Nagasaki-Hiroshima CML city differences

The `SEERaBomb` function `blood2012fig4()` uses the model

$$E_i = \left(e^{c0+k*age_i} + D_i e^{L_{j,}}\right) PY_i \qquad (1)$$

which states that for the ith data cell, incidence multiplied by person-years (PY_i) at risk equals the expected number of CML cases E_i (the observed number is then an integer sampled from a Poisson distribution with this mean). This model makes two reasonable assumptions: the background incidence increases exponentially with age and induced CML risk is linear in the radiation dose (Di) in Sieverts. It is flexible in that it allows the data to speak regarding the shape of the radiation-to-CML waiting time distribution by fitting separate dose-response linear slope parameters e^{Lj} to each of 5 time-since-exposure groups (obtained by pairwise binning of 10) of the 1950-1987 dataset. This model represents a null hypothesis of no city differences. An alternative hypothesis is that a virus depleted the hematopoietic stem cell (HSC) reserve of the Nagasaki population to X% of normal levels at the time of the exposure, and as a result, background and radiation-induced CML incidence in Nagasaki is only X% of that in Hiroshima. Suppose this Nagasaki virus is sexually transmitted such that only those older than 10 years of age at the time of bombing were protected from CML by it. The demo script `nagaCML.R` compares this hypothesis to Eq. (1): the deviance is reduced by more than 3.84 (chi-squared with 1 df) so the city effect is statistically significant. This script also estimates X as 20-30%.