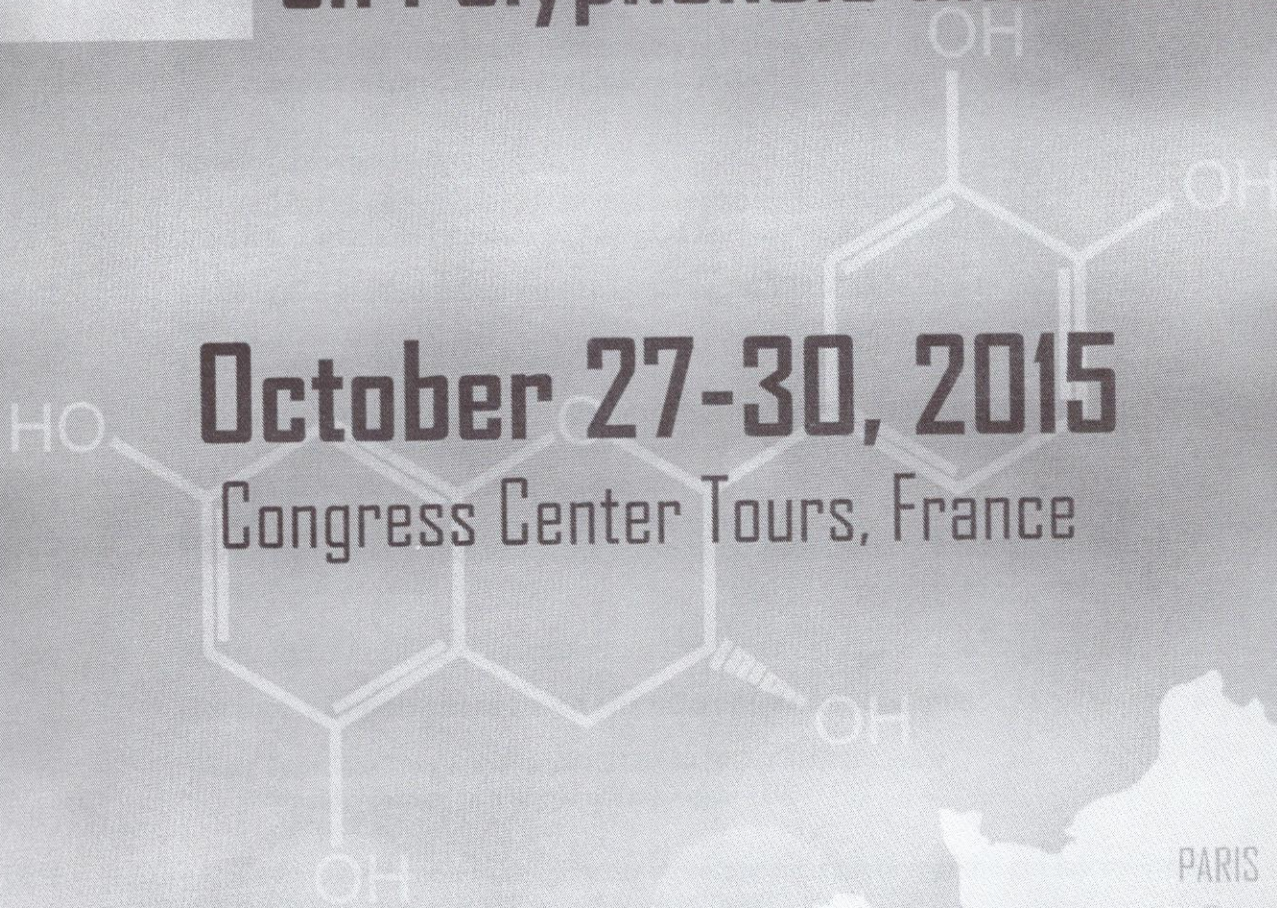


**7<sup>th</sup>**  
ICPH

# International Conference on Polyphenols and Health

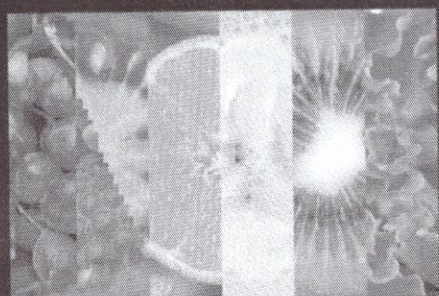
**October 27-30, 2015**

Congress Center Tours, France



Global Photo: www.icph2015.com - Calaisan - Elements/1274

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# 7<sup>th</sup> ICPH

## International Conference on Polyphenols and Health



INRA  
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www.icph2015.com

October 27-30, 2015  
Congress Center Tours, France

Clermont-Ferrand, June 15, 2015

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Dear Vladimir Ajdžanović,

The Organizing and Scientific Committees of the **7<sup>th</sup> International Conference on Polyphenols and Health**, are pleased to announce you that your abstract: *Soy isoflavones and membrane steroid receptors: a new horizon*, has been selected for an oral communication at the conference.

