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MOLECULAR ASPECTS INVOLVED IN THE MODULATION OF THE INFLAMMATORY RESPONSE BY INSULIN

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Insulin is a key regulator of the glucose metabolism and has an important anabolic function throughout the body. Insulin controls glucose uptake by many different cells and can modulate various processes where there is need for energy, such as mitogenesis, gene transcription and autophagy. Under certain conditions, for example, diabetes mellitus, the homeostasis of many tissues and organs are affected, leading to an increased mortality due to an enhanced susceptibility to infections. This vulnerability to infection may be partially explained by an inefficient inflammatory response. Several studies in animal models and patients have demonstrated that diabetic individuals have shown ineffective inflammatory response. This deficiency is reflected by a decrease in chemotaxis and neutrophils recruitment, altered production of inflammatory mediators such as cytokines and chemokines, changes in expression of adhesion molecules, the latter two on both: protein synthesis and gene expression. In addition, macrophages from diabetic animals showed decreased phagocytic and microbicidal activities. In most of the parameters studied on this animal model, once the insulin therapy is introduced, these parameters can be reverted. To explore the susceptibility to infections in diabetic patients, the role of insulin in natural immunity against pathogens and inhibiting/reduction of deleterious effects of inflammation, is the nature of my line of research.

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