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Time trends in mobile phone use and glioma incidence among males in the Nordic Countries, 1979–2016

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ABSTRACT

Introduction: In the Nordic countries, the use of mobile phones increased sharply in the mid-1990s especially among middle-aged men. We investigated time trends in glioma incidence rates (IR) with the perspective to inform about the plausibility of brain tumour risks from mobile phone use reported in some case-control studies.
Methods: We analysed IR of glioma in Denmark, Finland, Norway, and Sweden among men aged 40–69 years, using data from national cancer registries and population statistics during 1979–2016, using log-linear joinpoint analysis. Information on regular mobile phone use and amount of call-time was obtained from major studies of mobile phones in these countries. We compared annual observed incidence with that expected under various risk scenarios to assess which of the reported effect sizes are compatible with the observed IR. The expected numbers of cases were computed accounting for an impact of other factors besides mobile phone use, such as improved cancer registration.
Results: Based on 18,232 glioma cases, IR increased slightly but steadily with a change of 0.1% (95 %CI 0.0%; 0.3%) per year during 1979–2016 among 40–59-year-old men and for ages 60–69, by 0.6 % (95 %CI 0.4; 0.9) annually. The observed IR trends among men aged 40–59 years were incompatible with risk ratios (RR) 1.08 or higher with a 10-year lag, RR $\geq$ 1.2 with 15-year lag and RR $\geq$ 1.5 with 20-year lag. For the age group 60–69 years, corresponding effect sizes RR $\geq$ 1.4, $\geq$2 and $\geq$ 2.5 could be rejected for lag times 10, 15 and 20 years.
Discussion: This study confirms and reinforces the conclusions that no changes in glioma incidence in the Nordic countries have occurred that are consistent with a substantial risk attributable to mobile phone use. This particularly applies to virtually all reported risk increases reported by previous case-control studies with positive findings.

1. Introduction

Public concern remains regarding a possible effect of radiofrequency electromagnetic fields from mobile phone on risk of cancer. Glioma has been a focus of both concern and research, as the head receives most of the exposure from use of mobile phones and malignant brain tumors constitute a serious condition with major health impact as no curative treatment exists.

Only two large cohort studies have evaluated the association between mobile phone use and brain tumour risk, and analyses of glioma risk have specifically been reported. No increased risk of glioma was reported (relative risk (RR) = 0.89 (0.78; 1.02)) for 10 years or more of mobile phone use based on 540 exposed female cases in the UK million women cohort (Schüz et al., 2022). Likewise, no increased glioma risk was reported in the Danish early mobile phone subscribers cohort, with RR = 1.04 (0.85 to 1.26) for men with 10 years or more of mobile phone subscription based on 117 exposed cases, and corresponding RR = 1.04 (0.56; 1.95) for women based on 10 exposed cases

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(Frei et al., 2011).

Nevertheless, some case-control studies have reported elevated odds ratios (OR) for glioma risk related to mobile phone use. In the large Interphone study with 13 countries and 2708 glioma cases, the results were inconsistent, showing a reduced glioma risk for ever use of mobile phones and no gradient with time since start of use, but an increased risk in the highest call-time decile (OR = 1.40, 95% CI 1.03; 1.89 for 1640 h or more of lifetime use compared to non-regular users) (Interphone Study Group, 2010). Analyses of the Nordic subset of the Interphone study reported neither excess risks for subjects who had started using their phone 10 years or more before their glioma diagnosis (OR = 0.95, 95% CI 0.74; 1.23) nor for those having accumulated more than 503 h of use (OR = 0.90, 95% CI 0.73; 1.10)(Lahkola et al., 2007). The case-control studies have used detailed exposure metrics, but a major limitation has been the potential for substantial recall and selection biases (Deltour et al., 2012, Johansen et al., 2017, Roosli et al., 2019). In the Interphone study, the inverse association of glioma risk with ever use of mobile phone (OR = 0.81 95 %CI 0.70; 0.94) was considered biologically implausible and most likely due to selection bias related to the low participation among controls especially among mobile phone non-users, and prodromal symptoms preventing yet to be diagnosed patients from becoming new mobile phone users (Olsson et al., 2019, Vrijheid et al., 2009). The elevated risk restricted to the highest decile of cumulative call-time without any consistent gradient has been attributed to recall bias (Vrijheid et al., 2006).

In Sweden, a series of case-control studies have been conducted and a pooled analysis of the risk for glioma including 1,498 cases and 3,430 controls indicated elevated ORs for mobile phone use with latencies as short as one year and effect size reaching OR 1.5 for 5-year latency (Hardell and Carlberg, 2015). A small French case-control study with 253 glioma cases showed a non-significantly increased OR of 1.24 (95% CI 0.86; 1.77) for ever use of mobile phone and the OR was significantly increased risk for the heaviest users (OR of 2.89, 95% CI 1.41; 5.93 related to 896 h of lifetime use compared to non-users) (Coureau et al., 2014). Analyses of the Canadian subset of the Interphone study also reported elevated risks for subjects who had accumulated 558 lifetime hours of use or more (OR = 2.0, 95% CI 1.2; 3.4) (Momoli et al. 2017).

Validation studies comparing self-reported mobile phone use with objective data from network operators have generally shown modest agreement, at most. They also indicated that recall accuracy appeared to deteriorate over time, with a tendency to overestimation of high use and underestimation of low use, and some differences between cases and controls with more frequent overestimation of use among cases (Aydin et al., 2011a, b, Kiyohara et al., 2016, Pettersson et al., 2015, Toledano et al., 2018).

If a genuine increased risk of gliomas is associated with mobile phone use, it should be reflected in the incidence of the exposed population over time. As practically 100% of the population is nowadays using a mobile phone, this should be detectable in the overall population rates, be first discernible in the population segment that first adopted mobile phone use and expected to be most evident among those with the heaviest exposure. Conversely, an absence of changes in the time trends, after an appropriate latency period, would constitute evidence against such an effect of the exposure under study, given the high prevalence of the exposure.

Cancer registry data provide excellent opportunities for analysing the level and changes in incidence rates of glioma, and surveillance studies have been conducted using data for the Nordic countries (Deltour et al., 2009, Deltour et al., 2012) and elsewhere, e.g. (Karipidis et al., 2018, Villeneuve et al., 2021, Sato et al., 2019). In the Nordic countries, the incidence trends of adult gliomas showed no increase that would parallel the increasing prevalence of mobile phone use with no changes apparent in the long-term time trends, but a slight secular increase starting well before the mobile phone era (Deltour et al., 2012). Most other studies have reported either no increases in the incidence of glioma or malignant brain tumours overall (Villeneuve et al., 2021), or increases unrelated to the changes in popularity and penetration of mobile phone technology in the population of (Karipidis et al., 2018, Sato et al., 2019).

