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Increased Zaltoprofen-Irinotecan Interaction in the Patients with Colon Cancer

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***SUMMARY*. Irinotecan is the first-line anti-tumor drug, and zaltoprofen is a nonsteroidal anti-inflammation drug. Irinotecan-zaltoprofen interaction was determined in this study, and the irinotecan-zaltoprofen interaction was compared between normal human intestinal microsomes (nHIMs) and tumor human intestinal microsomes (tHIMs) incubation system. Human intestinal microsomes (HIMs) were prepared from colon cancer and surrounding tissues, and the inhibition of zaltoprofen on the glucuronidation of irinotecan active metabolite SN-38 was determined. In the incubation system with normal human intestinal microsomes, 100 μM of zaltoprofen inhibits 45.8% activity of SN-38 glucuronidation catalyzed by normal human intestinal microsomes (nHIMs). Compared with nHIMs incubation system, the inhibition of zaltoprofen on the glucuronidation of SN-38 was stronger in tumor human intestinal microsomes (tHIMs), with 81.5% activity inhibited. Furthermore, UGT1A1-catalyzed metabolism of 4-MU was significantly ($p < 0.001$) inhibitied by 100 μM of zaltoprofen. Much attention should be given for zaltoprofen-irinotecan interaction in the patients with colon cancers.**

RESUMEN. Irinotecan es un fármaco antitumoral de primera línea y zaltoprofeno un anti-inflamatorio no esteroide. En este estudio se determinó la interacción irinotecan-zaltoprofeno y se comparó el sistema de incubación entre microsomas intestinales humanos normales (nHIMs) y microsomas intestinales humanos tumorales (tHIMs). Los microsomas intestinales humanos (HIMs) se prepararon a partir de cáncer de colon y los tejidos circundantes y se determinó la inhibición de zaltoprofeno en la glucuronidación del metabolito activo de irinotecan, SN-38. En el sistema de la incubación con microsomas intestinales humanos normales, 100 μM de zaltoprofeno inhibe la actividad de 45,8% de glucuronidación de SN-38 catalizada por microsomas intestinales humanos normales (nHIMs). En comparación con el sistema de incubación nHIMs, la inhibición de zaltoprofeno en la glucuronidación de SN-38 fue más fuerte en microsomas intestinales de tumor humanos (tHIMs), con actividad de inhibición del 81,5%. Además, el metabolismo de 4-MU catalizado por UGT1A1fue significativamente ($p < 0,001$) inhibido por 100 μM de zaltoprofeno. Mucha atención se debe prestar a la interacción zaltoprofeno-irinotecán en pacientes con cáncer de colon.

KEY WORDS: colon cancers, drug-drug interaction, irinotecan, zaltoprofen.

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