7th Obesity & Endocrinology Specialists Congress

October 10-12, 2016 Manchester, UK

Keynote Forum

(Day 1)

Maria Fernanda Cury-Boaventura, J Obes Weight Loss Ther 2016, 6:8(Suppl)
http://dx.doi.org/10.4172/2165-7904.C1.038

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Maria Fernanda Cury-Boaventura

University of Cruzeiro do Sul, Brazil

Obesity and immune cells

Obesity is a major medical problem and causes the development of chronic low-grade metabolic inflammation. The high incidence of metabolic disorders is associated with white adipose tissue (WAT) surrounding intra-abdominal organs. It is believed that the initial cause of metabolic inflammation is adipocyte hypertrophy in visceral adipose tissue (VAT). In the hypertrophic adiposity, non-esterified fatty acids (NEFA) induces local macrophages to produce high levels of TNFa which in turn promote a positive feedback inducing more NEFA, pro-inflammatory cytokines, acute phase proteins and chemokines, which attract more monocytes/macrophages resulting in dysregulation of pro-inflammatory mediators andlow-grade inflammation. Hypertrophic adipose expansion also induces hypoxia that promotes angiogenic factors, hypoxia- inducible factor (HIF) 1 and inflammatory response-associated genes upregulation resulting in fibrosis. Lean adipose tissues have various anti-inflammatory immune cells, such as eosinophils, M2 macrophages, Th2 cells, iNKT cells, and Treg cells. In obese adipose tissue, the numbers of pro-inflammatory immune cells, including neutrophils, M1 macrophages, mast cells, Th1 cells, and CD8 T cells, are elevated. Simultaneously, reduced number of anti-inflammatory immune cells accelerates pro-inflammatory response and adipose tissue dysfunction. During the last decade, it was also identified that the vast majority of obese are characterized with a gut microbiota dysbiosis. Metabolic diseases are associated with cellular changes in the innate immune compartment of the intestine. The first line of intestinal defense is based on the secretion of defensins and IgA by intestinal epithelial cells, which are reduced in obese patients. The high fat diet induces the translocation of bacterial components such as LPS. The translocation of LPS or bacteria to tissues is a physiological mechanism, however, when unregulated, leads to a state of chronic inflammation that depends from the immune and epithelial cells response. A subpopulation of dendritic cells expressing CX3CR1 and innate lymphoid cells 3 are involved in the impairment of appropriate response. This mechanism is linked to the production of large numbers of cytokines such as IL-6, IL-17, IL-22, GM-CSF and TNF.

Biography

Maria Fernanda Cury-Boaventura has completed her PhD in Human Physiology from University of São Paulo and Post-doctoral studies from University of São Paulo. She is a Professor and Researcher at Institute of Physical Activity and Sport Sciences since 2007. She has published more than 40 papers in reputed journals and has been serving as an Editorial Board Member of repute.

mafecuryb2009@hotmail.com

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Thais Cesar, J Obes Weight Loss Ther 2016, 6:8(Suppl)
http://dx.doi.org/10.4172/2165-7904.C1.038

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Thais Cesar

Sao Paulo State University (UNESP), Brazil

Orange juice, as a natural source of bioactive compounds, protects against cardiometabolic risks in overweight and obese subjects

