4. *Prevalence* could be measured as a point prevalence (estimated at a particular point in time) or period prevalence (estimated over a period).

In this study, the prevalence measure is a closed system or cohort, and the prevalence estimation is a 'point prevalence'. It requires only the assessment of the prevalence of thyroid cancer, moth/tongue/lip cancer, and heart disease outcome among the target population in the US who participated in the 2012 NHIS study (point in time).

Research Question #1— The Assessment of the Difference in the Prevalence of Thyroid Cancer between the Exposed and Non-exposed.

With research question #1, I evaluated the difference in the prevalence of thyroid cancer between individuals who received all or almost all calls on cell phones, and participants who received very few or no calls on cell phones. Table 21 is the contingency table, which represents the participants' cell phone use behavior. For those that received calls on cell phones often (All the time/almost all the time), 843 participants were told by a doctor that they had cancer, and 3,802 subjects did not have any cancer. With individuals who 'sometimes received calls on cell phones', 712 participants reported to have been told by a doctor that they had cancer and 6,025 individuals have never been told by a doctor they had any cancer. In contrast, for the group that received very few calls on cell phones 'rarely' or those that do not' receive calls on cell phones, 385 participants have been told by a doctor that they had cancer. Also, for rare or no cell phone users, 4,684 participants reported that they have never been told by a doctor they had any cancer..

Table 21

All Cancer Outcome Distribution

Contingency Table for Hosmer and Lemeshow Test

		Ever told by a doctor you had cancer = Yes		*Ever told by a doctor you had cancer = No*		
		Observed	*Expected*	*Observed*	*Expected*	*Total*
Step 1	All/almost all calls on cell phones	843	843	3802	3802	4645
	Some calls on cell phone	712	712	6025	6025	6737
	Very few/no calls on cell phone	385	385	4684	4684	5069

In Table 22, individuals with a 'very few/no calls' received on cell phones was the reference group when comparing the effects of the number of calls received to all cancer outcomes. Individuals or group who received 'all/almost all calls' via the cell phones (phoneuse 1 (PU1)) had a statistical significance difference for all cancer outcomes when compared to individuals or group that received 'very few or no calls' on cell phones or 'rare/no cell phone' users (phoneuse (PU group)). The 'PU1' group significantly predicted all cancer outcomes, $\beta = 0.992$, $W(1,2) = 231.155$, $OR = 2.698$, $p < .001$, 95% CI [2.374, 3.066]. Similarly, the group that received calls 'sometimes' on cell phones (phoneuse 2 (PU2)) as shown in Table 22 had a statistical significance difference for all cancer outcomes when compared to the group that received very few or no calls on cell phones. The 'PU2' group also significantly predicted all cancer outcomes, $\beta = 0.629$, $W(1,2) = 131.135$, $OR = 1.876$, $p < .001$, 95% CI [1.685, 2.090]. The odds ratio estimate for the 'PU1' group was 2.698, meaning that individuals who

received all or almost all calls on cell phones were 2.7 times more likely to develop cancer outcomes than those who received very few or no calls on cell phones. Similarly, the odds ratio estimate for the 'PU2' was 1.876, indicating that individuals who received some calls on cell phones were 1.9 times more likely to develop a cancer outcome than those who received very few or no calls on cell phones.

Table 22

All Cancer Outcome Analysis

Variables in the Equation

								95% C.I.for EXP(B)	
		B	*S.E.*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower*	*Upper*
Step 1[a]	Very Few Calls (PU)			264.829	2	.000			
	All Calls (PU1)	.992	.065	231.155	1	.000	2.698	2.374	3.066
	Some Calls (PU2)	.629	.055	131.135	1	.000	1.876	1.685	2.090
	Constant	1.506	.038	1565.618	1	.000	4.510		

a. Variable(s) entered on step 1: Very few Calls/no calls (PHONEUSE).

Table 23 is the description of the inferential analysis which included the independent variable (IV), confounders (gender, race, age, marital status, and employment status) and covariates (menopausal problem and smoking status) evaluated in this study. As shown in Table 22, the PU1 and PU2 alone significantly predicted all cancer outcomes. However, when gender, race, age, marital status, employment status, menopausal problem, and smoking status were added in the statistical analysis, none of the phoneuse group (PU1 or PU2) significantly predicted all cancer outcomes, β = *-1.075, W(1,2) = 2.224, OR = 0.341, p = 0.136, 95% CI [0.083, 1.402]* and β = *-0.305,*

W(1,2) = 0.175, OR = 0.737, p = 0.676, 95% CI [0.177, 3.074] respectively. Also, the odds ratio estimate for participants in the 'PU1 group was 0.341, indicating that individuals who received all calls or almost all calls on cell phones have 0.341 likelihood of developing cancer compared to those who received very few or no calls on cell phones after accounting for gender, race, age, marital status, employment status, menopausal problem, and smoking status. Similarly, the odds ratio estimate for participants in the 'phoneuse2 group was 0.737, suggesting that individuals who received some calls on cell phones have 0.737 likelihood of developing cancer when compared to those who received very few or no calls on cell phones after accounting for gender, race, age, marital status, employment status, menopausal problem, and smoking status. None of the covariates shown in Table 23 produced a statistically significant p-value when all the confounding variables were added in the regression model..

Table 23

All Cancer Outcome Analysis including Confounders/Covariates

Variables in the Equation

							95% C.I.for EXP(B)	
	B	*S.E.*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower*	*Upper*
Step 1[a] Very Few Calls (PU)			3.405	2	.182			
All Calls (PU1)	-1.075	.721	2.224	1	.136	.341	.083	1.402
Some Calls (PU2)	-.305	.729	.175	1	.676	.737	.177	3.074
Race	.451	.552	.668	1	.414	1.570	.532	4.630
Age	-.116	.072	2.604	1	.107	.891	.774	1.025
Marital status	-.052	.095	.296	1	.587	.950	.788	1.144
Job status	.321	.494	.423	1	.515	1.379	.524	3.632
Menopausal	-.068	.480	.020	1	.887	.934	.365	2.391
Smoke status	-.516	.330	2.443	1	.118	.597	.312	1.140

	Constant	9.510	4.654	4.175	1	.041	13496.814

a. Variable(s) entered on step 1: PHONEUSE, MRACRPI2, AGE_P, R_MARITL, BUSINC1A, MENOYR, SMKSTAT2.

Table 24 shows the correlational matrix of cell phone use (PU1 and PU2), confounders, and covariates. Race correlated positively to PU1, PU2, age, current job status, and menopausal problems. Smoke and marital statuses correlated negatively to race. Marital status correlated positively to PU1, PU2, age, menopausal problem, and smoke status, but negatively to current job status. Current job status showed a positive correlation to PU1, PU2, race, age, and smoke status, but negatively correlated to marital status and menopausal problems. The menopausal problem was positively correlated with race, age, and marital status, but negatively correlated to PU1, PU2, current job status, and smoke status. Smoke status was positively correlated to PU1, PU2, age, marital status, and current job status, but negatively correlated to race and menopausal problems (see Table 24 for more detail)..

Table 24

Correlation Matrix of All Cancer Outcomes

Correlation Matrix

		Constant	*Phoneuse(1)*	*Phoneuse(2)*	*Race*	*Age*	*Marital status*	*Current Job status*	*Menopausal problems*	*Smoke status*
Step 1	Constant	1.000	-.386	-.254	-.240	-.910	-.224	-.275	-.215	-.364
	Phoneuse(1)	-.386	1.000	.757	.050	.279	.038	.143	-.016	.039
	Phoneuse(2)	-.254	.757	1.000	.059	.144	.059	.066	-.009	.007
	Race	-.240	.050	.059	1.000	.113	-.050	.065	.080	-.039
	Age	-.910	.279	.144	.113	1.000	.137	.073	.068	.116
	Marital Status	-.224	.038	.059	-.050	.137	1.000	-.016	.014	.232

	Current Job status	-.275	.143	.066	.065	.073	-.016	1.000	-.032	.079
	Menopausal problems	-.215	-.016	-.009	.080	.068	.014	-.032	1.000	-.058
	Smoke status	-.364	.039	.007	-.039	.116	.232	.079	-.058	1.000

Thyroid Cancer Inferential Assessment

Table 25 and 26 shows the proportion of individuals who reported being told by a doctor that they have thyroids cancer or those that have not been told they have thyroid cancer. The proportion estimate was tabulated based on three levels of cell phone use behaviors (all or almost all calls received on cell phones; some received on cell phones, and very few or no cell phone). The total number of individuals who either mentioned they had thyroid cancer or those that did not mention thyroid cancer outcome and who either used cell phones often or sometimes or rarely is about 1,936 participants. In total, 50 participants mentioned they had been told by a doctor they had thyroid cancer, and 1,886 individuals reported they had not been told by a doctor they had thyroid cancer, see Table 26.

Partitioning these individuals to the level of cell phone use as shown in Table 25 and 26, about 11 (2.9%) individuals who received all or almost all calls on cell phones mentioned they had thyroid cancer. While 373 (97.1%) participants who received all or almost all calls on cell phones did not mention they had thyroid cancer. For those who received some calls on cell phones, 22 (3.1%) individuals mentioned they had thyroid cancer while 689 (96.9%) participants did not mention they had thyroid cancer. With participants who received very few calls or no calls on cell phones, 17 (2.0%) people

mentioned they had thyroid cancer while 824 (98.0%) participants did not mention they had thyroid cancer. Table 27 shows there is no statistical significant difference for any of the correlational approaches used for the Table 25 and 26 comparisons.

Table 25

Thyroid Cancer Analysis

Case Processing Summary

		N	*Marginal Percentage*
What kind of cancer ... Thyroid	Mentioned	50	2.6%
	Not mentioned	1886	97.4%
Received calls cell/landline/both	All or almost all calls received on cell phones	384	19.8%
	Some received on cell phones and some on regular phones	711	36.7%
	Very few or none on cell phones	841	43.4%
Valid		1936	100.0%
Missing		41409	
Total		43345	
Subpopulation		3	

Prevalence = Number of cases/Total population size:

Using Table 26, the prevalence of thyroid cancer per 1000 participants for PU1, PU2, and PU3 groups are as follows:

PU1-Thyroid Cancer Prevalence = (11/1936) *1000 = 5.68 per 1000

PU2-Thyroid Cancer Prevalence = (22/1936) *1000 = 11.36 per 1000

PU3-Thyroid Cancer Prevalence = (17/1936) *1000 = 8.78 per 1000

The total prevalence of thyroid cancer for the studied population among those with complete information/response is as follows:

PU1-3: Thyroid Cancer Prevalence = (50/1936) *1000 = 25.83 per 1000

The Thyroid Cancer Prevalence for PU1 and 2 is as follows:

PU1 and 2-Thyroid Cancer Prevalence = (11+22/1936) *1000 = 17.05 per 1000

From the prevalence estimation, the prevalence of thyroid cancer among individuals who received all calls or almost all calls on cell phones (PU1 group) is the lowest (5.68 per 1000) among the exposed group. The prevalence of thyroid cancer among individuals who received some calls on cell phones (PU2 group) is the highest (11.36 per 1000) among the exposed group. For the control group (PU3 group), the prevalence of thyroid cancer among individuals who received very few calls or no calls on cell phones (PU3 group) is the second lowest (8.78 per 1000).

Table 26

Level of Cell Phones Calls Received and Thyroid Cancer Outcome

*Received calls cell/landline/both * What kind of cancer ... Thyroid Crosstabulation*

			What kind of cancer ... Thyroid		
			Mentioned	*Not mentioned*	*Total*
Received calls cell/landline/both	All or almost all calls received on cell phones (phoneuse1)	Count	11	373	384
		% within Received calls cell/landline/both	2.9%	97.1%	100.0%
	Some received on cell phones and some on regular phones (phoneuse 2)	Count	22	689	711
		% within Received calls cell/landline/both	3.1%	96.9%	100.0%
		Count	17	824	841

	Very few or none on cell phones (phoneuse3)	% within Received calls cell/landline/both	2.0%	98.0%	100.0%
Total		Count	50	1886	1936
		% within Received calls cell/landline/both	2.6%	97.4%	100.0%

The cell phone use or levels of cell phone exposure's correlation matrix link to thyroid cancer outcomes in Table 27 presented the correlational information about the association between cell phone use and thyroid cancer. The correlational value of PU1 (All/almost all calls received on cell phones) and PU2 (Some calls received on cell phones) regarding thyroid cancer outcomes is 0.468. Table 28 shows the model used in this statistical assessment was a fitted model for the analysis. Hence, $p = 0.377$ was not statistically significant.

