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· 科研新闻速递 ·

葡萄糖-6-磷酸脱氢酶缺乏症与脓毒症引起的炎症反应有关

葡萄糖-6-磷酸脱氢酶(G-6-PD)缺乏症是人类常见遗传多态现象。一方面此多态性可以减少疟疾发生率，另一方面严重创伤后缺乏 G-6-DP 会使临床病情恶化。最近美国科研人员用体外和体内实验研究了 G-6-PD 缺乏症与脂多糖(LPS)/细菌性脓毒症[盲肠结扎穿孔术(CLP)]后细胞因子和死亡率改变之间的关系。实验采用等位基因特异性聚合酶链反应(PCR)法检测了动物的基因型，采用基因序列分析、酶联免疫吸附法(ELISA)和流式细胞术(FACS)测定了巨噬细胞和脾细胞对内毒素的反应性，并观察了不同处理组动物的死亡率。实验用野生型和 G-6-PD 突变型小鼠进行杂交构建 G-6-PD 缺乏症动物模型，其 G-6-PD 缺乏程度类似于非洲人种中的 G-6-PD 缺乏症患者(正常值的 20%)。结果显示：与野生型小鼠相比，LPS 刺激后(10～35 mg/kg 腹腔注射)，G-6-PD 缺乏型小鼠血清和腹腔液中的白细胞介素-1β(IL-1β)、IL-6 和 IL-10 水平均明显升高，其死亡率也明显升高(40%～70%比 5%～40%)。但 CLP 诱导的脓毒症中，G-6-PD 缺乏症小鼠与野生型小鼠无论是否给予补液或抗生素治疗，其死亡率差异均无显著性。G-6-PD 缺乏症小鼠和野生型小鼠的脾脏和血液巨噬细胞在体外对 LPS 的反应性均降低。因此研究者认为，G-6-PD 缺乏使动物细胞因子受炎症刺激后的反应性升高，但 G-6-PD缺乏对生存率的不利影响会被严重脓毒症后出现的免疫抑制状态所掩盖。

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