Previous studies have associated orange juice consumption with prevention of oxidative stress and systemic inflammation, which may improve insulin sensitivity and reduce the risk of diabetes and cardiovascular disease. These effects are attributed to the bioactive compounds in orange juice, such as flavonoids, carotenoids and vitamin C, which protect the body against oxidative stress and reduce serum lipid levels. Other studies have consistently shown that regular orange juice consumption reduces the serum levels of total cholesterol and LDL-cholesterol and improves the endothelial function, reducing the risk of atherosclerosis. We have conducted several studies to evaluate the effect of regular orange juice consumption on the risk factors for metabolic syndrome and cardiovascular disease. In all these studies, the participants have consumed orange juice daily, for two to three months of period. Anthropometric, hemodynamic, biochemical, inflammatory and oxidative statuses were assessed at baseline and at the end of the intervention period. No changes in the patient's body weight, percentage of fat mass, and waist circumference was shown after the regular consumption of orange juice, suggesting that orange juice did not contribute to weight or fat mass gain. In fact, the consumption of orange juice improved the diet quality by adding important nutrients as folate, vitamin C, and calcium. Also, it has showed that orange juice significantly reduced blood cholesterol levels, LDL-C, insulin, HOMA-IR, C-reactive protein, blood pressure and, increased serum antioxidant activity by more than a 100 percent. On another transversal study, we also verified that long term orange juice consumption (one year) was associated with low LDL-C and apolipoprotein B in normal and moderately hyper-cholesterolemic subjects. In conclusion, our studies showed that orange juice consumption promoted lipid-lowering, anti-inflammatory and antioxidant activities, which contribute to the prevention of the oxidative stress and the risk factors for diabetes and cardiovascular diseases.

Biography

Thais Cesar is an Associate Professor of Nutrition, Faculty of Pharmaceutical Sciences, Sao Paulo State University (UNESP), Brazil. She has a BSC in Biology and PhD in Food Science and Nutrition, University of Sao Paulo, Brazil. She did her Post-doctoral at the Boston University and at the Citrus and Subtropical Products Research Laboratory, ARS-USDA. Her scientific focus is investigating the nutritional and metabolic properties of citrus fruits in clinical studies and animal models, regarding to the effect of its bioactive compounds as a protection factor against the development of chronic diseases.

tcesar@fcfar.unesp.br

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Keynote Forum

(Day 2)

Don S Schalch, J Obes Weight Loss Ther 2016, 6:8(Suppl)
http://dx.doi.org/10.4172/2165-7904.C1.038

7th Obesity & Endocrinology Specialists Congress

Don S Schalch

University of Wisconsin School of Medicine and Public Health, USA

Food deserts and swamps impacting on health: Tale of two cities

Availability of various foods is an important determinant of what people buy and eat, thus impacting on their health. This is illustrated in our studies of two cities: one large; i.e., Cleveland, Ohio, replete in "food swamps," and one small; i.e., Madison, Wisconsin, site of many "food deserts," food swamp which is readily-accessible convenience stores and fast food restaurants; food desert where it is difficult-to-buy fresh fruits and vegetables. Food swamps and deserts often co-exist. Availability of nutritious food is one determinant of people's diet; others are cost, cultural, racial, ethnic, habits, and inadequate transportation in low-income areas. Fast foods in restaurants and junk foods in convenience stores, rich in carbohydrates, fats and sugar, are associated with increased risk of being overweight/obese and increased prevalence of type 2 diabetes, hypertension, heart disease, and cancer. Recent WHO European Region report indicated poor diet, overweight and obesity which contribute to a large proportion of cardiovascular diseases and cancer, the two main killers in the Region. Lack of essential nutrients/minerals, lead to growth impairment, organ dysfunction, and failure in maintaining normal nitrogen balance in the body. Using housing- and food-outlet data (Cleveland) and UW APL, UWSMPH, and WI Depts. HS/U&RP data (Madison), employing GIS, food swamps in the former and food deserts in the latter have been mapped, corresponding to areas of poverty, people of color, and increased prevalence and poorly-controlled diabetes. Emphasizing complexity of poor diet choices, a refrigerated 40-foot trailer offering fresh fruits and vegetables in 8 Madison food deserts was unsustainable after 2 years because initial consumer interest declined.

Biography

Don S Schalch has completed his MD from the University of Cincinnati, OH, in 1960. He completed his Medicine Residency and Fellowship in Endocrinology and Metabolism from the University of Rochester, NY, in 1964. He was a Visiting Scientist at Erasmus University in Rotterdam and at Kantonspital in Zürich in 1972-73, and joined the University of Colorado Medical School Faculty in Denver, CO in 1974. In 1982, he became the Chief of Endocrinology at the University of Wisconsin School of Medicine and Public Health. He has published 101 papers, has been a reviewer for 3 journals, and became an Emeritus Professor since 1999.

dss@medicine.wisc.edu

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