Pleural Plaques and Their Effect on Lung Function

Conclusions Based on Insufficient Power to Reject the Null

To the Editor:
I read with interest the article in CHEST (September 2014) by Clark et al evaluating pulmonary function measures with respect to pulmonary CT scan findings among former vermiculite workers who were exposed to amphibole fibers. The study shows no statistically significant differences in lung function parameters between the pleural plaques only (PPO) group and the normal CT scan (NCTS) group. The authors indicate confidence “that the potential for type 2 error was minimal.” Determination of whether differences between independent groups can be statistically detected (i.e., avoiding false negatives) depends upon the sample size, the true difference between groups, and the within-group SD. The mean percentage point difference between PPO and NCTS is 5.29, 5.72, 4.14, and 5.98 for FVC, FEV1, total lung capacity, and diffusing capacity of the lung for carbon monoxide, respectively. Based on the within-group SDs, a control to case ratio of 0.18, and an α level of 0.05, the given sample sizes for PPO and NCTS indicate that this study had only 15% to 34% power to detect the differences noted previously. This post hoc power analysis is limited to the data available in the published tables, and it could be further informed by the covariates in the model. Nevertheless, 80% is typically considered to be a minimal level of power necessary to avoid excess type 2 error.

The authors should be commended for attempting to determine if there are lung function parameter differences according to severity of pleural plaque findings. However, the statistical power problem indicated here is even more severe with the smaller group sizes. The monotonic response by severity status for FVC, FEV1, and residual volume suggests that an evaluation of trend or slope by subgroups may have been valuable. The authors also could have compared each of these PPO subgroups to the NCTS group to yield a more informative, albeit less statistically powerful, comparison.

Finally, for an occupational study, the exposure assessment was limited. Although the authors were able to adjust their models for length of employment and time since last occupational exposure, they do not account for variance in exposure potential according to job task and time period of employment. As noted in prior studies, this occupational cohort experienced a wide range of exposure potentials, and the amphibole exposure histories of workers who had resided locally are further complicated by environmental pathways. For these reasons, the conclusions by Clark et al should be interpreted with caution.

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REFERENCES:

Response

To the Editor:
We welcome the opportunity to respond to Dr Noonan’s letter regarding our recent article in CHEST. Statistical power is always a potential limitation in biomedical studies. This is especially true in retrospective observational studies, in which an a priori power analysis...
cannot be performed. As mentioned in our article, although our reference group of miners with normal high-resolution CT scan (NCTS) studies was relatively small, there was enough power to detect statistically significant differences in mean lung function data between the NCTS reference group and the miner group with pleural plaques and interstitial fibrosis and the miner group with other high-resolution CT (HRCT) scan findings. However, when we compared the miner group with pleural plaques only (PPO) with the NCTS reference group, we were unable to detect a statistical significance in mean lung function data, even though the PPO miner group had many more subjects than did the miner group with pleural plaques and interstitial fibrosis or the miner group with other HRCT scan findings. Information contained within the 95% CI for comparison of the PPO miner group with the NCTS reference group indicates that we should not reject the null hypothesis. Tukey analysis was also used to minimize type 1 error. Thus, our analysis supports the conclusion that pleural plaques alone have no significant effect on lung function in Libby vermiculite miners.

In his letter, Dr Noonan includes a post hoc power analysis. However, his analysis is meaningless with respect to the statistical significance of our results. Current statistical convention dictates that post hoc power analysis should not be performed because it is fundamentally flawed, does not provide any meaningful information, and may be misleading. There are a number of reasons for its inappropriateness. First, post hoc power analysis can mislead the reader into concluding that an observed result is a false-negative caused by type 2 error, by incorrectly implying that a low post hoc power is caused by "too few" subjects. Apparently, this is what Dr Noonan has attempted to imply. However, once a statistically insignificant result has been obtained, the effect of sample size and associated variance should be obtained from information within the 95% CI, not by post hoc power analysis. In addition, calculated power has relevance only when the null is known to be false. To calculate post hoc power for a statistically insignificant result requires one to make a biased assumption that the null is false, even though it is already known that this assumption is not supported by the data. This is "nonsensical." Furthermore, a statistically insignificant result will always result in a low post hoc power. That is, $P \geq .05$ will always produce a post hoc power $\leq 0.5$, regardless of how large or small the sample size is. This is because post hoc power is directly related to the $P$ value mathematically. In fact, the greater (less significant) the observed $P$ value, the lower the post hoc calculated power. Thus, the basic logic of using post hoc power analysis is convoluted and fundamentally flawed. In this regard, calculating post hoc power adds no new information once the $P$ value is known; it simply tells us what we already know. With respect to our study, Dr Noonan's post hoc power analysis simply tells us that our data analysis does not detect a statistically significant difference in mean lung function data between the miner group with pleural plaques alone and the miner group with normal HRCT scan studies. Again, this result is supported by information contained within the 95% CI.

Furthermore, based on the means in Table 3, we would not expect any significant lung function differences between the PPO group and the NCTS reference group if we were to conduct a new investigation with a larger sample size, with the possible exception of expiratory reserve volume. However, because elevated BMI is well known to reduce expiratory reserve volume, this possibility is most likely related to the high proportion of our cohort subjects with an elevated "obese range" BMI and not caused by any effect of pleural plaques.

For readers of CHEST who desire a clinically oriented explanation of the senselessness of post hoc power analysis, we recommend an excellent article by Goodman and Berlin. These authors state that post hoc power analysis is "like trying to convince someone that buying a lottery ticket was foolish (the before-experiment perspective) after they hit a lottery jackpot (the after-experiment perspective)." This is a good way to think about the impropriety of post hoc power analysis. These authors also state, "Once the data are in, the only way to avoid confusion is to not compress the results into dichotomous significance verdicts and to avoid post hoc power analyses entirely." In his letter, Dr Noonan characterizes our study as an "occupational study." We respectfully disagree with this characterization. Although the data for this study were derived from an occupational cohort of Libby vermiculite miners, the objective and design of the study were simply to determine the effect of pleural plaques, as detected by HRCT scan, on miner lung function. Therefore, our study is a physiology study and was appropriately published as such by the editors of CHEST. Our study does not address "causal association" of occupational exposures with either pleural plaques or lung function. It addresses the effect of an HRCT scan anatomic variant (pleural plaques) on lung function. We did adjust for the length of employment and the time since last occupational asbestos exposure, but neither covariate significantly contributed to lung function outcomes. We agree with the information that Dr Noonan has provided about the
basic elements of an "occupational study"; however, this information has no relevance to our study conclusion. We greatly appreciate Dr Noonan's thought-provoking comments. They have enabled us to further describe the nature of our physiology study and provide additional support for our study conclusion.

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References


