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摘要:

以培养血管平滑肌细胞(vascular smooth muscle cell, VSMC)为模型,观察了间硝苯地平(*m*-nifedipine, *m*-Nif)对血管紧张素Ⅱ(angiotensin II, ANG II)促进VSMC增殖和蛋白质合成的影响。结果表明,*m*-Nif抑制ANG II ($100\text{nmol}\cdot\text{L}^{-1}$)引起VSMC [^3H]thymidine和 [^3H]leucine参入,并呈剂量依赖性。*m*-Nif($2\times10^{-6}\text{mol}\cdot\text{L}^{-1}$)可抑制ANG II对VSMC的刺激、DNA及蛋白质合成速率,分别降低了46%, 58%, 53%。提示*m*-Nif可抑制ANG II对VSMC增殖和蛋白合成的促进作用。

关键词: 间硝苯地平 血管紧张素Ⅱ 血管平滑肌细胞 蛋白质合成

I N H I B I T O R Y E F F E C T S O F *M*-N I F E D I P I N E O N A N G I O T E N S I N I I S T I M U L A T E D P R O L I F E R A T I O N A N D P R O T E I N S Y N T H E S I S I N C U L T U R E D R A B B I T A O R T I C S M O O T H M U S C L E C E L L S

HB Gu; YH Tang and Y Xu

Abstract:

By using rabbit aortic smooth muscle cells(VSMC) in culture as a model, the effects of *m*-nifedipine(*m*-Nif) on the growth of VSMC were observed. The results showed that *m*-Nif inhibited [^3H]-thymidine and [^3H]-leucine incorporation into DNA and protein, decreased cell number in a concentration-dependent manner in angiotensin II (ANG II) induced VSMC. At the concentration of $2\ \mu\text{mol}\cdot\text{L}^{-1}$, *m*-Nif was found to reduce the ANG II ($100\ \text{nmol}\cdot\text{L}^{-1}$) stimulated [^3H]-thymidine, [^3H]-leucine incorporation and cell number by 46%, 58% and 53%, respectively. Our results suggest that *m*-Nif can inhibit ANG II stimulated proliferation and protein synthesis in cultured rabbit aortic VSMC.

Keywords: Angiotensin II Smooth muscle cell Protein synthesis *m*-Nifedipine

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