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Platelet-Derived Growth Factor-B Receptor/P38 Pathway May Be the Potential Liver Damage Mechanisms Caused by Saikosaponin D

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Abstract : SaikosaponinD (SSD) is a major component of saikosaponins isolated from Bupleurumfalactum. Our current study was to examine the toxic effect of SSD on liver cells and explore the possible mechanism. The results demonstrated that SSD induced mouse liver injury and led to apoptosis in LO2 cells. HE staining and TUNEL analyses showed that SSD stimulated liver injury and hepatocyte apoptosis in vivo. Subsequent experiments showed that SSD down-regulated Bcl-2 but up-regulated Bax. In vitro, SSD-treated LO2 cells exhibited apparent down-regulated expression of p-p38. Moreover, PDGF-βR agonist PDGF-BB alone significantly upregulated p38 phosphorylation, while combined with SSD, p38 phosphorylation expression was reduced. Furthermore, shRNA-mediated PDGF-βR knockdown augmented the inactivation of p-p38 and Bcl2 but abrogated the activation of Bax, these results were more obvious when shRNA combined with SSD. These data indicated that SSD stimulated liver injury and apoptosis in hepatocytes and PDGF-βR /p38 pathway may be the potential mechanistic.

Keywords : saikosaponin D, hepatotoxicity, liver injury, apoptosis, platelet-derived growth factor-β receptor, p38
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