Evolving metabolism of 2,4-dinitrotoluene triggers SOS-independent diversification of host cells

The molecular mechanisms behind the mutagenic effect of reactive oxygen species (ROS) released by defective metabolism of xenobiotic 2,4-dinitrotoluene (DNT) by a still-evolving degradation pathway were studied. To this end, the genes required for biodegradation of DNT from Burkholderia cepacia R34 were implanted in Escherichia coli and the effect of catabolizing the nitroaromatic compound monitored with stress-related markers and reporters. Such a proxy of the naturally-occurring scenario faithfully recreated the known accumulation of ROS caused by faulty metabolism of DNT and the ensuing onset of an intense mutagenesis regime. While ROS triggered an oxidative stress response, neither homologous recombination was stimulated nor the recA promoter activity increased during DNT catabolism. Analysis of single-nucleotide changes occurring in rpoB during DNT degradation suggested a relaxation of DNA replication fidelity rather than direct damage to DNA. Mutants frequencies were determined in strains defective in either converting DNA damage into mutagenesis or mediating inhibition of mismatch repair through a general stress response. The results revealed that the mutagenic effect of ROS was largely SOS-independent and stemmed instead from stress-induced changes of rpoS functionality. Evolution of novel metabolic properties thus resembles the way sub-lethal antibiotic concentrations stimulate the appearance of novel resistance genes.

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