We hypothesized that mobile phone use had increased glioma risk in accordance with the results from some case-control studies in men aged 40 to 69 years in Denmark, Finland, Norway and Sweden. Under this hypothesis, we computed expected number of cases, during the years 1979–2016 based on hypothetical effect sizes, induction periods (latencies or lag times), and adjusted for other factors potentially influencing incidence rate changes. We tested if the expected numbers of cases were statistically compatible with the observed numbers of cases in these population groups over this period to assess the consistency of the published results of those epidemiological studies with the observed time trends.

2. Material

2.1. Data

Numbers of primary gliomas in male patients aged 40 to 69 years at diagnosis in 1979–2016 were obtained from the national cancer registries of Denmark, Finland, Norway and Sweden. We collected nationwide annual data by 5-year age groups from each participating country. Male population sizes at risk for these age groups were acquired from the national population registers for each calendar year.

We included all gliomas defined according to the International Classification of Disease for Oncology version 3, located in the brain (topography code C71), with morphology codes 938–946, excluding mixed neuronal-glial tumours. We used corresponding codes in the International Classification of Diseases version 7 for the early period, when ICD-O codes were not available (See Annex Table 1 for the list of specific morphological codes). The primary analyses were performed on all gliomas combined to allow for changes in reporting practices for subgroups of gliomas.

2.2. Statistical methods

2.2.1. Scenarios of hypothetical mobile phone risk

If the radiofrequency electromagnetic field (RF EMF) emitted by mobile phones caused glioma, the marked increase in prevalence of mobile phone use in the general population over the past decades would eventually result in an increased occurrence of gliomas. To evaluate this issue, we examined the observed numbers of glioma cases and incidence rates among men in the age groups 40–59 years and 60–69 years. Men aged 35–44 years around 1990 were the first to start using mobile phones in the early years of mobile telephony (Cardis et al., 2007). Therefore, this population group had, on average, the longest period of exposure and likely also the highest cumulative exposures. To assess their subsequent glioma incidence after accruing meaningful amounts of exposure and allowing for latency, we should focus on rates when they have reached older ages, say after year 2000. For instance, men aged 35–44 in 1990 were in the ages 60–69 years in 2015.

We developed simple, hypothetical scenarios of glioma risk related to mobile phone use, based on the results of case-control studies reporting increased risks or investigating hypothetical lower risk levels (Table 1). Each of the scenarios comprised an effect size (risk ratio = 1.05, 1.08, 1.1, 1.2, 1.3, 1.4, 1.5, 2.0, or 2.5 (the maximum used as the simulations showed this was already unrealistically high so there was no need to simulate even larger effect sizes)), used as a coefficient for the baseline incidence rate. Each scenario also comprised either an induction period (5, 10, 15 and 20 years) or an effect limited to a subgroup with heavy exposure (those who had accumulated $\geq$ 339 h, $\geq$558 h, $\geq$896 h or $\geq$ 1640 h of mobile phone use). The Universal Mobile Telecommunications System (UMTS; 3rd generation) network was launched in 2003, and the Long Term Evolution (LTE; 4th generation) network was launched in 2009 (https://www.fjarskiptastofa.

Table 1
Selected elevated risks of glioma or of malignant brain tumours reported in the scientific literature.

Identification of reference	Period of case recruitment	Size of case-control study	Exposure definition	OR	95% CI	Label of analysis for cross-reference
Hardell and Carlberg (2015)	1997–1999, 2000–2003, and 2007–2009	1498 malignant brain tumours cases; risk analysis on 1380 glioma cases and 3430 controls	use of mobile phones more than one year	1.3	1.1 to 1.6	1
Hardell and Carlberg (2015)	1997–1999, 2000–2003, and 2007–2009	1498 malignant brain tumours cases; risk analysis on 1380 glioma cases and 3430 controls	10 to 15 years after first using a mobile phone [a]	1.4[a]	1.1 to 1.9	2
Coureau et al. (2014) Cerenat	2004–2006	253 gliomas cases and 892 matched controls	self-reported lifetime cumulative mobile phone conversations ≥ 339 hours	1.78	0.98 to 3.24	3
Coureau et al. (2014) Cerenat	2004–2006	253 gliomas cases and 892 matched controls	self-reported lifetime cumulative mobile phone conversations ≥ 896 hours	2.89	1.41 to 5.93	4
Momoli et al. (2017) (Interphone Canada)	2001–2004	170 gliomas cases and 653 controls	>558 hours of cumulative use	2.0	1.2 to 3.4	5
Interphone international study (13 countries) (2010)	2000–2004	2708 gliomas cases and 2972 matched controls	self-reported lifetime cumulative mobile phone conversations ≥ 1640 hours	1.40	1.03 to 1.89	6

Notes: OR: Odds-ratio; CI: confidence interval. a: higher central estimate of risk reported for persons exposed longer.

is/library/Skrar/Innflutt/PDF/Norr%C3%A6n%20GSM%20sk%C3%BDrsla%20-%20loka%C3%BAtg%C3%A1fa.pdf, https://en.wikipedia.org/wiki/LTE_(telecommunication)). In both networks there are lower output emissions of mobile phones as compared to previous generation (Roser et al., 2015). In view of this, we developed a scenario assuming that the risk was restricted to the time period when people were using mainly the Global System for Mobile Communications (GSM; 2nd generation of mobile phone technology) or the earlier generation: this GSM-only exposure scenario was modelled by having the size of the exposed group not increasing after 2003.

2.2.2. Exposure distributions

Prevalence and amount of mobile phone use (i.e., the exposed population) were estimated using two sources of information. The first one was the control group of the Interphone study, restricted to men from the Nordic countries (N = 997) interviewed about mobile phone use in 2000–2003 at ages 18–63 years (Lahkola et al., 2007). We also used data from the Danish participants of the large prospective Cosmos cohort study (N = 25,907) collected in 2007–2009 (Schuz et al., 2011, Toledano et al., 2018).

From each of these sources, we abstracted the proportion of men aged 40–59 years and 60–69 years who used mobile phones, adjusting for the induction period of the scenario. We also abstracted the proportions of heavy users in the scenarios (with criteria listed above). The structure of the datasets did not cover the use of mobile phone in the distant past for men aged 60–69 years, and those were extrapolated from younger age groups. In the Interphone dataset, for the period 2003–2016, we extrapolated the use in 2002 assuming a linear increase, with the slope becoming weaker in the later years to reflect market saturation. For the Cosmos dataset, information was elicited for past use in the years 1987, 1990, 1995, 2000, 2005, 2007–2009, and conservatively estimated between these time points; it was similarly extrapolated to the periods not included in the data.

