Bridges in Life Sciences 8th Annual Scientific Conference
"Laugh and Be the Best in Research and Patient Care" ™

RECOOP HST ASSOCIATION

Hotel ILF Prague
April 5–7, 2013
RECOOP HST Association

Association for Regional Cooperation in the Fields of Health, Science and Technology

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Visegrad Fund

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Cedars-Sinai Medical Center, Los Angeles, California, USA

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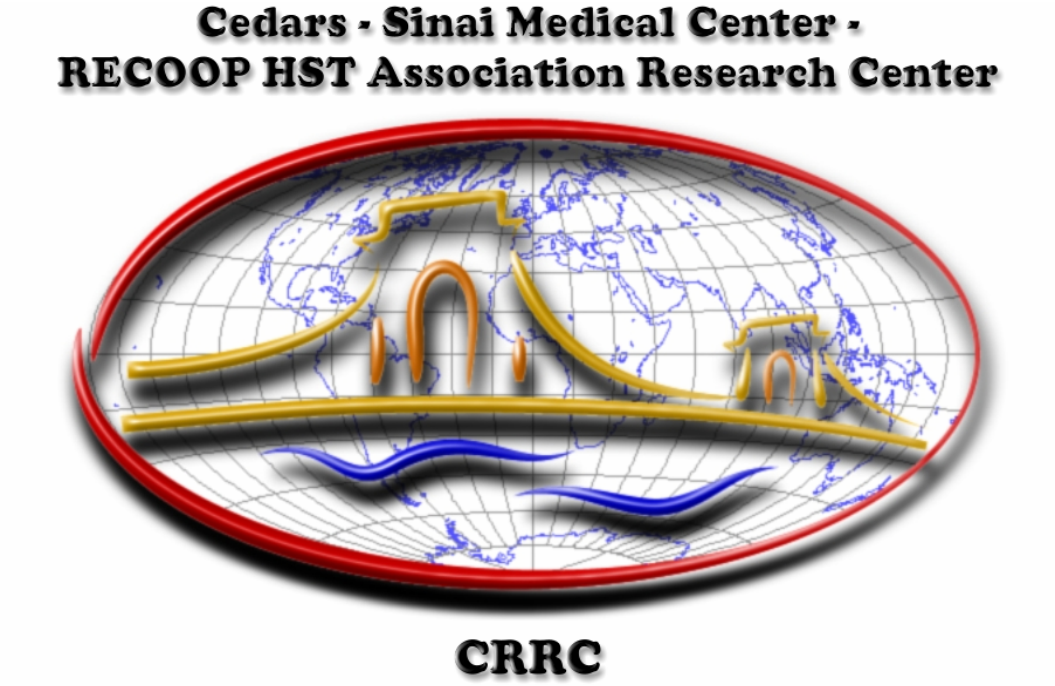

In 2012 the RECOOP HST Association is integrated the multidisciplinary, multicenter research studies of the RECOOP Research Networks in the RECOOP Life Science Research Platform and formed 17 **CSMC RECOOP Research Centers (CRRC)** from 7 countries (Croatia, Czech Republic, Hungary, Poland, Romania, Slovak Republic, and Ukraine) working on translational and clinical research in the field of Genomics – Proteomics, Epigenetics, Metagenomics, Molecular Biology, Metabolomics and Nano-biotechnology.

Poster Sessions

Partner Organizations

Poster Session Schedule

18:00 – 20:00 **Poster Sessions -** ***Conference Rooms # 4 and 5***
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Preterm Birth
Cardiovascular Diseases

11:30 – 14:00 **Poster Sessions -** ***Conference Room # 6***
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Cancer Research
NanoBioTechnology

11:00 – 12:00 **Poster Sessions –** ***Conference Rooms # 7***
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Stress, Obesity and Metabolic Diseases
Neurological Disorders and Brain Research

Poster Sessions

Stress, Obesity and Metabolic Diseases

Metabolic corrections of rat liver damage under diabetes

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Prevention of the mitochondrial oxidative damage is a therapeutic strategy in diabetes and mitochondria-targeted antioxidants are to have a therapeutic potential in diabetes. The aim of the present work was to investigate the role of a functional damage in rat liver mitochondria during diabetes as well as to evaluate the possibility of metabolic and antioxidative correction of liver disorders. Earlier we demonstrated an impairment of the antioxidative defence system during long-term streptozotocin-induced diabetes (9 weeks) (Lapshina et al., 2006). We observed some improvement of liver metabolism due to the long-term acetylsalicylic acid (ASA) administration that led to some decrease in the level of haemoglobin glycation and affected the activities of different enzymes in the liver tissue, reverting the decreased GSHPx and G6PDH activities in the diabetic rats.

Melatonin treatment (10 mg/kg b.w., 30 days, daily) under streptozotocin – induced diabetes in rats did not influence the level of hyperglycemia or glycated hemoglobin but partially reversed both the activities of the pentose phosphate pathway enzymes, G6PDH and transketolase, and GSHPx activity in the diabetic liver.

The most pronounced effect of the melatonin administration was prevention of an increase in nitric oxide levels in blood plasma and aortic tissue during diabetes. Melatonin might be considered as a factor regulating glucose metabolism by affecting glucose – metabolizing enzyme activities, restoring tissue redox-balance and nitric oxide bioavailability. The melatonin administration during diabetes reversed the decreased mitochondrial ADP-dependent respiration rate and the acceptor and respiration control ratios, demonstrating mitochondria – specific activity.

The effects of melatonin might be due to its radical scavenging properties, its metabolic effects and specific interaction with complexes of the respiratory chain. Our results suggest that melatonin regulating mitochondrial function may have a therapeutic potential for correction of diabetic liver damages.

Key words: diabetes, liver damage, mitochondria, melatonin, acetylsalicylic acid