



Review

Gut Microbiota and Critically Ill Patients: Immunity and Its Modulation via Probiotics and Immunonutrition

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Abstract: Critically ill patients have a hyper-inflammatory response against various offending injuries that can result in tissue damage, organ failure, and fatal prognosis. The origin of this detrimental, uncontrolled inflammatory cascade can be found also within our gut. In detail, one of the main actors is our gut microbiota with its imbalance, namely gut dysbiosis: learning about the microbiota’s dysfunction and pathophysiology in the frame of critical patients is of crucial and emerging importance in the management of the systemic inflammatory response syndrome (SIRS) and the multiple organ dysfunction syndrome (MODS). Multiple pieces of evidence indicate that the bacteria that populate our gut efficiently modulate the immune response. Treatment and pretreatment with probiotics have shown promising preliminary results to attenuate systemic inflammation, especially in postoperative infections and ventilation performance. Finally, it is emerging how immunonutrition may exert a possible impact on the health status of patients in intensive care. Thus, this manuscript reviews evidence from the literature on gut microbiota composition, its derangement in critically ill patients, its pathophysiological role, and the described and emerging opportunities arising from its modulation.

Keywords: intensive care; gut microbiota; inflammation; probiotics; antibiotics; nutrition



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1. Introduction

Patients affected by critical illnesses develop a potentially lethal body reaction to damage (e.g., infection, trauma, burning). The tissue damage may worsen up to the point where it causes deterioration of the organs’ function and eventually leads to their failure. Thus, the scythe of death comes closer and closer. For example, almost 50 million patients with sepsis are reported throughout the world, with a mortality of about 20% [1]. In addition, trauma is the leading cause of death in younger age [2,3]. These conditions ignite a physiological innate and adaptive immune systems response, able to manage pathogens proliferation across tissues, organs, and the whole organism. In detail, some main players of this systemic inflammatory response to damage have been described: pathogen-associated pattern molecules (PAMPs) or damage-associated pattern molecules (DAMPs). These are involved in the systemic inflammatory response syndrome (SIRS), the multiple organ dysfunction syndrome (MODS), and the related compensatory anti-inflammatory response syndrome (CAIRS) [3,4]. Indeed, prolonged SIRS suppresses Th1-type immune and increases Th2-type responses. Altogether, both features of SIRS can lead

Gut Dysfunction In Critically Ill Patients

Sebastian Brünink



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