Response

Contrary to what Morgan et al. claim, pilocytic astrocytoma was included in our study, as clearly stated in the Methods. Regarding participation rates, Morgan et al. confused noneligibility with nonparticipation. Reasons for noneligibility include lack of language skills, diagnosis of neurofibromatosis, or tuberous sclerosis. Reasons for nonparticipation include refusal to participate or inability to be contacted. We identified 529 case and 1052 control subjects during the study period from which 423 and 909 were eligible, respectively. Participation rates are calculated in reference to eligible subjects.

We stated in the article that operator records regarding the amount of time since the start of the phone users' first subscriptions were available for 35% of case and 34% of control subjects with a mobile phone subscription. It is obviously impossible to retrieve operator data for subjects without any subscription. In total, operator data were available for 80 case and 141 control subjects, and we included all subjects whose subscriptions started before the reference date in the respective analysis. As stated in the footnote of table 4, categories were not mutually exclusive because the reference category always included 123 case and 233 control subjects who were never regular uses and reported to have no subscription. Dropping these nonexposed subjects from the analysis would therefore create bias. Similarly, not all categories in the laterality analyses were mutually exclusive as explained in the footnote of table 5. For laterality analysis, we applied standard definitions and methods defined in the INTERPHONE study (1).

The statistically significant exposure–response association for operator-recorded time since first mobile phone use is comprehensively discussed in the article. This twofold increased risk after approximately 3 years of regular mobile phone use is inconsistent with observed brain tumor incidence rate trends in the Nordic Countries (Figure 1). Neither Morgan et al. nor Söderqvist et al. in a recent commentary (2) have provided a plausible explanation for this inconsistency. Because of the limitations of retrospectively retrieved operator data and self-reported wireless phone use, it is essential to check the consistency of those results with observed time trends of incidence rates to avoid coming to the wrong conclusions (3).

Assuming a short latency of a few years, increased brain tumor risks should be detectable in the incidence data that are already available because of the steep increase in wireless phone use among adolescents during the last two decades. For this reason, we restricted our analysis of cordless phone use to the first 3 years of use. Because most children and adolescents in CEFALO had used cordless phones earlier in life than mobile phones, we could address the effects of microwave radiation with longer latency time periods or with exposure at a young age. However, it was strikingly difficult for many participating families to recall the amount of cordless phone use early in life; some did not feel comfortable about answering questions about amount, duration, or years since first use.

We emphasize that all issues that may be perceived as conflicts of interest were declared. No cell phone company was involved in this study nor provided any funding. Firewalls, as established in this study, have shown to be effective in preventing biased study results (4).

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Notes

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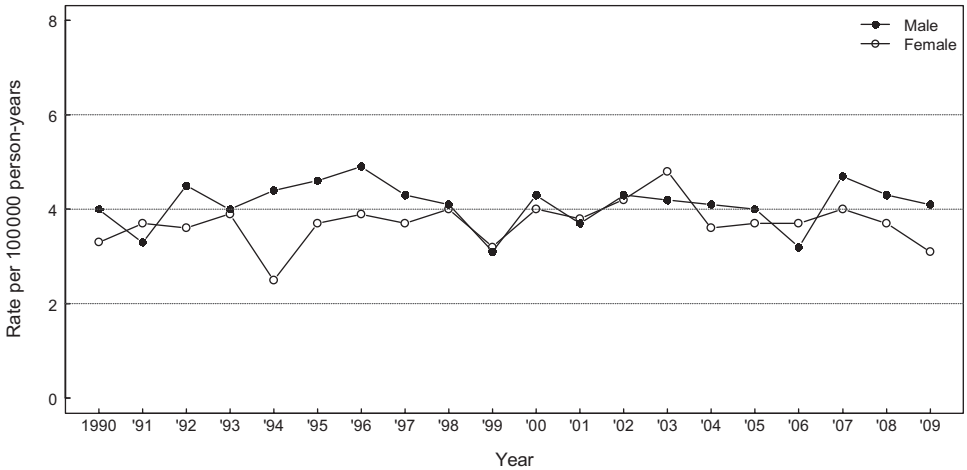

Figure 1. Age-standardized incidence rates for brain and central nervous system tumors for children and adolescents aged 5–19 years living in the Nordic Countries (obtained from NORDCAN [http://www-dep.iarc.fr/nordcan/English/frame.asp]).

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