Table 27

The Correlation Matrix of Thyroid Cancer vs. Cell Phone use

Asymptotic Correlation Matrix

			What kind of cancer ... Thyroid Mentioned		
What kind of cancer ... Thyroid[a]		*Intercept*	*[PU1]*	*[PU2]*	*[PU3]*
Mentioned	Intercept	1	-.625	-.749	.[b]
	[PU1]	-.625	1	.468	.[b]
	[PU2]	-.749	.468	1	.[b]
	[PU3]	.[b]	.[b]	.[b]	.[b]

a. The reference category is: Not mentioned.

b. One or both parameter estimates are redundant.

Table 28

Thyroid Cancer Regression Model

Model Fitting Information

Model	*Model Fitting Criteria* *-2 Log Likelihood*	*Likelihood Ratio Tests* *Chi-Square*	*df*	*Sig.*
Intercept Only	15.737			
Final	13.787	1.950	2	.377

The parameter estimate table below shows the statistical relationship between PU1 and thyroid cancer outcomes. The relationship between PU2 and thyroid cancer outcome was shown in Table 29 as well. Based on the analysis, there is no statistically significant relationship between PU1 and thyroid cancer outcome or PU2 and thyroid cancer outcome. The reference variable used in this analysis was PU3, which represented individuals who received very few calls/no calls on cell phones. For this analysis, PU1 and PU2 did not significantly predict thyroid cancer outcomes, $\beta = 0.357$, *W(1) = 0.831, OR = 1.429, p = 0.362, 95% CI [0.663, 3.082] and* $\beta = 0.427$, *W(1) = 1.784, OR = 1.548, p = 0.182, 95% CI [0.815, 2.938]*. Therefore, the null hypothesis for the research question #1 will not be rejected. See Table 29 for more detail.

Table 29

Thyroid Cancer Parameter Estimates Without Confounders/Covariates

Parameter Estimates

What kind of cancer ... Thyroid[a]	*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B)* *Lower Bound*	*Upper Bound*

Mentioned	Intercept	-3.881	.245	250.875	1	.000			
	[PHONEUSE=1]	.357	.392	.831	1	.362	1.429	.663	3.082
	[PHONEUSE=2]	.437	.327	1.784	1	.182	1.548	.815	2.938
	[PHONEUSE=3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Confounder and Covariate Inferential Analysis for Thyroid Cancer

Based on the statistical analysis shown in Table 30, age is a strong predictor of thyroid cancer. The Wald estimation with *W= 27.8*, *OR = 0.945*, and *p < 0.001* supported the age-thyroid cancer relationship.

Table 30

Thyroid Cancer Parameter Estimates with all Confounders and Covariates

Parameter Estimates

								95% Confidence Interval for Exp(B)	
What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower Bound*	*Upper Bound*
Mentioned	Intercept	-.114	.710	.026	1	.873			
	AGE_P	-.056	.011	27.792	1	.000	.945	.926	.965
	[PHONEUSE=1]	-.430	.424	1.028	1	.311	.651	.283	1.494
	[PHONEUSE=2]	.093	.335	.077	1	.781	1.098	.569	2.117
	[PHONEUSE=3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Based on the statistical analysis shown in Table 31, gender is a strong predictor of thyroid cancer. As well, the Wald estimation with *OR* value of *3.637* and *p = 0.001*, supported the gender-thyroid relationship observed.

Table 31

Thyroid Cancer Parameter Estimates with Gender Alone

Parameter Estimates

What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
Mentioned	Intercept	-6.154	.764	64.794	1	.000			
	GENDER	1.291	.389	11.005	1	.001	3.637	1.696	7.800
	[PHONEUSE=1]	.398	.393	1.027	1	.311	1.490	.689	3.220
	[PHONEUSE=2]	.511	.328	2.422	1	.120	1.667	.876	3.174
	[PHONEUSE=3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Table 32 represents the distribution and proportion of gender based on calls received on cell phones and the corresponding responses to the question on thyroid cancer status.

Table 32

Thyroid Cancer Distribution Categorized by Gender

Observed and Predicted Frequencies

Gender	Received calls cell/landline/both	What kind of cancer ... Thyroid	*Frequency*		*Pearson Residual*	*Percentage*	
			Observed	*Predicted*		*Observed*	*Predicted*

Male	All or almost all calls received on cell phones	Mentioned	1	1.753	-.572	0.6%	1.1%
		Not mentioned	153	152.247	.572	99.4%	98.9%
	Some received on cell phones and some on regular phones	Mentioned	3	3.907	-.462	1.0%	1.3%
		Not mentioned	304	303.093	.462	99.0%	98.7%
	Very few or none on cell phones	Mentioned	4	2.340	1.089	1.3%	0.8%
		Not mentioned	301	302.660	-1.089	98.7%	99.2%
Female	All or almost all calls received on cell phones	Mentioned	10	9.247	.253	4.3%	4.0%
		Not mentioned	220	220.753	-.253	95.7%	96.0%
	Some received on cell phones and some on regular phones	Mentioned	19	18.093	.218	4.7%	4.5%
		Not mentioned	385	385.907	-.218	95.3%	95.5%
	Very few or none on cell phones	Mentioned	13	14.660	-.440	2.4%	2.7%
		Not mentioned	523	521.340	.440	97.6%	97.3%

The percentages are based on total observed frequencies in each subpopulation.

Table 33 represents the thyroid cancer parameter estimates based on marital status. The statistical analysis in Table 33 shows that marital status is not a predictor of thyroid cancer outcome. An estimated Wald value of *0.122, OR = 1.023,* and $p = 0.727$ was not statistically significant and supports these findings.

Table 33

Thyroid Cancer Parameter Estimates with Marital Status

Parameter Estimates

What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
Mentioned	Intercept	-3.947	.312	160.513	1	.000			
	R_MARITL	.022	.064	.122	1	.727	1.023	.902	1.159
	[PHONEUSE=1]	.352	.392	.806	1	.369	1.422	.659	3.068
	[PHONEUSE=2]	.444	.328	1.839	1	.175	1.560	.820	2.965
	[PHONEUSE=3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Table 34 represents the distribution and proportion of all the categories of marital status defined or specified in this study. The observed thyroid cancer outcomes among the participants are stratified based on marital status and call received on cell phones.

Table 34

Marital Status and Thyroid Cancer Proportion

Observed and Predicted Frequencies

Marital Status	Received calls cell/landline/both	What kind of cancer ... Thyroid	*Frequency* *Observed*	*Predicted*	*Pearson Residual*	*Percentage* *Observed*	*Predicted*
Married - spouse in household	All or almost all calls received on cell phones	Mentioned	4	5.381	-.604	2.0%	2.7%
		Not mentioned	193	191.619	.604	98.0%	97.3%
		Mentioned	17	13.086	1.099	3.9%	3.0%

	Some received on cell phones and some on regular phones	Not mentioned	421	424.914	-1.099	96.1%	97.0%
	Very few or none on cell phones	Mentioned	7	8.036	-.369	1.7%	1.9%
		Not mentioned	408	406.964	.369	98.3%	98.1%
Married - spouse not in household	All or almost all calls received on cell phones	Mentioned	0	.140	-.379	0.0%	2.8%
		Not mentioned	5	4.860	.379	100.0%	97.2%
	Some received on cell phones and some on regular phones	Mentioned	0	.397	-.640	0.0%	3.1%
		Not mentioned	13	12.603	.640	100.0%	96.9%
	Very few or none on cell phones	Mentioned	0	.119	-.348	0.0%	2.0%
		Not mentioned	6	5.881	.348	100.0%	98.0%
Widowed	All or almost all calls received on cell phones	Mentioned	2	1.458	.456	4.0%	2.9%
		Not mentioned	48	48.542	-.456	96.0%	97.1%
	Some received on cell phones and some on regular phones	Mentioned	0	3.315	-1.851	0.0%	3.2%
		Not mentioned	104	100.685	1.851	100.0%	96.8%
	Very few or none on cell phones	Mentioned	3	4.921	-.875	1.3%	2.1%
		Not mentioned	235	233.079	.875	98.7%	97.9%
Divorced		Mentioned	3	2.055	.669	4.3%	3.0%

	All or almost all calls received on cell phones	Not mentioned	66	66.945	-.669	95.7%	97.0%
	Some received on cell phones and some on regular phones	Mentioned	3	2.606	.248	3.8%	3.3%
		Not mentioned	77	77.394	-.248	96.3%	96.7%
	Very few or none on cell phones	Mentioned	5	2.092	2.032	5.1%	2.1%
		Not mentioned	94	96.908	-2.032	94.9%	97.9%
Separated	All or almost all calls received on cell phones	Mentioned	0	.335	-.588	0.0%	3.0%
		Not mentioned	11	10.665	.588	100.0%	97.0%
	Some received on cell phones and some on regular phones	Mentioned	0	.233	-.491	0.0%	3.3%
		Not mentioned	7	6.767	.491	100.0%	96.7%
	Very few or none on cell phones	Mentioned	1	.302	1.283	7.1%	2.2%
		Not mentioned	13	13.698	-1.283	92.9%	97.8%
Never married	All or almost all calls received on cell phones	Mentioned	2	.995	1.023	6.3%	3.1%
		Not mentioned	30	31.005	-1.023	93.8%	96.9%
	Some received on cell phones and some on regular phones	Mentioned	1	1.564	-.459	2.2%	3.4%
		Not mentioned	45	44.436	.459	97.8%	96.6%
		Mentioned	0	1.214	-1.114	0.0%	2.2%

	Very few or none on cell phones	Not mentioned	55	53.786	1.114	100.0%	97.8%
Living with partner	All or almost all calls received on cell phones	Mentioned	0	.636	-.810	0.0%	3.2%
		Not mentioned	20	19.364	.810	100.0%	96.8%
	Some received on cell phones and some on regular phones	Mentioned	1	.799	.229	4.3%	3.5%
		Not mentioned	22	22.201	-.229	95.7%	96.5%
	Very few or none on cell phones	Mentioned	1	.316	1.231	7.1%	2.3%
		Not mentioned	13	13.684	-1.231	92.9%	97.7%

The percentages are based on total observed frequencies in each subpopulation.

The statistical analysis shown in Table 35 represents the parameter estimates of thyroid cancer outcomes among the participants based on the levels of cell phone use. For this analysis, the data were stratified by the race groups. Based on the statistical estimate, race was not a predictor of thyroid cancer outcomes. The estimated Wald value for this analysis is *W=1.034, OR=1.053*, and *p=0.309*, which is not statistically significant.

Table 35

Phoneuse, Thyroid Cancer and Marital Status Parameter Estimates

Parameter Estimates

							95% Confidence Interval for Exp(B)	
What kind of cancer ... Thyroid[a]	*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower Bound*	*Upper Bound*

Mentioned	Intercept	-3.957	.258	235.231	1	.000			
	MRACRPI2	.052	.051	1.034	1	.309	1.053	.953	1.165
	[PHONEUSE=1]	.338	.393	.738	1	.390	1.401	.649	3.027
	[PHONEUSE=2]	.429	.327	1.720	1	.190	1.536	.809	2.917
	[PHONEUSE=3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Table 36 shows the distribution and proportion of all the categories of the racial groups of individuals represented in the study. The observed thyroid cancer outcomes among the participants were stratified by racial groups and calls received on cell phones.

