MUTATOR AND UV-RESISTANCE PLASMIDS FROM INCOMPATIBILITY GROUPS OTHER THAN N

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The sensitivity of the Ames test for chemical mutagens has been increased by transfer of the N incompatibility group plasmid pKM101 into the tester strains (McCann and others, 1975). Some other N group plasmids increase UV or chemical-mutagenesis in Salmonella or Escherichia coli strains and also confer UV resistance on their hosts (Drabble and Stocker, 1968; MacPhee, 1973; Tribe and Pinney, 1977). To determine how universal the plasmid mediated mutator effect is, and in an attempt to find plasmids with even greater mutator activities than pKM101, plasmids from 19 incompatibility groups have been investigated for mutator and UV-protecting activity.

The ability of a plasmid to cause mutation was tested by exposing plasmid-containing (P^+) strains of E.coli 343/113 lys arg. to 40 J/m^2 of UV light as described by Tribe and Pinney (1977), and comparing the frequency of reversion to lysine independence with that obtained in the plasmid-less (P^-) strain. UV protection was tested by comparing survival of washed overnight broth cultures of p^+ and P^- strains after exposure to a UV dose of 240 J/m^2.

Plasmids from twelve incompatibility groups showed no significant increase in mutagenic activity or UV resistance. These were: FI, FII, A, C, H1, H2, I2, P, S, T, W and X. Plasmids from seven groups significantly increased post-UV reversion to lysine prototrophy in E.coli strain 343/113 and also decreased the UV sensitivity of this strain. These were: FIV, M, B, I1, I_γ, N and O. Greatest increase in mutation frequency was found with plasmid pKM101, which fully justifies its use in the Ames tester strains. The N group plasmid R390 was exceptional in that although it increased mutation frequency, it exerted no UV-protecting activity on its host. A series of deletion mutants was obtained and tested for all known phenotypic traits of R390. The following order of linkage was determined:

-Cm-Tc-Tra-(Rep-Ap)-Mut-(Mod-Res)-(Sm-Su)-

This agrees well with the R390 gene map of Coetzee, Datta and Hedges (1972) and extends it to include the position of the mutator (Mut) gene. Ampicillin resistance (Ap) is carried on transposon A (TnA) in R390 (Heffron and others, 1975), and its proximity to Mut may be significant. MacPhee (1974) has suggested that UV protection conferred by another N group plasmid R-Utrecht is due to a plasmid-coded DNA polymerase. If the mutator effect of R390 is also due to plasmid-mediated error-prone DNA polymerase activity, then the insertion of TnA into Mut may have destroyed the UV repair activity of the Mut gene product without abolishing its mutagenic function.

I thank Drs R.W. Hedges and H.R. Smith for plasmids, and am most grateful to Joyce Dunachie for excellent technical assistance.

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