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CoMBoS2

Content

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

CoMBoS2

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Abstracts

Session MOLECULAR BIOMEDICINE

AUTOPHAGY RECEPTOR P62 REGULATES SARS-COV-2-INDUCED INFLAMMATION IN COVID-19

Danijela Stevanovic,[1] Verica Paunovic,[1] Ljubica Vucicevic, [2] Maja Misirkic Marjanovic,[2] Vladimir Perovic,[1] Biljana Ristic,[1] Mihajlo Bosnjak,[1] Milos Mandic,[1] Ljubica Harhaji-Trajkovic,[2] Kristina Janjetovic,[2] Milica Kosic,[1] Jovan Lalosevic,[3,4] Milos Nikolic, [3,4] Branka Bonaci-Nikolic ,[4,5] Vladimir Trajkovic[1]

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Introduction: Since the interaction between autophagy and virus-induced inflammation is complex, we investigated the interplay between autophagy and inflammation in COVID-19 patients and THP-1 cells expressing SARS-Cov2 proteins NSP5 and ORF3a.

Methods: Autophagy markers in blood from 19 control subjects and 26 COVID-19 patients at hospital admission and one week later were measured by ELISA, while cytokine levels were examined by flow cytometric bead immunoassay. The level of p62 in cells and its concentration in cell culture supernatants was measured by immunoblot/ELISA. The mRNA levels of proinflammatory cytokines were measured by RT-qPCR.

Results: IFN-α, TNF, IL-6, IL-8, IL-17, IL-33, and IFN-γ were elevated in COVID-19 patients at both time points, whereas IL-10 and IL-1β were elevated at admission and one week later, respectively. Autophagy markers LC3 and ATG5 were unchanged in COVID-19. The concentration of autophagic cargo receptor p62 was significantly lower and positively correlated with TNF, IL-10, IL-17, and IL-33 at hospital admission, returning to normal levels after one week. The expression of SARS-CoV-2 proteins NSP5 or ORF3a in THP-1 cells caused an autophagy-independent decrease/autophagy-inhibition-dependent increase of intracellular and secreted p62. This was associated with an NSP5-mediated decrease in TNF/IL-10 mRNA and an ORF3a-mediated increase in TNF/IL-1β/IL-6/IL-10/IL-33 mRNA levels. A genetic knockdown of p62 mimicked the immunosuppressive effect of NSP5, while a p62 increase in autophagy-deficient cells mirrored the immunostimulatory action of ORF3a.

Conclusion: The autophagy receptor p62 is reduced in acute COVID-19, and the balance between autophagy-independent decrease and autophagy blockade-dependent increase of p62 levels could affect SARS-CoV-induced inflammation.

Key words: inflammation; COVID19; p62; NSP; ORF3a

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