Table 36

Proportion of Thyroid Cancer with Cell Phone use and Race

Observed and Predicted Frequencies

Race coded to single/multiple race group - MRACRPI2	Received calls cell/landline/both	What kind of cancer ... Thyroid	*Frequency*			*Percentage*	
			Observed	*Predicted*	*Pearson Residual*	*Observed*	*Predicted*
White	All or almost all calls received on cell phones	Mentioned	9	8.895	.036	2.8%	2.7%
		Not mentioned	315	315.105	-.036	97.2%	97.3%
	Some received on cell phones and some on regular phones	Mentioned	20	18.937	.248	3.2%	3.0%
		Not mentioned	611	612.063	-.248	96.8%	97.0%
	Very few or none on cell phones	Mentioned	17	14.809	.575	2.3%	2.0%
		Not mentioned	733	735.191	-.575	97.7%	98.0%
		Mentioned	0	1.242	-1.131	0.0%	2.9%

Black/African American	All or almost all calls received on cell phones	Not mentioned	43	41.758	1.131	100.0%	97.1%
	Some received on cell phones and some on regular phones	Mentioned	1	1.705	-.548	1.9%	3.2%
		Not mentioned	53	52.295	.548	98.1%	96.8%
	Very few or none on cell phones	Mentioned	0	1.371	-1.183	0.0%	2.1%
		Not mentioned	66	64.629	1.183	100.0%	97.9%
Indian (American), Alaska Native	All or almost all calls received on cell phones	Mentioned	0	.030	-.177	0.0%	3.0%
		Not mentioned	1	.970	.177	100.0%	97.0%
	Some received on cell phones and some on regular phones	Mentioned	0	.033	-.185	0.0%	3.3%
		Not mentioned	1	.967	.185	100.0%	96.7%
	Very few or none on cell phones	Mentioned	0	.087	-.299	0.0%	2.2%
		Not mentioned	4	3.913	.299	100.0%	97.8%
Asian Indian	Some received on cell phones and some on regular phones	Mentioned	0	.135	-.375	0.0%	4.5%
		Not mentioned	3	2.865	.375	100.0%	95.5%
	Very few or none on cell phones	Mentioned	0	.089	-.303	0.0%	3.0%
		Not mentioned	3	2.911	.303	100.0%	97.0%
Chinese		Mentioned	0	.086	-.300	0.0%	4.3%

	All or almost all calls received on cell phones	Not mentioned	2	1.914	.300	100.0%	95.7%
	Some received on cell phones and some on regular phones	Mentioned	0	.188	-.445	0.0%	4.7%
		Not mentioned	4	3.812	.445	100.0%	95.3%
	Very few or none on cell phones	Mentioned	0	.125	-.359	0.0%	3.1%
		Not mentioned	4	3.875	.359	100.0%	96.9%
Filipino	All or almost all calls received on cell phones	Mentioned	0	.182	-.436	0.0%	4.5%
		Not mentioned	4	3.818	.436	100.0%	95.5%
	Some received on cell phones and some on regular phones	Mentioned	1	.396	.984	12.5%	5.0%
		Not mentioned	7	7.604	-.984	87.5%	95.0%
	Very few or none on cell phones	Mentioned	0	.230	-.487	0.0%	3.3%
		Not mentioned	7	6.770	.487	100.0%	96.7%
Other Asian (See file layout)	All or almost all calls received on cell phones	Mentioned	2	.443	2.409	25.0%	5.5%
		Not mentioned	6	7.557	-2.409	75.0%	94.5%
	Some received on cell phones and some on regular phones	Mentioned	0	.543	-.760	0.0%	6.0%
		Not mentioned	9	8.457	.760	100.0%	94.0%
		Mentioned	0	.201	-.457	0.0%	4.0%

	Very few or none on cell phones	Not mentioned	5	4.799	.457	100.0%	96.0%
Primary race not releasable (See file layout)	Some received on cell phones and some on regular phones	Mentioned	0	.063	-.260	0.0%	6.3%
		Not mentioned	1	.937	.260	100.0%	93.7%
Multiple race, no primary race selected	All or almost all calls received on cell phones	Mentioned	0	.122	-.361	0.0%	6.1%
		Not mentioned	2	1.878	.361	100.0%	93.9%
	Very few or none on cell phones	Mentioned	0	.089	-.305	0.0%	4.4%
		Not mentioned	2	1.911	.305	100.0%	95.6%

The percentages are based on total observed frequencies in each subpopulation.

Shown in Table 37 is the parameter estimates of thyroid cancer and levels of calls received on cell phones. The thyroid cancer risk estimate given the exposure to cell phone use was stratified by current job status. It shows that current job status is not a predictor of thyroid cancer outcomes, with a Wald value of 0.000, OR = 1.007, and p = 0.994. Therefore, the analysis was not statistically significant.

Table 37

Parameter Estimates of Thyroid Cancer Categorized by Phoneuse and Employment

Parameter Estimates

							95% Confidence Interval for Exp(B)	
What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower Bound*	*Upper Bound*
Mentioned	Intercept	-3.700	1.628	5.169	1	.023			

	BUSINC1A	.007	.881	.000	1	.994	1.007	.179	5.656
	[PHONEUSE=1]	-.118	1.239	.009	1	.924	.889	.078	10.078
	[PHONEUSE=2]	.553	.928	.356	1	.551	1.739	.282	10.712
	[PHONEUSE=3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Table 38 shows the distribution and proportion of all the current job status of individuals represented in this study. The observed thyroid cancer outcomes among the participants were stratified by current job status and call received on cell phones.

Table 38

Distribution of Thyroid Cancer Categorized by Cell Phone use and Current Employment

Observed and Predicted Frequencies

Current job an incorporated business	Received calls cell/landline/both	What kind of cancer ... Thyroid	*Frequency*			*Percentage*	
			Observed	*Predicted*	*Pearson Residual*	*Observed*	*Predicted*
Yes	All or almost all calls received on cell phones	Mentioned	1	.325	1.199	6.7%	2.2%
		Not mentioned	14	14.675	-1.199	93.3%	97.8%
	Some received on cell phones and some on regular phones	Mentioned	1	.996	.005	4.2%	4.1%
		Not mentioned	23	23.004	-.005	95.8%	95.9%
	Very few or none on cell phones	Mentioned	0	.680	-.835	0.0%	2.4%
		Not mentioned	28	27.320	.835	100.0%	97.6%
No		Mentioned	0	.675	-.831	0.0%	2.2%

All or almost all calls received on cell phones	Not mentioned	31	30.325	.831	100.0%	97.8%
Some received on cell phones and some on regular phones	Mentioned	2	2.004	-.003	4.2%	4.2%
	Not mentioned	46	45.996	.003	95.8%	95.8%
Very few or none on cell phones	Mentioned	2	1.320	.599	3.7%	2.4%
	Not mentioned	52	52.680	-.599	96.3%	97.6%

The percentages are based on total observed frequencies in each subpopulation.

The statistical analysis shown in Table 39 represents the parameter estimates of thyroid cancer outcomes among the participants based on the levels of cell phone use. The analysis was stratified by smoking status. Based on the statistical estimates, smoking status is not a predictor of thyroid cancer outcomes, with an estimated Wald value of *2.197, OR = 1.327,* and *p = 0.138,* an estimate which is not statistically significant.

Table 39

Parameter Estimates of Thyroid Cancer Categorized by Cell Phone use and Smoking Status

Parameter Estimates

								95% Confidence Interval for Exp(B)	
What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower Bound*	*Upper Bound*
Mentioned	Intercept	-4.820	.708	46.330	1	.000			

	SMKSTAT2	.283	.191	2.197	1	.138	1.327	.913	1.929
	[PU1]	.351	.392	.799	1	.371	1.420	.658	3.064
	[PHU2]	.412	.327	1.586	1	.208	1.510	.795	2.869
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Table 40 shows the distribution and proportion of the smoking status of individuals represented in this study. The observed thyroid cancer outcomes among the participants were stratified by the smoking status and call received on cell phones.

Table 40

Proportion of Thyroid Cancer Categorized by Cell Phone use and Smoking Status

Observed and Predicted Frequencies

Smoking Status: Recode	Received calls cell/landline/both	What kind of cancer ... Thyroid	*Frequency*			*Percentage*	
			Observed	*Predicted*	*Pearson Residual*	*Observed*	*Predicted*
Current every day smoker	All or almost all calls received on cell phones	Mentioned	1	.719	.334	2.1%	1.5%
		Not mentioned	47	47.281	-.334	97.9%	98.5%
	Some received on cell phones and some on regular phones	Mentioned	1	.764	.272	2.1%	1.6%
		Not mentioned	47	47.236	-.272	97.9%	98.4%
	Very few or none on cell phones	Mentioned	1	.826	.192	1.3%	1.1%
		Not mentioned	77	77.174	-.192	98.7%	98.9%
		Mentioned	1	.237	1.581	8.3%	2.0%

Current some day smoker	All or almost all calls received on cell phones	Not mentioned	11	11.763	-1.581	91.7%	98.0%
	Some received on cell phones and some on regular phones	Mentioned	0	.336	-.586	0.0%	2.1%
		Not mentioned	16	15.664	.586	100.0%	97.9%
	Very few or none on cell phones	Mentioned	0	.266	-.520	0.0%	1.4%
		Not mentioned	19	18.734	.520	100.0%	98.6%
Former smoker	All or almost all calls received on cell phones	Mentioned	2	2.974	-.572	1.8%	2.6%
		Not mentioned	112	111.026	.572	98.2%	97.4%
	Some received on cell phones and some on regular phones	Mentioned	7	7.643	-.236	2.5%	2.8%
		Not mentioned	269	268.357	.236	97.5%	97.2%
	Very few or none on cell phones	Mentioned	5	5.775	-.326	1.6%	1.9%
		Not mentioned	307	306.225	.326	98.4%	98.1%
Never smoker	All or almost all calls received on cell phones	Mentioned	7	7.070	-.027	3.4%	3.4%
		Not mentioned	199	198.930	.027	96.6%	96.6%
		Mentioned	14	13.257	.208	3.8%	3.6%

Some received on cell phones and some on regular phones	Not mentioned	350	350.743	-.208	96.2%	96.4%
Very few or none on cell phones	Mentioned	11	10.132	.276	2.7%	2.4%
	Not mentioned	404	404.868	-.276	97.3%	97.6%

The percentages are based on total observed frequencies in each subpopulation.

Shown in Table 41 is the parameter estimates of thyroid cancer and levels of calls received on cell phones. The thyroid cancer risk estimates given the exposure to cell phone use was stratified by the menopausal status of the female participants. It shows that among the female participants, menopausal status is not a predictor of thyroid cancer outcomes, with a Wald value of *0.452, OR = 0.681*, and *p = 0.50*.

Table 41

Parameter Estimate of Thyroid Cancer including Cell Phone use and Menopausal Problem

Parameter Estimates

What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B)* *Lower Bound*	*Upper Bound*
Mentioned	Intercept	-20.147	.879	525.307	1	.000			
	MENOYR	-.385	.572	.452	1	.501	.681	.222	2.089
	[PU1]	17.611	.674	683.210	1	.000	44501189.08	11881272.9	166678758.8

	[PU2]	18.791	.000	.	1	.	144822526.2	144822526.2	144822526.3
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

If the gender were not differentiated for the menopausal problems analyzing the interactive effects of the menopausal problems among the selected population would be problematic, and the lack thereof may confound and distort the findings because menopausal problems are linked to women, not men. I stratified the participant by gender by performing the split functions using the SPSS software. See the Table 42 and 43 below.

Table 42

Proportion of Thyroid Cancer Categorized by Gender/female Stratification

Case Processing Summary[a]

		N	*Marginal Percentage*
What kind of cancer ... Thyroid	Not mentioned	8	100.0%
Working cell during land-line outage	Yes	8	100.0%
Valid		8	100.0%
Missing		19244	
Total		19252	

a. Gender = Female

Table 43

Thyroid Cancer proportion including Cell Phone use and Menopausal Problems

Observed and Predicted Frequencies

Menopausal problems, past 12 months	Received calls cell/landline/both	What kind of cancer ... Thyroid	*Frequency*			*Percentage*	
			Observed	*Predicted*	*Pearson Residual*	*Observed*	*Predicted*
Yes	All or almost all calls received on cell phones	Mentioned	1	1.585	-.477	3.2%	5.1%
		Not mentioned	30	29.415	.477	96.8%	94.9%
	Some received on cell phones and some on regular phones	Mentioned	7	6.415	.250	16.3%	14.9%
		Not mentioned	36	36.585	-.250	83.7%	85.1%
	Very few or none on cell phones	Mentioned	0	.000	.000	0.0%	0.0%
		Not mentioned	22	22.000	.000	100.0%	100.0%
No	All or almost all calls received on cell phones	Mentioned	2	1.415	.501	5.0%	3.5%
		Not mentioned	38	38.585	-.501	95.0%	96.5%
	Some received on cell phones and some on regular phones	Mentioned	4	4.585	-.289	9.3%	10.7%
		Not mentioned	39	38.415	.289	90.7%	89.3%
	Very few or none on cell phones	Mentioned	0	.000	.000	0.0%	0.0%
		Not mentioned	34	34.000	.000	100.0%	100.0%

The percentages are based on total observed frequencies in each subpopulation.

For the PU1 or 2 group for the research question #1, only age and gender predicted thyroid cancer. Therefore, age and gender were added in the regression model. See the regression model formula and Table 44 below.

