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CoMBoS2

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WELCOME SPEECH



Professor Dušanka **Savić-Pavićević** President of the Serbian Society for Molecular Biology



Dr. Melita **Vidaković** President of the Steering Committee of the Serbian Society for Molecular Biology

Dear colleagues and friends,

On behalf of the Serbian Society for Molecular Biology (MolBioS), we warmly welcome you to Belgrade for the Second Congress of Molecular Biologists of Serbia (CoMBoS2).

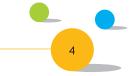
The congress is gathering almost 250 participants from 13 countries (Sweden, United Kingdom, Italy, Switzerland, USA, Australia, Hungary, Czech Republic, Romania, Montenegro, Croatia, Bosnia and Herzegovina, and Serbia).

The program covers various fields of Molecular Biology, including Molecular Biomedicine, Molecular Biotechnology and Molecular Cell Biology, and consists of plenary and invited lectures, the MolBioS award winner lecture, poster sessions and the project corner. Special attention is paid to students and young scientists through the MolBioS Student Session, flash presentations and workshops on state-of-the-art molecular biology methods.

We wish you to be inspired by exciting and outstanding lectures given by renowned scientists and experts, exchange ideas, find opportunities for new collaborations, and have good fun.

WELCOME TO





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METABOLIC DISTURBANCES IN ANIMAL MODEL OF POLYCYSTIC OVARY SYNDROME: IMPACT OF EARLY POSTNATAL OVERFEEDING

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Introduction: Polycystic ovary syndrome (PCOS) is a common endocrine disorder that affects women's fertility and metabolic health throughout their life time. Insulin resistance and obesity, in conjunction with excess androgens, are undeniably involved in its development. We aimed to elucidate how hyperandrogenemia and prepubertal adiposity contribute to the development of metabolic disturbances in rat model of PCOS.

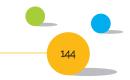
Methods: The animal model of PCOS induced by 5a-dihydrotestosterone (DHT) was additionally challenged by litter size reduction (LSR) during suckling period, to ensure overfeeding and development of prepubertal adiposity. Systemic parameters of insulin sensitivity, along with markers of energy sensing, insulin signaling, and lipid metabolism were analyzed in visceral adipose tissue (VAT) and skeletal muscle.

Results: The combination of treatments led to hyperinsulinemia and impaired systemic insulin sensitivity. This was not accompanied with altered insulin signaling in the VAT, in spite of observed adipocytes hypertrophy probably due to activation of AMPK and restrained lipogenesis in this tissue. On the other hand, insulin signaling in skeletal muscle was impaired, which resulted in increased muscle fatty acid uptake and oxidation after combined treatment. The switch to fatty acids oxidation subsequently led to oxidative stress and inflammation, which was followed by adaptive activation of AMPK and increased expression of its targets involved in antioxidant protection and mitochondrial biogenesis.

Conclusion: Our results suggest that prepubertal weight gain predisposes to insulin resistance development in androgen-excess PCOS. The protective activation of AMPK in VAT and muscle makes it a potential therapeutic target for insulin-resistant PCOS patients.

Key words: polycystic ovary syndrome; early postnatal overfeeding; insulin resistance; adipose tissue; skeletal muscle

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Session MOLECULAR MECHANISMS OF CELL FUNCTIONS