

## Response to “Fruits, fish, and mercury: further considerations”

We read the comments by Dórea and Barbosa on the article “Eating Tropical Fruit Reduces Mercury Exposure from Fish Consumption in the Brazilian Amazon,” by Passos et al. (2003a) with much interest. Here, we would like to respond to some of the points raised in their letter. First, there seems to be a misunderstanding with respect to the information that was collected and the analyses that were performed. All dietary items, including specific fruit, were recorded daily. Every woman was provided with a detailed questionnaire (type of meat, fish, fruit, etc.), which she filled out. Every afternoon, for a period of 1 year, the information was collected and verified by the village midwife. The same procedure was carried out for fruit, as for fish; i.e., if a woman had checked “fruit,” she would then write the specific fruit. The use of a daily diary served to minimize under- or over-reporting due to recall bias. This provided us with extensive data points for 365 days (1095 meals plus items that were eaten between meals).

Dórea and Barbosa correctly point out that there are important seasonal differences in both fish and fruit consumption. Our results confirm this and a detailed description of the specific dietary items within each category and seasonal variations is presented elsewhere (Passos et al., 2003b). Indeed, we carried out the study over an entire year and determined segmental-Hg analyses in order to be able to analyze and take into account seasonal variations in diet. We state in the section on “statistical analysis”: “The relationship between food intake and hair-Hg was examined annually and seasonally.” We divided the seasons according to water levels: increasing (December–February), high (March–May), descending (June–August), and low (September–November). Not only are there seasonal differences in the type of fruit, but also in fruit consumption over time; fruit consumption is lowest in the high-water season. Dórea and Barbosa suggest that the observed differences with respect to fruit consumption may be due to seasonal differences in fruit consumption. The seasonal results for the multiple regression models, which were mentioned but not included in the Passos et al. (2003a) article, are presented below. The explicative variables

are number of fish meals and number of meals with fruit within each season. Fruit consumption is significantly and negatively correlated with total hair-mercury levels in all seasons, with the exception of the rising water season, where a tendency is observed (Table 1).

With regard to specific fruit, we indicate in the article (Passos et al., 2003a) that ingá and banana seem to have the strongest effect and we present the data for these fruit. Other items in the diet, such as manioc, rice, dairy products, etc., were also collected. Their intake does not vary much over time (Passos et al., 2003b) and no relation (positive or negative) was observed with Hg.

Dórea and Barbosa mention that several studies show a decrease in Hg levels during pregnancy and we have recently confirmed this, even at very low levels of exposure (Morrissette et al., 2004). They suggest that pregnancy may explain these results. We wish to assure them that none of these women were pregnant over this period.

Our analyses were based on multiple regression models, using continuous variables, as well as analysis of covariance. We did not base our conclusions solely on the findings that those who ate more than one fruit/day had lower hair-Hg levels for the same amount of fish consumption compared to those who ate less than one fruit/day. This was a convenient way of presenting the results.

We do not wish to overstate the case. However, the strength of our study is its longitudinal design and our results are consistent throughout the year. Although diet has been suggested as a possible modulator of the relation between Hg and effect (for review see Clarkson and Strain, 2003), this is the first study to demonstrate that fruit consumption may indeed influence mercury exposure.

There is always a trade-off between the amount of data that can be collected and the size of the population. In the Passos et al. (2003a) study, we opted for a large amount of longitudinal data from a small population in order to identify the relevant food items that could then be used in a study with a much larger population. We are currently examining the possible interactions between specific fruit consumption, fish consumption, and Hg in a large population study along the Tapajós River.

Table 1

Beta coefficients and *P*-value results of multiple regression models for mean total hair-Hg for different seasons with respect to fish consumption

	High-water $\beta$ coeff ( <i>P</i> )	Descending-water $\beta$ coeff ( <i>P</i> )	Low-water $\beta$ coeff ( <i>P</i> )	Rising-water $\beta$ coeff ( <i>P</i> )
<i>F</i> for model	5.70	17.59	4.094	5.86
Fish consumption	0.077 (0.06)	0.123 (<0.001)	0.058 (0.03)	0.074 (0.003)
Fruit consumption	−0.056 (0.05)	−0.026 (0.01)	−0.028 (0.02)	−0.023 (0.09)

Fish is a very healthy food and a dietary mainstay of this population; we hope that these studies will serve to further our knowledge on how to maximize the nutritional input from traditional food consumption and minimize toxic risk.

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## Respiratory impairment due to asbestos exposure in brake-lining workers

The stated purpose of the article by Erdiç et al. (2003) was “to assess the long-term effects of chrysotile asbestos exposure on lung function and the risk of asbestos-related diseases in brake-lining production workers.” The authors concluded that “even in the absence of radiographic asbestosis,  $T_L$ ,  $CO$  and  $K_{CO}$  may decrease after a mean 10-year duration of exposure to asbestos in brake-lining workers and this is more noticeable with cigarette burden.” However, because of flaws in the study design, execution, and analyses, these conclusions are not supported by the study.

This was a prospective study among active workers employed by a single brake-lining production facility. Subjects completed pulmonary function tests, specifically spirometry and diffusion capacity, in 1992 and 1999. Exposed workers were employed processing asbestos brake linings; unexposed workers were employed in offices. The asbestos-exposed group was divided based on smoking status: current smokers ( $n = 49$ ) and nonsmokers ( $n = 25$ ). All unexposed workers were current smokers ( $n = 12$ ). There are a number of features of the study group that are unexplained and problematic. First, there were no ex-smokers among the 86 subjects. Ex-smokers usually comprise a large fraction of working populations, which

suggests that a substantial number of workers were excluded from the study. Second, the number of unexposed workers was too small for statistical analyses. Third, there were no nonsmokers among the unexposed subjects, which would limit the ability to measure the independent effect of smoking on pulmonary function among subjects. Fourth, there was no mention of workers who completed testing in 1992, but who did not complete testing in 1999. It is important to know whether workers who completed follow-up testing differed at baseline from subjects who did not complete the second round of testing. These flaws in selection and/or description of the study population suggest the possibility of substantial bias.

There are a number of problems with the pulmonary function tests. It was stated that “nobody had smoked at least 3 h before the testing procedure.” The half-life of carbon monoxide in blood while breathing room air is approximately 4 h (Rom, 1998). Since testing for diffusion capacity (i.e., transfer factor) was performed as little as 3 h after smoking, or less than one half-life, measurements in smokers were likely biased (i.e., they would appear more impaired) due to carbon monoxide backpressure. Though the authors acknowledged the possibility of a problem with carbon monoxide back-