**Summary**

The cells in a multicellular organism communicate a wide variety of mechanisms which include not only the direct transfer from cell-cell contact, but also the secretion of molecules and vesicles traveling through other cells. Mammalian cells secrete a variety of extracellular vesicles (EV). Exosomes are extracellular vesicles with a size between 20-100 nanometers that can transfer information as nucleic acids and proteins from different cells, so they are recognized as a powerful mechanism for intercellular communication. Such vesicles is under intense investigation not only in the process of tumor metastasis, as it is believed that exosomes are related to the extent of tumor to organs not related, but in the field of regenerative medicine, as they are considered potential candidates in the paracrine mechanisms induced by stem cells. Therefore, in this study we analyzed the therapeutic potential of mesenchymal stem cells (MSC) derived exosomes. Thus, we hypothesized that MSCs were able to transfer exosomes to cells that repair tissues. Recent work by our research group showed that overexpression of hypoxia-inducible factor 1α (HIF-1α) in MSC (MSC-HIF) enhanced the therapeutic potential of MSCs to improve cardiac function after acute myocardial infarction (IAM). Accordingly, we studied the content of microRNAs in exosomes derived from MSC and MSC-HIF. We showed that microRNAs populations present in exosomes derived from MSC and MSC-HIF reproducibly changed depending on the oxygen availability for the cells, which we interpret as a cellular response mechanism mediated by exosomes. Moreover, we observed that in the MSC-HIF the combining overexpression of HIF-1α with low oxygen tension generated exosomes loaded with microRNAs with therapeutic potential for the IAM treatment. However, we were unable to demonstrate the therapeutic potential of exosomes isolated from these cell populations in an experimental IAM model in rat, which could be caused by improper design of the experimental procedure. This caused a shift in the thesis orientation and we decided to focus on the cellular mechanisms induced by exosomes in the cardiac environment. In this context, recently described the presence of exosomes in human cardiomyocytes (CM). At ultrastructure level, anatomy of the heart tissue reveals an intrinsic relationship between CM and endothelial cells (ECs) composing the coronary microvascular endothelium, which is responsible for nurturing the CM by transporting metabolic fuel from the blood into CMs. The perivascular space between the CM of ECs is only 1 µm, allowing the flow of information to short distance between the CM and ECs. Keep this anatomical disposition is essential to achieve a metabolic coupling between both cell types. The heart does not have significant reserves of metabolic fuel, so the supply of nutrients and oxygen must be continuous and regulated. Therefore we think that the energy contribution of the endothelium to the heart should be finely coordinated not only by exogenous control of metabolism throughout the body but also for a mechanism in which the cardiomyocyte itself regulates the nutrient transportation of their associated ECs. Also known to the ECs release factors that alter the activity of the CM, and in the same way that the CM releasing factors that alter the ECs. However, little is known about the mechanisms that regulate the flow of nutrients from the ECs to the CM, especially in acute stress situations where an active local regulating endothelial transport mechanism is required. In this work we studied how exosomes derived from CM altered glucose transport in ECs. First we show evidence that glucose starvation increases the synthesis and secretion of exosomes in cultured neonatal rat CM. Secondly we show that these CM derived exosomes are internalized by ECs in a glucose availability dependent manner. Finally we provide evidence that exosomes derived from CM cultured under glucose starvation were able to transfer glucose transporters (GLUTs) to ECs, where these GLUTs transferred via exosomes increased the glucose uptake and the glycolytic activity of ECs. Taking together the results, in this thesis we propose a model of communication between CMs and ECs, in which protein trafficking mediated by exosomes from CM to ECs would move the metabolic needs of the CM to the ECs which are in direct contact with the coronary nutrients flow. This novel short distance action mechanism reveals an intrinsic relationship between glucose demand of CM and glucose transport of ECs allowing a quick response from ECs by increasing the amount of glucose transporters without de novo synthesis or modification of gene transcription profiles, which certainly increases the efficiency of cellular communication process.