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Use of Mobile and Cordless Phones and the Association with Prostate Cancer

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Abstract

Exposure to radiofrequency (RF) radiation in the frequency range 30 kHz–300 GHz was in 2011 evaluated by the International Agency for Research on Cancer (IARC) at the World Health Organization (WHO) to be a 'possible' human carcinogen, Group 2B. This was based on epidemiological results on increased risk for glioma and acoustic neuroma. Results on other cancer types are sparse. An increased incidence in male rats of proliferative lesions in the prostate gland induced by RF radiation was found in the US NTP study. Thus, it was pertinent to study an association with prostate cancer in human studies. We analyzed data in two of our previous studies, one on brain tumors (only deceased subjects; those who died from prostate cancer were defined as cases) and another on prostate cancer (living subjects) that included similar questions on use of mobile phones or cordless phones. The pooled analysis gave for mobile phone use OR = 1.8, 95 % CI = 1.01-3.1, increasing in the >10 year latency group to OR = 2.8, 95 % CI = 1.5-5.3. Also, use of the cordless phone gave increased risk, although not statistically significant. Dose-response analysis gave highest risk for >2,000 h use of the mobile phone with OR = 2.4, 95 % CI = 1.2-5.1. The cordless phone yielded highest risk in the group 1001-2000 h with OR = 2.3, 95 % CI = 1.01-5.4. Lower OR was seen for use > 2,000 h but based on low numbers. Higher risk was seen in cases with more aggressive cancer based on Gleason score, PSA, and high risk profile, and among subjects with heredity for prostate cancer.

Keywords: Mobile phone, cordless phone, prostate cancer, risk factors

Introduction

The carcinogenic potential of radiofrequency (RF) radiation has been discussed during a long time (1). The brain is the most exposed human organ during the use of the handheld wireless phone; both mobile phone and cordless phone (DECT). An increased risk for glioma and acoustic neuroma has been shown in epidemiological studies, for overview see (2,3). The International Agency for Research on Cancer (IARC) at the World Health Organization (WHO) classified in May 2011 RF radiation in the frequency range 30 kHz–300 GHz as a 'possible' human carcinogen, Group 2B based on e.g. the epidemiological findings on brain tumour risk (4,5).After the IARC evaluation the RF radiation carcinogenesis has been strengthened by the Ramazzini Institute study in Italy on rats (6). A statistically significant increase in the incidence of malignant Schwannoma in the heart was found in male rats at the highest dose, 50 V/m, corresponding to 0.66 mW/cm^2 and a whole body SAR of 0.1 W/Kg. Increased incidence of heart Schwann cells hyperplasia was observed in treated male and female rats at the highest dose (50 V/m), but was not statistically significant. In treated female rats at the highest dose (50 V/m) the incidence of malignant glial tumors was increased,

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although not statistically significant. The study showed increased incidence of tumor types similar to those in human epidemiology studies associated with use of wireless phones; glioma and acoustic neuroma.

The NTP study from USA gave additional confirmation of the carcinogenicity of RF radiation (7,8). An increased incidence of malignant schwannoma in the heart and brain glioma in male rats exposed either to GSM-modulated or CDMA modulated cell phone RF radiation for two years was found. There were also increased incidences of some other tumor types and diseases (9). Of special interest in this context was the increased incidence in male rats of proliferative lesions (neoplasms and/or preneoplastic epithelial hyperplasia) in the prostate gland induced by GSM- and CDMA-modulated cell phone RF radiation. The review panel concluded that there was equivocal evidence of prostate carcinogenicity (11 yes votes, 0 no vote). Equivocal evidence of carcinogenic activity is interpreted as showing a marginal increase of neoplasms that may be test-agent related (7-10). Although the definitions typically are applied to chemical agents, NTP also uses them for physical agents like cell phone radiation. The Nordic countries were among the first in the world to widely adopt wireless telecommunications technology. Analogue phones (NMT, Nordic Mobile Telephone System) were introduced in the early 1980s using both 450 and 900 Megahertz (MHz) frequencies. NMT 450 was used in Sweden from 1981, but closed down on 31 December, 2007; NMT 900 operated during 1986-2000.