Variable added in the Model:

1) $Y_1 = \beta_0 + Bx_{PU1} + \beta X_{age} + \beta X_{gender} + \varepsilon$

2) $Y_2 = \beta_0 + \beta X_{PU2} + \beta X_{age} + \beta X_{gender} + \varepsilon$

Table 44

Parameter Estimates of Thyroid Cancer including Cell Phone use, Gender and Age only

Parameter Estimates

What kind of cancer ... Thyroid[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
Mentioned	Intercept	-2.339	1.059	4.879	1	.027			
	Gender	1.136	.392	8.407	1	.004	3.116	1.445	6.717
	AGE_P	-.053	.011	23.602	1	.000	.949	.929	.969
	[PU1]	-.371	.427	.756	1	.384	.690	.299	1.593
	[PU2]	.157	.337	.216	1	.642	1.170	.604	2.266
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Research Question #2—The Assessment of the Difference in the Prevalence of Mouth/Tongue/Lip cancer between the Exposed and Non-exposed.

For the research question #2 inquiry, the comparative assessment of the difference in the prevalence of mouth/tongue/lip cancer between participants who received all or almost all calls on cell phones (PU1) versus those who received very few or no calls on cell phones (PU3) was analyzed. The difference in the prevalence of mouth/tongue/lip cancer between participants who received some calls on cell phones (PU2) versus those who received very few or no calls on cell phones (PU3) was analyzed as well. In Table 45, out of a total of 1,936 participants who owned and used cell phones, 10 people (0.5%) reported that they have been told by a doctor that they had mouth/tongue/lip cancer. In contrast, 1,926 (99.5%) individuals indicated that they have not been told they had mouth/tongue/lip cancer. About 41,409 individuals did not report any information or provide any response to either cell phone use or mouth/tongue/lip cancer and thus are considered missing values. Even when there is a large number of missing value, there was enough sample size from those who reported cell phone use/exposure to establish sufficient statistical power for the data analysis. In this study, the G*power sample size estimation with a 95% statistical power requires a minimum total sample size of 1,188 participants. For the statistical analysis, 1,936 participants had a complete response to cell phone use/exposure. See Table 4, Table 45, and Figure 19 for more detail.

Table 45

Distribution of Mouth/Tongue/Lip Cancer Categorized by Cell Phone Use/Exposure

Case Processing Summary

		N	*Marginal Percentage*
What kind of cancer ... mouth/tongue/lip	Mentioned	10	0.5%
	Not mentioned	1926	99.5%
Received calls cell/landline/both	All or almost all calls received on cell phones	384	19.8%
	Some received on cell phones and some on regular phones	711	36.7%
	Very few or none on cell phones	841	43.4%
Valid		1936	100.0%
Missing		41409	
Total		43345	
Subpopulation		3	

There were 384 participants who received all calls or almost all calls on cell phones (see Table 46). Among these individuals, 3 (0.8%) mentioned that at least a doctor had told them they had mouth/tongue/lip cancer while 381 (99.2%) participants did not have mouth/tongue/lip cancer. Among individuals who reported receiving some calls on cell phones, out of 711 participants, 5 (0.7%) reported that they had been told by a doctor that they had mouth/tongue/lip cancer. In contrast, 706 (99.3%) participants did not have mouth/tongue/lip cancer. Out of 841 people under the control group, those that rarely received calls on cell phones or those that did not receive any calls on cell phones, 2 (0.2%) people reported that they had been told by a doctor that they had

mouth/tongue/lip cancer. About 99.8% (839) of the control group did not have mouth/tongue/lip cancer.

The following is the prevalence estimation formula used for the analysis:

Prevalence = Number of cases/Total population size:

Using Table 45 and 46, the prevalence of mouth/tongue/lip cancer per 1000 participants for PU1, PU2, and PU3 groups could be calculated as follows:

PU1-Mouth/Tongue/Lip Cancer Prevalence = (3/1936) *1000 = 1.55 per 1000

PU2-Mouth/Tongue/Lip Cancer Prevalence = (5/1936) *1000 = 2.58 per 1000

PU3-Mouth/Tongue/Lip Cancer Prevalence = (2/1936) *1000 = 1.03 per 1000

The total prevalence rate of mouth/tongue/lip cancer among the studied population for participants with complete response is as follows:

PU1-3: Mouth/Tongue/Lip Cancer Prevalence = (10/1936) *1000 = 5.17 per 1000

The Mouth/Tongue/Lip Cancer Prevalence for PU1 and 2 is as follows:

PU1 & 2- Mouth/Tongue/Lip cancer Prevalence = (3+5/1936) *1000 = 4.13 per 1000

The prevalence estimation among the control group, which are participants who received very few calls or no calls on cell phones (PU3 group) had the lowest rate (1.03 per 1000). The prevalence rate of Mouth/Tongue/Lip cancer among participants who received all calls or almost all calls on cell phones (PU1 group) was the second to the lowest (1.55 per 1000). The prevalence rate of Mouth/Tongue/Lip cancer among the participants who received some calls on cell phones (PU2 group) was the highest (2.58 per 1000).

Table 46

Distribution of Thyroid Cancer Categorized by the Level of Cell Phone Use/Exposure

Observed and Predicted Frequencies

		Frequency			*Percentage*	
Received calls cell/landline/both	What kind of cancer ... mouth/tongue/lip	*Observed*	*Predicted*	*Pearson Residual*	*Observed*	*Predicted*
All or almost all calls received on cell phones	Mentioned	3	3.000	.000	0.8%	0.8%
	Not mentioned	381	381.000	.000	99.2%	99.2%
Some received on cell phones and some on regular phones	Mentioned	5	5.000	.000	0.7%	0.7%
	Not mentioned	706	706.000	.000	99.3%	99.3%
Very few or none on cell phones	Mentioned	2	2.000	.000	0.2%	0.2%
	Not mentioned	839	839.000	.000	99.8%	99.8%

The percentages are based on total observed frequencies in each subpopulation.

Table 47 shows the parameter estimate of the effect of cell phone use on mouth/tongue/lip cancer prevalence. PU3 represent individuals who rarely receive calls on cell phones or individuals who do not receive calls on cell phones. Individuals who received all or almost all calls on cell phones are represented as 'PU1'. Individuals who received some calls on cell phones were represented as 'PU2'. In this study, PU1 did not statistically predict mouth/tongue/lip cancer outcomes, $\beta = 1.195$, $W(1) = 1.705$, $OR = 3.303$, $p = 0.192$, *95% CI [0.550, 19.849]*. Similarly, the 'PHONUSE=2' did not predict mouth/tongue/lip cancer outcomes, $\beta = 1.089$, $W(1) = 1.688$, $OR = 2.971$, $p = 0.194$, *95% CI [0.575, 15.360]*. Therefore, the null hypothesis for research question #2 is not rejected. Even when the estimated p-values in Table 47 are not statistically significant,

the effect sizes (odds ratios) for both statistical estimates (PU1 and 2) were very interesting. The odds ratio for the 'PU1' was 3.303, which means that participants who received all calls or almost all calls on cell phones are over 3 times more likely to have mouth/tongue/lip cancer outcomes. Similarly, the odds ratio for the 'PU2' was 2.971, suggesting that participants who received some calls on cell phones were about 2.9 times more likely to have mouth/tongue/lip cancer outcomes. The estimated p-values, 0.192 and 0.194 as shown in Table 47 for PU1 and 2 respectively for research question #2 are not statistically significant. Therefore, there is no justification to perform covariate and confounder interaction analysis. The estimated p-values for the analysis on the effects of cell phone use on mouth/tongue/lip cancer are not statistically significant. Hence, the null hypothesis was not be rejected. Also, in this analysis, Table 48 shows the correlation matrix of the associations between PU1 and 2 and the prevalence of mouth/tongue/lip cancer..

Table 47

Parameter Estimates of Mouth/Tongue/Lip Cancer Categorized by Cell Phone Use/Exposure

Parameter Estimates

What kind of cancer ... mouth/tongue/lip[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
Mentioned	Intercept	-6.039	.708	72.767	1	.000			
	[PU1]	1.195	.915	1.705	1	.192	3.303	.550	19.849
	[PU2]	1.089	.838	1.688	1	.194	2.971	.575	15.360
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Not mentioned.

b. This parameter is set to zero because it is redundant.

Table 48

Correlation Matrix of Mouth/Tongue/Lip Cancer Categorized by Cell Phone Use/Exposure

Asymptotic Correlation Matrix

		What kind of cancer ... mouth/tongue/lip Mentioned			
What kind of cancer ... mouth/tongue/lip[a]	*Intercept*	*[PU1]*	*[PU2]*	*[PU3]*	
Mentioned	Intercept	1	-.774	-.845	.[b]
	[PU1]	-.774	1	.654	.[b]
	[PU2]	-.845	.654	1	.[b]
	[PU3]	.[b]	.[b]	.[b]	.[b]

a. The reference category is: Not mentioned.

b. One or both parameter estimates are redundant.

Research Question #3—Difference in the Prevalence of Heart Condition between the Exposed and Non-exposed

The inferential difference in the prevalence of heart conditions/disease between participants who received all or almost all calls on cell phones and individuals who received very few or no calls on cell phones was evaluated using multiple logistic regression. The relationship between the three levels of cell phone use behavior/exposure specified in Table 7 or 21 were evaluated. Out of a total of 16,446 participants that had a complete response to the questions ‘Ever been told you had a heart condition/disease’ and who also received or did not receive calls on cell phones (see Table 49). A total of 1,447 (8.8%) individuals reported that they had been told that they had heart condition/disease

and 14,999 participants (91.2%) indicated that they had not been told they had heart condition/disease (see Table 49).

Also, out of the 16,446 participants with a complete response to the questions in this section shown in Table 49, approximately 5,068 participants (30.8%) received all calls or almost all calls on cell phones. About 6,734 participants (40.9%) received some calls on cell phones while 4,644 respondents (28.2%) reported receiving very few calls or no call on cell phones. There were 26,899 respondents reported as missing cases. Even with a large number of missing cases, there was enough sample size from those with complete responses on cell phone use and heart condition/disease to generate enough statistical power for the inferential analysis for research question #3. Notably, the G*power sample size estimation generated for a 95% statistical power for this analysis required a total sample size of 1,188 subjects, but 16,446 participants with a complete response to the cell phone use and heart condition/disease questions were used for the statistical analysis for this research inquiry, see Table 4, Table 49 and 50, and Figure 19 for more detail.

Table 49

Distribution of Heart Condition/Disease Categorized by Cell Phone Use Exposure

Case Processing Summary

		N	*Marginal Percentage*
Ever been told you had a heart condition/disease	Yes	1447	8.8%
	No	14999	91.2%
Received calls cell/landline/both	All or almost all calls received on cell phones	5068	30.8%

	Some received on cell phones and some on regular phones	6734	40.9%
	Very few or none on cell phones	4644	28.2%
Valid		16446	100.0%
Missing		26899	
Total		43345	
Subpopulation		3	

Prevalence = Number of cases/Total population size:

Using Table 49 and 50 the prevalence of heart condition/disease per 1000 participants for PU1, PU2, and PU3 groups could be calculated as follows:

PU1-Heart condition/disease Prevalence = (322/16446) *1000 = 19.58 per 1000.

PU2-Hear condition/disease Prevalence = (525/16446) *1000 = 31.92 per 1000.

PU3-Heart condition/disease Prevalence = (600/16446) *1000 = 36.48 per 1000.

The total prevalence of heart condition/disease for the studied population for participants with complete information/responses is as follows:

PU1-3: Heart condition/disease Prevalence = (1447/16446) *1000 = 87.97 per 1000.

The heart condition/disease Prevalence for PU1 and 2 is as follows:

PU1 & 2-Heart condition/disease Prevalence = (322+525/16446) *1000 = 51.50 per 1000.

Based on the prevalence estimation, the control group, which represent individuals who received very few calls or no calls on cell phones (PU3 group) had the highest rate (36.48 per 1000) for the heart condition/disease. The prevalence rate of heart condition/disease among participants who received some calls on cell phones (PU2

group) is the second highest (31.92 per 1000). The prevalence rate of heart condition/disease among participants who received all calls or almost all calls on cell phones (PU1 group) is the lowest (19.58 per 1000), see Table 49 and 50 for more detail.

Table 50

Distribution of Heart Condition/Disease Categorized by Cell Phone Use/Exposure

*Received calls cell/landline/both * Ever been told you had a heart condition/disease Crosstabulation*

Count

		Ever been told you had a heart condition/disease		
		Yes	*No*	*Total*
Received calls cell/landline/both	All or almost all calls received on cell phones	322	4746	5068
	Some received on cell phones and some on regular phones	525	6209	6734
	Very few or none on cell phones	600	4044	4644
Total		1447	14999	16446

Table 51 shows the likelihood ratio tests for 'PU' on whether cell phone use is associated with heart condition/disease. Based on the likelihood ratio test (LRT) estimation, $LRT\ (2) = 160.86, \chi^2 = 137.196, ***p < 0.001$. Based on the p-value estimation which is less than the predetermined alpha value of 0.05, the null hypothesis is rejected. Therefore, there is an association between cell phone use and heart condition/disease. Similarly, the prevalence of heart condition/disease between

participants who received all calls on cell phones or individuals who received some calls on cell phones differ compared to individuals who received very few or no calls on cell phones. See Table 52 for more detail.