2.3. Statistical analysis

Annual percent changes (APC) in incidence rates were estimated using the Join-point regression program, with a log-linear model of the rates (Joinpoint Regression Program, Version 4.8.0.1; Statistical Methodology and Applications Branch, Surveillance Research Program, National Cancer Institute). The expected numbers of cases assuming an effect of mobile phone use were computed for each risk scenario and exposure distribution, also accounting for an impact of other factors, such as improved cancer registration. Standardised incidence ratio (SIR) analyses compared the observed incidence with the expected number of cases for each risk scenario (See annex for details of the statistical analysis). The main analyses used risk scenarios modelling half of the IR increase as attributable to mobile phone use (and the remainder to other factors), and a sensitivity analysis was carried out, in which a quarter of the IR increase was modelled as attributable to mobile phone use. These values were chosen based on the conclusions of previous work, whereby mobile phone would not explain the total increase observed so far (Deltour et al., 2012).

The Poisson distributional assumption was tested and tests showed a good fit of a time-adjusted Poisson model in both age groups, thereby justifying the use of Poisson distributions in the statistical modelling.

2.4. Ethics and data protection

The IARC Ethics committee approved the study protocol (reference 15–39). In Denmark, all data shared for the present project were aggregated data not requiring ethical approval and the Data Protection Agency approved the COSMOS project. In Finland, permission for collecting the data was obtained from the National Institute for Health and Welfare, but no evaluation of an ethical committee was required. In Norway and Sweden, de-identified data (i.e. anonymized) where no link is made with other registries do not need ethical approval, and the project was approved by the Data Delivery Unit at the Cancer Registry of Norway and Sweden, respectively.

3. Results

This study was based on 18,232 male glioma cases diagnosed during 1979–2016 in the Nordic countries (Table 2). The IR of gliomas increased during the study period. In the age group 40–59 years, the male IR of gliomas increased slightly and consistently by an APC = 0.1% per year over 1979–2016 (95% CI 0.0%; 0.3%) with IR reaching 9.25 per 100,000 person-years over the last 10 years. The increase was more pronounced among men in their sixties, with APC = 0.6% per year, reaching 18.34 per 100,000 person-years over the last 10 years. The trends in IR were smooth in all countries (Fig. 1, Fig. 2), except in Sweden, where the incidence increased among men aged 40–59 years in 1979–1984 and decreased thereafter until 1991 (Fig. 1).

3.1. Mobile phone use

We analysed the prevalence of mobile phone use in study populations from two studies (Interphone, Cosmos-Denmark). The prevalence of use and of heavy mobile phone use increased markedly over time (Figs. 3, 4). The prevalence of mobile phone use increased from <

Table 2
Combined and country specific total number of cases, total population at risk, incidence rate in the last 10 years and annual percent change in incidence rates estimated using joinpoint analysis, among men 40–69 years old, by age group in the Nordic countries.

	Men 40–59 years old						
	Cases (total N)	Population at risk (total PY)	IR (2007–2016)	(95 %CI of IR)	Period	APC	(95% CI of APC)
Nordic countries	10,668	117,208,217	9.2	(8.9; 9.6)	1979-2016	0.1	(0.0; 0.3)
Denmark	2,676	26,525,415	9.9	(9.2; 10.7)	1979-2016	0.0	(-0.3; 0.3)
Finland	1,956	26,108,320	8.3	(7.6; 8.9)	1979-2016	0.6	(0.2; 1.0)
Norway	2,015	21,230,877	9.7	(8.9; 10.4)	1979-2016	0.4	(-0.2; 0.9)
Sweden	4,021	43,343,605	9.2	(8.7; 9.8)	1979-1984[A]	7.9	(2.3; 13.8)
					1984[A]-1991[B]	−3.3	(-6.7; 0.4)
					1991[B]-2016	0.1	(-0.3; 0.5)
	Men 60–69 years old						
	Cases (total N)	**Population at risk (total PY)**	**IR (2007–2016)**	**(95 %CI of IR)**	**Period**	**APC**	**(95% CI of APC)**
Nordic countries	7,564	44,624,456	18.4	(17.7; 19.1)	1979-2016	0.6	(0.4; 0.9)
Denmark	1,836	9,941,801	20.0	(18.5; 21.6)	1979-2016	0.5	(0.1; 1.0)
Finland	1,223	9,265,303	14.6	(13.3; 16.0)	1979-2016	0.9	(0.4; 1.4)
Norway	1,441	7,891,072	21.7	(19.9; 23.6)	1979-2016	1.4	(0.9; 1.9)
Sweden	3,064	17,526,280	18.2	(17.1; 19.4)	1979-2016	0.4	(0.0; 0.7)

Notes: PY: person-years; IR: incidence rate; CI: confidence interval; APC: annual percent change.
A: 95 %CI around jointpoint: 1981–1987. B: 95 %CI around joinpoint: 1986–1999.

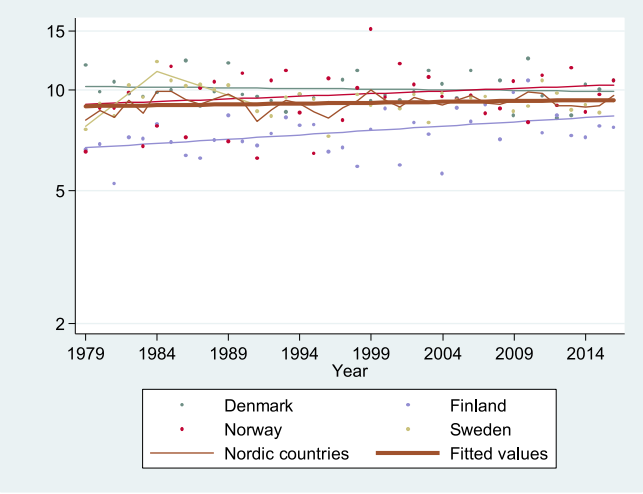

Fig. 1. Observed annual IR of gliomas among 40–59 years old men in Denmark, Finland, Norway and Sweden (dots), and in the combined dataset (thin brown line), and best fitting join-point regression lines.

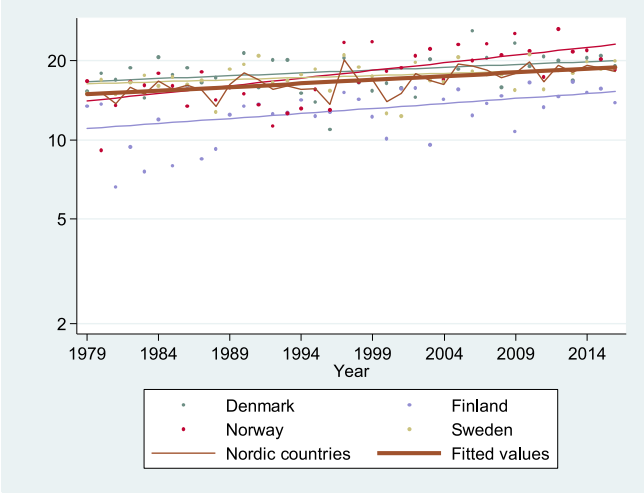

Fig. 2. Observed annual IR of gliomas among 60–69 years old men in Denmark, Finland, Norway and Sweden (dots), and in the combined dataset (thin brown line), and best fitting join-point regression lines.

