We develop and analyze a reaction-diffusion model to investigate the dynamics of the lifespan of a bystander signal emitted when cells are exposed to radiation. Experimental studies by Mothersill and Seymour 1997, using malignant epithelial cell lines, found that an emitted bystander signal can still cause bystander effects in cells even 60h after its emission. Several other experiments have also shown that the signal can persist for months and even years. Also, bystander effects have been hypothesized as one of the factors responsible for the phenomenon of low-dose hyper-radiosensitivity and increased radioresistance (HRS/IRR). Here, we confirm this hypothesis with a mathematical model, which we fit to Joiner's data on HRS/IRR in a T98G glioma cell line. Furthermore, we use phase plane analysis to understand the full dynamics of the signal's lifespan. We find that both single and multiple radiation exposure can lead to bystander signals that either persists temporarily or permanently. We also found that, in a heterogenous environment, the size of the domain exposed to radiation and the number of radiation exposures can determine whether a signal will persist temporarily or permanently. Finally, we use sensitivity analysis to identify those cell parameters that affect the signal's lifespan and the signal-induced cell death the most.