Editorial

“Melatonin and Mitochondria Interact in Diseases”

The Turkish Journal of Biology aims to publish special issues that focus on research topics of common interest on a regular basis. To that end, the journal presents this special issue on “Melatonin and Mitochondria Interact in Diseases”. Melatonin (N-acetyl-5-methoxytryptamine) is a low molecular weight indoleamine ubiquitously present in all living organisms. In humans, as in other vertebrates, melatonin is mainly synthesised in the pineal gland from tryptophan or formed as the metabolic end product of serotonin, although many other cells have now been shown to produce melatonin. Additionally, melatonin is a nonenzymatic antioxidant and neuroprotective agent and can be an effective scavenger of reactive oxygen species. Melatonin and its metabolites can also stimulate the activities of several antioxidant enzymes including superoxide dismutase, catalase, glutathione peroxidase, and glutathione reductase. Thus, this hormone can restore the imbalance between prooxidant and antioxidant systems during oxidative stress. Mitochondrial function is essential for cell survival because cells critically depend on ATP synthesis generated by mitochondrial oxidative phosphorylation. Mitochondrial depolarisation activity depends on Ca$^{2+}$ and is fuelled either by Ca$^{2+}$ from the extracellular space when triggered by neuronal activity or by Ca$^{2+}$ released from the endoplasmic reticulum and taken up through a specialised contact site between the endoplasmic reticulum and mitochondria known as mitochondrial associated endoplasmic reticulum membranes. The intrinsic caspase cascade is initiated following diabetes for mitochondrial release of cytochrome c into the cytosol and sequential activation of caspase-3 and caspase-9 for neuronal death. Extensive studies have already confirmed the activation of caspases of the intrinsic pathways for modulation of apoptosis in different cells. For this special issue we have invited expert reviews or original research papers on therapeutic targets of melatonin regarding the roles of mitochondrial function and apoptosis. We hope that the research presented here will be both informative and stimulating for the scientific community in general and for the readers of the Turkish Journal of Biology.