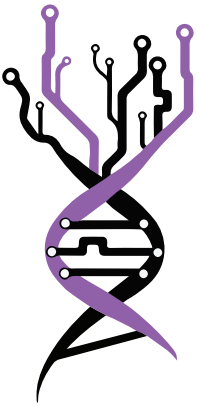
#BelBi2023 • Belgrade, Serbia

BOOK OF ABSTRACTS



4th Belgrade Bioinformatics Conference

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EDITORS
Dr. Ivana Morić
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FOREWORD

Dear colleagues and friends,

The 4th Belgrade Bioinformatics Conference - BelBi2023, where many high-quality scientific contributions were presented, has just ended. With great thanks to all participants, we now proudly present a book of abstracts that both reflects the scientific abundance and diversity of the conference and serves as a reminder of a memorable event.

Several research institutions, faculties, and scientific societies from Serbia joined forces in organizing this international conference, which covered numerous topics in computational biology, bioinformatics, and biomedical and health informatics. The main goal of BelBi2023 was to foster contact between scientists, both early stage career and senior researchers, allowing them to share experiences and latest advances in their fields. We sincerely hope that BelBi2023 has served as a platform for researchers from around the world to meet, initiate new collaborations, and expand professional contacts, and that all of you would become a part of the growing BelBi community.

We are grateful and proud to have welcomed more than 250 researchers from 21 countries. We have had 28 scientific sessions, consisting of more than 60 lectures (including eight Keynote talks), 47 presented posters, as well as three workshops and one satellite event – COST action. We have also organized seven industry lectures, including the NGS Challenge, two Meet the Expert Sessions, and one Business Coffee Break where ten start-up companies took part. And finally, the future BIO4 campus was presented and first panel on Serbia's resources for storage and analyses of genetic data was organized.

We would like to thank all the members of the International Advisory Board and the International Program Committee for their efforts and help in making this event a success. We are very grateful to the Ministry of Science, Technological Development and Innovation of the Republic of Serbia, SAIGE project, and UNDP-Serbia for their support. Finally, the Local Organizing Committee is very grateful to all the sponsors of the conference - BGI, Illumina & Elta'90MS, PacBio & East Diagnostics, ThermoFisher Scientific & Vivogen, Huawei, Labena, DSP Chromatography, RNIDS, Telekom Srbija, Alfa Genetics, Kefo and Superlab, hoping that they will stay with us for many years to come.

Looking forward to seeing you again at the 5th Belgrade Bioinformatics Conference.

Belgrade, July 2023

Dr. Valentina Đorđević
& Dr. Ivana Morić,
On behalf of BelBi2023
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POSTER PRESENTATION

Cell-type-specific mechanistic drivers of progressive multiple sclerosis lesions

Elkjaer ML*[1,2,3], Hartebrodt A[4,8], Oubounyt M[5], Weber A[1,2,3], Vitved L[3], Reynolds R[6], Thomassen M[2,7], Rottger R[8], Baumbach J[5,8], and Illes Z[1,2,3]

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Understanding the drivers of compartmentalized and sustained inflammation in the brain of progressive multiple sclerosis (PMS) remains elusive. To investigate the interplay between inter- and intra-cellular molecular mechanisms in white matter (WM) lesions, we integrated single-cell transcriptome and chromatin accessibility data from PMS lesions with spatial transcriptomics of chronic active lesion borders. We identified a PMS-specific oligodendrocyte genetic program governed by the Krüppel-like factor and specificity protein (KLF/SP) gene family, implicated in myelination and stress-induced iron uptake. Additionally, we found high expression of transferrin gene (TF) and its receptor megalin (LRP2) across lesion types, suggesting autocrine communication of iron uptake potential related to iron rim lesion in smoldering MS. Additionally, inflammatory phenotype of oligodendrocytes expressing osteopontin gene and complement were observed at chronic active lesion edges. Inside the chronic active lesion, the axonal damage biomarker, neurofilament light (NFL) gene expression was upregulated, and an astrocytic-neuronal axis through fibroblast growth factor (FGF) signaling (FGFR3-FGF13) was present. Additionally, a metabolic astrocyte phenotype at the lesion border potentially segregates inflammation areas. We also identified two distinct B cell co-expression networks with different locations and gene expressions, preferring different lesion types. Overall, single-cell multi-omics enabled the identification of specific cell types with unique molecular profiles, cell-cell communications, and spatial context, contributing to lesion fate.

Keywords: white matter lesions, single-cell multi-omics, progressive multiple sclerosis, spatial transcriptomics, iron metabolism, FGF signaling, chronic active lesion

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