PLASMIDS FROM EIGHT INCOMPATIBILITY GROUPS REQUIRE SIMILAR HOST DNA REPAIR FUNCTIONS FOR UV-PROTECTION AND MUTAGENESIS

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The presence of plasmid pKM101 in the Salmonella strains used in the Ames test for the detection of chemical mutagens significantly increases the sensitivity of the test (McCann et al 1975). Many plasmids increase survival and mutagenesis following DNA damage (Pinney 1980). The genetic interactions of pKM101 with repair-deficient hosts suggests that it participates in "error-prone" DNA repair (Dobson & Walker 1980). We now report experiments with plasmids known to increase mutagenesis from eight incompatibility groups, designed to show if they all interact with error-prone DNA repair, or if some are dependent upon error-free repair pathways for activity.

Four error-free DNA repair mutants of Escherichia coli were used. These were the strains AB1886 uvrA, JG138 polA, JC3890 uvrB and AB2470 recB. Six E.coli mutants were also tested that were defective in error-prone DNA repair. These were the strains AB2463 recA, JC9239 recF, TK501 uvrB umuC, JC8471 recL, KMBL91 uvrE and N14-4 uvrD. Plasmid transfer was by conjugation from stock laboratory E.coli strains. Sensitivity to ultraviolet light (UV) and UV-induced mutagenesis was measured as described by Upton & Pinney (1981). All eight plasmids increased UV-induced mutagenesis in the repair-proficient E.coli strain AB1157 (Table 1). Seven of these also increased UV resistance but R391 made AB1157 significantly more sensitive to UV. The plasmids behaved similarly in all four of the error-free DNA repair E.coli mutants, which suggests that none of these plasmids are dependent upon the error-free DNA repair pathways tested for activity.

Table 1. Plasmid-mediated UV protection and mutagenesis in E.coli AB1157

Plasmid	R391	R805a	R46	R16	R124	JR66a	R621a	R446b
Incompatibility group	J	Iζ	N	B	FIV	Iα	Iγ	M
*[1] Fold increase in post-UV survival	0.19	11.5	10.5	7.1	7.0	11.6	5.7	6.5
*[2] Fold increase in post-uv mutagenesis	5.2	3.4	9.5	5.2	4.1	3.6	2.9	7.2

*Compared with survival and mutagenesis in AB1157R⁻.[1]after 100 J/m².[2]after 8 J/m²

None of the plasmids protected or increased mutagenesis in the recA strain, whereas all plasmids fully restored UV mutability to the umuC mutant, which is completly deficient in UV-induced mutagenesis and sensitive to UV. Although the seven UV-protecting plasmids restored UV resistance to the umuC strain, R391 still sensitized it to UV. It has been reported (Kuemmerle & Masker 1980) that uvrD, uvrE, and recL are distinct alleles of a single gene, with differing effects on DNA repair. However, Todd and Glickman (1979) found that pKM101 increased mutation in uvrD, recL and uvrE strains, but that only uvrD strains were protected from UV damage. Our results for plasmid-mediated mutagenesis agree with those of Todd and Glickman (1979), but we found that the plasmids tested protected both the uvrD and the recL strains from UV. Three plasmids (R16, R805a and R446b) also gave a low but consistant level of protection to the uvrE strain. Plasmid R391 sensitized these three strains to UV.

Thus, no plasmid tested is dependent upon error-free DNA repair for activity. Since their effect is similar in error-prone repair mutants, it appears that all eight plasmids affect both post-UV survivaland mutagenesis by interaction with common host repair functions.

Dobson, P.P., Walker, G.C. (1980) Mutat. Res.71 : 25-41
Kuemmerle, N.B., Masker, W.E. (1980) J. Bacteriol. 142 : 535-546
McCann, J. et al (1975) Proc. Natl. Acad. Sci. U.S.A. 72 : 979-983
Pinney, R.J. (1980) Mutat. Res. 72 : 155-159
Todd, P.A., Glickman, B.W. (1979) Mutat. Res. 62 : 451-457
Upton, C., Pinney, R.J. (1981) J. Gen. Microbiol. in press

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