5% before 1987 to 95% in 2016. The younger age group aged 40–59 years tended to report higher prevalence of heavy use than those aged 60–69 years, especially since the early 2000 s (Fig. 4, appendix Figures). In 2016, the prevalence of 40–59-year-old men having accumulated more than 1640 h of use was higher in the Nordic Interphone controls (49 %) than in the Cosmos-Denmark participants (34%). The differences between the datasets were small in the age group 60–69 years (prevalence of use in 2016: 12% in the Nordic Interphone controls and 12% in the Cosmos Denmark participants).

3.2. Analyses of observed and expected numbers of cases

We predicted the expected number of cases for each risk scenario (given presumed effects sizes and latencies), and compared it to the observed number of gliomas to obtain a SIR, with values <1 indicating fewer observed cases and >1 indicating larger numbers of cases than expected based on the scenario (Table 3). For instance, an SIR of 0.60 was obtained for men aged 40–59 years indicating that the observed number of cases was 60% of the expected, in the scenario where the RR was assumed to be 2.5, the prevalence of use was equal to that of the Nordic Interphone participants, the group at risk was all mobile phone users with a 5-year induction period, and half of the baseline increase was modelled. The confidence interval of the SIR (0.59; 0.62) did not include 1, indicating that the expected number of cases, computed for this scenario, was not statistically compatible with the observed number of cases, hence the scenario with such excess risk attributable to mobile phone use was not consistent with the observed data.

The main analyses showed that among men aged 40–59 years, the observed incidence was incompatible with risk ratios >1.05 with a 5-year latency, >1.08 with 10-year latency, >1.1 with 15 years and >1.4 assuming a 20-year latency (Table 3, Annex Table 2, Fig. 5). Analyses of men aged 60–69 years, in contrast, showed that the scenarios with low risks and long induction times underestimated the observed numbers of gliomas, as an increasing incidence trend was observed in this age group. On the other hand, risk ratios exceeding 1.3 with five and ten-year latencies, as well as 1.5 at 15 years or 2.0 at 20 years could be ruled out as inconsistent with the observed incidence.

The analyses of scenarios where the risk was assumed to be limited to heavy users showed that rate ratios exceeding 1.1 for >339, 558 h of cumulative use, ≥1.2 for 896 h of use, ≥1.3 for 1640 h of use were

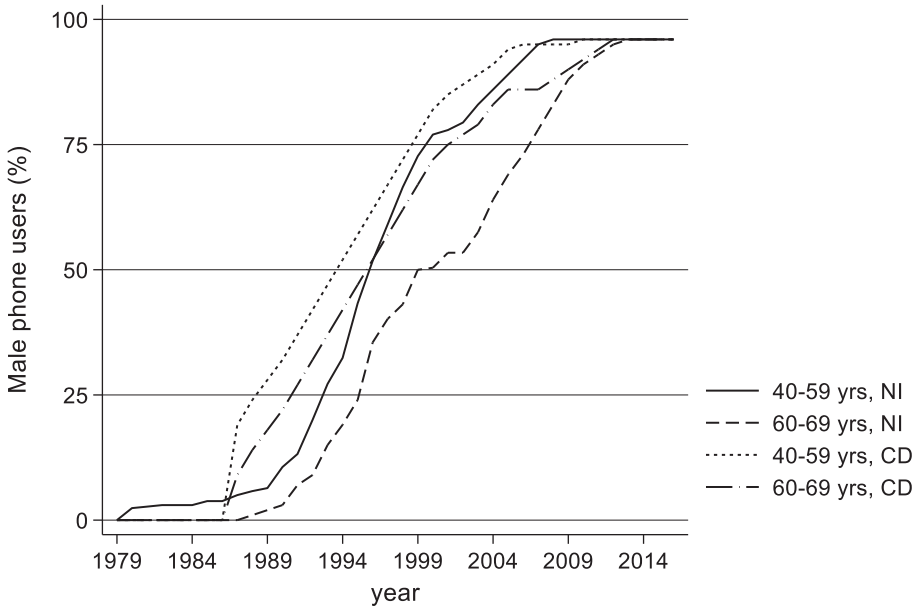

Fig. 3. Proportion of users among men aged 40–59 years old and 60–69 years old in the Nordic Interphone (NI) and in the Cosmos Denmark (CD) studies.

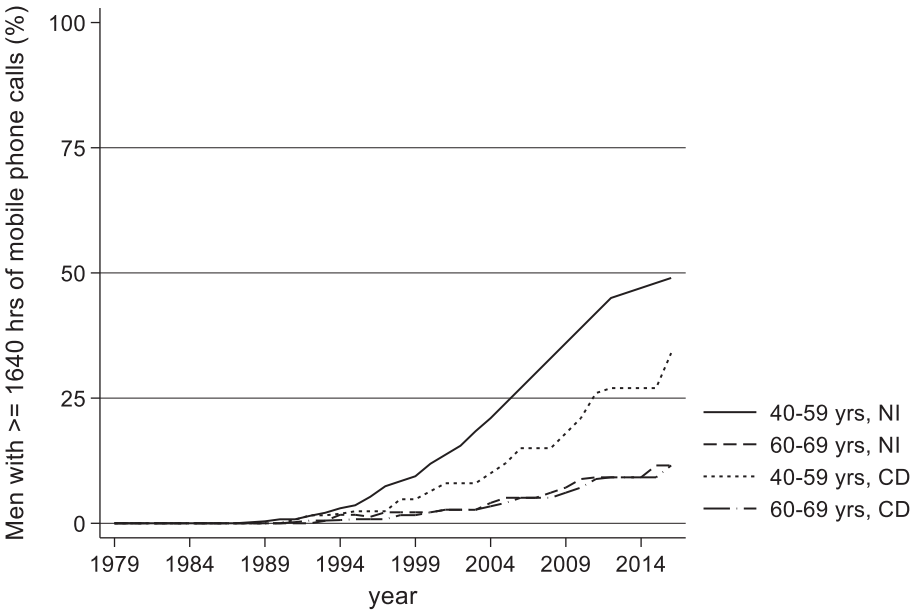

Fig. 4. Proportion of users having accumulated at least 1640 years of mobile phone use, among men aged 40–59 years old and 60–69 years old in the Nordic Interphone (NI) and in the Cosmos Denmark (CD) studies. In the Cosmos questionnaire, responses were elicited using categorical answers (including 30–59 minutes / week, 1–3 hours / week, 4–6 hours /week, >6 hours / week). For this reason, only approximate definition of heavy users could be abstracted and conservative assumptions were used. We assumed that on average, subjects kept the same use between data points and we used the midpoint of the interval as representative of the interval's range of values. For example, subjects who reported that they used their phone 1–3 hours per week in 2000, were assumed to have accumulated 339 hours of use or more by 2002.

inconsistent with the observed incidence rates in the age group 40–59 years (Table 4, Annex Table 3). RRs exceeding 1.5 for heavy users of the GSM technology were also inconsistent with the observed IR of men aged 40–59 years (Annex Table 3). For men aged 60–69 years, only risk ratios exceeding 1.5 for 339 h of use, ≥2.0 for 558 h of use, and ≥2.5 for 896 h were incompatible with the observed incidence, while most of the lower RRs underestimated the observed incidence due to an increasing trend in the background rates.