Table 51

Likelihood Ratio of Heart Condition/Disease Categorized by Cell Phone Use/Exposure

Likelihood Ratio Tests

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	23.664[a]	.000	0	.
PU	160.860	137.196	2	.000

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

Table 52 shows more elaborate information on the differential effects of cell phone use on heart condition/disease. Individuals who received all or almost all calls on cell phones were represented as ‘PU1’while individuals who received some calls on cell phones were represented as ‘PU2’. PU3 represent individuals who received very few calls or no calls on cell phones. Based on the information provided in Table 52, ‘PU1’ significantly predicted heart condition/disease outcomes, *β = 0.782, W(1) = 117.054, OR = 2.187, ***p < 0.001, 95% CI [1.898, 2.520]*. Similarly, ‘PU2’ significantly predicted heart condition/disease outcomes, *β = 0.562, W(1) = 79.446, OR = 1.755, ***p < 0.001, 95% CI [1.551, 1.986]*. Again, based on the statistical analysis, the null hypothesis was rejected. The effect size estimates (odds ratios) for the ‘PU1’ and ‘PU2’ group were 2.187 and 1.755 respectively. For ‘PU1’ group, the *OR* value of *2.187* suggest that

participants who received calls on cell phones all the time or almost all the time are more than twice likely to have heart condition/disease. Similarly, for 'PU2' group with the *OR* value of *1.755*, it suggests that individuals who received calls on cell 'sometimes' have about twice the chance of developing heart condition/disease.

Table 52

The variable in the Equation for Heart Disease Categorized by Cell Phone Use/Exposure

Variables in the Equation

								95% C.I.for EXP(B)	
		B	*S.E.*	*Wald*	*df*	*Sig.*	*Exp(B)*	*Lower*	*Upper*
Step 1[a]	PU			140.278	2	.000			
	PU1	.782	.072	117.054	1	.000	2.187	1.898	2.520
	PU2	.562	.063	79.446	1	.000	1.755	1.551	1.986
	Constant	1.908	.044	1902.191	1	.000	6.740		

a. Variable(s) entered on step 1: PHONEUSE.

Confounder and Covariate Inferential Analysis for Heart Condition/Disease:

Table 53 showed case summary of the participants when the possible confounders and covariates were included in the analysis. Out of 2,206 subjects who had a complete response to the heart disease question, there were 139 participants (6.3%) who indicated that they had been told they had a heart condition/disease. A total of 2067 (93.7%) individuals reported that they had never been told that they had heart condition/disease. Individuals who received all calls or almost all calls on cell phones were 752 (34.1%). Those that received some calls on cell phones was 1,007 (45.6%) while 447 (20.3%) participants received very few or no calls on cell phones. Overall, there were 41,139 subjects with missing data.

Table 53

Distribution of Heart Condition/Disease Categorized by Cell Phone Use/Exposure

Case Processing Summary

		N	*Marginal Percentage*
Ever been told you had a heart condition/disease	Yes	139	6.3%
	No	2067	93.7%
Received calls cell/landline/both	All or almost all calls received on cell phones	752	34.1%
	Some received on cell phones and some on regular phones	1007	45.6%
	Very few or none on cell phones	447	20.3%
Valid		2206	100.0%
Missing		41139	
Total		43345	
Subpopulation		1315[a]	

a. The dependent variable has only one value observed in 1242 (94.4%) subpopulations.

The likelihood ratio test shown in Table 54 contains the confounders and covariates included in this analysis together with the independent variable (PU). Based on the likelihood ratio test (LRT) estimation on whether 'PU' with the covariates/confounders inclusion in the model is associated with heart condition/disease. Out of the 7 covariate/confounders listed in Table 54, race (MRACRP12) and the menopausal problem (MENOYR), the LRT and $\chi 2$ was statistically significant $p < 0.05$ (see Table 52 for more information). However, with the inclusion of the

covariates/confounders, the 'PU p-value in Table 51, was no longer statistically significant. With the inclusion of the covariates, the estimated p-value of 'PU' was 0.720 (see Table 54).

Table 54

Likelihood Ratio of Heart Disease including Cell Phone Use/Exposure and all Covariates/Confounders

Likelihood Ratio Tests

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	804.323[a]	.000	0	.
AGE_P	804.324	.001	1	.977
GENDER	804.323[a]	.000	0	.
R_MARITL	804.835	.512	1	.474
MRACRPI2	809.606	5.283	1	.022
WRKLONGH	804.328	.005	1	.946
SMKSTAT2	805.224	.901	1	.342
MENOYR	825.867	21.543	1	.000
PHONEUSE	804.979	.656	2	.720

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimate table below shows corresponding beta, Wald, sig., odds ratio, and CI values which were relevant in making inferential conclusions. When the confounders/covariates (age, gender, marital status (R-MARITL), race (MRACRPI2), employment status (WRKLONGH), and smoking status (SMKSTAT2)) were added in the model, the 'PU1' and 'PU2' analysis did not produce statistically significant p-values (*p =0.602 and p=0.068* respectively) as shown in Table 55 when compared the p-values

produced previously in Table 52 for ‘PU1’ and ‘PU2’ with both p-values less than *0.001*, an estimate generated without the addition of the covariates/confounders in the model. Therefore, using the Table 55 estimation, the null hypothesis was rejected. With the inclusion of the specified covariates/confounders in the model, there is no association between cell phone use and heart condition/disease when age, gender, marital status, race, employment status, and smoking status are all included in the logistic regression model. Hence, PU1 and 2 group did not predict heart condition when the confounders/covariates were added in the model; β *= -0.56, W(1) = 0.271, OR = 0.946, p = 0.602, 95% CI [0.766 1.167]* and β *= -0.186, W(1) = 3.333, OR = 0.830, p = 0.602, 95% CI [0.680, 1.014]* respectively. The *OR* estimation for PU1 and 2 were not much different than the control/reference group. Therefore, when the specified covariates are accounted for, the prevalence of heart condition/disease between participants who received all calls on cell phones or individuals who received some calls on cell phones was not different compared to individuals who received very few or no calls on cell phones.

Table 55

Parameter Estimated of Heart Disease including levels of Cell Phone Use/ Exposure and all Confounders and Covariates

Parameter Estimates

Ever been told you had a heart condition/disease[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
Yes	Intercept	-4.290	.301	202.521	1	.000			
	AGE_P	.036	.003	117.285	1	.000	1.036	1.030	1.043
	GENDER	.057	.080	.503	1	.478	1.059	.904	1.239

	R_MARITL	.020	.016	1.604	1	.205	1.020	.989	1.051
	MRACRPI2	-.049	.018	7.610	1	.006	.952	.920	.986
	WRKLONGH	.121	.080	2.284	1	.131	1.129	.965	1.322
	SMKSTAT2	-.114	.037	9.703	1	.002	.892	.831	.959
	[PU1]	-.056	.108	.271	1	.602	.946	.766	1.167
	[PU2]	-.186	.102	3.333	1	.068	.830	.680	1.014
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: No.

b. This parameter is set to zero because it is redundant.

The LRT in Table 56 shows the effects of age and phoneuse with LRT and χ^2 values with a statistically significant *p-value (p <0.001* and *p = 0.05* respectively).

Table 56

Likelihood Ratio Effects of Age and Cell Phone Use/Exposure

Likelihood Ratio Tests

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	866.572[a]	.000	0	.
AGE_P	1386.391	519.819	1	.000
PU	872.506	5.934	2	.051

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimate table shows the effects of age and PU1 and 2 on heart condition/disease. When age is included in the model as shown in Table 57, 'PU1' did not predict heart condition/disease, *β = 0.067, W(1) = 0.696, OR = 1.069, p = 0.404, 95% CI [0.914, 1.251],* while 'age' and 'PU2' predicted heart condition/disease outcomes; *β = -0.044, W(1) = 459.189, OR = 0.957, ***p < 0.001, 95% CI*

[0.953,0.961] and $\beta = 0.160$, *W(1) = 5.782, OR = 1.174, p = 0.016, 95% CI [1.030, 1.337]* respectively. Using Table 57 that shows 'age' alone as a covariate, the null hypothesis should be rejected, suggesting that only PU2 exposure predicted heart disease.

Table 57

Parameters Estimated of Heart Disease Categorized by Cell Phone Use and Age

Parameter Estimates

Ever been told you had a heart condition/disease[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	4.805	.149	1043.828	1	.000			
	AGE_P	-.044	.002	459.189	1	.000	.957	.953	.961
	[PU1]	.067	.080	.696	1	.404	1.069	.914	1.251
	[PU2]	.160	.067	5.782	1	.016	1.174	1.030	1.337
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

The LRT shown in Table 58 demonstrates the effects of gender and phoneuse with LRT and χ^2 values with a statistically significant p-value ($p = 0.028$ and $p < 0.001$ respectively).

Table 58

Likelihood Ratio of the Effects of Gender and Cell Phone Use

Likelihood Ratio Tests

Effect	*Model Fitting Criteria* *-2 Log Likelihood of Reduced Model*	*Likelihood Ratio Tests* *Chi-Square*	*df*	*Sig.*
Intercept	44.308[a]	.000	0	.
GENDER	49.148	4.840	1	.028
PU	183.851	139.543	2	.000

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimate table shows the effects of gender, PU1 and PU2 on heart condition/disease. With this estimation, when the covariate-gender is included in the model as shown in Table 59, 'gender', 'PU1', and 'PU2' predicted heart condition/disease outcomes; *β = 0.123, W(1) = 4.859, OR = 1.131, p = 0.028, 95% CI [1.014, 1.261], β = 0.791, W(1) = 119.160, OR = 2.205, ***p < 0.001, 95% CI [1.913, 2.541],* and *β = 0.568, W(1) = 80.832, OR = 1.764, ***p < 0.001, 95% CI [1.559, 1.997*] respectively.

Table 59

Parameter Estimates Categorized by Gender and Levels of Cell Phone Use/Exposure

Parameter Estimates

Ever been told you had a heart condition/disease[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	1.711	.099	298.879	1	.000			
	GENDER	.123	.056	4.859	1	.028	1.131	1.014	1.261
	[PU1]	.791	.072	119.160	1	.000	2.205	1.913	2.541
	[PU2]	.568	.063	80.832	1	.000	1.764	1.559	1.997
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

The LRT shown in Table 60 represents the effects of marital status and phoneuse. The LRT and χ^2 had a statistically significant p-value (*p = 0.011* and *p < 0.001* respectively).

Table 60

Likelihood Ratio on the Effects of Marital Status and Cell Phone Use/Exposure

Likelihood Ratio Tests

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	261.868[a]	.000	0	.
R_MARITL	268.367	6.499	1	.011
PU	397.413	135.545	2	.000

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimate table shows the effects of marital status, PU1, and PU2 on heart condition/disease. For this parameter estimation, when marital status together with the primary independent variable (PU) were included in the model as shown in Table 61; 'marital status' (R-MARITL), 'PU1', and 'PU2' predicted heart condition/disease outcomes; *β = 0.028, W(1) = 6.418, OR = 1.029, p = 0.011, 95% CI [1.006, 1.051], β = 0.774, W(1) = 114.181, OR = 2.168, ***p < 0.001, 95% CI [1.881, 2.498],* and *β = 0.567, W(1) = 80.607, OR = 1.763, ***p < 0.001, 95% CI [1.557, 1.995*] respectively.

Table 61

Parameter Estimated of Heart Disease Categorized by Marital Status and Cell Phone Use/Exposure

Parameter Estimates

Ever been told you had a heart condition/disease[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	1.821	.055	1086.640	1	.000			
	R_MARITL	.028	.011	6.418	1	.011	1.029	1.006	1.051
	[PU1]	.774	.072	114.181	1	.000	2.168	1.881	2.498
	[PU2]	.567	.063	80.607	1	.000	1.763	1.557	1.995
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

The LRT shown in Table 62 demonstrates the effects of race and phoneuse. The LRT and χ^2 had a statistically significant p-value ($p < 0.001$ and $p < 0.001$ respectively).

