In Reply We appreciate the opportunity to respond to the letters from Peacock and colleagues and Grady and colleagues regarding our Original Investigation. The aim of our study was to investigate the dose-response relationship between estimated organ doses and cancer mortality risks in patients treated with radioactive iodine (RAI) for hyperthyroidism. This was the major strength of our study compared with earlier analyses of this and other cohorts, which relied on the cruder approach of comparing risks in exposed and unexposed patients. Dose-response relationships are less likely to be explained by confounding and, thus, provide stronger evidence in support of a causal relationship. These estimates also quantify the risk per unit dose, which can be translated into estimated absolute risks. In the article, we emphasized that the magnitude of the risk associated with current typical treatment doses is small (20-30 lifetime excess cancer deaths per 1000 patients treated with RAI).

In response to Peacock and colleagues, any uncertainties in the dosimetry were mainly expected to have biased our dose-response estimates toward the null, and statistical tests showed that the linear dose-response model provided our dose-response estimates toward the null, and statistical ties in the dosimetry were mainly expected to have biased per 1000 patients treated with RAI.

In response to the question of how to answer these questions because of dramatic changes in antithyroid drug formulations since the 1940s to 1960s—one of the major strengths of our study compared with other studies—dose-response relationships are less likely to be explained by confounding and, thus, provide stronger evidence in support of a causal relationship. These estimates also quantify the risk per unit dose, which can be translated into estimated absolute risks. In the article, we emphasized that the magnitude of the risk associated with current typical treatment doses is small (20-30 lifetime excess cancer deaths per 1000 patients treated with RAI).

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