Introduction
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The pancreas is a unique organ, comprising both endocrine and exocrine cells. The endocrine cells are grouped in the islets of Langerhans, which were discovered in 1869 by the German pathological anatomist Paul Langerhans (Kloppe., 1969). The islets of Langerhans are dispersed into millions of microorgans scattered among the 100-fold more abundant exocrine tissue. Insulin is a natural peptide hormone made by the pancreas and whose major function is to control tissue uptake and storage of glucose (Jurdjevic and Tillman., 2004). In general, both insulin resistance and impaired insulin secretion are required for manifest type 2 diabetes to occur. However, the mechanisms involved in islet dysfunction remain elusive.

The endogenous islets are richly irrigated, i.e. they have a well-developed vascular network in order to supply oxygen and nutrients to the islets. Notably, capillaries feeding the islets have highly fenestrated endothelium and blood flow is high compared to exocrine pancreas, thus allowing for rapid and efficient delivery of nutrients to islets. These capillaries, resembling a glomerulus, course through the islet in a tortuous manner that is ideal for blood-cell and cell-blood interactions. In addition, the blood flow to the islets has been found to be disproportionately large (~ 10 % of the pancreatic blood flow for the 1-2 % of pancreatic volume). These features create a favorable environment for delivery of substances from the blood to the islet cells. To what extent alterations in pancreatic islet microcirculation contribute to the development of the disturbed glucose homeostasis and insulin secretion in diabetes is unknown at present. This thesis studied the microcirculation of pancreatic islets and insulin secretion in normal and diabetic rats.