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论著

葡萄糖PDS对单层人腹膜间皮跨细胞电阻及迁移修复能力的影响

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摘要
目的：观察不同浓度葡萄糖PDS（PDS）对单层人腹膜间皮细胞（HPMCs）跨细胞电阻（TER）以及迁移修复能力的影响。方法：用不同葡萄糖浓度PDS(1.5%,2.5%,4.25%)分别与DMEM以1∶1比例混合后体外培养HPMCs。四甲基偶氮唑盐（MTT）比色法测定细胞增殖。使用双池培养皿(transwell)构建HPMCs单层细胞模型。采用TER技术测定PDS对HPMCs通透性的影响。划痕损伤实验观察葡萄糖对HPMCs修复再生能力的影响。结果：不同浓度葡萄糖PDS(1.5%,2.5%,4.25%)干预均可明显地抑制HPMCs的增殖(P＜0.05)。TER值随PDS干预时间的延长而逐渐下降，且与葡萄糖浓度呈负相关(P＜0.01)。葡萄糖PDS可明显抑制HPMCs迁移修复能力，且与葡萄糖浓度相关。结论：高糖PDS液抑制细胞增殖，降低单层HPMCs的TER值，并抑制HPMCs损伤后迁移修复能力。提示PDS中高糖成分是导致腹透患者腹膜高通透性和超滤衰竭的重要因素。

关键词 人腹膜间皮细胞；跨细胞电阻；细胞迁移；腹膜透析
分类号

Effect of glucose peritoneal dialysates on the transmesothelial electrical resistance and cellular migration of monolayer human peritoneal mesothelial cell

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Abstract
ObjectiveTo investigate the effect of different concentrations of glucose peritoneal dialysates (PDS) on monolayer transmesothelial electrical resistance (TER) and migration ability of cultured human peritoneal mesothelial cells (HPMCs) to clarify the cause of peritoneal hyperpermeability state and ultrafiltration failure during prolonged peritoneal dialysis. MethodsHPMCs were cultured in a 1∶1 mixture of DMEM and PDS containing 1.5%, 2.5%, and 4.25% glucose. Methyl thiazolyl tetrazolium (MTT) assay and TER were measured to determine the effect of glucose PDS on the proliferation and permeability of human peritoneal mesothelial monolayers, respectively. Wound-healing assay was used to confirm whether glucose could do harm to the migration of cells. ResultsProliferation of HPMCs was significantly suppressed by different glucose concentrations at 24 hours. TER decreased in a time- and concentration-dependent manner after culture with different concentrations of glucose PDS. Cells lost migration in the presence of high glucose after 24 hours, and most cells lost their normal morphology and became detached from plates after 48 hours of wounding. ConclusionHigh glucose in PDS can cause peritoneal damage by suppressing cell proliferation, inducing increase in paracellular permeability of HPMCs and inhibiting cell migration after damage, which may be responsible for peritoneal hyperpermeability and the development of ultrafiltration failure.

Key words human peritoneal mesothelial cell transmesothelial electrical resistance cell migration peritoneal dialysis
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