In the sensitivity analyses, where only a quarter of the increase in incidence rates was attributed to mobile phones, the results for the different latencies investigated were largely comparable to the main analyses: risk scenarios with similar or lower risk ratios than the equivalent scenario in the main analyses were compatible with the observed data (Table 5, Annex Table 4). The results of the sensitivity analyses of the scenarios where the risk was assumed to be limited to heavy users were also largely similar to the main analyses in the age group 40–59 years, while among 60–69-year-old men, lower risks were compatible with the observed IRs in the sensitivity than in the main analyses (Table 6, Annex table 5). Our assessment of risks, based on the main analyses, were therefore likely conservative.

4. Discussion

Our analyses of mobile phone use patterns and incidence trends of glioma in the Nordic countries during 1997–2016 among men aged 40–59 years indicate that the population rates of glioma were not compatible with increased risks from mobile phone use such as those reported in Swedish and French case-control studies, or in general, rate ratios exceeding 1.1 to 1.4 with latencies up to 20 years. For cumulative hours of use, rate ratios higher than 1.2 were not compatible with the observed incidence trends in this age group. For the age group 60–69 years, an increasing underlying trend rendered both very high and very

Table 3
SIR and 95% CI calculated as the ratio of observed numbers of cases to that expected from various risk scenarios with hypothetical risks and lag periods, using the exposure distribution of the Nordic Interphone controls, a model attributing half of the baseline increase in incidence rate to mobile phone use.

	Men 40–59 years old when all mobile phone users were at risk after a lag period							
	5 years lag		**10 years lag**		**15 years lag**		**20 years lag**	
Hypothetical RR	SIR	95% CI	SIR	95% CI	SIR	95% CI	SIR	95 % CI
2.5	0.60	(0.59; 0.62)	0.69	(0.67; 0.70)	0.79	(0.78; 0.81)	0.91	(0.90; 0.93)
2	0.70	(0.69; 0.71)	0.77	(0.76; 0.78)	0.85	(0.84; 0.87)	0.94	(0.93; 0.96)
1.5	0.83	(0.81; 0.84)	0.87	(0.86; 0.89)	0.93	(0.91; 0.94)	0.98	(0.96; 1.00)
1.4	0.86	(0.84; 0.87)	0.90	(0.88; 0.92)	0.94	(0.92; 0.96)	**0.98**	**(0.96; 1.00)**
1.3	0.89	(0.88; 0.91)	0.92	(0.91; 0.94)	0.96	(0.94; 0.98)	**0.99**	**(0.97; 1.01)**
1.2	0.93	(0.91; 0.95)	0.95	(0.93; 0.97)	0.98	(0.96; 0.99)	**1.00**	**(0.98; 1.02)**
1.1	0.97	(0.95; 0.99)	0.98	(0.96; 1.00)	**0.99**	**(0.97; 1.01)**	**1.00**	**(0.99; 1.02)**
1.08	0.98	(0.96; 1.00)	**0.99**	**(0.97; 1.01)**	**1.00**	**(0.98; 1.02)**	**1.01**	**(0.99; 1.03)**
1.05	**0.99**	**(0.97; 1.01)**	**1.00**	**(0.98; 1.02)**	**1.00**	**(0.98; 1.02)**	**1.01**	**(0.99; 1.03)**
	Men 60–69 years old when all mobile phone users were at risk after a lag period							
2.5	0.68	(0.67; 0.70)	0.73	(0.72; 0.75)	0.84	(0.82; 0.86)	0.97	(0.95; 0.99)
2	0.78	(0.76; 0.80)	0.83	(0.81; 0.84)	0.91	(0.89; 0.93)	**1.01**	**(0.99; 1.03)**
1.5	0.91	(0.89; 0.93)	0.94	(0.92; 0.96)	**0.99**	**(0.97; 1.02)**	1.05	(1.03; 1.08)
1.4	0.94	(0.92; 0.97)	0.97	(0.95; 0.99)	**1.01**	**(0.99; 1.04)**	1.06	(1.04; 1.09)
1.3	**0.98**	**(0.96; 1.00)**	**1.00**	**(0.98; 1.02)**	1.03	(1.01; 1.06)	1.07	(1.05; 1.10)
1.2	1.02	(0.99; 1.04)	1.03	(1.01; 1.05)	1.05	(1.03; 1.08)	1.08	(1.06; 1.10)
1.1	1.06	(1.03; 1.08)	1.06	(1.04; 1.09)	1.08	(1.05; 1.10)	1.09	(1.07; 1.11)
1.08	1.06	(1.04; 1.09)	1.07	(1.05; 1.10)	1.08	(1.06; 1.11)	1.09	(1.07; 1.12)
1.05	1.08	(1.05; 1.10)	1.08	(1.06; 1.11)	1.09	(1.06; 1.11)	1.09	(1.07; 1.12)

Notes: RR: relative risk; SIR: standardised incidence ratio; CI: Confidence Interval. In bold, non-significant SIR, which indicated compatibility between the expected values and the observed data.

