Welcome to the 3rd ISAA, October 2012 in Burgas, Bulgaria!

SOS FOR HOMO SAPIENS OBESUS


George N. Chaldakov¹, Luigi Aloe², Anton B. Tonchev¹, Gorana Rančić³, Mariyana G. Hristova⁴, Neše Tunçel⁵, Dimitar D. Kostov⁶, Marco Fiore², Vesselka Nikolova⁷, and Vladmila Bojanić⁸

¹Laboratory of Cell Biology, Medical University, Varna, Bulgaria, ²Institute of Neurobiology and Molecular Medicine, National Research Council (CNR), Rome, Italy, ³Department of Histology and Embryology, Niš University Medical Faculty, Niš, Serbia, ⁴Department of Endocrinology, Private Policlinic, Varna, Bulgaria, ⁵Department of Physiology, Medical Faculty, Eskişehir Osmangazi University, Eskişehir, Turkey, ⁶Department of Internal Medicine, University St Marina Hospital, Varna, Bulgaria, ⁷Intensive Cardiology Unit, University St Marina Hospital, Varna, Bulgaria, and ⁸Department of Pathophysiology, Niš University Medical Faculty, Niš, Serbia

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Correspondence: Dr George N. Chaldakov, Laboratory of Cell Biology, Medical University, BG-9002 Varna, Bulgaria. E-mail: chaldakov@yahoo.com
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The adipose tissue in the human body is there for the best, the bad, and the worse!

Paraphrased from Charles Lapiere and Erik Maquoi (1)

Published on 1 December 1994 issue of Nature (2), the Jeffrey Friedman’s discovery “gave leptin in the beginning” of the endocrine saga of adipose tissue. Onwards, studies on this tissue have enjoyed an explosive growth that conceptualized a novel field of research, adipobiology (3). Arguably, in the heart of adipobiology and adipopharmacology are studies focusing on the pathogenesis, prevention and therapy of cardiometabolic diseases (CMD) including atherosclerosis, hypertension, obesity, type 2 diabetes, metabolic syndrome (global cardiometabolic risk), and lipodystrophies.

The 2nd International Symposium on Adipobiology and Adipopharmacology (ISAA) was organized by the Bulgarian Society for Cell Biology. Scientists from 15 countries presented 34 state-of-the-science (SOS) lectures.

Gema Frühbeck (Pamplona, Spain) highlighted the significance of adipokines represented by a large number of signaling proteins, e.g. pro- and anti-inflammatory interleukins, also leptin, adiponectin, resistin, tumor necrosis factor-α, renin, angiotensin II, and the “newcomers” retinol-binding protein-4, angiopoietin-like protein-2 (Angpl-2) and Angpl-4 (fasting-induced adipose factor), visfatin, vaspin, pigment epithelium-derived factor, apelin, chemerin, adiponectin, resistin, tumor necrosis factor-α, renin, and nerve growth factor (NGF) (4). She also introduced caveolins and aquaporin-7 (AQP-7) into the pathogenesis of CMD.

Caroline Pond (Milton Keynes, UK) presented her SOS on paracrine interactions between perinodal white adipose tissue and lymph nodes. She demonstrated that adipocytes associated with lymphoid structures are specialised to supply fatty acids, and possibly other precursors, to lymphoid cells. Such mechanisms emancipate the immune system from fluctuations in the composition and quantity of dietary lipids and are particularly important during immune responses, when perinodal adipose tissue remote from the site of immune stimulation as well as that around the locally stimulated nodes are activated. Caroline Pond also provided evidence that defective perinodal adipose tissue may be central to Crohn’s disease (5).

Harold Sacks (Memphis, Tennessee, USA) presented results about paracrine secretion of adipokines by epicardial adipose tissue and its significance for cardiovascular disease (6). His talk as well as that of Caroline Pond stressed the relevance of adipotopography in pursuing a hidden H. obesus (7), a phenotype of TOFI (thin outside, fat inside), a term coined initially by Jimmy Bell (Imperial College, Hammersmith Hospital, London, UK), to emphasize that we cannot not rely only on BMI and other “classical” anthropometric measurements when imaging techni-
nal exposure of mice to ethanol, but not red wine, causes memory deficit expressed by reduced presence of brain NGF, BDNF and choline acetyltransferase reactivity in the offspring (18).

