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A sodium fluoride sensitive mutant of Aspergillus nidulans

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Abstract
Fluoride is a widely spread naturally occurring substance in many foods and is used extensively for industrial purposes. The addition of fluoride to drinking water has been assumed to be safe. However, a number of studies have indicated that sodium fluoride is both genotoxic and cytotoxic to mammalian cells (Tsutsui et al. 1984 Mut. Res. 139:193-198). There is conflicting evidence suggesting that NaF is not genotoxic (Kram et al. 1978 Mut. Res. 57:51-55; Martin et al. 1979 Mut. Res. 66:159-167; Li et al. 1987 Mut. Res. 192:191-202) and can suppress the activity of polyfunctional alkylating agents (Obe and Slacik-Erben 1973 Mut. Res. 18:369-371).

A sodium fluoride sensitive mutant of Aspergillus nidulans

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Fluoride is a widely spread naturally occurring substance in many foods and is used extensively for industrial purposes. The addition of fluoride to drinking water has been assumed to be safe. However, a number of studies have indicated that sodium fluoride is both genotoxic and cytotoxic to mammalian cells (Tsutsui et al. 1984 Mut. Res. 139:193-198). There is conflicting evidence suggesting that NaF is not genotoxic (Kram et al. 1978 Mut. Res. 57:51-55; Martin et al. 1979 Mut. Res. 66:159-167; Li et al. 1987 Mut. Res. 192:191-202) and can suppress the activity of polyfunctional alkylating agents (Obe and Slacik-Erben 1973 Mut. Res. 18:369-371).

Here we report that the alkylating agent-sensitive strains of Aspergillus nidulans L452 (pabaA1;wA3;sagB2), isolated in this laboratory (Swirski et al. 1988 Mut. Res. 193:255-268), also showed an increase in sensitivity to the presence of NaF in the growth medium. In mapping experiments using strain L452 and the derivative strain L455 (yA2;pyroA4;sagB2), it was found that hyphal growth of these strains was completely inhibited by 75 mM NaF. The parent strain L20 (pabaA1;wA3) grew on 75 mM NaF at 50- 60% the rate of the NaF-free controls. L452 and L455 also showed an increased sensitivity to KF. Addition of NaCl or KCl to the growth medium to 100 mM in excess of the normal concentrations had no effect on the growth rates of strains L452, L455, and L20.

The concurrent appearance of an NaF sensitive phenotype in the alkylation repair defective strains of A. nidulans suggested that the effect might be due to the presence of the sagB2 mutations. The sensitivity to NaF and KF of L452 and L455 is not a pleiotropic effect of the sagB2 mutation; this was demonstrated by the separation of the two phenotypes in crosses (R.F.=27-47%). Haploidization studies showed that the mutation which confers sensitivity to NaF is located on chromosome VIII: sagB2 is also located on chromosome VIII. The sensitivity to NaF and KF of these strains is functionally recessive to the wild-type.

Other notable NaF sensitive mutants of A. nidulans are the palB strains (e.g. G832 - galH7;facB101 riboB2 palB7 chaA1); the sensitivity in this case is due to a defective alkaline monophosphoesterase activity (Dorn 1965 Genet. Res. Camb. 6:13- 26). Our strains showed wild-type acid and alkaline monophosphoesterase activities as judged by histological staining methods. Unlike the palB strains, the internal pH of L20, L452, and L455 was found to be wild type using the method of Caddick et al. 1984 Mol. Gen. Genet. 203:346-353. The wild-type NaF response of an L455::G832 diploid strain provided further evidence that our mutation was not a variant of the palB locus mutation. Furthermore, both L452 and L455 showed a wild-type response to X-irradiation in an oxygen-free environment. This suggests that the mutation conferring NaF sensitivity on these strains is unlikely to cause a defect in the radiation response/repair systems.

In conclusion, we have revealed a functionally recessive mutation on chromosome VIII of the A. nidulans strain L452 which results in an increased sensitivity to NaF. The data show that this mutation probably arose by an independent event during the mutagenesis procedure used to induce the sagB2 mutation. This new locus has been designated nfsA; its function is as yet unknown.

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