ABSTRACT

Heart failure is a condition in which the mechanical pumping function of the blood is impaired and the oxygen and nutrients delivery to the tissue is not adequate to meet the needs of the body. PPAR receptors function as nuclear transcription factors of energy metabolism-related genes in cardiomyocytes. The PPARα isoform is a central regulator of myocardial fatty acid metabolism involved in the pathogenesis of heart failure. Activation of PPARα by specific ligands promotes fatty acid uptake, utilization, and catabolism by increasing the expression of genes involved in fatty acid transport, binding, activation, and β-oxidation. Studies to date have highlighted changes in PPARα expression, the influence of a non-functional gene studied in animal models with gene deletion, reduced expression of the coactivator PGC-1α, the influence of PPARα agonists, and the impact of these factors in the development of heart failure. The current results suggest that the level of PPARα expression may become a metabolic marker of cardiomyocytes. This bachelor thesis aims to summarize the current knowledge on PPAR receptors, with a focus on the PPARα isoform, especially with regard to its role in myocardial metabolism during heart failure.

Keywords: nuclear receptor PPARα, metabolism, heart failure