Table 62

Likelihood Ratio on the Effects of Race and Cell Phone Use

Likelihood Ratio Tests

Effect	*Model Fitting Criteria -2 Log Likelihood of Reduced Model*	*Likelihood Ratio Tests Chi-Square*	*df*	*Sig.*
Intercept	134.095[a]	.000	0	.
MRACRPI2	159.378	25.284	1	.000
PU	264.806	130.711	2	.000

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimate table shows the effects of race, PU1, and PU2 on heart condition/disease. For this parameter estimation, when race together with the primary independent variable (phoneuse) were included in the model as shown in Table 63; 'race' (MRACERP12), 'PU1', and 'PU2' predicted heart condition/disease outcomes; β =

*0.057, W(1) = 20.935, OR = 1.059, ***p < 0.001, 95% CI [1.033, 1.086], β = 0.765, W(1) = 111.591, OR = 2.149, ***p < 0.001, 95% CI [1.865, 2.477],* and *β = 0.549, W(1) = 75.495, OR = 1.731, ***p < 0.001, 95% CI [1.530, 1.959*] respectively.

Table 63

Parameter Estimated of Heart Disease Categorized by Race and Cell Phone Use

Parameter Estimates

Ever been told you had a heart condition/disease[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	1.817	.048	1456.376	1	.000			
	MRACRPI2	.057	.013	20.935	1	.000	1.059	1.033	1.086
	[PU1]	.765	.072	111.591	1	.000	2.149	1.865	2.477
	[PU2]	.549	.063	75.495	1	.000	1.731	1.530	1.959
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

The case summary table below shows the distribution of participants included in the analysis. Out of 11,585 individuals who had a complete response to the heart disease question, there were 702 participants (6.1%) who indicated that they had been told they had a heart condition/disease. A total of 10,883 participants (93.9%) reported that they had never been told that they had heart condition/disease. Individuals who received all calls or almost all calls on cell phones were 4,356 (37.6%) in total. Those that received some calls on cell phones was 4,977 (43.0%), while 2,252 (19.4%) participants received very few or no calls on cell phones. A total of 31,760 participants were missing data. See Table 64 for more detail.

Table 64

Distribution of Heart Disease Categorized by Cell Phone Use and Employment Status

Case Processing Summary

		N	*Marginal Percentage*
Ever been told you had a heart condition/disease	Yes	702	6.1%
	No	10883	93.9%
Received calls cell/landline/both	All or almost all calls received on cell phones	4356	37.6%
	Some received on cell phones and some on regular phones	4977	43.0%
	Very few or none on cell phones	2252	19.4%
Valid		11585	100.0%
Missing		31760	
Total		43345	
Subpopulation		6	

The LRT shown in Table 65 shows the effects of employment status and PU. The LRT and χ^2 values had a statistically significant *p-value* ($p = 0.042$ and $p < 0.001$ respectively).

Table 65

Likelihood Ratio on the Effects of Current Employment/Longest Held and Cell Phone Use

Likelihood Ratio Tests

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	44.323[a]	.000	0	.
WRKLONGH	48.476	4.153	1	.042
PU	62.736	18.413	2	.000

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimates in Table 66 shows the effects of employment status (WRKLONGH), PU1, and PU2 on heart condition/disease. With this parameter estimation, when employment status together with the primary independent variable (PU) were included in the model as shown in Table 66; employment status, 'PU1', and 'PU2' predicted heart condition/disease outcomes; *β = -0.162, W(1) = 4.189, OR = 0.850, p = 0.041, 95% CI [0.728, 0.993], β = 0.408, W(1) = 16.027, OR = 1.504, ***p < 0.001, 95% CI [1.232, 1.837]*, and *β = 0.383, W(1) = 14.999, OR = 1.466, ***p < 0.001, 95% CI [1.208, 1.780*] respectively.

Table 66

Parameter Estimates of Heart Disease Categorized by Employment Status /Longest Held and Cell Phone Use/Exposure

Parameter Estimates

Ever been told you had a heart condition/disease		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	2.661	.137	375.369	1	.000			
	WRKLONGH	-.162	.079	4.189	1	.041	.850	.728	.993
	[PU1]	.408	.102	16.027	1	.000	1.504	1.232	1.837
	[PU2]	.383	.099	14.999	1	.000	1.466	1.208	1.780
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

The LRT in Table 67 shows the effects of smoking status and PU. The LRT and χ^2 values for smoking status is not statistically significant, $p = 0.057$ while the LRT and χ^2 values for PU is statistically significant, $p < 0.001$ respectively).

Table 67

Likelihood Ratio on the Effects of Smoking Status and Cell Phone Use/Exposure

Likelihood Ratio Tests

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	150.222[a]	.000	0	.
SMKSTAT2	153.831	3.609	1	.057
PHONEUSE	279.817	129.595	2	.000

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimates in Table 68 shows the effects of smoking status, PU1, and PU2 on heart condition/disease. For this parameter estimation, when smoking status together with the primary independent variable (PU) were included in the model as shown in Table 68; smoking status did not predict heart condition/disease outcomes; *β = 0.053, W(1) = 3.678, OR = 1.054, p = 0.055, 95% CI [0.999, 1.113]*, but 'PU1', and 'PU2' predicted heart condition/disease outcomes; *β = 0.766, W(1) = 111.330, OR = 2.152, ***p < 0.001, 95% CI [1.867, 2.482]*, and *β = 0.548, W(1) = 74.437, OR = 1.729, ***p < 0.001, 95% CI [1.527, 1.959*] respectively.

Table 68

Parameter Estimates of Heart Disease Categorized by Smoking Status and Cell Phone Use/Exposure

Parameter Estimates

Ever been told you had a heart condition/disease[a]		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	1.745	.100	305.465	1	.000			
	SMKSTAT2	.053	.028	3.678	1	.055	1.054	.999	1.113
	[PU1]	.766	.073	111.330	1	.000	2.152	1.867	2.482
	[PU2]	.548	.063	74.437	1	.000	1.729	1.527	1.959
	[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

For the assessment of the effect of menopausal problems together with phone use on heart condition/disease, the dataset was split or stratified by gender. The analysis included only the women and excluded all men. The case summary in Table 69 below represented the distribution of women included in the analysis. Out of 2,350 women who had a complete response to the heart disease questionnaire question, there were 145 participants (6.2%) who indicated that they had been told they had a heart condition/disease. A total of 2,205 women (93.8%) reported that they had never been told that they had heart condition/disease. Women who received all calls or almost all calls on cell phones were 804 (34.2%) in total. The total number of women who received some calls on cell phones was 1,059 (45.1%). A total of 487 (20.7%) women received very few or no calls on cell phones. There were 16,902 women with missing data, see Table 69 for more detail.

Table 69

Distribution of Heart Disease Categorized by Menopausal Problems of Gender/Female

Case Processing Summary[a]

		N	*Marginal Percentage*
Ever been told you had a heart condition/disease	Yes	145	6.2%
	No	2205	93.8%
Received calls cell/landline/both	All or almost all calls received on cell phones	804	34.2%
	Some received on cell phones and some on regular phones	1059	45.1%
	Very few or none on cell phones	487	20.7%
Valid		2350	100.0%
Missing		16902	
Total		19252	
Subpopulation		6	

a. Gender= Female

The LRT shown in Table 70 is the effects of menopausal problems (MENSYR) and phoneuse. The LRT and χ^2 values for MENSYR is statistically significant, ***$p < 0.001$ while the LRT and χ^2 values for PU after accounting for MENSYR was not statistically significant, $p < 0.584$ respectively).

Table 70

Likelihood Ratio on the Effects of Menopausal Problems (Gender=Women) and Cell Phone Use/Exposure

Likelihood Ratio Tests[a]

	Model Fitting Criteria	*Likelihood Ratio Tests*		
Effect	*-2 Log Likelihood of Reduced Model*	*Chi-Square*	*df*	*Sig.*
Intercept	29.734[b]	.000	0	.
MENSYR	56.423	26.689	1	.000
PU	30.810	1.076	2	.584

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

a. Gender = Female

b. This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom.

The parameter estimates in Table 71 shows the effects of menopausal problems, PU1, and PU2 on heart condition/disease among women participants. When menopausal problem together with the primary independent variable (PU) was included in the model as shown in Table 71; menopausal problems predicted heart condition/disease outcomes among women.; *β = 0.429, W(1) = 6.962, OR = 1.535, p = 0.008, 95% CI [1.117, 2.110],* but the inclusion of the menopausal variable, 'PU1' and 'PU2' did not predict heart condition/disease outcomes among women participants in this study; *β = 0.168, W(1) = 0.816, OR = 1.182, p = 0.366, 95% CI [0.822, 1.701],* and *β = 0.358, W(1) = 3.527, OR = 1.430, p = 0.060, 95% CI [0.985, 2.078*] respectively. Based on these findings, the two regression models "The variable in the Model with effects" equation below shows the appropriate variables needed to be included in the model for the heart condition/disease analysis based on cell phone use exposure.

Table 71

Parameter Estimates on the Effects of Menopausal Problems (Gender=Women) and Cell Phone Use/Exposure

Parameter Estimates

Ever been told you had a heart condition/disease		*B*	*Std. Error*	*Wald*	*df*	*Sig.*	*Exp(B)*	*95% Confidence Interval for Exp(B) Lower Bound*	*Upper Bound*
No	Intercept	2.019	.329	37.636	1	.000			
	MENSYR	.429	.162	6.962	1	.008	1.535	1.117	2.110
	[PU1]	.168	.185	.816	1	.366	1.182	.822	1.701

[PU2]	.358	.191	3.527	1	.060	1.430	.985	2.078
[PU3]	0[b]	.	.	0	.	.	.	.

a. The reference category is: Yes.

b. This parameter is set to zero because it is redundant.

Variable in the Model with Effects

1) $Y_1 = \beta_0 + Bx_{PU1} + Bx_{\text{marital status}} + \beta X_{\text{gender}} + \varepsilon$

2) $Y_2 = \beta_0 + Bx_{PU2} + \beta X_{\text{age}} + \beta X_{\text{gender}} + Bx_{\text{memoyr}} + \varepsilon$

Summary

Chapter 4 of this dissertation contains the results of the descriptive and inferential statistics, which provided the estimation of the difference in the prevalence of thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease. The prevalence analysis between individuals who received all calls or almost all calls on cell phones (PU1) and individuals that received very few calls or no calls on cell phones (PU3) was evaluated. The prevalence difference between individuals who received some calls on cell phones (PU2) and individuals that received very few calls or no calls on cell phones (PU3) were evaluated.

The prevalence of thyroid cancer for this population among those with complete response was 25.83 per 1000. Thyroid cancer prevalence for the PU1 group was 5.68 per 1000. The thyroid cancer prevalence rate estimate for the PU2 group was 11.36 per 1000, while the prevalence rate for thyroid cancer among the PU3 group was 8.78 per 1000. The prevalence of thyroid cancer among individuals who received all calls or almost all calls on cell phones (PU1 group) was lower than the prevalence rate of thyroid cancer of individuals who received some calls on cell phones (PU2 group). Also, lower

for the control group (PU3 group), individuals who received very few calls or no calls on cell phones.

The prevalence rate of mouth/tongue/lip cancer for the PU1, PU2, and PU3 groups was 5.17 per 1000. The prevalence rate estimate for mouth/tongue/lip cancer among the PU1 group was 1.55 per 1000. The estimated mouth/tongue/lip cancer prevalence rate among the PU2 group was 2.58 per 1000. On the other hand, the mouth/tongue/lip cancer prevalence rate among the PU3 group was 1.03 per 1000. Based on the prevalence rate estimation, participants who received very few calls or no calls on cell phones (PU3 group) had the lowest prevalence rate compared to those who received all calls or almost all calls on cell phones (PU1 group). Individuals who received some calls on cell phones (PU2 group) had lower prevalence rate as well.

The estimated prevalence rate for heart condition/disease was 87.97 per 1000. The heart condition prevalence was 19.58 per 1000 for the PUI group, 31.92 per 1000 among the PU2 group, and 36.48 per 1000 for the PU3 group. Based on the information, the heart disease prevalence rate estimate among participants who received very few calls or no calls on cell phones (PU3 group) was higher compared to those who received some calls on cell phones (PU2 group). Participants who received all calls or almost all calls on cell phones (PU1 group) had the lowest prevalence rate, 19.58 per 1000. This estimation is consistent with the parameter estimation in Table 52. It showed a negative association. These findings, its implications, limitations, and relevance will be discussed in detail in Chapter 5.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

Uncertainty exists in the published body of literature on the adverse health effects of long-term cell phone use. Several researchers demonstrated some adverse correlational or causal health effects of cell phone-driven radiation on either humans or animals (Balmori, 2016; Fehske et al., 2011; Kesari et al., 2013; NTP, 2016; Tkalec et al., 2008). The objective of this epidemiological inquiry was to assess differences in the prevalence rate of thyroid cancer, mouth/tongue/lip cancer, and heart conditions among individuals who are heavy cell phone users and individuals who do not use cell phones or who rarely receive calls on cell phones. The prevalent nature of cell phone use in the US and other parts of the world facilitated this study. The current findings could advance future short and long-term health promotion measures focused on reducing adverse health impacts of cell phone use.

