

ITC CONFERENCE GRANT SCIENTIFIC REPORT

This report is submitted for approval by the grantee to the MC Chair.

Action number: CA15203

Conference title: 13th Conference on Mitochondrial Physiology: The role of mitochondria in health, disease and drug discovery - COST MitoEAGLE perspectives and MitoEAGLE WG and MC Meeting

Conference start and end date: 18/09/2018 to 21/09/2018

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Grantee name: AVRAM VLAD FLORIAN

ACTIVITIES DURING YOUR ATTENDANCE AT THIS CONFERENCE:

(max.500 words)

During the attendance at this conference I have attended all the conference sessions. On the 19th of September I have attended the Mitochondrial Physiology General Assembly meeting and I took part in the talks and the voting process. On the 20th of September, I presented the unpublished collaborative work done between the team in Timisoara and the team in Lund entitled "Cell-permeable succinate bypasses statin-induced mitochondrial complex I inhibition in human platelets" in order to start disseminating our data as we proposed to do in the STSM report from June 2018. On the 21st of September, I took part in the discussions of the four working groups (WG1, WG2, WG3, WG4) and after this I took part in the MC meeting and the voting process.

IMPACT ON YOUR RESEARCH AND FUTURE COLLABORATIONS (if applicable)

(max.500 words)

The collaboration between The Pathophysiology Department from "Victor Babeş" University of Medicine and Pharmacy Timișoara, Romania and The Department of Clinical Sciences, Mitochondrial Medicine, Lund Univ, Sweden will continue with finalizing the experiments and the future joint publication of the final results on statin-induced mitochondrial dysfunction in human thrombocytes and the effects of the cell permeable succinate under the umbrella of the COST action MITOEAGLE. After this we envision a future project in which we propose to assess the effect on thrombocyte mitochondrial function in patients chronically treated with statins and whether the cell permeable succinate may be useful in the treatment of statin-induced mitochondrial dysfunction in patients.