Dragan Djuric (Belgrade, Serbia) focused on the role of folate acid and homocysteine, which is also “secreted” by adipose tissue (19), in coronary artery functions evaluated by coronary flow and oxidative stress biomarkers. Bilge Pehlivanoglu (Ankara, Turkey) associated obesity with immune cell responses to stress. Hiroshi Yamamoto (Kanazawa, Japan) looked for the “enemies and friends within” the mechanisms of diabetic angiopathy. He portrayed the AGE-RAGE-esRAGE system, that is, advanced glycation end-products and their receptors, and an endogenous secreted splice variant coding for a decoy form of RAGE, respectively (20). Neşe Tunçel (Eskişehir, Turkey) implicated white- and brown adipose tissue-derived nitric oxide and associated mast cells in cold stress-mediated vascular contractility in rats (21), referring also to the recent challenge in brown adipobiology (22).

Sukhinder Cheema (St. John’s, Newfoundland, Canada) highlighted the paradigm of nutrition-related developmental programming as related to the prevention of CMD, showing that a maternal high-fat diet alters the lipid metabolism, including hepatic LDL receptor expression, of their adult male offspring (23). Vladmila Bojanić (Niš, Serbia) reviewed data showing the effects of placenta- and breast milk-derived leptin on mother’s and offspring’s health, the so-called lepin-mediated metabolic programming (see 10). Collectively, CMD prevention should start during in utero and suckling life of the human. Likewise, Harpal Buttar (Ottawa, Ontario, Canada) advocated that “prevention is better than cure”. There is an overwhelming evidence that the Mediterranean-type diets which are high in fibre, low in saturated fat and glycemic load are associated with the decreased prevalence of metabolic syndrome and diabetes mellitus as well as improved lipid homeostasis and reduced risk of CMD. Of note, Harpal Buttar mentioned recent data of Yuji Matsuzawa’s group demonstrating that smoking cessation is associated with increased plasma levels of adiponectin, an anti-atherogenic and anti-diabetic adipokine (24). Richelle McCullough (Winnipeg, Manitoba, Canada) presented findings demonstrating that flaxseed is an excellent source of alpha linolenic acid (ALA), which is stored in adipose tissue, and that dietary flaxseed inhibits cardiac arrhythmias and reduces atherosclerosis (25).

Gorana Rančić (Niš, Serbia) implicated adipose-pineal network in the chronobiology of CMD. Luigi Aloe and George Chaldakov raised a hypothesis of adipose tissue as a third brain (26) - this sophisticated tissue is a source of and target for a variety of hypothalamus-pituitary hormones, releasing factors, neuropeptides and neurotrophic factors, which play a pivotal role in lipid, glucose and energy homeostasis as well as learning, memory and other neural functions. This may open a novel field of research, neuroadipology, which may contribute to further study on the pathogenesis of various diseases (26-28; for leptin’s neurotrophic action, see 10).

As we started this Report, “the adipose tissue in the human body is there for the best, the bad and the worse” (1). “The best”, because adipose tissue produces (i) anti-obesity, anti-inflammatory and anti-atherogenic factors such as adiponectin, NGF, brain-derived-neurotrophic factor (BDNF), interleukin-10, IL-1 receptor antagonist, (ii) vascular relaxants such as adipose-derived relaxing factors, nitric oxide (NO), hydrogen sulfide (H2S), and adiponectin, and (iii) neurotrophic factors such as NGF, BDNF, ciliary neurotrophic factor, leptin, and NPY and other neuropeptides. “The bad”, because adipose tissue accumulation stimulates the secretion of disease-associated (proinflammatory) adipokines. “The worse”, because adipose tissue disappearance results in lipodystrophies.

Altogether, adipose tissue may be a crossroad of CMD and neurodegenerative diseases. Further progress in adipobiology and adipopharmacology holds much promise for understanding how to walk on that road. Finally, all participants broadcasted the signal of “SOS for Homo sapiens obesus”, to hopefully reach politicians and businessmen, and make them altruistic to science and human health.

The 3rd ISAA will be held in October 2012 in Burgas, Bulgaria. Welcome!

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