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Title	Metabolism of N-nitrodialkylamines
Sub Title	
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Publisher	共立薬科大学
Publication year	1984
Jtitle	共立薬科大学研究年報 (The annual report of the Kyoritsu College of Pharmacy). No.29 (1984.) ,p.101- 101
JaLC DOI	
Abstract	
Notes	学会講演要旨
Genre	Technical Report
URL	https://koara.lib.keio.ac.jp/xoonips/modules/xoonips/detail.php?koara_id=AN00062898-00000029-0106

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A similar order was observed also in the cytotoxicity. Mutagenic and cytotoxic potencies of these α-hydroxy *N*-nitrosamines in V79 cells were well correlated not only with those of model compounds, α-acetoxy and α-hydroperoxy *N*-nitrosamines, but with their alkylating ability measured by alkylation of thiophenol. The mutagenic activity of the α-hydroxy *N*-nitrosamines in V79 cells was shown to be in parallel with that in *Salmonella typhimurium* TA1535 and with that of *N*-nitrosodialkylamines in V79 cells after metabolic activation by rat hepatocytes. The results obtained here supported further that the α-hydroxy *N*-nitrosamine is the active species in the metabolic activation of carcinogenic and mutagenic *N*-nitrosodialkylamines.

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Metabolism of N-Nitrodialkylamines

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〔8th International Meeting on *N*-Nitroso Compounds (Banff, Canada, September, 1983) で発表〕

In vitro and *in vivo* metabolism of *N*-nitramines were investigated to compare their mode of action with that of *N*-nitrosamines. *N*-Nitrodibutylamine and *N*-nitrodiethylamine were incubated with rat liver microsomes and hepatocytes, and the products were analyzed by HPLC and GLC. The *in vitro* metabolic pattern of these *N*-nitramines was quite similar to that of the corresponding *N*-nitrosamines except that *N*-nitromonoalkylamines (produced by α-hydroxylation) were isolated and characterized in the incubation with the *N*-nitrodialkylamines. Seven *N*-nitramines including glucuronides were isolated and identified from urine of rats given *N*-nitrodibutylamine, which were produced by ω, ω-1, and α oxidations of the *N*-nitramine. The *in vivo* metabolic pattern of *N*-nitrodibutylamine was also similar to that of *N*-nitrosodibutylamine, except that *N*-nitromonobutylamine (a product of α-hydroxylation) was isolated and characterized.

N-Nitramines were mutagenic to *E. coli* WP2 *hcr*$^-$ but not to *S. typhimurium* TA1535. *N*-Nitrodibutylamine and *N*-nitrodiethylamine were mutagenic only in the presence of hepatic microsomes, while *N*-nitromonobutylamine and *N*-nitromonoethylamine were direct mutagens. Thus, the *N*-nitrodialkylamine is also metabolically activated to a mutagen through α-hydroxylation.

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