PROPERTIES OF CLONED DOMINANT Gam43 ALLELE

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E. coli Gam^{r}444 mutant is super-radioresistant (survival S = 10^{-3} at a dose 3D = 5.0 kGy) and contains several mutations relevant to enhanced repair. It was found that the level of DNA degradation is significantly lower in UV- or gamma-irradiated cells of Gam^r mutants as compared with the wild-type strain. It was suggested that enhanced radioresistance of Gam^r mutants was due to decreased formation of enzymatically induced double stranded breaks (dsb) as a consequence of moderated DNA degradation under the action of repair exonucleases and also of enhanced efficiency of dsb repair [1].

Dominant plasmids *pGam26* and *pGam43* carrying mutant loci from Gam^{r}444 strain mediate RecA-independent inhibition of post-irradiation DNA degradation [2].

Measurement of 3'-^{3}H-end labelled DNA degradation in purified cell lysates [3] showed that major DNA exonuclease activity in Gam^{r}444 mutant is 2 times lower than that in wild-type cells. Unknown inhibitor is produced constitutively because relative efficiency of plating of T4 *2*$^-$ mutant phages with defective protein protecting linear T4 DNA [4] on unirradiated Gam^{r}444 host appeared to be as high as on *recB*$^-$ strain (Table). It is possible that *gam43* mutation enhances efficiency of the inhibitor.

Thus, RecA-independent constitutive inhibitor of DNA degradation is a part of repair system of radioresistant strain.

Table

The efficiency of plating (EOP) of T4 *2*$^-$ on *E. coli*

Strain	Relevant genotype	Relative EOP of T4 2$^-$
WA235	*recA56 recB21 sup*$^+$	1
WA237	*recB*$^+$ *sup*$^+$	0.000065
Gamr445	*gam12 gam**	0.0353
Gam^{r}444	*gam12 gam18 gam43*	0.489
AB1157	*recB*$^+$ *supE37*	0.0011
GH12/pGam12	*recB*$^+$ *gam12*	0.00152
AB1157/pGam18	*recB*$^+$ *gam18*	0.000441
AB1157/pGam^s 18::Tn5	*recB*$^+$ *gam*^{s} *18::Tn5*	0.00670
AB1157/pGam43	*recB*$^+$ *gam43*	0.990

* – unidentified.

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