BOOK OF ABSTRACTS

6. CONGRESS
OF THE SERBIAN GENETIC SOCIETY

2019 | October 13–17

VRNJAČKA BANJA • SERBIA

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Abstracts of the 6th CONGRESS OF THE SERBIAN GENETIC SOCIETY

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WELCOME TO VI CONGRESS OF THE SERBIAN GENETIC SOCIETY!

Dear colleagues,

Welcome to the 6th Congress of the Serbian Genetic Society. The Serbian Genetic Society (SGS) has been founded in 1968 and the first Congress organized by the SGS was held in 1994 in Vrnjacka Banja. Since then, the Congress of Serbian Genetic Society is held every five years. Over the past years, the Congress has grown from a national to an international meeting.

The experience of the past meetings motivated our efforts to continue with this series with a clear tendency to strengthen the scientific connections among researchers from different European countries.

The Congress will focus on the most recent advances in genetics and on wide range of topics organized in 9 sessions and two workshops. Many of the presentations will be in lecture-like settings, but we hope that there will also be ample opportunities for informal interaction outside the scheduled sessions.

The successful organization of the Congress has required the talents, dedication and time of many members of the Scientific and Organizing committees and strong support from our sponsors. I hope that you will find the Congress both pleasant and valuable, and also enjoy the cultural and natural beauty of Vrnjacka Banja.

Yours sincerely,

Branka Vasiljevic
President of the Serbian Genetic Society

02 – 29 Poster

ARCA3 IN SERBIAN ROMANI FAMILY CAUSED BY FOUNDER MUTATION IN ANO10 – A GENETIC APPROACH

Milica Mihajlović [1], Vanja Tanasić [1], Dušan Keckarević [1], Jelena Mladenović [2], Miljana Kecmanović [1], Milica Keckarević Marković [1]

[1] Faculty of Biology, University of Belgrade, Belgrade, Serbia
[2] Clinic for Neurology and Psychiatry for Children and Adolescents, University of Belgrade, School of Medicine, Belgrade, Serbia

milica.mihajlovic@bio.bg.ac.rs

Introduction: Autosomal-recessive cerebellar ataxias (ARCAs) are a heterogeneous group of disorders with different genetic causes. Recently discovered ARCA3 is caused by mutations in ANO10 gene and most frequently associated with c.1150_1151del [p.Leu384fs], founder mutation, originated in Romani population. Although genetic causes of ARCAs are different, ARCA3 could be misdiagnosed as Friedreich ataxia or some other type of ARCA, so genetic analysis should be done in order to assess the right diagnosis and adequate therapy. Based on these facts, in this study, we analyzed 55 individuals: 16 ARCA patients previously subjected and negative for Friedreich ataxia, 38 Serbian Roma with no apparent neurological disease and one Romani woman (53 years old) with an undiagnosed neurological disorder.
Methods: Genomic DNA was isolated from buccal swabs. A region within exon 6 of ANO10, bearing founder mutation, was PCR amplified with fluorescently labeled primers. Fragment analysis was performed using ABI3130 Genetic Analyzer and analyzed using the GeneMapper® software.
Results: Patients negative for Friedreich ataxia were also negative for ANO10 founder mutation. A woman with the undiagnosed neurological disorder was found to be a recessive homozygote for c.1150_1151del, and her sister to be a carrier of the mutation. No other analyzed Roma were found to be the carriers.
Conclusion: Knowing that Romani people are marginalized and often disregarded in terms of social and health care, especially in not so developed countries, one should consider a genetic approach that could potentially facilitate the diagnosis of inherited disorders caused by recessive founder mutations originated in Romani population.

AUTOSOMAL-RECESSIVE CEREBELLAR ATAXIAS (ARCAs), ROMANI POPULATION, ANO10

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02 – 30 Poster

DETECTION OF DNA METHYLATION PROFILES IN MOUSE EMBRYONIC FIBROBLASTS USING FOURIER TRANSFORM-INFRARED SPECTROSCOPY

Ana Sarić [1], Anja Tolić [1], Nevena Grdović [1], Svetlana Dinić [1], Jovana Rajić [1], Miloš Đorđević [1], Marija Đorđević [1], Jelena Arambašić Jovanović [1], Mirjana Mihailović [1], Goran Poznanović [1], Aleksandra Uskoković [1], Melita Vidaković [1], Tanja Dučić [2]

[1] Department of Molecular Biology, Institute for Biological Research, University of Belgrade, Belgrade, Serbia
[2] CELLS – ALBA, Cerdanyola del Vallès, Barcelona, Spain

anasaric29@gmail.com

Epigenetic processes orchestrate the cell type-specific use of the genetic information essential for normal development and for maintaining the overall integrity of the genome. DNA methylation is probably the most extensively studied epigenetic mark and represents the covalent attachment of a methyl group to cytosine that is located next to guanine within the genomic DNA. The alteration of DNA methylation patterns by hyperglycaemia, oxidative stress and inflammation may have potential epigenetic impacts on gene regulation in diabetic individuals. Further research devoted to improve the steps that could be undertaken in the early diagnosis, prevention and treatment of diabetes and its complications is a scientifically and socially significant task. We used the Fourier transform-infrared (FTIR) spectroscopy (ALBA synchrotron, Cerdanyola del valles, Spain) for qualitative spectral analysis of normomethylated DNA from mouse fibroblast cells (NIH3T3) and hypomethylated DNA from PARP-1 knockout mouse fibroblast cells (PARP-/-). We obtained the global information regarding the DNA methylation profiles in mouse fibroblast cells by FTIR spectroscopy that was visualised by immune-fluorescent staining using anti-methyl cytosine (5mC) antibody. Some differences in DNA methylation profiles between examined cell lines were observed in spectral region significant for cytosine (990-1250 nm). The most interesting picks were observed approximately at wavelength: 1240 nm, 1150 nm, 1110 nm and 1010 nm. These results are in the same time a verification of the proof of principle for FTIR based analysis of the differences between normomethylated and hypomethylated genomic DNA which could be set as a potential predictive and diagnostic tool in future. To our knowledge this is a first time that synchrotron-based FTIR micro-spectroscopy is used for detection of the presence of 5mC and changes in the DNA methylation profile in cells.

EPIGENETICS, DNA METHYLATION, FT-IR SPECTROSCOPY