8 MUTATION INDUCTION IN BACTERIA AFTER HEAVY ION IRRADIATION

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From a compilation of experimental data on the mutagenic effects of heavy ions in bacteria [2], [3] main conclusions have been drawn as follows:

- The mutagenic efficacy of heavy ions in bacteria depends on physical and biological variables. Physical variables are the radiation dose, energy and charge of the ion; the biological variables are the bacterial strain, the repair genotype of bacteria, and the endpoint investigated (type of mutation, induction of enzymes related to mutagenesis).

- The responses on dose or fluence are mainly linear or linear quadratic. The quadratic component, if found for low LET radiation, is gradually reduced with increasing LET.

- At low values of Z and LET the cross section of mutation induction σ_m (as well as SOS response, σ_{SOS}, and λ phage induction, σ_λ) versus LET curves can be quite consistently described by a common function which increases up to approximately 100 keV/μm. For higher LET values, the σ_m versus LET curves show the so-called "hooks" observed also for other endpoints [1].

- For light ions ($Z \leq 4$), the cross sections mostly decrease with increasing ion energy, which is probably related to the decrease of the specific energy departed by the ion inside the sensitive volume (cell). For ions in the range of Z=10, σ_m is nearly independent on the ion energy. For heavier ions ($Z \geq 16$), σ_m increases with the energy up to a maximum or saturation around 10 MeV/u. The increment becomes steeper with increasing atomic number of the ion. It correlates with the increasing track radius of the heavy ion.

- The mutagenic efficiency per lethal event changes slightly with ion energy, if Z is small indicating a rough correlation between cellular lethality and mutation induction, only. For ions of higher Z this relation increases with energy, indicating a change in the "mode" of radiation action from "killing-prone" to "mutation-prone".

- Repair genotype substantially influences the radiation induced mutagenesis. Different mechanisms of mutation induction and/or different types of biologically significant lesions in wild type cells compared to repair deficient strains are a likely explanation.

The observed results suggest the following interpretation. For a bacterial cell, affected by a heavy ion, the injury will be either "killing-prone" of "mutation-prone". In the track core of densely ionizing radiation, the cells will be inactivated with high probability and mutations are unlikely to be produced. Mutations are most likely to be produced by δ-rays. Therefore, in the cross section of a track, one can imagine a "zone" between the track core and the track edge where mutations are induced with high probability. This "mutagenic belt" is restricted to an area where the density of the departed energy is low

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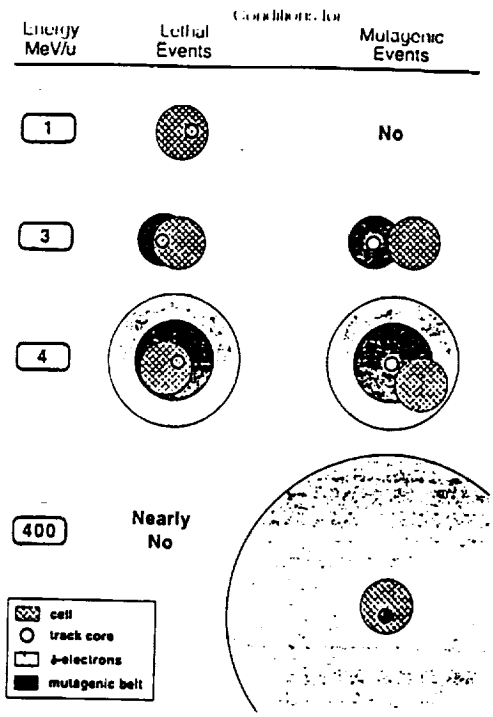

Figure 1: The "mutagenic belt" as a interpretation of mutagenesis by heavy ions in bacteria.

enough in order not to kill the cells and high enough to produce mutations (Figure 1). The "mutagenic belt" can be reduced if the density of departed energy is increased -from one side - or decreased - from the other side. For light ions, the "mutagenic belt" includes the track core owing to the low density of departed energy even in the track core which, in this case, decreases with increasing energy. Hence, the mutagenic efficacy decreases with increasing energy of light ions. For ions of high Z, there is no "mutagenic belt" if the energy is low owing to the very high concentration of energy in the track core and a very short range of δ-electrons. Therefore, no mutations are induced by those ions of high Z at low energies. Increasing energy leads to a growth in the "mutagenic belt" which should be more pronounced the greater the Z of the ion. This "mutagenic belt" interpretations demonstrates the important role of δ-electrons in heavy ion mutagenesis. A theoretical approach for interpretation of this "mutagenic belt" phenomenon is in preparation.

References

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