REDOXBA **2023**

Workshop on Redox Nutrition and Toxicology

School of Pharmacy and Biochemistry,
University of Buenos Aires.
Buenos Aires, Argentina

November **12-14, 2023**

Organizing Committe

Silvia Alvarez

Pablo Evelson

Cesar G. Fraga

Mónica Galleano

Local comittee

Mariana Garcés

Natalia Magnani

Virginia Vanasco

Tamara Zaobornyj

Oxygen Club of California

.UBA UNIVERSIDAD DE BUENOS AIRES

.UBA FARMACIA Y BIOQUÍMICA

Society for Free Radical Research International

Society for Free Radical Research Europe

Food & Function

Brassica Protection Products

Avoiding one-electron oxidation of tyrosine by DOPA

Neyra Recky J. R., Serrano M. P., Dántola M. L., **Lorente C.**

INIFTA, Facultad de Ciencias Exactas, Universidad Nacional de La Plata. CCT-La Plata CONICET

Photosensitized oxidation of proteins induced intermolecular and/or intramolecular cross-linking, depending on the protein nature and the environment conditions. Several amino acids participate in cross-linking due to the formation of a covalent bond between two identical or different amino acids. The amino acids involved in crosslinking are mainly tyrosine, histidine, tryptophan, lysine, cysteine. From a biomedical point of view, cross-linking of proteins is involved in pathologies such as cataracts, photoaging, and inactivation of enzymes.
Tyrosine is an amino acid related to crucial physiological events and its oxidation, that produce beneficial or detrimental effects on biological systems, has been extensively studied. Degradation of tyrosine often begins with the loss of an electron in an electron transfer reaction in the presence of a suitable electron acceptor. The reaction is facilitated by excited states of the acceptor in photosensitized processes. Several products of tyrosine oxidation have been described, the main ones being 3,4-dihydroxy-L-phenylalanine (commonly known as DOPA) and tyrosine dimers. The latter are responsible of protein cross-linking.
We have observed that tyrosine is recovered from tyrosyl radical, after one-electron oxidation, in the presence of DOPA. We propose that under high oxidative stress the oxidation of tyrosine may be controlled, in part, by one of its oxidation products. Also, we present strong evidence of antioxidant action of DOPA by preventing tyrosine dimerization, one of the most serious oxidative protein modifications, and the origin of structural modifications leading to the loss of protein functionality.
In consequence, under oxidative stress the formation of DOPA, avoids tyrosine dimer formation between two neighboring tyrosine residues.

Maternal metabolic syndrome affects the progeny's redox balance and increases neuroinflammation with neurodevelopmental and metabolic adverse consequences

Facundo H., Prado Spalm*, Marié L., Cuervo Sánchez*, Natalia E., Furland, Ana S., Vallés[1].

Nutrition and Neurodevelopmental Laboratory, INIBIBB-CONICET-UNS. Camino La Carrindanga Km. 7, B8000FWB Bahía Blanca, Argentina.
** Both authors contributed equally to this work.*
[1] *Corresponding author: sofiavalles@gmail.com*

Various lifestyle factors, including diet, can impact on redox balance and brain health. Consumption of fructose-sweetened beverages has drastically increased in the last decades and is widely associated with metabolic disease, systemic proinflammatory status and adverse transgenerational effects. To date, the impact of maternal fructose intake in brain redox balance and function of the offspring is less explored. We investigated whether the progeny of mothers with Metabolic Syndrome (MetS), induced by *ad libitum* consumption of a 20% fructose solution, present any redox alteration in the brain as a consequence of being gestated in a metabolic altered intrauterine environment.
Wistar rats were randomly separated into two groups with access to water or fructose (20% w/v in water) for 10 weeks. After MetS was confirmed, dams were mated with control males and continued drinking water or fructose solution during gestation. At postnatal day (PN) 1, a subgroup of offspring of each sex was sacrificed and brains were dissected for oxidative stress and inflammatory status analysis. The developmental milestones and behavioral test were also evaluated (PN3-PN100) in another subgroup of offspring to identify any long-term consequence to being gestated by a dam with MetS.
Maternal MetS affects the redox balance and increases neuroinflammation in female offspring at birth. Sexually dimorphic effects were also found on the progeny's acquisition of neurodevelopmental milestones and in their psychiatric, cognitive and metabolic state.
Although direct extrapolation of our findings cannot be made to humans, the results presented herein reinforce the necessity of considering the potentially negative effects of fructose-induced MetS prior to, and during pregnancy in offspring’s brain and metabolic physiology.