**Abstract**

Inhibitory factor 1 (IF1) is one of the major regulators of mitochondrial ATP synthase activity, a key enzyme of energy metabolism. Its inhibitory effects are known in conditions such as hypoxia or starvation, but the hypothesis that IF1 inhibits ATP synthase activity even under physiological conditions is still not entirely accepted. Disorders of ATP synthase regulation can be fatal to the cell and have been described, for example, in carcinogenesis and ischemia. It has also been found that silencing of the IF1 gene in pancreatic β-cells increases insulin secretion, and thus, IF1 may be important in the pathogenesis of type 2 diabetes. The goal of this work is to summarize the current knowledge about the IF1 protein and to obtain new results that will help elucidate the mechanism by which this protein regulates mitochondrial ATP synthase. Specifically, this work deals with the ratio of IF1 protein to ATP synthase in pancreatic β-cells, depending on different culture conditions. It further investigates the occurrence of post-translational modifications of the IF1 protein in pancreatic β-cells (INS-1E model cells), which may play a role in the regulation of IF1 activity. It also deals with the cellular ATP/ADP ratio, which is one of the key factors for insulin secretion by pancreatic β-cells. An important part of this work are also experiments on isolated liver mitochondria of whole-body IF1-knockout mice, in which respiration is examined in the presence of various energy substrates.

**Key words**

IF1, ATP synthase, post-translational modifications, PKA, ATP, mitochondria