

Response to Moskowitz and Birnbaum, Taylor, Baldwin, et al.

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In our large prospective study of 776 000 UK women, 3268 developed a brain tumor during the 14 years after they reported their cellular telephone use in median year 2001. There was no increase in the incidence of brain tumors in cellular telephone users compared with nonusers, overall (relative risk = 0.97, 95% confidence interval = 0.90 to 1.04) or by tumor subtype (including glioma and acoustic neuroma). Nor was there an increased risk of tumors in the parts of the brain most likely to be exposed to radio-frequency electromagnetic fields from the telephones. This prospective design overcomes the obvious potential recall bias in retrospective studies [called “case-control” by Moskowitz (1)], where information on cellular telephone use is reported after the brain tumors are diagnosed and use is then compared with information reported by people who do not have brain tumors. Validation studies of exposure assessment in such retrospective studies suggest that such recall bias may have created spurious associations, especially for heavy cellular telephone use (2), which is why the evidence in humans in the hazard identification of radio-frequency electromagnetic fields by the International Agency for Research on Cancer has been classified as “limited” (3).

Moskowitz raises concerns that our exposure assessment may be the reason for the null findings (1). As we already discussed, some misclassification is inevitable in any study, and it is true that random misclassification of telephone use would attenuate any increased relative risk toward 1.00. However, given that our findings were essentially null, even among daily users, there would have to have been substantial exposure misclassification to mask any meaningful excess risk, which is unlikely given that when resurveyed 10 years later, more than 40% of daily users reported talking on a cellular telephone for 30 minutes or more each week compared with just 9% of never-users. So far, all epidemiological studies that assessed cellular telephone use relied on self-reported use by study participants. Furthermore, comparisons of self-reported cellular telephone use with objectively recorded traffic records by network operators have shown that questionnaires using categorical options for cellular telephone use (4), as in our study, had higher validity than asking about duration of calls in open-ended questions, as in some retrospective studies (5). Moskowitz’s claim

that there was a high attrition in our study is incorrect; indeed, of the women who reported mobile phone use in 2001, only 1% had been lost to follow-up in the subsequent 14 years.

We do agree, however, with both Moskowitz (1) and Birnbaum et al. (6) that our study does not include many heavy users of cellular phones. This study reflects the typical patterns of use by middle-aged women in the UK starting in the early 2000s. In the largest retrospective study to date, a modestly increased relative risk for glioma was reported in heavy users; this heavy-user group was less than 5% of their study population selected from the “. . . age-range aimed to maximise the likelihood of exposure” (5), but reporting bias cannot be excluded. A large international prospective study of cellular telephone users including also men and younger women is underway (7), but results have not been published yet. Overall, our findings and those from other studies support our carefully worded conclusion that “cellular telephone use *under usual conditions* [our emphasis] does not increase brain tumor incidence.” However, advising heavy users on how to reduce unnecessary exposures remains a good precautionary approach.

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Data Availability

Please refer to the data availability statement in the authors' original article (<https://doi.org/10.1093/jnci/djac042>).

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