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1-磷酸鞘氨醇对过氧化氢所致微血管通透性增高的影响

Effect of Sphingosine 1-Phosphate on the Increased Microvessel Permeability Induced by H_2O_2

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中文关键词： 血管通透性 1-磷酸鞘氨醇 过氧化氢 静水传导性 血管内皮细胞钙粘蛋白 肌动蛋白

英文关键词:microvessel permeability sphingosine 1-phosphate hydrogen peroxide hydraulic conductivity vascular endothelial-cadherin actin

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中文摘要:

目的 研究1-磷酸鞘氨醇(S1P)对氧化应激状态下过氧化氢引起的大鼠微血管通透性增高的影响。方法 应用大鼠在体肠系膜微灌注的方法，通过外源性给予过氧化氢模拟病理条件下氧化应激状态，并通过测定微静脉的静水传导性(hydraulic conductivity，p)，观察S1P对过氧化氢引起的微血管通透性增高的影响；并利用在体免疫荧光组织化学技术，观察S1P对过氧化氢引起的内皮细胞连接主要成分钙粘蛋白(VE-Cadherin)、F-肌动蛋白变化的影响。结果 过氧化氢可增加微血管Lp至正常对照的6.13±0.87倍(P<0.0微血管经S1P预处理后，再给予过氧化氢未出现Lp的明显变化，与对照值比较无显著性差异。免疫荧光组织化学检测结果显示：过氧可改变内皮细胞F-肌动蛋白形态及分布，细胞F-肌动蛋白分布紊乱不规则，细胞内部有密集的应力纤维形成。过氧化氢也可影响VE-erin正常结构，导致粘附连接断裂，细胞间隙形成。而S1P预处理可抑制过氧化氢对VE-Cadherin和F-肌动蛋白的影响。结论 S1P可过氧化氢引起的血管通透性增高，其机制可能与S1P抑制应力纤维形成、加强内皮细胞间粘附连接，抑制细胞间隙形成有关。

英文摘要:

OBJECTIVE To study the effect of sphingosine 1-phosphate (S1P) on the increased microvessel permeability of r induced by hydrogen peroxide(H2O2). METHODS The method of rat mesenteric microvascular perfusion in vivo was used. Microvessel permeability was assessed by measuring hydraulic conductivity(Lp). The microvessel were stained with immunofluorescence technique and examined with laser confocal microscopy to observe the effect of S1P on the change vascular endothelial-cadherin (VE-Cadherin) and F-actin caused by H2O2. RESULTS H2O2 increased Lp to 6.1±0.9 times the control(P<0.01), but after pretreatment with S1P, H2O2 did not give rise to a further significant change. Immunofluorescence study showed that H2O2 could change F-actin cytoskeletal architecture. F-actin arranged disorder and irregularly. Formation of stress fiber was observed in the middle part of cell. H2O2 could also restructure VE-Cadherin. Detachment of adherent junction and formation of endothelial gap was observed. Pretreatment with S1P coul inhibit the change of VE-Cadherin and F-actin induced by H2O2. CONCLUSION S1P can improve the increased microvessel permeability caused by H2O2, which might mediated by inhibiting the formation of stress fibre and endothelial gaps, strengthening adherent junction.

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