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Author	武田, 啓(Takeda, Kei) 鵜川, さと子(Ukawa, Satoko) 望月, 正隆(Mochizuki, Masataka)
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Inhibition by Fatty Acids on Direct Mutagenicity of *N*-Nitroso Compounds*

Kei TAKEDA, Satoko UKAWA and Masataka MOCHIZUKI

武田　啓，鵜川さと子，望月正隆

N-Nitroso compounds are suspected to be one of the causes of human cancer. It is important to find out suppressive factors of mutagenicity by *N*-nitroso compounds in view of protecting human from cancer. We found that fatty acids suppressed direct acting mutagenicity of *N*-nitroso compounds in *S. typhimurium* TA1535, *E. coli* WP2 and WP2 *hcr*$^-$, and *E. coli* H/r30R (wild) and Hs30R (*uvr* A). The suppressive activity was dependent on the concentration of fatty acids, and fatty acids with longer alkyl chain showed stronger suppression. The suppression was observed in α-oxygenated derivatives of *N*-nitrosodialkylamines with hydroxy or hydroperoxy groups and in *N*-nitroso-*N*-alkylureas, but hardly observed in those with acetoxy and phosphonooxy groups. The suppressive effect was strongest in α-hydroxy nitrosamines, active metabolites of *N*-nitrosodialkylamines. A half-life of decomposition of α-hydroxy nitrosamines or *N*-nitroso-*N*-alkylureas was similar in phosphate buffer and in fatty acids solutions. Partitioning property of the mutagens was altered by the addition of fatty acids, but its degree was not enough to explain the amount of suppression. No significant difference in the alkylating activity of the *N*-nitroso compounds in phosphate and in acetate buffer was also observed. A stronger suppression of mutagenicity by butylating mutagen was detected in *E. coli* WP2 than in WP2 *hcr*$^-$ and *E. coli* H/r30R than in Hs30R, suggesting an involvement of excision repair as a possible mechanism of suppression. Mutagenicity and cytotoxicity of α-hydroxy nitrosamines in Chinese hamster V79 cells were also suppressed by acetic acid.

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