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论著

低浓度甲基硝基亚硝胍激活p38MAPK信号转导通路

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摘要 在低浓度甲基硝基亚硝胍（MNNG）诱发猴肾vero细胞遗传不稳定的实验模型中，曾经证明受试细胞中酪氨酸磷酸化蛋白谱的改变和JNK/SAPK信号通路的激活. 同样条件下，现又发现p38MAPK及其上游激酶MKK3/MKK6，以及JNK/SAPK的上游激酶SEK1/MKK4的磷酸化程度增高，提示低浓度MNNG可通过激活MAPK家族的两条应激信号转导通路诱导细胞的应激反应，且不同通路之间可能存在交互作用。

关键词 应激, 细胞 信号转导 甲基硝基亚硝胍

分类号 Q257

Low concentration *N*-methyl-*N*′-nitro-*N*-nitroguanidine activates p38MAPK signal transduction pathway

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Abstract

An experimental model in which alkylating agent *N*-methyl-*N*′-nitro-*N*-nitroguanidine (MNNG) with low concentration was employed to induce genetic instability of a m*onkey* kidney vero cell line, it was proved that there were changes in the patterns of protein tyrosine residue phosphorylation and the activation of stress activated kinase(JNK/SAPK). Now with the same experimental conditions, it is discovered that the phosphorylation degree increased in p38MAPK and its upstream kinase MKK3/MKK6 and upstream activator SEK1/MKK4 of JNK/SAPK, suggesting that both stress signaling pathways in mitogen activated protein kinase(MAPK) family be served by MNNG to activate cellular stress response and there may be cross talks between different pathways.

Key words stress cells signal transduction N-methyl-N′-nitro-N-nitroguanidine

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