Oregonin from Alnus incana preserves from atherosclerosis by preventing endothelial dysfunction through nutrigenomic and epigenetic regulations
Laurent-Emmanuel Monfoulet, Sylvie Mercier, Desislava Abadjieva, Elena Stoyanova, Liga Lauberte, Galina Telesheva, Jelena Krasilnikova, Elena Kistanova

To cite this version:
Laurent-Emmanuel Monfoulet, Sylvie Mercier, Desislava Abadjieva, Elena Stoyanova, Liga Lauberte, et al.. Oregonin from Alnus incana preserves from atherosclerosis by preventing endothelial dysfunction through nutrigenomic and epigenetic regulations. 10th International Conference on Polyphenols and Health, Apr 2022, Londres, United Kingdom. hal-03687784

HAL Id: hal-03687784
https://hal.inrae.fr/hal-03687784
Submitted on 3 Jun 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L’archive ouverte pluridisciplinaire HAL, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d’enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.
Oregonin from Alnus incana preserves from atherosclerosis by preventing endothelial dysfunction through nutrigenomic and epigenetic regulations

Laurent-Emmanuel Monfoulet¹, Sylvie Mercier¹, Desislava Abadjieva², Elena Stoyanova², Liga Lauberte³, Galina Telesheva³, Jelena Krasilnikova³, Elena Kistanova³

¹ Université Clermont Auvergne, INRAE, UNH, F-63000 Clermont-Ferrand, France
² Institute of biology and immunology of reproduction, Bulgarian Academy of Sciences, Sofia, Bulgaria
³ Latvian State Institute of Wood Chemistry, 27 Dzerbenes Str. LV 1006 Riga, Latvia
⁴ Department of Human Physiology and Biochemistry, Stradiņš University, 16 Dzirciema Street LV-1007 Riga, Latvia

Background: Scientific evidence from experimental to clinical studies support a beneficial effect of polyphenols on cardiometabolic health. Oregonin is a polyphenol present in different parts of plants from genus Alnus that exhibits anti-inflammatory and anti-adipogenic properties. However, the underlying mechanisms of its effects are still not known. This research aimed to analyse pathways of oregonin effects on endothelial cells and its ability to preserve endothelial function under an inflammatory stress.

Methods: Human endothelial cells (HUVECs) were exposed to oregonin extract from bark of Alnus incana or control medium for 48 hours and then stimulated with tumor necrosis factor alpha (TNF-α) for 4 hours. Expression of adhesion (ICAM-1, VCAM-1) as well as monocyte adhesion were investigated. Methylation specific PCR for analysing ICAM-1 and VCAM1 methylation indexes was applied. The expression of total DNA-methyltransferase-1 (DNMT1), and mitochondrial DNA-methyltransferase-1 (mtDNMT1) and mitochondrial transcription factor (TFAM) by RT-PCR was analysed.

Results: The increased expression of vCAM-1 was inhibited by 50% with a pre-incubation of HUVECs with 7.5 µM oregonin while iCAM-1 expression did not change. Oregonin exposition significantly decreased the adhesion of monocytes to endothelial cell surface. A strong, and negatively dependent to oregonin concentration, correlation between monocyte adhesion and the expression of CAM proteins was observed. No differences were defined between methylation indexes of iCAM-1 and vCAM-1 in control and oregonin-exposed cells despite a significant decrease of DNMT1 in oregonin pre-incubated cells. The expression of DNMT1 was not changed after TNF-α cell activation. However, a significant decrease of TFAM and mtDNMT1 mRNA expression was established in oregonin exposed cells.

Conclusion: All together these results show the ability of oregonin to counteract endothelial inflammation by alleviating pro-inflammatory genes expression and monocyte adhesion, by and to regulating of DNMT1 and mtDNMT1 expression make this polyphenol an interesting candidate to prevent cell-specific epigenetic change in atherosclerosis.

The author(s) would like to acknowledge networking support by the COST Action FA 1403 POSITIVe (Interindividual variation in response to consumption of plant food bioactives and determinants involved), supported by COST (European Cooperation in Science and Technology) and the European Twinning Project PhenolAcTwin coordinated by the Tubitak Institute (Turkey) for supporting the attendance of L-Em. Monfoulet at ICPH 2022.