The digital system (GSM, Global System for Mobile Communication) using dual band, 900 and 1,800 MHz, started to operate in 1991. The third generation of mobile phones, 3G or UMTS (Universal Mobile Telecommunication System), using 1,900/2,100 MHz RF fields was introduced worldwide, in Sweden in 2003. The fourth generation, 4G established in Sweden in 2009/2010 operating at 800/2,600 MHz, and Trunked Radio Communication (TETRA 380-400 MHz) were introduced in Sweden stepwise between 2005 and 2010. Currently the fifth generation, 5G, is implemented in Sweden as well as in many other countries since 2019/2020. In this article we present results for RF radiation exposure and the risk for prostate cancer based on two previous case-control studies. The results on the risk of prostate cancer have not been published previously. In the first case-control study we investigated use of mobile and cordless phones and the risk for malignant brain tumors among deceased patients using deceased persons as the control group (11). The inclusion period was 1997-2003.

In the second study we investigated perfluoroalkyl substances (PFAS), environmental contaminants, and the association with prostate cancer (12). The inclusion period was 2007-2011. An increased risk was found for certain PFAS in the case group with heredity as a risk factor for prostate cancer. Also use of wireless phones was assessed. Prostate cancer is the most common malignant disease in Sweden with 12,066 new cases in 2022 constituting 30.9 % of all male cancer (13). Risk factors are numerous and heterogeneous. They include genetic, inflammatory and infectious, androgen-related, dietary, age-related, and ethnic factors that contribute to prostate cancer susceptibility (14).

Materials and Methods

Study I (1997-2003)

The first study in this article included patients with histopathological confirmed brain tumour diagnosed during 1997-2003 and aged 20-80 years (11). They were all deceased. Controls were drawn from the Swedish Death Register, one group of control subjects that had died from other types of malignant diseases than brain tumour and another group that had died from other diseases than cancer, for further details see (11). Next of kin to both cases and controls were identified from the Swedish Population Registry and the Swedish Tax Agency. Exposure to various agents was assessed through mailed questionnaire to the closest relative; wife, husband, child, parent, sibling or other relative. This study was based on patients in the control group that had died from prostate cancer (defined as cases in this study) and subjects that had died in other diseases than cancer (defined as controls in this study). This part included use of analogue and digital 2G mobile phones, and also DECT. The control group consisted of 619 deceased subjects; 67 % response rate. In this control group 51 were deceased with prostate cancer and 150 men that had died in another disease than cancer.

Study II (2007-2011)

Patients with prostate cancer admitted to the Department of Oncology, University Hospital at Örebro, Sweden during 2007-2011 were recruited for a study on persistent organic pollutants (12). They were asked to give a blood sample for chemical analysis. Blood was drawn before any treatment with cytostatic drugs or radiotherapy. Of the 252 consecutive patients 200 (79 %) participated. To each case one population-based control matched on age (date of birth) and geographical area (Örebro County) was selected from the Swedish population register. Of these, 93 subjects that did not want to participate were replaced. Two control subjects that turned out to have prostate cancer were included in the case group. The final control group consisted of 186 (54 %) participating controls and 202 participating cases with prostate cancer.

This study included 51 subjects from study I and 202 subjects from study II with prostate cancer; 253 in total. The control persons included 150 persons from study I and 186 persons from study II; 336 control subjects in total.

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Assessment of exposure

Exposure was assessed using a mailed questionnaire sent to each person, or next of kin. Similar questionnaire was used in both studies. Regarding use of a mobile or cordless phone, the time of average use (min per day) was asked for as well as time period for use of the wireless phone. The technology has changed since the first introduction of mobile phones. The first generation was analogue phones with an output power of 1 W at about 900 MHz and was followed by the 2nd generation GSM phones (2G) with either 900 or 1,800 MHz frequency and with a pulsed output power. The mean output power was of the order of tens of mW. In the 3rd generation phones (3G) the output is more to be characterized as amplitude modulated than pulsed and the output power is of the order of tens of μW. The type of mobile phone was recorded and checked by the prefix for the phone number; 010 for analogue phones and 07 for digital phones (2G, 3G). Some special questions covered the extent of use in a car with an external antenna, and use of a hands-free device, both regarded as no exposure to RF-EMF. Use of cordless desktop phones was covered by similar questions, e.g. years for use, and average daily use. The procedure was conducted without knowledge of the status of the study subject.

Statistical methods

StataSE 12.1 (Stata/SE 12.1 for Windows; StataCorp., College Station TX) was used for the analyses. Odds ratios (OR) and 95% confidence intervals (CI) were calculated using unconditional logistic regression. Subjects that reported no use of mobile or cordless phones constituted the control group. Latency (time from first use) was defined as the year of first use of a wireless phone to the year of diagnosis. The cumulative number of hours of use was calculated (number of years multiplied by average time per year based on use per day). Use in a car with external antenna was disregarded, as was use of a hands-free device. A minimum latency period of ≤1 year of exposure was adopted, less than that was included in the unexposed category. The same year as for diagnosis of each case was used for the corresponding control as the cut-off for exposure accumulation. Note that latency was calculated separately for the used phone type or combination of phones that were analysed. Adjustment was made for age (as a continuous variable), year of diagnosis, and study. Latency was analysed using the periods, >1-5 years, >5-10 years, >10 years. Cumulative use of the different phone types was analysed in two groups based on the median number of hours among the controls, and for 1-1,000, 1,001-2,000, and >2,000 hours of use.

