TWO AUTHORS REPLY

We thank Stang (1) for his comments on our paper (2). Stang raised concerns about the following aspects of our study: highly skewed data distribution, quantitative evaluation of data, and the possibility of misclassification of study subjects.

As Stang noted in his letter, our data on hair nicotine concentration had a highly skewed distribution. Because our data were still skewed in distribution after logarithmic transformation, we approached our data with a more robust method that had no assumption of a specific distribution throughout the entire analytical process. This is the reason why we provided the median of each subgroup and also used a nonparametric test for the statistical significance of differences observed among subgroups. The mean and standard deviation can provide useful information on data distribution if they are normally distributed. With highly skewed data, however, the median may be preferable to the mean as a measure of central tendency, and it may be used with nonparametric methods of analysis (3). Therefore, we presented the mean and standard deviation as a conventional measure and box plots with the median and interquartile range as a more appropriate measure for highly skewed data. We had expected that readers could be provided more accurate information about our skewed data through 2 different measures.

Stang also asserted that our study focused only on statistical significance and bypassed quantitative evaluation of data. It is not clear what he meant by “quantitative evaluation of data for a bare-bones dichotomy” (1, p. 1531). Gardner and Altman (3) recommended the confidence interval as a more informative approach rather than the limited-value level of statistical significance. In the case of normally distributed data, the confidence interval can be
calculated for the sample mean and the difference between sample means. In the case of nonnormal data, the confidence interval can be calculated for the median. In our series, the median and 95% confidence interval of neonatal hair nicotine concentrations of each subgroup were as follows: 0.10 ng/mg (95% confidence interval (CI): 0.00, 0.10) in the nonsmoker group, 0.10 ng/mg (95% CI: 0.00, 0.10) in the outdoor-smoker group, and 0.20 ng/mg (95% CI: 0.10, 1.16) in the indoor-smoker group. The confidence interval in the nonsmoker group was equal to that in the outdoor-smoker group. However, the respective upper limits of the 95% confidence interval in the nonsmoker group and the outdoor-smoker group were equal to the lower limit of the 95% confidence interval in the indoor-smoker group, supporting a significant difference between the nonsmoker and indoor-smoker group or between the outdoor-smoker and the indoor-smoker group.

Individual hair nicotine concentrations could be affected by various factors from tobacco exposure to nicotine metabolism. Therefore, it is important to assess the possible factors for differences between outdoor-smoker and indoor-smoker groups. Especially, the paternal smoking quantity can be the most potent factor and can overwhelm all other factors, even smoking location. We conducted multiple regression analysis using SPSS, release 12.0, software (SPSS, Inc., Chicago, Illinois) to provide a more complete exploration of the association of neonatal nicotine concentrations with other variables, such as the maternal or paternal nicotine concentration, paternal smoking quantity, and paternal smoking location. Among those variables, the neonatal nicotine concentration was significantly associated with only paternal smoking location (adjusted $R^2 = 22.4\%$). In addition, there are no significant associations between neonatal nicotine and the maternal or paternal nicotine concentration. Our conclusion, “differences . . . are not due to differences in paternal smoking but to exposure at home” (1, p. 1143), pointed out that paternal smoking location rather than paternal smoking quantity is a more important determinant of neonatal nicotine concentrations on the basis of these analyses.

Stang also suggested the considerable potential of misclassification of smoking group in our study, especially in the case of fathers who smoked on a veranda. The apartment’s structure has to be considered to determine whether these subjects must be classified into the outdoor-smoker or the indoor-smoker group. Because the veranda in the regional apartment generally has sliding windows on the outside as well as the inside, it is apparently different from the windowless terrace of townhouses that it is arguably classified as “outdoors.” Accordingly, we classified it as indoors. In addition, we contacted all families with fathers who smoked and all families with biomarker status in discord with self-reported smoking status, and we confirmed their smoking status to minimize misclassification by self-report.

Consequently, although our data are few and highly skewed, the conclusion that results from these robust analyses is expected to provide important information about fetal exposure in relation to paternal smoking.

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REFERENCES


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