#### Please, note the following information:

- You will present your abstract in the Session G: Mechanisms of action of Polyphenols
- Presentation time: 10 minutes
- Questions: 5 minutes

#### Please be aware that:

- ***You must be registered for the conference***  
*The acceptance of your oral communication does not include a free registration*
- The deadline for early registration is July 15, 2015.

**Please provide the confirmation of your acceptance  
before June 22, 2016 to [icph2015@agence-mo.com](mailto:icph2015@agence-mo.com)**

Congratulations and we look forward to seeing you in Tours for the ICPH 2015 Conference.

The Scientific and Organizing Committees

17:30

## PARALLEL SESSION G - Auditorium Ronsard

## New insights into mechanisms of action of polyphenols

Chairs: **Mayte Garcia Conesa** - *CEBAS-CSIC of Murcia - Spain*  
**Dragan Milenkovic** - *INRA Clermont-Ferrand - France*

15:30



Dietary flavonoids induce autophagic degradation in macrophages.  
**Yoshichika Kawai** - *University of Tokushima - Japan*

16:00



Procyanidin-membrane interactions in the regulation of cell signaling.  
**Patricia Oteiza** - *University of California - USA*

*Selected communications*

16:30

Soy isoflavones and membrane steroid receptors: a new horizon.  
**Vladimir Ajdžanović** - *Institute for Biological Research "Siniša Stanković" - Serbia*

16:45

Delphinidin inhibits tumor growth by acting on VEGF signalling in endothelial cells.  
**Claire Lugnier** - *INSERM - France*

17:00

Potent inhibition of VEGF activity by pomegranate polyphenols: A novel mechanism for the atheroprotective effects observed in epidemiology.  
**Rebecca Edwards** - *Institute of Food Research - UK*

17:15

(Poly)phenols from quasi-isogenic raspberries: attenuation of neuroinflammation.  
**Gonçalo Garcia** - *iBET - Portugal*

15:30-17:30

## PARALLEL SESSION H - Auditorium Descartes

## Hot topics in polyphenols and gut microbiota interactions

Chairs: **Francisco Tomas-Barberan** - *CEBAS-CSIC of Murcia - Spain*  
**David Vauzour** - *University of East Anglia - UK*

15:30



A role for polyphenols in shaping the structure and function of the gut microbiome.  
**Kieran Tuohy** - *Foundation Edmund Mach of Trento - Italy*

16:00



Dietary modulation of gut microbial metabolome.  
**Wendy Russell** - *Rowett Institute of Nutrition and Health - UK*

*Selected communications*

16:30

Identification of novel flavonoid O- and C-glycosidases in gut bacteria.  
**Annett Braune** - *German Institute of Human Nutrition Potsdam-Rehbruecke - Germany*

16:45

Quantification of microbial polyphenol uptake, kinetics and metabolism using mass spectrometry.  
**Nikolai Kuhnert** - *Jacobs University Bremen - Germany*

17:00

Importance of the colonic microbiota in the bioavailability of orange juice (poly)phenols: in vivo and in vitro studies.  
**Gema Pereira-Caro** - *Andalusian Institute of Agricultural and Fishing Research and Training - Spain*

17:15

Short chain fatty acids affect hesperetin transport and phase II metabolism in Caco-2 cells.  
**Evelien Van Rymenant** - *Ghent University - Belgium*

17:30-18:30

## REFRESHMENTS - POSTER SESSION

18:30

GALA DINNER - *Departure in bus in front of the Congress Center*



## PARALLEL SESSION G - Auditorium Ronsard

## New insights into mechanisms of action of polyphenols

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**Soy isoflavones and membrane steroid receptors: a new horizon**Ajdzanovic<sup>1</sup>\*, I. Jaric<sup>1</sup>, J. Živanovic<sup>1</sup>, M. Mojic<sup>2</sup>, N. Ristic<sup>1</sup>, B. Filipovic<sup>1</sup>, V. Milošević<sup>1</sup><sup>1</sup> Department of Cytology, Institute for Biological Research "Siniša Stankovic", University of Belgrade, Despot Stefan Blvd. 142, 11060 Belgrade, Serbia., \*avlada@ibiss.bg.ac.rs<sup>2</sup> Department of Immunology, Institute for Biological Research "Siniša Stankovic", University of Belgrade, Despot Stefan Blvd. 142, 11060 Belgrade, Serbia

Soy isoflavone's (especially genistein and daidzein) health effects have been studied for decades, while their mechanisms of action remain complex, involving modulation of gene expression and enzyme activity. Currently there is a need for refreshed approach in this field considering identification of isoflavone's new molecular target. The cell membrane constituents-mediated effects of soy isoflavones are worthy of special attention from the perspective of cancer metastasis or cardiovascular diseases management. Specifically, the expanding concept of membrane steroid receptors and rapid signalling from the cell surface may include the prominent role of these steroid-like compounds.