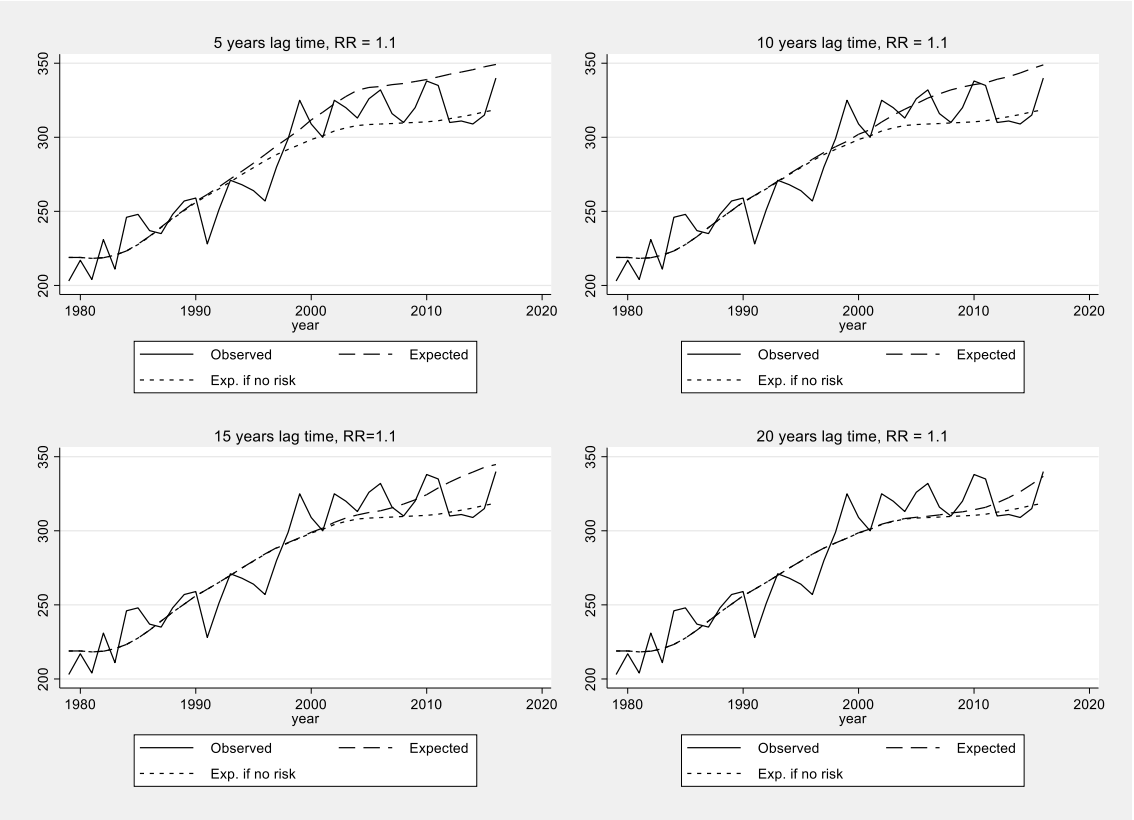

Fig. 5. Graphical illustration of SIR analyses. Graphs of observed numbers of cases and expected number of cases over time when half of the baseline increase is explained by the mobile phone related risk and the other half is unexplained, for scenarios of exposure based on the Nordic Interphone controls male 40–59 years old, with a RR of 1.1, and 5 (top left), 10 (top right), 15 (low left), and 20 (low right) years of lag time. Observed (respectively Expected) indicates the observed (respectively expected) number of cases, and "Exp. if no risk" indicates the expected number of cases if the RR was equal to 1. The expected number of cases were statistically significantly different and higher from the observed number of cases in the upper panel, and were not statistically significantly different in the lower panels. It can be seen that towards the end of the period of observation, the expected number of cases was higher than the observed for the upper panels, while observed and expected aligned better in the lower panels.

Table 4
SIR and 95% CI calculated as the ratio of observed numbers of cases to that expected from various risk scenarios with hypothetical risks for heavy users using the exposure distribution of the Nordic Interphone controls, a model attributing half of the baseline increase in incidence rate to mobile phone use.

	Men 40–59 years old when users having accumulated above threshold lifetime hours of use were at risk									
	>= 339 h		**>= 558 h**		**>= 896 h**		**>= 1640 h**		**>= 1640 h before 2003**	
Hypothetical RR	SIR	95% CI	SIR	95% CI	SIR	95% CI	SIR	95% CI	SIR	95% CI
2.5	0.67	(0.66; 0.69)	0.72	(0.71; 0.73)	0.75	(0.74; 0.77)	0.80	(0.79; 0.82)	0.89	(0.88; 0.91)
2.0	0.76	(0.74; 0.77)	0.80	(0.78; 0.81)	0.82	(0.81; 0.84)	0.86	(0.85; 0.88)	0.93	(0.91; 0.95)
1.5	0.87	(0.85; 0.88)	0.89	(0.87; 0.91)	0.91	(0.89; 0.92)	0.93	(0.91; 0.95)	0.97	(0.95; 0.99)
1.4	0.89	(0.88; 0.91)	0.91	(0.90; 0.93)	0.93	(0.91; 0.94)	0.95	(0.93; 0.96)	**0.98**	**(0.96; 1.00)**
1.3	0.92	(0.90; 0.94)	0.94	(0.92; 0.95)	0.95	(0.93; 0.96)	0.96	(0.94; 0.98)	**0.99**	**(0.97; 1.00)**
1.2	0.95	(0.93; 0.97)	0.96	(0.94; 0.98)	0.97	(0.95; 0.99)	0.98	(0.96; 1.00)	**0.99**	**(0.98; 1.01)**
1.1	0.98	(0.96; 1.00)	**0.99**	**(0.97; 1.00)**	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.98; 1.01)**	**1.00**	**(0.98; 1.02)**
1.08	**0.99**	**(0.97; 1.00)**	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.97; 1.01)**	**1.00**	**(0.98; 1.02)**	**1.00**	**(0.99; 1.02)**
1.05	**1.00**	**(0.98; 1.01)**	**1.00**	**(0.98; 1.02)**	**1.00**	**(0.98; 1.02)**	**1.00**	**(0.98; 1.02)**	**1.01**	**(0.99; 1.03)**
	Men 60–69 years old when users having accumulated above threshold lifetime hours of use were at risk									
2.5	0.89	(0.87; 0.91)	0.94	(0.92; 0.96)	0.98	(0.95; 1.00)	1.04	(1.01; 1.06)	1.07	(1.05; 1.10)
2.0	0.95	(0.93; 0.97)	**0.99**	**(0.97; 1.01)**	**1.01**	**(0.99; 1.04)**	1.06	(1.03; 1.08)	1.08	(1.06; 1.11)
1.5	**1.02**	**(1.00; 1.04)**	1.04	(1.02; 1.06)	1.05	(1.03; 1.08)	1.08	(1.05; 1.10)	1.09	(1.07; 1.11)
1.4	1.03	(1.01; 1.06)	1.05	(1.03; 1.08)	1.06	(1.04; 1.09)	1.08	(1.06; 1.11)	1.09	(1.07; 1.12)
1.3	1.05	(1.03; 1.07)	1.06	(1.04; 1.09)	1.07	(1.05; 1.10)	1.09	(1.06; 1.11)	1.09	(1.07; 1.12)
1.2	1.07	(1.04; 1.09)	1.07	(1.05; 1.10)	1.08	(1.06; 1.11)	1.09	(1.07; 1.12)	1.10	(1.07; 1.12)
1.1	1.08	(1.06; 1.11)	1.09	(1.06; 1.11)	1.09	(1.07; 1.11)	1.09	(1.07; 1.12)	1.10	(1.07; 1.12)
1.08	1.09	(1.06; 1.11)	1.09	(1.06; 1.11)	1.09	(1.07; 1.12)	1.10	(1.07; 1.12)	1.10	(1.07; 1.12)
1.05	1.09	(1.07; 1.12)	1.09	(1.07; 1.12)	1.09	(1.07; 1.12)	1.10	(1.07; 1.12)	1.10	(1.07; 1.12)

Notes: RR: relative risk; SIR Standardised incidence ratio; CI: Confidence Interval; h: hours. In bold, non-significant SIR indicating compatibility of the expected values with the observed data.

Table 5
SIR and 95% CI calculated as the ratio of observed numbers of cases to that expected from various risk scenarios with hypothetical risks and lag periods, using the exposure distribution of the Nordic Interphone controls, attempting to model a quarter of the baseline increase in incidence rate.

