measurement) of Rowland et al. (1983). For the carcinomas, Mays et al. used the function derived by Rowland et al. (1978), which again was linear. Since drinking water normally contains low levels of radium, lifetime accumulations in the body remain very low, and the use of linear dose-response functions for the very low dose range was judged to be appropriate. Mays et al. found that, for an intake of 5 pCi per day over a lifetime of 75 years, about 9 radium-induced sarcomas and 12 radium-induced carcinomas would occur among 1,000,000 exposed individuals. The same population would experience about 750 bone sarcomas and 375 carcinomas due to natural causes.

These calculations certainly indicate that exposure to the stated level of ^{226}Ra is relatively safe, but the use of linear dose-response functions implies that, at very low radium levels, the carcinomas of the paranasal sinuses and mastoid air cells are more likely to be observed than the bone sarcomas. Since this implication is in direct opposition to the observations made on a large number of radium cases (at much, much higher radium levels), the conclusion should be viewed with suspicion. The dose-squared function, which appears to fit the sarcoma data so well, would predict even fewer bone sarcomas at these very low doses, suggesting that the true description of radium-induced carcinomas might fall off much faster than linearly at the lower doses. With so few of these rare carcinomas as a basis for a dose-response function, this question will probably remain unanswered.

After the radium program at Argonne had ended, Rowland (in press) presented an invited paper on dose-response relationships at an international seminar on radium and thorium (in Heidelberg, April 1994). This new analysis considered all female dial workers with body burden measurements who entered the industry before 1950, a total of 1,530 individuals. In this cohort, 46 women had bone sarcomas, and 19 had head carcinomas; 3 women experienced both a bone sarcoma and a head carcinoma.

Incorporation of the revised estimates of systemic intake into the new calculations significantly changed the dose-response functions. Rowland (in press) found no satisfactory fits with the previously used form of the dose-response function for bone sarcomas (Equations 6 and 9) when the very small constant term indicating the natural rate of sarcomas in an unexposed population was ignored. However, changing the exponent on the systemic intake term (D in Equation 9) from 2 to 3.15 (a parameter determined from the data) produced a good fit. When the exponent was specified instead, satisfactory fits were obtained with exponents of 2.7-4.1. Alternatively, when a threshold was assumed for bone sarcomas and the exponent was set at 3.15, a solution was obtained with a threshold of 79 μCi. The mechanistic meaning of the exponent value of 3.15 is not obvious, suggesting that, at least for radium-induced bone sarcomas in humans, a threshold hypothesis is as good as any other.