We have shown *in vitro* that genistein inhibits LNCaP prostate cancer cells invasiveness by decreasing the membrane fluidity. This is complemented with genistein-caused immobilization of the androgen receptor containing membrane lipid rafts and down regulation of the androgen receptors/Akt signalling in mentioned metastatic prostate cancer cell line. On the other hand, it was observed that daidzein strongly couples with membrane estrogen receptors in adrenal medullary cells. At low doses (10 – 1000 nM) daidzein was found to stimulate catecholamine synthesis via extracellular signal-regulated kinase 1/2 or protein kinase A pathways, but at higher doses it inhibited catecholamine synthesis and secretion induced by acetylcholine.

Given that catecholamine excess can contribute to the cardiovascular pathologies and that catecholamine lack may lead to depression, daidzein application promises to have a wide range of therapeutic effects.

These data are supportive in development of the molecular pharmacotherapy pertinent to balanced soy isoflavone treatment of steroid-related malignancies as well as cardiovascular and psychiatric diseases.

Financial support: This work was supported by the Ministry of Science, Education and Technological Development of the Republic of Serbia, Grant number 173009.

S30

**Delphinidin inhibits tumor growth by acting on VEGF signaling in endothelial cells**T. Keravis<sup>1,†</sup>, L. Favot<sup>1,3,†</sup>, A. Abusnina<sup>1,5,†</sup>, A. Anton<sup>1,6</sup>, R. Soleti<sup>2</sup>, R. Andriantsitohaina<sup>2,4</sup>, C. Lugnier<sup>1\*</sup><sup>1</sup> Laboratoire de Biophotonique et de Pharmacologie, CNRS UMR 7213, Université de Strasbourg, France.<sup>2</sup> LUNAM, INSERM, U1063, Université d'Angers, Angers, France Present addresses:<sup>3</sup> Laboratoire Inflammation, Tissus Epithéliaux et Cytokines EA 4331 Pôle Biologie Santé, Université de Poitiers, France.<sup>4</sup> Centre Hospitalo-Universitaire, Angers, France.<sup>5</sup> Tripoli University, Faculty of Veterinary Medicine, Tripoli, Libya.<sup>6</sup> ALL.DIAG, Instruments and Reagents, Strasbourg, France.<sup>†</sup> T. Keravis, L. Favot and A. Abusnina participated equally to this paper.

\* claire.lugnier@unistra.fr

The vasculoprotective properties of delphinidin are driven mainly by its action on endothelial cells. Moreover, delphinidin displays anti-angiogenic properties in both *in vitro* and *in vivo* angiogenesis models and thereby might prevent the development of tumors associated with excessive vascularization. This study was aimed to test the effect of delphinidin on melanoma-induced tumor growth with emphasis on its molecular mechanism on endothelial cells. Delphinidin treatment significantly decreased *in vivo* tumor growth induced by B16-F10 melanoma cell xenograft in mice. *In vitro*, delphinidin was not able to inhibit B16-F10 melanoma cell proliferation but it specifically reduced basal and VEGF-induced endothelial cell proliferation. The anti-proliferative effect of delphinidin was reversed in the presence either of the MEK1/2 MAP kinase inhibitor, U-0126, or the PI3K inhibitor, LY-294002. VEGF-induced proliferation was reduced either by U-0126 or LY-294002. Under these conditions, delphinidin failed to decrease further endothelial cell proliferation. Delphinidin prevented VEGF-induced phosphorylation of ERK1/2 and p38 MAPK and decreased the expression of the transcription factors, CREB and ATF1. Finally, delphinidin was more potent in inhibiting *in vitro* cyclic nucleotide phosphodiesterases (PDEs), PDE1 and PDE2, compared to PDE3-PDE5. Altogether delphinidin reduced tumor growth of melanoma cell *in vivo* by acting specifically on endothelial cell proliferation. The mechanism implies an association between inhibition of VEGF-induced proliferation via MAPK, PI3K and at transcription level on CREB/ATF1 factors, and the inhibition of PDE2. In conjunction with our previous studies, we demonstrate that delphinidin is a promising compound to prevent pathologies associated with generation of vascular network in tumorigenesis.

Financial support: This work was supported in part by the French Centre National de la Recherche Scientifique, the INSERM, the University of Strasbourg, the University of Angers and APRIFEL/Ligue Contre le Cancer, France.