**RecX stimulates homologous recombination by modulating RecA activities**

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Annex 1. The absence of RecX does not increase the spontaneous mutation rate.

To determine whether the reduced threshold for SOS response in Δ*recX* could facilitate the emergence of error-prone DNA repair [[reviewed by 1](#_ENREF_1)] and indirectly contribute to sexual isolation (see Introduction) the spontaneous mutation rate (to rifampicin resistance or reversion of the *metB*5 point mutation to *met*+ auxotrophy) under conditions of full SOS induction of *rec*+ cells (0.6 μM MMC) was measured. The frequency of spontaneous mutations obtained in *rec*+, *recX*342 or Δ*recX* cells in either presence or absence of MMC was not significantly different (data not shown), suggesting that the potential contribution of error-prone repair, if any, is not significant.

Annex 2. *recP*149 mutation maps is the *recA* gene

Three qualitatively distinct situations may arise when the frequency of recombinational repair of single and double mutant strain deficient in recombination (*rec*-) are analyzed: (i) the survival frequency may be equal to that of the more deficient single-mutant parent (equal epistatic group), (ii) it may be equal to the sum of each of the single-mutant parents (different group, additive effect) or (iii) it may be greater than the sum of each of the single-mutant parents (different group, synergistic effect) [[see 2](#_ENREF_2)]. However, these analyses are not valid for *recA* mutants, because homologous recombination is going trough the RecA pathway [[3](#_ENREF_3)]. In other words, the survival frequency of any mutant in concert with a mutation in *recA* must be equal to that of the more deficient single-mutant parent (*recA*). In early nineties the *recH*342 (*recX*342) and *recP*149 mutations were tentative classified together within epistatic group  [[2](#_ENREF_2)]. Both mutants lacked a direct selection, and the double mutant could not be constructed, because the *recP*149 mutation (mapped in the *purA* - *cysA* region, 11º interval, by PBS1 transduction) was incorrectly mapped (Manfredi et al., to be published elsewhere). Recently, the *recP*149 mutation was mapped within the *recA* gene, at the 150º interval, hence *recP*149 was renamed as *recA*149 (Manfredi et al., to be published elsewhere), and withdrawn from epistatic group γ.

**References**

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