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ABSTRACTS E-BOOK

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DEHYDROEPIANDROSTERONE SUPPLEMENTATION IMPROVES CELLULAR AND BIOCHEMICAL MARKERS OF OBESITY

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Introduction: The obesity pandemic is a major worldwide health concern that predisposes to a higher risk of metabolic and cardiovascular diseases (CVD). In menopausal women the *decline* of *ovarian steroidogenesis* is associated to a high prevalence of CVD and obesity. Steroid hormones have a pivotal role in the regulation of angiogenesis. Vascularization of the adipose tissue modulates adipogenesis, lipids storage through a complex interplay not fully understood--. According to intracrinology, DHEA can be converted into active sex steroids in peripheral tissues, avoiding their systemic exposure. DHEA supplementation is proposed as a low risk therapy for the prevention of postmenopausal diseases.

Objectives: The aim of this work was to study: a) the effect of DHEA administration on the metabolic profile and on oxidative stress markers related to obesity using using a murine model of obesity and hipoestrogenism; b) the role of DHEA on angiogenesis.

Methods: Ovariectomized Wistar rats feed with standard diet (ND) (4%fat) or high fat (HF) diet (27%fat) received daily injections of vehicle (C) or DHEA (1mg/kg.day) for 8 weeks. Angiogenic effect of DHEA was evaluated *in vitro* using primary cultures of endothelial cells (EC) and *ex vivo,* using the rat aortic ring assay.

Results: Caloric intake was 22% higher in HF vs ND groups, with an increase of body weight and adiposity index. No significative differences were detected in glucose, cholesterol, HDL-cholesterol and triglycerides levels (table). In contrast, DHEA induced a reduction of Cholesterol/HDL index in ND group, and of serum ROS (H2-DCFDA) both in NF and HF groups.

Nitric oxide production *ex vivo* by rat aortic rings was enhanced by DHEA in both ND and HF (130% and 138% vs C, $p<0.01$), effect dependent on DHEA convertion to more active steroids since it was abolished in presence of a 3β-HSD inhibitor.

The angiogenic process requires ECs proliferation, migration and organization. In ECs in vitro treatment with DHEA increased cell proliferation (130% above C $p<0.01$) and enhanced cell migration (15% above C $p<0.05$). Indeed, DHEA stimulated capillary tube formation when ECs were cultured in a fibrin matrix. Consistently, *ex vivo* assays showed that DHEA (20 and 200nM) stimulated capillary tubes formation around aortic ring (8.5 vs 28.1, 29.2µm, C vs 20nM, 200nM-DHEA $p<0.05$).

Image:

	ND-Vehicle	ND-DHEA	HF-Vehicle	HF-DHEA
Initial Weight (g)	292.3±14.5	306.3±19.1	311.8±20.9	305.2±26.2
Final Weight (g)	334.7±17.1	342.6±15.7	368.9±25.5*	366.1±24.5*
Adiposity index	5.7±0.4	5.6±0.6	6.5±0.5*	6.8±0.4*
Food intake (g/kg.day)	53.7±5.1	52.9±5.6	41.8±4.0	42.0±6.0
Caloric intake (Kcal/kg)	166.5±15.8	164.1±17.4	200.8±19.2*	201.4±28.8*
Liver (g)	12.1±2.1	12.2±2.1	12.0±2.3	12.1±2.2
Heart (g)	1.1±0.1	1.2±0.1	1.2±0.1	1.2±0.1
Abdominal White fat (g)	10.0±1.1	11.2±1.3	18.1±3.3*	17.6±3.1*
Brown fat (g)	0.8±0.1	0.9±0.1	0.6±0.2	0.8±0.1
Glucose (mg/dl)	112±9	120±11	118±13	110±11
Cholesterol (mg/dl)	52±12	46±8	50±11	54±15
HDL Cholesterol (mg/dl)	11±3	13±3	9±1	12±4
Cholesterol/HDL	4.7±0.8	3.5±0.7*	5.6±0.9	4.9±0.8
Triglycerides (mg/dl)	72±21	74±23	70±23	69±21
ROS	680±23	420±37*	720±29	530±52[a]

Mean ± SD of n=5 rats/group. *p<0,05 vs. ND-Vehicle [a]p<0,05 vs. HF Control