NATURAL PLASMID pSD89 (Cm^r) FROM *SALMONELLA DERBY* K89 PROTECTS *ESCHERICHIA COLI* K-12 STRAINS AGAINST RADIATION

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A few natural plasmids increasing resistance of *Escherichia coli* and *Salmonella typhimurium* to lethal effects of UV-light irradiation and some chemical agents are carrying *E. coli umuDC* analogs and their effects depend on *recA*$^+$ and *lexA*$^+$ genes [1]. To determine molecular mechanisms responsible for radiation-resistance of cells with natural and recombinant plasmids, new objects should be studied.

Table

The effect of pSD89 (Cm^r) plasmid on sensitivity of *S. derby* and *E. coli* strains to UV light and γ-rays

Strain	UV light		γ-rays	
	D_{37} J/m^2)	DMF	D_{37} (Gy)	DMF
S. derby:				
K89	25		0.11	
K82	25		0.10	
K82/pSD89 Cm^r	125	5	0.33	3.3
E. coli K-12				
AB1157	126		0.17	
AB1157/pSD89 Cm^r	–		0.21	1.2
Z805 (*r*$^-$ *m*$^+$ *recA13*)	–		0.025	
Z805 (*r*$^-$ *m*$^+$ *recA13*)/pSD89 Cm^r	–		0.05	2.0
P108 (*polA6*)	28		0.03	
P108 (polA6)/pSD89 Cm^r	97	3.5	0.175	5.8
AB2463 (*recA13*)	4		0.025	
AB2463 (*recA13*)/pSD89 Cm^r	97	24.3	0.14	5.6
TK603 (*uvrA umuC*$^+$)	4		–	
TK603 (*uvrA umuC*$^+$)/pSD89 Cm^r	6	1.5	–	
TK610 (uvrA6 *umuC36*)	4		–	
TK610 (*uvrA6 umuC36*)/pSD89 Cm^r	6	1.5	–	

Natural plasmid pSD89 (Cm^r) was extracted from clinical isolate of *Salmonella derby* K89 [2] which was resistant to several antibiotics. Strains of *S. derby* K89 and K82 (cured of plasmids) manifest the same resistance to UV-light but original strain is more sensitive to ionizing radiation. Several plasmids with molecular mass 2 - 60 Md were found by means of electrophoresis in agarose gel. Cm^r transformants which were more resistant to gamma-rays and UV-light were selected after transformation of *S. derby* K82. Plasmid pSD89 (Cm^r) was modified in *E. coli* K-12 strain Z805 (r^- m^+). Varios repair-deficient *E. coli* strains were transformed with pSD89 (Cm^r) and their radiation-sensitivities were evaluated. Plasmid pSD89 (Cm^r) increases radiation-resistance of *E. coli* wild-type and compensates defect in great extent in *polA*, *recA* mutants but not in *uvrA* and *umuC* mutants (Table).

It is likely that plasmid pSD89 (Cm^r) from *S. derby* K89 is carrying locus determining product which does take part in repair but is not identical to known MucA and MucB proteins.

REFERENCES

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KARYOTYPING OF INDIVIDUAL CELLS WITH FLOW CYTOMETRY

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As the study of metaphase chromosomes with flow cytometry presupposes mixing all chromosomes from many cells before analysis, important information is lost. To overcome this limitation we have developed a novel cell-oriented flow cytometric method for chromosome analysis [1-3].

A flow cytometer supplied with special Device for disruption of metaphase chromosomes is the heart of the method (Fig.1). Cells with stained chromosomes are pressed into the Disruption Device and then converted in batches of chromosomes. Chromosome fluorescence and time intervals between fluorescence pulses are registered. There are large time intervals between neighboring batches because a sparse cell suspension is used and, in turn, there are small time intervals inside batches. Taking into account of time intervals it is possible to identify individual cell flow karyotypes (Fig.2 and 3).

The novel method possesses the following features:

- Unique ability to obtain individual cell flow karyotypes (Fig.3B and 3C);
- High technologic principle based on ordinary flow cytometer modified with one accessory - Disruption Device;
- Small number of cells needed for analysis (some thousands);
- Automatical acquisition and analysis of data;
- High rate of acquisition, 100-200 cells/min;