

Abstract

Monocytes represent a component of the non-adaptive part of the immune system. During their life cycle, they can differentiate into other cell types, such as macrophages, dendritic cells, osteoclasts or microglia. Their activity is associated with a wide variety of signaling mechanisms, mostly with the expression of cell surface Toll-like receptors (TLR) or production of mediators (IL-6 or IL-10). Activation of these pathways is mediated by an external stimulus, which may be a fluctuation in the blood glucose level. Monocytes respond to changes in glucose levels in many ways, overexpression of TLRs, adhesion molecules and release of proinflammatory mediators among them, which makes both, hyperglycemia and hypoglycemia to be proinflammatory states. It increases the risk of pathologies, especially in subjects with impaired glycemetic control – in diabetic patients. These pathologies mainly include cardiovascular diseases (CVD). Thus, cellular and molecular mechanisms described in this thesis contribute to understanding why diabetic patients suffer from a significantly higher CVD risk when compared to healthy non-diabetic subjects.