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Possible Correlation Between Transformability and Deficiency in Error-Prone Repair

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We have investigated the relationship between UV-induced mutability (as a measure of an error-prone repair process) and the genetic transformability of transformable and nontransformable bacterial strains. The data suggest a correlation between chromosomal transformability and a deficiency in an error-prone repair system in bacteria.

Genetic transformability at a high level is confined to a small number of bacterial species (19). These bacteria must be able to perform all the steps required to obtain stable genetic recombinants, from the binding and entry of donor DNA to its integration and expression in the recipient cells. Nontransformability can thus result from many factors that may interfere with any of these steps and that depend mostly upon membrane properties, the presence of DNAses, and recombination proteins. One possible mechanism is the elimination by some repair process of the new information given by the donor DNA. We have investigated the relationship between UV-induced mutability, as a measure of an error-prone repair process, and the genetic transformability of transformable and nontransformable bacterial strains.

Wild-type strains of *Streptococcus pneumoniae* are not mutable by UV irradiation (3, 13), thymine starvation, or transformation (3). Moreover, there is no increased survival of UV-irradiated phage occurring when the host is also irradiated before infection. This suggests that in pneumococci, there is no efficient, inducible repair process similar to the SOS repair described for *Escherichia coli* (3). We have shown that a variety of transformable *Streptococcus sanguis* strains related to group H pneumococci do not respond to UV induction of mutations (17). Irradiation with doses sufficient to reduce survival to a few percent causes no detectable increase in the frequency of aminopterin-, rifampin-, or streptomycin-resistant mutants. Similar experiments were performed with two strains of *Streptococcus mutans*, and similar results were observed for both strains. All these strains are transformable and are comparable in their inability to produce mutants after UV treatment. On the other hand, it has been reported that UV irradiation induces streptomycin-resistant mutations in five different nontransformable streptococcal strains of group A under experimental conditions in which no such mutations could be detected in pneumococci (13).

The UV-induced mutability in such nontransformable strains and the absence of mutations in transformable strains pose the question of a correlation between the processes of transformability and mutability. This led us to extend our observations to a wide variety of strains. A summary is given in Table 1. The absence of UV-induced mutability has been found in several transformable strains of *Haemophilus influenzae* (7, 8) and *Micrococcus radiodurans* (18). No UV-induced mutants have been reported for the genus *Neisseria*. For the genus *Rhizobium*, only one brief mention of the use of UV irradiation to produce mutants has been made (12), and it has been shown that the efficiency of the repair system is weak (10). Conversely, for *E. coli*, which is transformable by chromosomal DNA at a low efficiency even after drastic treatment to force the membrane barrier (9), and *Salmonella typhimurium*, for which only transfection is decribed, UV mutability is well established (2, 20). *Proteus mirabilis*, for which no transforming activity has been reported, is not UV mutable, although it possesses functions homologous to those controlled in *E. coli* by the *recA* and *lexA* genes (5). The nonmutability after UV exposure results from the lack of functions comparable to those coded by *umuC* (5). For *Bacillus subtilis*, in which an inducible system comparable to the SOS system has been observed (21) and mutants have been obtained (14, 23), only one strain, *B. subtilis* 168, is transformable (4). Moreover, the efficiency of the error-prone repair seems to be weak; Weigle reactivation is limited to lesions containing thymine dimers, and no mutation

TABLE 1. Genetic transformability and UV-induced mutability in different bacterial strains

Strain	Transformability of chromosomal DNA[a]	UV mutability	References
Streptococcus pneumoniae	+	−	3, 13
Streptococcus sanguis	+	−	See text
Steptococcus mutans	+	−	See text
Streptococcus sp. group A	−	+	13
Haemophilus influenzae	+	−	7, 8
Micrococcus radiodurans	+	−	18
Rhizobium spp.	+	Low, if any	12
Neisseria spp.	+	−	22
Escherichia coli	Low	+	20
Salmonella typhimurium	Low?	+	2
Proteus mirabilis	−	−	5
		Presence of *umuC*-like mutation	
Bacillus subtilis 168	+	+	14, 23

[a] For review on transformability of strains, see reference 1.

occurs on the phage (R. E. Yasbin, 6th European Meeting on Transformation and Transfection, Lisbon, Portugal, 1982).

All these data suggest that transformable bacterial strains are deficient in an error-prone repair system and that conversely, nontransformable strains have such a system. A correlation has already been made between genetic transformability and non-photoreactivability (6). Despite speculation on the subject (16), the basis of this correlation remains unclear. Another correlation has been noted between genetic transformability and the ability of various bacterial species to release dimers into the medium (15). It concerns some peculiarity of the cell wall which facilitates the uptake of DNA (15). Indeed, the penetration of DNA is one of the major requirements for many strains to transform. We would like to point out here the possible role of repair in chromosomal transformation. It has been recently shown that recombination proteins are induced during competence in *Streptococcus sanguis* (11). A repair system is also induced by transformation in competent *B. subtilis* (14, 22). However, transformation and error-prone repair seem to act through different processes. Indeed, many nontransformable mutants of *S. pneumoniae*, lacking recombination proteins, are not UV sensitive (D. Morrison, 6th European Meeting on Transformation and Transfection, Lisbon, Portugal, 1982), although *recA* mutants of *E. coli* that are incapable of SOS repair are sensitive to UV exposure. Does this mean that a nonspecific process of correction eliminates part of the new donor DNA in the process of recombination? The absence of such a system would lead to the high frequency of transformants observed in *S. pneumoniae* and in other fully transformable strains. The efficiency of conjugation in many enteric organisms could involve a different mechanism of recombination, despite the fact the heteroduplex products are similar. The efficiency of transfection in most bacteria (1) would be explained if this process is not dependent on the repair of bacterial DNA.

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