RECOMBINANT PLASMIDS CARRYING MUTANT LOCI *GAM* INCREASE RESISTANCE OF *ESCHERICHIA COLI* WILD TYPE CELLS TO IONIZING RADIATION

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To undestand factors limiting repair system of *Escherichia coli* wild-type strain mutant loci *gam* were cloned from hyperradioresistant mutant of *E. coli* Gam^r444 using vector MudII4042.

The recessive *gam12* mutation results in constitutive expression of heat-shock regulon as we tested with *lacZ* gene under control of heat-shock promoter p_{htpG}. On the other hand, induction of abnormal protein (human prourokinase) titrating the DnaK increases radioresistance of *E. coli* wild type cells harboring pUK-O2pmO.

Dominant mutation *gam18* compensates defect in *uvrD* gene partly. Random insertions of mini-Tn10-Km^r transposon into pGam18 by hop-transposition from λ_{1105} vector were obtained and plasmid which had lost Gam^r phenotype was selected. Transduction of *kan* marker by Kohara phages revealed homology of plasmid-borne allele with helicases which have 3'→5' polarity. Dominant plasmid of another type pGam43 leads to RecA-independent inhibition of post-irradiation DNA-degradation. Unknown inhibitor is produced constitutively because relative efficiency of plating of T4 2^- mutant phages appeared to be as high as in *recB* strain.

Our data show that enhanced radioresistance of *E. coli* wild type cells harboring recombinant plasmids with mutant loci *gam* is due to unusual involvement of heat-shock proteins in repair and encreased activities of SOS-repair system and specifically RecF-way of recombination-repair.