Daniel Martin Arsanios et al., J Diabetes Metab 2018, Volume 9
DOI: 10.4172/2155-6156-C1-078

Insulin, more than a metabolic hormone: Focus in sepsis

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Sepsis is defined as deleterious response of host towards a pathogen, in which the number of physiopathological phenomenon implied lead to cell dysfunction, multi-organ failure and death. In this unregulated response, hormones change its functions and metabolism. One example is insulin, a peptide hormone which is not only implied on the glucose's homeostasis and growth pathways but also function as a mediator of inflammatory, hemodynamic and micro-vascular processes involving sepsis. Insulin inhibits the production and secretion of substances implied in systemic inflammatory response syndrome like tumor necrosis factor, reactive oxygen species and nuclear factor-kB (NF-kB). Also, it is able to activate protective molecules such as Janus kinase by boosting a counter-regulation mechanism to the damage induced by NF-kB and to the phosphorylation of pathways related to mitogen-activated protein kinases, inhibiting pro-inflammatory proteins (ERK, P38) involved in cell damage, by activation of P21-RAS. Evidence supports the contribution of insulin resistance to the inflammatory phenotype that is similar in obesity microvasculature. This also leads to endothelial cell dysfunction, manifested as an impaired capacity of microvasculature to relax in response to endothelium-dependent vasodilators, favoring the binding of both leukocytes and platelets to the vascular wall. Insulin administration can attenuate this response as well as suppress genes commanding inflammatory cytokines. Based on its anti-inflammatory, hemodynamic, hormonal and regulating mechanisms of fatty acids, carbohydrates and proteins, attempts have been made to support the use of insulin during sepsis and septic shock in practice. On one side, there is evidence of mortality reduction rate of up to 3% in the ICU patient after its use greater than 28 days additionally showed a reduction in morbidity by decreasing renal injury until 8.9%, mechanical ventilation and ICU stay (p=0.05). It is important to know all the possible pathways where the insulin works to improve the utility in septic patient.

Biography

Daniel Martin Arsanios is a Doctor at Sabana University in Colombia. He has experience in molecular biology and in conjunction with issues of sepsis and sought to go deeper into issues related to the development and complications of microvasculature in sepsis.

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