· SHOCK 速递 ·

《SHOCK》2024 年第 6 期新观点

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本期（SHOCK）杂志刊出了 18 篇文章，其中综述 1 篇，临床研究 5 篇，基础研究 12 篇。研究内容聚焦脓毒症急性肾损伤、危重症患者运输和热损伤等领域，在脓毒症药物研发、蛋白质酰基化修饰和代谢重编程等方面提供了新观点。

脓毒症（sepsis）是一种危及生命的感染性疾病，其发病机制尚不完全清楚。姜黄素具有抗炎作用，可能对脓毒症的治疗有效。Tao 等总结了目前使用姜黄素治疗脓毒症的应用现状[1]。

侵袭性真菌感染诊断延迟与患者不良预后高度相关。在这项回顾性研究中，Cao 等人基于机器学习算法开发了重症患者侵袭性真菌感染的临床预测模型[2]。

触珠蛋白（haptoglobin）可清除溶血过程中释放的无细胞血红蛋白，溶血是脓毒症、急性呼吸窘迫综合征（acute respiratory distress syndrome，ARDS）或体外膜氧合患者的常见并发症。Bünger 等人研究了触珠蛋白水平与 ARDS 患者预后之间的关系[3]。

正确使用强心药物是早期治疗感染性休克患者的关键。Qu 等研究了中心静脉-动脉血二氧化碳分压差对早期感染性休克患者强心药物使用的意义[4]。

急性肾损伤是脓毒症的主要并发症。在这项研究中，Wang 等人研究了维生素 B6 的应用是否能减轻脓毒症患者的肾损伤[5]。

MacArthur 等评估了新型息肉蛋白拮抗剂对创伤患者血浆中凝血酶生成的影响[6]。

危重症患者在航空医疗运送过程中的低压低氧环境可导致肺损伤。Yin 等利用模拟航空医疗后运送急性肺损伤大鼠模型，发现线粒体自噬可能在航空医疗诱导的继发性肺损伤中起重要作用[7]。

在宿主对感染的反应中观察到不同的体温调节趋势（高温和低温）。在本研究中，Moretti 等人证明，低温并不依赖于与脑氧合的反馈，而是依赖于一种前馈机制，即在预测脑氧合变化时即触发低温[8]。

治疗延迟对战伤救治效果有重要影响。Penn 等研究了人白蛋白是否可以改善创伤和出血大鼠模型的血流动力学[9]。

热损伤引起的高代谢重编程被认为是导致发病率和死亡率增加的原因。Bieerkehazhi 等研究了一种可逆的翻译后修饰-蛋白质 s-酰基化（S-acylation），并探究了其是否有助于烧伤诱导的高代谢重编程[10]。

Fanous 等利用盲肠结扎和穿刺的新生猪模型，发现早期实施复苏可减轻炎症、急性肾损伤和急性肝损伤[11]。

Shi 等通过孟德尔随机化研究（mendelian randomization，MR）方法，研究了单纯疱疹病毒 1 型（Herpes Simplex Virus 1，HSV-1）对脓毒症的影响[12]。

Meng 等利用非靶向代谢组学，证明甘草酸二铵通过影响灭活 Toll 样受体 3（toll-like receptor 3，TLR3）的表达，调节多个代谢通路并缓解病毒 dsRNA 合成模拟物聚肌胞（Polyinosinic-polycytidylic acid，poly（I：C））诱导的肺炎[13]。

脓毒症诱导心肌细胞凋亡。Xing 等通过盲肠结扎穿孔脓毒症大鼠模型，证明内源性 β3 肾上腺素的激活可减少脓毒症诱导的心肌细胞凋亡，并维持固有的心肌收缩功能[14]。

脓毒症与调节性 T 细胞中腺苷浓度和叉头框蛋白（FoxP3）表达增加相关。Zhang 等使用了脓毒症的盲肠结扎模型，发现腺苷受体 A2A 诱导转录因子 CREB 的磷酸化，从而增加调节性 T 细胞中 FoxP3 表达的证据[15]。

心脏骤停后复苏与心脏骤停后综合征导致的死亡风险增加相关。Li 等研究了高氧和目标温度管理对大鼠心脏停搏后综合征的影响[16]。

Huang 等研究了 miR-208a-3p 在缺血/再灌注诱导的急性肾损伤中的作用。作者证明 miR-208a-3p 可以影响多种细胞过程，最终减少组织损伤[17]。

Chen 等发现，Mst1/ERS 信号通路参与了血管内皮细胞功能障碍和急性肺损伤的发展，此外，复方葛根素通过抑制 Mst1/ERS 通路，减轻内皮细胞功能障碍和肺损伤[18]。

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