壓力導致的糖類代謝改變

Stress-induced Alterations in Glucose Metabolism in Critical Illness

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Acute illness such as trauma, burn, sepsis, and pancreatitis is associated with development of hyperglycemia. Often referred to as stress-induced diabetes, traumatic diabetes, or diabetes of injury, this condition is different from diabetes mellites in that it is normally presented with normal or elevated blood insulin levels and with increased peripheral insulin resistance. The metabolic response to injury/stress is divided into hypo- (ebb phase) and hyper-metabolism (flow phase). Hyperglycemia during ebb phase is mainly due to hepatic and muscle glycogenolysis, while hyperglycemia during flow phase is mainly due to hepatic gluconeogenesis (as a result of protein catabolism) and insulin resistance to adipose and skeletal muscle tissues. Current standard of care involves tight control of blood sugar between 140-180 using insulin. However, new researches provided evidence pointing to the possibility of modulating glucagon and manipulating the 'unstoppable' hepatic gluconeogenesis and skeletal muscle catabolism, thereby controlling stress-induced hyperglycemia from the perspective of reducing glucose production.

This lecture will focus on the three systems responsible for translating the initial insult into the stress response, namely the central nervous, the endocrine, and the humoral (inflammatory cytokines) system. Possible mechanisms involved in hyperglycemia due to insuppressible hepatic gluconeogenesis and peripheral insulin resistance involving GLUT4 will also be discussed.