	Men 40–59 years old when all mobile phone users were at risk after a lag period							
	5 years lag		**10 years lag**		**15 years lag**		**20 years lag**	
Hypothetical RR	SIR	95% CI	SIR	95% CI	SIR	95% CI	SIR	95% CI
2.5	0.60	(0.59; 0.61)	0.68	(0.67; 0.69)	0.79	(0.77; 0.80)	0.91	(0.89; 0.92)
2.0	0.69	(0.68; 0.71)	0.76	(0.75; 0.78)	0.85	(0.83; 0.86)	0.94	(0.92; 0.95)
1.5	0.82	(0.80; 0.83)	0.87	(0.85; 0.88)	0.92	(0.90; 0.94)	0.97	(0.95; 0.99)
1.4	0.85	(0.83; 0.87)	0.89	(0.87; 0.91)	0.93	(0.92; 0.95)	0.98	(0.96; 0.99)
1.3	0.88	(0.87; 0.90)	0.92	(0.90; 0.93)	0.95	(0.93; 0.97)	**0.98**	**(0.96; 1.00)**
1.2	0.92	(0.90; 0.94)	0.94	(0.93; 0.96)	0.97	(0.95; 0.99)	**0.99**	**(0.97; 1.01)**
1.1	0.96	(0.94; 0.98)	0.97	(0.95; 0.99)	**0.99**	**(0.97; 1.00)**	**1.00**	**(0.98; 1.02)**
1.08	0.97	(0.95; 0.99)	**0.98**	**(0.96; 1.00)**	**0.99**	**(0.97; 1.01)**	**1.00**	**(0.98; 1.02)**
1.05	**0.98**	**(0.96; 1.00)**	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.98; 1.01)**	**1.00**	**(0.98; 1.02)**
	Men 60–69 years old when all mobile phone users were at risk after a lag period							
2.5	0.64	(0.63; 0.66)	0.70	(0.68; 0.71)	0.79	(0.78; 0.81)	0.92	(0.90; 0.95)
2.0	0.74	(0.72; 0.76)	0.78	(0.77; 0.80)	0.86	(0.84; 0.88)	0.96	(0.94; 0.98)
1.5	0.87	(0.85; 0.89)	0.90	(0.88; 0.92)	0.95	(0.93; 0.97)	**1.00**	**(0.98; 1.03)**
1.4	0.90	(0.88; 0.92)	0.92	(0.90; 0.94)	0.97	(0.94; 0.99)	**1.01**	**(0.99; 1.04)**
1.3	0.93	(0.91; 0.95)	0.95	(0.93; 0.97)	**0.99**	**(0.96; 1.01)**	**1.02**	**(1.00; 1.04)**
1.2	0.97	(0.95; 0.99)	**0.98**	**(0.96; 1.00)**	**1.01**	**(0.98; 1.03)**	1.03	(1.01; 1.05)
1.1	**1.01**	**(0.98; 1.03)**	**1.01**	**(0.99; 1.04)**	1.03	(1.00; 1.05)	1.04	(1.02; 1.06)
1.08	**1.01**	**(0.99; 1.04)**	**1.02**	**(1.00; 1.04)**	1.03	(1.01; 1.05)	1.04	(1.02; 1.06)
1.05	1.03	(1.00; 1.05)	1.03	(1.01; 1.05)	1.04	(1.01; 1.06)	1.04	(1.02; 1.07)

Notes: RR: relative risk; SIR Standardised incidence ratio; CI: Confidence Interval. In bold, non-significant SIR indicating compatibility of the expected values with the observed data.

low risks incompatible with the observed data. So far, there has been no evidence that mobile phone related risks would be different between these age groups, and therefore we expected the risk to be similar.

When half of the IR increases were attributed to other factors, none of the scenarios with mobile phone related risks were compatible with the data, while under the assumption that a quarter of these increases were attributable to an effect of mobile phone use, small excess risks (RR = 1.08 applying to all users) or risks after very long latencies (20 years) could not be entirely dismissed. Further work on these scenarios and continued follow up could shed more light on the remaining uncertainties.

Our analyses are based on 18,232 male glioma cases, which occurred in the male population aged 40–69 years in Denmark, Finland, Norway and Sweden with 162 million person-years at risk. Rates of glioma showed a slow and constant increase with no marked changes in the trend in the recent years. We analysed the incidence rates of glioma in the Nordic countries, with very high standards of cancer care including diagnosis, comprehensive public health care, and a long tradition of

Table 6
SIR and 95% CI calculated as the ratio of observed numbers of cases to that expected from various risk scenarios with hypothetical risks for heavy users, using the exposure distribution of the Nordic Interphone controls, attempting to model a quarter of the baseline increase in incidence rate.

	Men 40–59 years old when users having accumulated above threshold lifetime hours of use were at risk									
	>= 339 h		>= 558 h		>= 896 h		>= 1640 h		>= 1640 h before 2003	
Hypothetical RR	SIR	95% CI	SIR	95% CI	SIR	95% CI	SIR	95% CI	SIR	95% CI
2.5	0.67	(0.65; 0.68)	0.71	(0.70; 0.73)	0.75	(0.73; 0.76)	0.80	(0.78; 0.81)	0.89	(0.87; 0.90)
2.0	0.75	(0.74; 0.77)	0.79	(0.77; 0.80)	0.82	(0.80; 0.83)	0.85	(0.84; 0.87)	0.92	(0.91; 0.94)
1.5	0.86	(0.84; 0.88)	0.88	(0.87; 0.90)	0.90	(0.88; 0.92)	0.92	(0.91; 0.94)	0.96	(0.94; 0.98)
1.4	0.88	(0.87; 0.90)	0.90	(0.89; 0.92)	0.92	(0.90; 0.94)	0.94	(0.92; 0.96)	0.97	(0.95; 0.99)
1.3	0.91	(0.89; 0.93)	0.93	(0.91; 0.95)	0.94	(0.92; 0.96)	0.95	(0.94; 0.97)	0.98	(0.96; 1.00)
1.2	0.94	(0.92; 0.96)	0.95	(0.93; 0.97)	0.96	(0.94; 0.98)	0.97	(0.95; 0.99)	**0.99**	**(0.97; 1.00)**
1.1	0.97	(0.95; 0.99)	0.98	(0.96; 1.00)	0.98	(0.96; 1.00)	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.98; 1.01)**
1.08	**0.98**	**(0.96; 1.00)**	**0.98**	**(0.96; 1.00)**	**0.99**	**(0.97; 1.00)**	**0.99**	**(0.97; 1.01)**	**1.00**	**(0.98; 1.02)**
1.05	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.97; 1.01)**	**0.99**	**(0.98; 1.01)**	**1.00**	**(0.98; 1.02)**
	Men 60–69 years old when users having accumulated above threshold lifetime hours of use were at risk									
2.5	0.85	(0.83; 0.87)	0.89	(0.87; 0.91)	0.93	(0.91; 0.95)	**0.99**	**(0.97; 1.01)**	1.02	(1.00; 1.05)
2.0	0.90	(0.88; 0.92)	0.94	(0.92; 0.96)	**0.97**	**(0.94; 0.99)**	1.01	(0.98; 1.03)	1.03	(1.01; 1.05)
1.5	0.97	(0.95; 0.99)	**0.99**	**(0.97; 1.01)**	1.01	(0.98; 1.03)	1.03	(1.00; 1.05)	1.04	(1.02; 1.06)
1.4	**0.99**	**(0.96; 1.01)**	1.00	(0.98; 1.03)	1.01	(0.99; 1.04)	1.03	(1.01; 1.06)	1.04	(1.02; 1.06)
1.3	**1.00**	**(0.98; 1.02)**	1.01	(0.99; 1.04)	1.02	(1.00; 1.05)	1.04	(1.01; 1.06)	1.04	(1.02; 1.07)
1.2	**1.02**	**(0.99; 1.04)**	1.02	(1.00; 1.05)	1.03	(1.01; 1.05)	1.04	(1.02; 1.06)	1.04	(1.02; 1.07)
1.1	1.03	(1.01; 1.06)	1.04	(1.01; 1.06)	1.04	(1.02; 1.06)	1.04	(1.02; 1.07)	1.05	(1.02; 1.07)
1.08	1.04	(1.01; 1.06)	1.04	(1.02; 1.06)	1.04	(1.02; 1.06)	1.04	(1.02; 1.07)	1.05	(1.02; 1.07)
1.05	1.04	(1.02; 1.06)	1.04	(1.02; 1.07)	1.04	(1.02; 1.07)	1.05	(1.02; 1.07)	1.05	(1.02; 1.07)