Results

Pooled analysis

In the pooled analysis of both studies, use of mobile phone yielded OR = 1.8, 95 % CI = 1.01-3.1, Table 1. The risk was highest in the longest latency croup, > 10 years yielding OR = 2.8, 95 % CI = 1.5-5.3. Similar results were found for both analogue and digital phones, and wireless phones in total. Also, use of the DECT phone increased the risk, although not statistically significant.

Table 2 gives the results based on two exposure groups, ≤ median number of hours or > median numbers of hours among the controls. In the highest cumulative use, statistically significant increased risk was found for mobile phones with OR = 2.0, 95 % CI = 1.1-3.7. Increased risk was seen both for analogue and digital phones, and for wireless phones in total. Also, DECT gave increased OR, although not statistically significant and of the same magnitude in both exposure groups.

Table 1: Odds ratios (OR) and 95 % confidence intervals (CI) for use of wireless phones in different latency groups. Numbers of exposed cases (Ca) and controls (Co) are given. The results are based on 253 cases and 336 controls in total. Adjustment was made for age at diagnosis, year of diagnosis, and study (deceased or living subjects). Subjects with latency ≤ 1 year were counted as unexposed.

	>1-5 year latency		>5-10 year latency		>10 year latency		Total, >1 year latency	
	Ca/Co	OR, 95 % CI	Ca/Co	OR, 95 % CI	Ca/Co	OR, 95 % CI	Ca/Co	OR, 95 % CI
Analogue	2/4	2.1	5/11	1.5	93/71	2.9	100/86	2.4
		0.3-13		0.5-5.0		1.3-6.3		1.2-4.8
Digital	10/33	0.8	58/66	2.0	122/91	2.5	190/190	1.6
		0.3-1.8		0.99-3.9		1.2-5.0		0.9-2.9
Mobile phone, total	9/33	0.8	39/55	1.8	155/117	2.8	203/205	1.8
		0.3-1.9		0.9-3.6		1.5-5.3		1.01-3.1
DECT	29/33	1.6	58/74	1.3	68/63	1.6	155/170	1.4
		0.7-3.3		0.7-2.5		0.8-3.2		0.8-2.7
Wireless phone	12/33	1.1	33/65	1.2	171/137	2.5	216/235	1.6
		0.5-2.5		0.6-2.4		1.4-4.7		0.95-2.8

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Table 2: Numbers of exposed cases (Ca) and controls (Co), odds ratios (OR), and 95 % confidence intervals (CI) for use of wireless phones. Exposure was divided in two groups of cumulative use in hours (h) based on median number among controls.

	≤ median, controls*			> median, controls*		
	Ca/Co	OR	95 % CI	Ca/Co	OR	95 % CI
Analogue	45/43	2.2	1.1 – 4.6	55/43	2.7	1.3 – 5.6
Digital	87/96	1.5	0.8 – 2.8	103/94	1.8	0.9 – 3.4
Mobile phone, total	97/106	1.7	0.9 – 3.0	106/99	2.0	1.1 – 3.7
DECT	85/86	1.5	0.8 – 2.8	70/84	1.4	0.7 – 2.7
Wireless phone	96/118	1.5	0.9 – 2.7	120/117	1.8	1.00 – 3.3

*Median for controls: Analogue=209 h; Digital=304 h; Mobile phone, total=383 h; DECT=365 h; wireless phone=624 h

Table 3: Odds ratio (OR) and 95 % confidence interval (CI) for cases (Ca) and controls (Co) and use of wireless phones in different groups of cumulative number of hours (h) for use of wireless phones.