The use of logistic regression analysis for the current comparative study facilitated the assessment of mediating or moderating effects of the confounders or covariates identified in this study. The findings suggested that there is an association between cell phone use and cancer outcomes. However, cell phone use did not significantly predict thyroid cancer or mouth/tongue/lip cancer outcomes. Rather, cell phone use is predictive of heart disease or condition.

Interpretation of the Findings

Balmori (2016) supported other published findings on the adverse health effects of cell phone-driven RFR exposure. The author concluded that cell phone-driven RFR

exposure or EMR is an environmental pollutant which adversely affects wildlife and other living things (Balmori, 2016). Balmori (2016) suggested that phone masts located around animal habitats irradiate species, which promotes long-term biological effects. Also, suggested the gap in the literature specifically on the long-term health effects of cell phone use or RFR exposure (Balmori, 2016). Many researchers reiterated the need for further epidemiological studies on cell phone use or cell phone-drive RFR exposure in humans as well (see Kesari et al., 2013; Kundi, 2009; Fehske et al., 2011).

In this study, I assessed the health effects of cell phone use or cell phone-driven RFR exposure through calls received. The three primary health effects evaluated were thyroid cancer, mouth/tongue/lip cancer, and heart disease. Thyroid cancer, mouth/tongue/lip cancer, and heart disease were measured by comparing the prevalence rates between individuals who either 'received all/almost all calls on cell phones' (PU1) or 'some calls on cell phones' (PU2) and those who 'received no/very few calls on cell phone' (PU3). Cell phone use behavior among individuals who received 'all/almost all calls' on cell phones significantly predicted cancer outcomes, $\beta = 0.992$, $W(1,2) = 231.155$, $OR = 2.698$, $***p < .001$, 95% CI [2.374, 3.066], compared to those who received no/very few calls. Cell phone use risk estimate between individuals who received 'some calls' on cell phones in comparison to those who received very few or no calls on cell phones significantly predicted cancer outcomes, $\beta = 0.629$, $W(1,2) = 131.135$, $OR = 1.876$, $***p < .001$, 95% CI [1.685, 2.090]. Individuals who received all/almost all calls on cell phones were 2.7 times more likely to have cancer than individuals who received no/very few calls on cell phones. Similarly, individuals who

received ‘some’ calls on cell phones were 1.9 times more likely to have cancer than individuals who received no/very few calls on cell phones (see Table 22). These findings addressed the gap in the literature about the risk of cell phone use or cell phone-driven radiation on thyroid cancer outcomes.

The risk rate of cancer observed in this study was similar and, in some cases, lower than the risk rates observed in other epidemiologic studies on the topic. Hardell, Carlberg, Söderqvist, Mild, and Morgan (2007) evaluated acoustic neuroma and brain tumor (glioma) risk among long-term cell phone users (i.e., >10 years and ipsilateral exposure) using a case-control study design. Their conclusion was similar to the observation made in this study. In the study, a two to three-fold increased risk of acoustic neuroma was observed among groups who had at least 10 years of cell phone use (Hardell et al., 2007). Hardell et al. (2007) found that the size of the tumors was significantly larger among users. The increased OR rate for cancers observed for ipsilateral exposure in the study was 5.4 (Hardell et al., 2007). The increased risk for the acoustic neuroma and glioma observed were mostly high-grade glioma and highest for ipsilateral exposure (Hardell et al., 2007).

In this current study, for thyroid cancer risk, cell phone use did not statistically predict thyroid cancer outcomes for either individuals who received all/almost all calls on cell phones compared to individuals who received no/very few calls on cell phones $\beta = 0.357$, $W(1) = 0.831$, $OR = 1.429$, $p = 0.362$, 95% CI [0.663, 3.082]. Hence, cell phone did not predict thyroid cancer in this study participants among those who received ‘some’ calls on cell phones in comparison to individuals who received no/very few calls on cell

phones *β = 0.427, W(1) = 1.784, OR = 1.548, p = 0.182, 95% CI [0.815, 2.938]* (See Table 29 for more detail). The assessment of thyroid cancer prevalence between the exposed groups was determined using the prevalence formula [*Prevalence = Number of cases/Total population size*]. Based on the formula computation, the prevalence of thyroid cancer for individuals who received all/almost all calls on cell phones (PU1 category) was 5.68 per 1000. The prevalence of thyroid cancer among individuals who received some calls on cell phones (PU2 category) was 11.36 per 1000, and the prevalence of thyroid cancer among individuals who received no/very few calls on cell phones (PU3 category) was 8.78 per 1000. The prevalence estimation is different and unique compared to the risk assessment observed in other studies because it specifically addressed the risk of thyroid cancer based on only cell phone use exposure and not the overall prevalence of thyroid cancer based on multiple exposure factors.

In another case-control study, Hardell, Mild, Carlberg, and Hallquist (2004) evaluated the association between brain tumors and mobile/cordless telephone use. The estimated OR in the study were 1.31 and 1.65 for brain tumors and ipsilateral use respectively (Hardell et al., 2004). Individuals between the age of 20-29 years had the highest risk (*OR = 5.91*) for ipsilateral use of analog cell phones (Hardell et al., 2004). The highest risk for brain tumor was associated with a 5-year or greater latency period in the 20-29 year age group for analog cell phone use, with an *OR* value of *8.17*, while individuals with cordless phones had an *OR* value of *4.30* (Hardell et al., 2004).

Myung, Ju, McDonnell, Lee, Kazinets, and Cheng (2009) used a case-control design and meta-analysis to demonstrate the association between cell phone use and risk

of tumors. They observed a positive association (harmful effect) in a random-effects meta-analysis of eight studies using a blinding approach, while a negative association (protective effect) was observed in a fixed-effects meta-analysis of 15 studies without a blinding approach (Myung et al., 2009). Long-term use of a cell phone for 10 years or longer was associated with a risk of tumors in 13 studies (*OR = 1.18*) (Myung et al., 2009). Myung et al. concluded that the studies provided possible evidence of cell phone use and increased risk of tumors. The OR estimation (1.18) Myung et al. (2009) reported was lower than the estimated OR (*1.4* and *1.5*) for the thyroid cancer outcomes observed in this study.

Similarly, Kan, Simonsen, Lyon, and Kestle (2008) demonstrated the association between brain tumors and cell phone use via a meta-analysis evaluation. The pooled OR for long-term users (i.e., ≥ 10 years) in the five studies analyzed using a random-effects model was 1.25 (Kan et al., 2008). The OR estimation was lower than the risk rate calculated in this current study. Kan et al. (2008) suggested that the potential elevated risk of brain cancers post long-term cell phone use exposures should be evaluated further. Assessment of mouth/tongue/lip cancer prevalence among cell phone users were evaluated to address the second research question and hypothesis. The second research question and hypothesis focused on the difference in the prevalence of mouth/tongue/lip cancer between individuals who received all/almost all calls on cell phones and those who received no/very few calls on cell phones or some calls on cell phones. Based on the findings, it was determined that cell phone use did not statistically predict mouth/tongue/lip cancer for individuals who received all/almost all calls on cell phones

when compared to individuals that received no/very few calls on cell phones, *β = 1.195, W(1) = 1.705, OR = 3.303, p = 0.192, 95% CI [0.550, 19.849]*, or for those that received 'some' calls on cell phones in comparison to individuals who received no/very few calls on cell phones *β = 1.089, W(1) = 1.688, OR = 2.971, p = 0.194, 95% CI [0.575, 15.360]* (see Table 46 for more detail). Even when there is no statistical difference in mouth/tongue/lip cancer outcomes between the exposure groups (PU1 vs PU3 and PU2 vs PU3 groups), the odds ratio for mouth/tongue/lip cancer outcome among individuals who received all/almost all calls on cell phones compared to individuals who received no/very few calls on cell phones was 3.3. Suggesting that it is 3.3 times more likely for individuals who received all/almost all calls on cell phones to develop mouth/tongue/lip cancer compared to individuals who received no/very few calls on cell phones. Similarly, the odds ratio for mouth/tongue/lip cancer among individuals who received some calls on cell phones compared to individuals who received no/very few calls on cell phones was 3.0. It also suggested that it is 3.0 times more likely for individuals who received some calls on cell phones to develop mouth/tongue/lip cancer compared to individuals who received no/very few calls on cell phones. Similarly, the prevalence estimation is different from the risk assessment observed in other studies perhaps because it uniquely addressed the risk of mouth/tongue/lip cancer upon repeated cell phone use or exposure rather than exploring multiple predictor factors. See Table 46 for more details.

Also, mouth/tongue/lip cancer prevalence estimation between the exposed groups (PU1, PU2, and PU3 groups) were determined [*Prevalence = Number of cases/Total population size*]. The prevalence rate of mouth/tongue/lip cancer among individuals who

received all/almost all calls on cell phones (PU1) was 1.55 per 1000. For the same health outcome, among individuals who received some calls on cell phones (PU2) it was 2.58 per 1000. While among individuals who received no/very few calls on cell phones (PU3) it was 1.03 per 1000.

The current study risk estimation is similar to the observations made by Lönn, Ahlbom, Hall, and Feychting (2004) on the effects of cell phone-driven radiofrequency exposure of the auditory nerve or tissue closest to the handheld cell phones using a case-control design. In the study, short-term cell phone use did not predict an increased risk of acoustic neuroma (Lönn, Ahlbom, Hall, & Feychting, 2004). However, cell phone use or exposure of at least 10 years showed an increased risk of acoustic neuroma (Lönn et al., 2004). The estimated OR for the acoustic neuroma among low cell phone users was 1.0 (Lönn et al., 2004). Individuals with at least 10 years exposure to cell phone-driven radiation or mobile phone use produced increased relative risk value of 1.9 (Lönn et al., 2004); while an ipsilateral use estimation yielded relative risk value of 3.9 (Lönn et al., 2004).

Hardell, Carlberg, and Mild (2006) demonstrated the association between cell phones use and cordless telephones, and the risk of malignant brain cancer. Using a case-control design, the authors explored similar findings that showed an increased risk of brain cancer among participants with long-term exposure to cell phone use or cordless phone use (>10 years) (Hardell, Carlberg, & Mild, 2006). The researchers showed that cumulative lifetime use (>2,000 hours) of analog cell phones produced an OR of 5.9, a value 3.7 for digital cell phone, and 2.3 for cordless phones (Hardell et al., 2006). They

concluded that ipsilateral exposure increased the risk for malignant brain cancers either for the analog cell phone, OR=2.1 or digital cell phone, OR=1.8 or cordless phone, OR=1.7 (Hardell et al., 2006). They also showed that individuals who used analog cell phones for latency period > 10 years, developed high-grade astrocytoma at an OR rate of 2.7, while digital phone produced an OR value of 3.8, and cordless phone yielded an OR value of 2.2 (Hardell et al., 2006). The multivariate estimation for all phone types showed an increased risk (Hardell et al., 2006). Malignant brain cancer estimation for subjects (<20 years of age) with first use was higher than observed in older individuals with an OR value of 3.7 for digital phones and an OR value of 2.1 for cordless phones (Hardell et al., 2006).

Hours, Bernard, Montetrucq, Arslan, Bergeret, Deltour, and Cardis (2007) demonstrated a contrast finding, yet indicated possible similarities to the current study. The authors evaluated the association between cell phone use and the brain tumor, central nervous system, gliomas, meningiomas, and neuromas of the cranial nerves in 13 countries using a case-control design (Hours, Bernard, Montetrucq, Arslan, Bergeret, Deltour, & Cardis, 2007). They determined that regular cell phone use was not associated with an increased risk of glioma (*OR=1.15*) or meningioma (*OR=0.74*) or neuroma (*OR = 0.92*) (Hours et al., 2007). However, they observed the tendency for an increased risk of glioma among heaviest users (Hours et al., 2007). The observation included individuals who received calls from cell phones often or almost all the times, which interestingly, was the basis of this dissertation. These users were classified as

long-term, heavy, and individuals with the largest numbers of telephones (Hours et al., 2007).