Notes: RR: relative risk; SIR Standardised incidence ratio; CI: Confidence Interval; h: hours. In bold, non-significant SIR indicating compatibility of the expected values with the observed data.

population-based cancer registration with nearly complete coverage (Engholm et al., 2010).

Our results extend those of previous studies of time trends in the Nordic countries up to 2008 by adding eight more calendar years of follow-up, which allowed us to examine longer latencies and obtain more precise estimates (Deltour et al., 2012). Over the period 1979 to 2008, the observed APC in glioma incidence rates in men aged 40–59 years (0.1%) is identical to the APC in this analysis (0.1%). The APC in the previous analysis was 0.9% in the age group 60–79 years, while here, we found a comparable APC of 0.6% at ages 60–69 years. In the previous study, the consistency check was performed on the age group 40–59 years only, and risks lower than 1.2 for any mobile phone use could not be excluded, nor risks of 1.5 or lower after an induction period of at least 15 years. In the present analysis, we extended these results: we found no changes in the underlying incidence trend up to 2016 indicating no population-level impact of increasing mobile phone use. We were also able to exclude smaller effect sizes from RR 1.2 to 1.08 and longer induction periods from 15 to 20 years. In the present study, none of the risk scenarios for heavy users were compatible with the data.

Our study has a number of strengths. To exclude secondary tumours related to treatment of an earlier cancer, we included only first primary cancers. We considered all cerebral gliomas (with topography C71 and morphologies 938–946 in the ICD-O-3 classification) to minimise any influence of changes over time in classification and diagnostic criteria and we excluded tumours of embryonal origin (medulloblastomas), neuronal and mixed neuronal-glial tumours, and dysembryoplastic neuroepithelial tumours.

Mobile phone use levels were obtained from two different populations covering a range of exposure situations. We considered possible lags, from 0 to 20 years and different risk groups among users based on prior studies, with a range of sizes of potentially at risk populations. While interviews of the controls for the Interphone study were conducted in 2000–2003 in these countries in a random sample of the general population, Cosmos-Denmark study was based on mobile phone users in 2007–2009. Only one study has attempted to validate the recall of the start of mobile phone use. It was conducted with data collected in 2007, and found the start date was reported by controls on average 0.71 years (8.4 months) earlier than the date registered by the operator, with large variability (SD = 4.17 years) (Pettersson et al., 2015). The results suggested a tendency for self-report to be later than the operator date for more recent start of use, while the self-reported start dates tended to be earlier than the operator dates for people with early start of use.

However, our study also has some limitations. The exposure prevalence was obtained from two sources with different recruitment methods, age and questionnaire characteristics; the prevalence in the age group 60–69 years was less accurately registered than for the age group 40–59 years, because both studies had small sample sizes in this age range at recruitment, and in addition, the data had to be extrapolated for the distant past. Therefore, using these estimates of exposure prevalence could at best provide a range of possible exposure distributions in the population. The use of hands-free devices was not accounted for, but this was not frequent in these populations (data not shown). Our study is not free of assumptions. The induction period for an effect of mobile phone use on glioma risk, if one exists, is unknown, as is the magnitude of the risk, if any, and the real patterns may be more complex than the scenarios that we simulated. In addition, there are several factors that we were not able to account for. The coverage of the Nordic cancer registries was not perfectly complete, some 1.5% to 10% of the malignant brain tumours were missed in these age groups, but there is no reason to believe this proportion has increased over time. In Sweden, it has been estimated that completeness would not have changed over the period 1998–2014 for the age group 20–69 years, but has increased among $\geq$ 70 year olds; completeness might have improved in the other countries due to introduction of automated registration routines (Tettamanti et al., 2019, Gjerstorff 2011, Leinonen et al., 2017, Larsen et al., 2009). Our analyses incorporated the possibility that other, currently unknown, risk factors, as well as improvement in glioma detection and reporting had a smooth, gradual impact, over the period 1979–2016, consistent with the gradually increasing IRs.

5. Conclusions

Our findings indicate that glioma incidence trends among men aged 40–59 years in the Nordic countries are not consistent with increased risks of moderate effect size (RR > 1.2–1.4) assuming latency up to 20 years. This means that increased risks reported in some case-control

studies are implausible and likely attributable to biases and errors in self-reported use of mobile phone. Our results were consistent with results from prospective cohort studies showing no association between mobile phone use and risk of glioma.

CRediT authorship contribution statement

Isabelle Deltour: Conceptualization, Investigation, Formal analysis, Methodology, Data curation, Project administration, Writing – original draft, Writing – review & editing. **Aslak Harbo Poulsen:** Conceptualization, Formal analysis, Investigation, Writing – review & editing. **Christoffer Johansen:** Conceptualization, Investigation, Writing – review & editing. **Maria Feychting:** Conceptualization, Investigation, Writing – review & editing. **Tom Børge Johannesen:** Conceptualization, Investigation, Resources, Writing – review & editing. **Anssi Auvinen:** Conceptualization, Investigation, Methodology, Writing – review & editing. **Joachim Schüz:** Conceptualization, Investigation, Supervision, Funding acquisition, Project administration, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2022.107487.

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