	1-1,000 h			1,001-2,000 h			>2,000 h		
	Ca/Co	OR	95 % CI	Ca/Co	OR	95 % CI	Ca/Co	OR	95 % CI
Analogue	87/73	2.4	1.2 – 4.8	9/8	2.6	0.8 – 8.3	4/5	1.8	0.4 – 8.3
Digital	142/143	1.6	0.9 – 2.9	18/21	1.4	0.6 – 3.3	30/26	2.3	0.9 – 4.3
Mobile phone, total	128/145	1.6	0.9 – 2.9	36/29	2.3	1.1 – 4.8	39/31	2.4	1.2 – 5.1
DECT	118/136	1.3	0.7 – 2.5	28/20	2.3	1.01 – 5.4	9/14	1.2	0.4 – 3.3
Wireless phone	117/146	1.5	0.9 – 2.6	43/42	1.8	0.9 – 3.7	56/47	2.2	1.1 – 4.3

Table 3 gives the results for cumulative use in hours of the phone in three exposure groups. The results for analogue phones were based on low numbers in the two groups with highest cumulative use. This reflects the shift to use of digital mobile phones. Thus, for that phone type highest OR was found for use > 2,000 h; OR = 2.0, 95 % CI = 0.9-4.3. A statistically significant risk was seen in that exposure group for all mobile phone use with OR = 2.4, 95 % CI = 1.2-5.1. Results for the DECT phone were based on low numbers in the highest exposure group compared with use 1,001-2,000 h yielding OR = 2.3, 95 % CI = 1.01-5.4. Clearly, for all wireless phone use, highest risk was seen in the group > 2,000 h with OR = 2.2, 95 % CI = 1.1-4.3.

Table 4: Interaction between risk factors for prostate cancer and use of wireless phones. Numbers of exposed cases/controls, odds ratios and 95% confidence inervals are given

Risk Factor	Mobile Phone Cases/Controls	Mobile Phone OR (95% CI)	DECT Cases/ Controls	DECT OR (95% CI)	Wireless Phone Cases/Controls	Wireless Phone OR (95% CI)
Gleason 2-6	62/158	1.1 (0.4-3.2)	49/135	1.0 (0.3-2.9)	65/172	1.0 (0.4-3.0)
Gleason 7-10	113/159	2.1 (0.7-6.0)	89/135	2.1 (0.7-6.2)	119/172	2.0 (0.7-6.0)
PSA ≤10	101/158	1.3 (0.5-3.4)	78/135	1.2 (0.4-3.1)	103/172	1.2 (0.5-3.2)
PSA >10	82/158	2.4 (0.7-8.8)	67/135	2.5 (0.7-9.0)	89/172	2.5 (0.7-8.9)
Low risk	39/158	1.1 (0.3-4.1)	31/135	1.0 (0.3-3.8)	40/172	1.0 (0.3-3.8)
Intermedium risk	76/158	1.8 (0.5-5.7)	54/135	1.5 (0.5-4.9)	78/172	1.7 (0.5-5.4)
High risk	60/158	1.7 (0.5-6.3)	53/135	2.0 (0.5-7.3)	66/172	1.8 (0.5-6.5)
No heredity (unexposed phone)	9/12	-	9/12	-	9/12	-
Heredity (unexposed phone)	1/2	0.6 (0.05-8.3)	1/2	0.7 (0.1-8.8)	1/2	0.7 (0.1-8.7)
No heredity (exposed phone)	144/139	1.4 (0.6-3.5)	113/118	1.3 (0.5-3.4)	152/150	1.4 (0.6-3.4)
Heredity (exposed phone)	39/19	2.8 (0.98-7.8)	32/17	2.7 (0.9-7.9)	40/22	2.5 (0.9-6.0)

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Other risk factors for prostate cancer based on study II

Grading of prostate cancer according to Gleason was lacking for 8 cases. Gleason grade 2-6 gave no increased risk, Table 4. For Gleason grades 7-10 increased ORs were found for all types of wireless phones with OR = 2.0, 95 % CI = 0.7-6.0. Separate analysis of mobile phone and DECT gave similar results. Also, for PSA highest OR was found in the group with most aggressive tumour. Thus PSA >10 gave for wireless phone use OR = 2.5, 95 % CI = 0.7-8.9, see Table 4. The cases were further divided into low risk: PSA $\leq$ 10 and Gleason = 2-6, and intermedium risk: PSA 11-19 and Gleason = 2-6, and high risk: PSA $\geq$20 (regardless of Gleason), or Gleason = 8-10 (regardless of PSA). Use of mobile phones or DECT was not associated with low-risk prostate cancer, Table 4. For intermedium and high-risk prostate cancer increased ORs were calculated.

Interaction between heredity for prostate cancer and use of wireless phones is presented in Table 4. Highest OR was found for both mobile phone use regardless of phone type and for DECT use for subjects with hereditary risk although not statistically significant interaction. Heredity and use of mobile phone gave increased risk of borderline statistical significance with OR = 2.8, 95 % CI = 0.98-7.8. Highest risks were found for all phone types in patients with heredity for prostate cancer. It is noteworthy that the results for the group with no heredity for prostate cancer and unexposed to wireless phones were based on few subjects.