Beyond cancers, Divan, Kheifets, Obel, and Olsen (2008) estimated the risk of prenatal and postnatal exposure to cell phone use or cell phone-driven radiofrequency radiation and behavioral problems in young children. The authors observed high odds ratio (*OR = 1.8*) for behavioral problems (emotional and hyperactivity problems) among children who had prenatal or postnatal exposure to cell phone use or cell phone-driven RFR exposure (Divan, Kheifets, Obel, & Olsen, 2008). They concluded that due to the widespread use of cell phone technology, the observed phenomenon is a public health concern (Divan et al., 2008). The findings in this study on OR estimation of cancers and heart disease among cell phone users similarly reflected an increased risk of behavioral problems observed among young children upon exposure to cell phone-driven radiation.

The third research question addressed in this study was whether there was any difference in the prevalence of heart disease among individuals with the condition based on prolonged cell phone use or cell phone-driven RFR exposure. Based on the findings, cell phone use statistically predicted heart disease. The difference in the rate of heart disease rate between individuals who received all/almost all calls on cell phones in comparison to individuals who received no/very few calls on cell phones was predictive, $\beta = 0.782, W(1) = 117.054, OR = 2.187, ***p < 0.001, 95\%\ CI\ [1.898, 2.520]$. The estimated beta value analysis is positive; therefore, there was a positive association between high cell phone use (PU1 group) and heart disease when compared to the control group (individuals who received no/very few calls on cell phones). The positive

association is statistically significant. Similarly, the difference in the rate of heart disease between individuals who received some calls on cell phones and those who received no/very few calls on cell phones was predicted by the level of cell phone use, $\beta = 0.562$, $W(1) = 79.446$, $OR = 1.755$, $***p < 0.001$, 95% CI [1.551, 1.986]. Since the beta value estimate is also positive (0.562), heart disease outcomes are positively association with cell phone use (PU2 group). The positive association is also statistically significant, a finding similar to the 2016 NTP study.

The assessment of heart disease prevalence between the exposed groups was determined using *[Prevalence = Number of cases/Total population size]* was estimated. The prevalence of heart disease for individuals who received all/almost all calls on cell phones (PU1 group) was estimated at the rate of 19.58 per 1000. The estimated prevalence of heart condition for individuals who received some calls on cell phones (PU2 group) was 31.92 per 1000. On the other hand, the estimated prevalence of heart disease/condition for individuals who received no/very few calls on cell phones (PU3 group) was 36.48 per 1000. The heart disease prevalence estimation in this study is similar to the risk assessment observed in many studies including the NTP 2016 experiment conducted in rats and mice. This current study uniquely addressed the risk of mouth/tongue/lip cancer in humans based on only on cell phone use exposure rather than the overall prevalence of thyroid cancer from multiple exposure factors.

The current findings regarding the effects of cell phone-driven RFR exposure to heart supported the observations described in the NTP 2016 experimental study on cell phone radiation effects on the heart, mice and rats. The NTP (2016) study demonstrated

that cell phone-driven RFR exposure was associated with Schwannoma and heart lesions in mice and rats. The IARC classified RF-EMF range 30 kHz-300 GHz as 'possibly' carcinogenic to humans (IARC, 2011; O'Neill et al., 2011). O'Neill et al. (2011) reported that many researchers had demonstrated that cell phone-driven RFR is associated with sperm damage, impairment of female fertility, and damage to unborn fetus. Genotoxic effects reported in the literature about cell phone exposure were sufficient evidence-based the assessment to promote future research on long-term health impact of cell phone use or cell phone-driven RFR exposure (O'Neill et al., 2011). According to O'Neill et al. (2011) account, cell phone-driven RFR exposure could damage the blood-brain barrier and reduce the melatonin levels in humans after 30 minutes of exposure.

In the current study, I demonstrated the inter-link between factors considered as social determinants of health which are elemental to the social, ecological theory and possible health outcomes. As described earlier in Chapter 1 of this dissertation, the fundamental operational elements of the social-ecological model are the micro-, meso- or exo- or macro-levels of individual or social constructs (Bronfenbrenner, 1979; 1986; 1994; 1995). These unique operational constructs used in the social, ecological model constitute the interactive features of the extrinsic and intrinsic elements of exposure (e.g. cell phone use) or event or outcome (e.g. thyroid cancer, mouth/tongue/lip cancer, and heart condition/disease). Cell phone use is currently a social trend that advances individual way of life either in the micro or meso- or exo or macro levels of the social-ecological model. The use of cell phone or reception of calls consequently interacts with

the intrinsic factors including the biological compositions of an individual and other social cues that may lead to increased risk of undesired adverse health outcomes.

Limitations of the Study

One of the limitations of the current study is that it is not possible to differentiate individuals who use headphones or cell phone speakers while receiving calls. The use of a built-in speakerphone or headphone while receiving calls minimizes direct cell phone-driven RFR exposure to the head area or heart area. Otherwise, such RFR exposure reduction would not be possible when a cell phone is placed directly close to the ear area when receiving a call. I could not differentiate individuals who use Bluetooth from the secondary data set. Bluetooth emits and transmits radio waves which also produces RFR. Unfortunately, the secondary data set did not contain measurements on either headphone, speakerphone or Bluetooth use.

This is a key factor in this study because lack of accurate measurement of the exposure level could produce an unreliable result and perhaps, confound or even bias the findings. As a result, a Type I (False Positive) or Type II (False Negative) result is likely. Due to lack of accurate measurement of direct exposure of RFR exposure to the head and heart area due to the use of a non-hand free device other than Bluetooth when receiving calls from cell phones, the internal and external validity of the study on the level of exposure may have been compromised. Therefore, the findings cannot be generalized to other settings or populations outside of this study target. Also, the study could only infer a correlational association and not a causal relationship because a cross-sectional research design was used in the study and not an experimental design.

There were a large number of missing data due to incomplete responses which may have compromised the reliability of the research findings. A large number of missing data may have led to an unintentional selection bias, which could have inherently produce either a Type I (False positive) or Type II (False Negative) result. When there is a large number of missing data or non-responses, the statistical power of the study is compromised. However, increasing the sample size to at least 80% is the standard recommended to minimize a Type I or II error.

The use of secondary data is a major limitation in this study because the primary purpose of the 2012 NHIS data may not adequately and sufficiently represent the primary purpose of the current study. Therefore, the key variables were not initially operationalized and tailored to the current study. The contents of some of the variables for this current research questions may have been measured or recorded in the dataset as an overview or demographic or supplementary information. For instance, if the 2012 NHIS data was specifically tailored to gather information on cell phone use or cell driven-RFR exposure and its effects on thyroid cancer, mouth/tongue/lip cancer, and heart conditions, the inquirers would likely collect information on the health outcomes' familial history and also use of Bluetooth, speakerphone, headphones (hand free device), and a non-hand free cell phone use. Unfortunately, in the 2012 NHIS dataset, critical information was not recorded or reported. Therefore, the totality of the integrity of the measured variables did not represent core elements of the research or tailored specifically to this current study as it should have if the research setting were conducted as a primary data approach rather than the use of secondary data.

Recommendations

The findings advanced in this study demonstrates the need for tailored research on exploring the health impacts of long-term exposure to non-hand free cell phone use or cell phone-driven RFR exposure. Even when there was no statistically significant link between thyroid cancer or mouth/tongue/lip cancer to the level of cell phone calls received, the effect size or magnitude of the effect estimation represented as an odds ratio was greater than 1, which indicates a potential risk of the exposure. The totality of the magnitude of the effect of the health outcomes and population health issues could be of essence to public health concerns. Furthermore, level of cell phone use exposure was shown in the current study to be positively associated with heart disease. To ensure that the estimated finding was reliable and did not occur due to Type I or Type II error, a tailored prospective cohort study is necessary to demonstrate if in fact high cell phone use or cell phone-driven RFR exposure is positively correlated to heart disease.

To advance meaningful conclusions on the biological effects of cell phone use (non-hand-free cell phone use exposure), a clear temporality sequence on the exposure relative to the onset of the health outcomes in question must be established in a study setting. Without such clarity, any possible association between cell phone use and the consequential health outcomes will be questionable. Unfortunately, this particular study did not demonstrate a clear spatiotemporal sequence of cell phone exposure and the health outcomes under investigation. This is the case because a cross-sectional research design was used in this study and it is inherently flawed and limited in establishing a clear spatiotemporal on exposure-health outcome sequence.

The adverse health effects of cell phone use should be rigorously evaluated in other locations and target population including children and teenagers. The risk of health outcomes among individuals that received calls on cell phones via a hand-free device or wired headphone should be compared to those without any hand-free device. Also, the risk of health outcomes among those that receive calls on cell phones via a Bluetooth should be evaluated compared to individuals who do not use Bluetooth or any hand-free accessories. Lifestyle factors such as education, and life course perspective or familial history of cancer, and heart disease should be considered in future studies.

Implications

The implications that could be advanced or explored from the findings of this study are invaluable in promoting health promotions measures associated with health-related effects of cell phone-driven RFR exposures. A meaningful advancement of effective health promotion measures or efforts could advance means for positive social change at the individual, family, organizational, and societal or policy or governmental levels. Cell phone-driven RFR is a preventable and correctable exposure. As well as the health and behavioral outcomes associated with it. With both formal and informal knowledge of the potential adverse effects of the exposure, individuals could make better-informed decisions on the safe use of the cell phone that would minimize their risk of RFR exposure. Cell phone companies could effectively implement knowledge from the evidence-based findings to develop safe cell phones that emit and transmit less RFR. Promotion awareness on the use of safe cell phone accessories such as cord-linked headphones, or non-radio wave-based headphones recommended guidelines by public

health professionals and cell phone manufacturers would be very helpful to consumer education and safety.

Indiscriminative exposure risks such as cell phone radiation that threatens the entire US population and global cell phone users regardless of age, gender, race, socio-economic status, etc. is a public health priority that requires increased attention. For this reason, policymakers should be in the position to act given the available research evidence on this issue. Such action may require the implementation of strict regulations limiting the levels of cell phone-driven RFR emission. In other words, regulatory guidelines are needed to monitor industrial adherence to RFR emission capability or miniaturization of RFR exposures.

The 2012 NHIS research was a cross-sectional design. Hence, only correlational associations could be drawn from this study. The objective nature of the current research questions which implicated the estimation of the difference in the prevalence of thyroid cancer, mouth/tongue/lip cancer, and heart disease could only be explained using a quantitative research method as demonstrated in this research setting. Similarly, the application of the social-ecological theory as the basis of the theoretical concept or/and the empirical aspects of the study was used on the basis that there is an interlink between intrinsic and extrinsic factors for the disease outcome onset. In this study, the intrinsic factors represented both the biological and personal effects of cell phone use or cell phone-driven RFR exposures while the extrinsic factors represented the societal norms and culture that facilitates the use of cell phone as the primary source of communication on a daily basis for either personal or business needs. Cell phone use or cell phone-

driven communication, on the global level, is a trending way of life and societal culture aimed to advance individual, organizational, and governmental needs.

Conclusion

Cell phone use or cell phone-driven RFR exposure is an increasingly adopted lifestyle behavior not just among the US population but globally. The adverse health outcomes associated with the societal norm of cell phone use if not minimized or controlled could lead to global health crises. Thyroid cancer, mouth/tongue/lip cancer, and heart disease are common health outcomes suggested to be associated with long-term cell phone use or cell phone-driven RFR exposure. In this current study, results showed that cell phone use/cell phone-driven RFR exposure was significantly associated with cancer outcomes, but it did not show that cell phone use or cell phone-driven RFR exposure was significantly linked to thyroid cancer or mouth/tongue/lip cancer. The evidence provided in the study suggests that the magnitude of effects in terms of odds ratio risk of thyroid cancer and mouth/tongue/lip cancer among individuals who are heavy cell phone users is two or three times higher than individuals who do not receive or rarely receive calls on cell phones respectively, see Table 22 and 47. The combined prevalence (PU1 & 2 categories) for thyroid cancer or mouth/tongue/lip cancer among cell phone users are higher, 17.05 per 1000 or 4.13 respectively, compared to the prevalence of none/rare cell phone users (PU3), 8.78 per 1000 or 1.03 per 1000 respectively.

Similarly, the combined prevalence (PU1 & 2 categories) of heart disease was 51.5 per 1000 for high cell phone users, which is almost twice that of the prevalence of

heart disease among none/rare cell phone users (PU3 category), which is 36.48 per 1000. Cell phone use significantly predicts heart disease, though, the predictive nature is a negative association, see Table 50. The odds ratio however for this effect, is very small, see Table 50. Based on these findings, there is enough evidence to suggest that cell phone use is a potential threat to some health outcomes and deserves further long-term prospective cohort or experimental studies to ascertain the validity of the proposed claim.

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