

论著

非律平和制霉菌素对人羊膜上皮细胞鞘脂类代谢的影响

刘广义¹, 马小琼², 沈静², 董正伟¹, 杨军^{1*}

(浙江大学 1. 公共卫生学院毒理学系, 2. 医学院病理及病理生理学教研室, 浙江 杭州 310058)

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摘要 目的 研究脂类干扰剂非律平和制霉菌素对人羊膜上皮细胞鞘脂类代谢的影响是否相同。方法 应用基质辅助激光解吸飞行时间质谱分析方法分析不同剂量非律平和制霉菌素对人FL细胞鞘脂类代谢的影响。结果 非律平和制霉菌素均可影响FL细胞鞘脂类代谢, 但是非律平(0.2 和10 $\mu\text{mol} \cdot \text{L}^{-1}$)诱导了多种新的神经酰胺类分子的合成, 推测主要影响神经酰胺类分子的代谢。而制霉菌素(0.054 和0.108 $\mu\text{mol} \cdot \text{L}^{-1}$)在诱导了新的神经酰胺分子合成的同时, 还导致鞘磷脂含量的增高, 0.108 $\mu\text{mol} \cdot \text{L}^{-1}$ 可诱导更多神经酰胺分子的合成, 推测制霉菌素可能对鞘磷脂类分子和神经酰胺类分子的代谢都有影响。结论 非律平和制霉菌素均干扰鞘脂类代谢, 但二者的靶点可能不同。

关键词 [非律平](#) [制霉菌素](#) [脂类组学](#) [神经酰胺类](#) [鞘磷脂类](#)

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Effects of filipin and nystatin on sphingolipids metabolism in human amnion epithelial cells

LIU Guang-Yi¹, MA Xiao-Qiong², SHEN Jing², DONG Zheng-Wei¹, YANG Jun^{1*}

(1. Department of Toxicology, School of Public Health, 2. Department of Pathology and Pathophysiology, School of Medicine, Zhejiang University, Hangzhou 310058, China)

Abstract

AIM To study whether macrolides polyene antibiotics filipin and nystatin have different effects on sphingolipids metabolism in human amnion epithelial (FL) cells. **METHODS** After FL cells were treated with two concentrations of filipin (0.2 and 10 $\mu\text{mol} \cdot \text{L}^{-1}$) and nystatin (0.054 and 0.108 $\mu\text{mol} \cdot \text{L}^{-1}$), respectively, sphingolipids were extracted and subjected to matrix-assisted, laser desorption-ionizing-time of flight mass spectrometry analysis. **RESULTS** Both filipin and nystatin affected sphingolipids metabolism. However, filipin (0.2 and 10 $\mu\text{mol} \cdot \text{L}^{-1}$) mainly affected ceramide, and induced the generation of many new ceramide species. On the other hand, nystatin (0.054 and 0.108 $\mu\text{mol} \cdot \text{L}^{-1}$) affected both ceramide and sphingomyelin. In addition to the induction of new ceramide species, nystatin also increased the concentration of several sphingomyelin species. Nonetheless, the higher concentration of nystatin (0.108 $\mu\text{mol} \cdot \text{L}^{-1}$) induced the generation of more ceramide species than the lower concentration of nystatin (0.054 $\mu\text{mol} \cdot \text{L}^{-1}$). **CONCLUSION** Both filipin and nystatin affect sphingolipids metabolism, however they may target different steps in the sphingolipids metabolism pathway.

Key words [filipin](#) [nystatin](#) [lipidomics](#) [ceramides](#) [sphingomyelins](#)

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通讯作者 杨军 gastate@zju.edu.cn

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