Discussion

This study was based on our two previous case-control investigations on risk factors for cancer. The first study was published in 2010 on use of mobile or cordless phones and the risk for brain tumours (11). It included only deceased subjects diagnosed during 1997-2003, aged 20-80 years. In previous publications on benign brain tumours (15) and malignant brain tumours (16) we published an increased risk for glioma and acoustic neuroma associated with use of cellular phones. The study on only deceased cases with brain tumour was initiated by a suggestion that exclusion of that case group would have biased the results (17). However, an increased risk for malignant brain tumors associated with use of wireless phones was also found in this study on only deceased cases and controls confirming our previous results. The subjects in the control group that had died with prostate cancer were included as cases in this study. The comparison group consisted of subjects that had died of other diseases than cancer. The second study was a case-control study on perfluorinated alkyl acids (PFAAS) and the risk of prostate cancer (12). Only living cases and controls aged 49-79 years were included. The results were based on chemical analysis of blood samples collected during 2007-2011. Blood was drawn at the time of the first visit of the case for treatment of the cancer and a similar time for the control subject. Other factors such as Gleason score, PSA, low, median, or high risk and heredity were assessed. Regarding use of mobile or cordless phones similar questions were used in both studies.

The main finding of this study was an increased risk for prostate cancer associated with use of mobile phones. The risk increased with latency and was highest in the >10 year latency period yielding OR = 2.8, 95 % CI = 1.5-2.3. Also, use of the cordless phone (DECT) gave highest risk in the same latency group, however not statistically significant (OR = 1.6, 95 % CI = 0.8-3.2). These results were based on rather high numbers or exposed cases and controls. Also, cumulative use in hours gave highest OR in the most exposed groups using median number of hours among the controls as cut off. Cumulative use was also divided in three groups yielding highest risk > 2,000 h for mobile phone use, OR = 2.4, 95 % CI = 1.2-5.1. Regarding the DECT phone statistically significant increased risk was obtained for 1,001-2,000 h cumulative use with OR = 2.3, 95 % CI = 1.01-5.4. Lower risk was seen for > 2,000 h cumulative use although based on low numbers.

The material in Study II was also analyzed according to the severity of prostate cancer. Both use of mobile and cordless phone gave OR = 2.1 for the most malignant type of prostate cancer with Gleason score 7-10, although not statistically significant. Similar results were seen for PSA > 10, but not statistically significant. The material was further divided into low risk profile for prostate cancer: PSA$\leq$10 and Gleason=2-6, intermedium risk: PSA<20 and Gleason=7; PSA=11-19 and Gleason=7-6, and high risk: PSA$\geq$20 (independent of Gleason score); Gleason=8-10 (independent of PSA level). Gleason score was not available for eight person that were excluded. No association was seen in the low risk group, whereas higher OR was found in both the intermedium and high risk groups, although not statistically significant. Highest risk was found in the group with heredity for prostate cancer and use of the wireless phone. Thus, mobile phone use yielded OR = 2.8, 95 % CI = 0.98-7.8 and cordless phone OR = 2.7, 95 % CI = 0.9-7.9, although no statistically significant interaction was found.

The literature on RF radiation and prostate cancer is sparse. The results in this study are supported by data from the UK Biobank Study (18). A statistically significant increased risk was found for prostate cancer with hazard ratio (HR) = 1.19, 95 % CI = 1.13-1.25. Length of mobile phone use gave highest risk for >8 years, HR = 1.33, 95 % CI = 1.17-1.52 with a statistically significant trend (p<0.001). The NTP study (7,8) gave support of an increased risk for prostate neoplasia. In conclusion this study showed an increased risk

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for prostate cancer associated with use of mobile or cordless phones. The risk increased with latency and cumulative use in hours. Furthermore, the risk was highest in cases with more aggressive cancer based on Gleason score, PSA, and high risk profile. Highest OR was found among subjects with heredity for prostate cancer.

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Availability of Data and Materials

The information generated and analyzed during the current study is available from the corresponding author on reasonable request.

Author's Contributions

LH and MC contributed to the conception, design and writing of the manuscript. Both authors read and approved the final manuscript.

Ethics Approval

Both studies were approved by the regional ethical committee.

Patient Consent for Publication: Not applicable

Competing Interests

The authors declare that they have no competing interests.

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