accumulation of glycated and fibrotic materials, what may result in treatment with pyridoxamine. (microvascular marker), what was partially reverted by the decrease of Bcl-2/Bax ratio (marker of apoptosis) and vWF staining of PAS and Masson Trichrome-positive components. MG caused a TGF-beta levels similar to W group and inhibited the accumulation fibrotic factor), as well as increased staining of PAS and Masson tissue levels of CEL (glycation marker) and TGF-beta precursor (pro

Results: α-MSH/MCSR role on lipid mobilization is mediated by HSL, ATGL and perilipins. Immunofluorescence microscopy of α-MSH-treated cells revealed that phosphorylated HSL clearly surrounds lipid droplets, in opposition to perilipins that leave the immediate periphery of lipids. These observations are lost in adipocytes with suppressed expression of MC5R. Moreover, α-MSH/MCSR decreases “de novo” synthesis of fatty acids through inactivation of ACC. Furthermore, fatty acid re-esterification is also impaired after α-MSH stimulation since a reduction on PEPCK activity was observed. Conclusion: Altogether these results indicate that, in adipocytes, α-MSH-activated MCSR regulates three tightly coupled pathways: lipolysis, lipid synthesis and re-esterification. The global effect is a decrease on adipocyte fat mass, important in strategies contributing to ameliorate obesity.

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CO062. PYRIDOXAMINE REDUCES THE MICROVASCULAR EFFECTS OF METHYLGLOXAL-INDUCED GLYCATION IN ADIPOSE TISSUE
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Introduction: Adipose tissue dysfunction results from many factors, including glycation-induced microvascular damages. Pyridoxamine is a scavenger of free radical and carbonyl species, able to protect the biological systems under glycatuting conditions. In this work we tested the usefulness of pyridoxamine treatment in inhibiting methylglyoxal-induced glycation in adipose tissue.

Methods: Wistar rats were treated daily with methylglyoxal (MG) during 8 weeks (75 mg/Kg/day, diluted in the water). After this time, half of the animals did not have any treatment (WM), while the other half (WMPyr) was treated during 4 weeks with pyridoxamine (1 g/day, diluted in water). Another group of Wistar rats with no treatment was used as control (W).

Results: WM group showed a decrease of HDL cholesterol and an increase of circulating free fatty acids, what was reverted by pyridoxamine (WMPyr group). MG also caused an increase of tissue levels of CEL (glycation marker) and TGF-beta precursor (pro fibrotic factor), as well as increased staining of PAS and Masson Trichrome-positive components. Pyridoxamine resulted in CEL and TGF-beta levels similar to W group and inhibited the accumulation of PAS and Masson Trichrome-positive components. MG caused a decrease of Bcl-2/Bax ratio (marker of apoptosis) and vWF staining (microvascular marker), what was partially reverted by the treatment with pyridoxamine.

Conclusion: Pyridoxamine prevents methylglyoxal-induced accumulation of glycated and fibrotic materials, what may result in better microvascular function in adipose tissue.

CO063. VASOPROTECTIVE EFFECTS OF ADIPONECtin IN HIGH-FAT FED WISTAR RATS
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Adiponectin (ApN) is the most abundant peptide secreted by adipocytes and has important effects on the cardiovascular and endocrine systems. It is involved in a wide variety of physiological processes including energy metabolism, inflammation, and vascular physiology. Despite being made by adipocytes, circulating adiponectin levels are paradoxically decreased in obesity and metabolic syndrome. Thus, we investigated the effects of chronic administration of ApN on vascular reactivity (contraction and relaxation) of rat aorta and mesenteric arteries in 12-month-old male Wistar rats fed with high fat diet. The effects of ApN were investigated on NO-dependent and independent vasorelaxation in isolated rat aortic and mesenteric arteries from 12-month-old male Wistar rats fed with high fat diet during 4 months (WHF) and compared them with WHF and with Wistar control rats fed with standard diet (W). Adiponectin (2.7 mg) was administered by continuous infusion with a minipump implanted subcutaneously on the back for 28 days before sacrifice. High fat diet induced significantly increased body weight and an increment in systemic levels of leptin and leptin/adiponectin ratio. It also significantly reduced the efficacy of NO-dependent vasorelaxation both in aorta and mesenteric arteries. Chronic ApN treatment significantly reduced body weight, leptin levels and the leptin/adiponectin ratio, normalizing endothelial function in both arteries. These results indicate that high-fat diet induced endothelial dysfunction in normal Wistar rats and that ApN was able to normalize endothelial function by a mechanism that likely includes an increment in NO bioavailability. Detailed characterization of the ApN signaling pathway in the vasculature and perivascular fat (as well as other metabolic tissues) is likely to provide novel tools in the management of atherosclerosis and metabolic disease.

CO064. POTENCIAL TERAPÉUTICO DA BERBERINA NA DISFUNÇÃO ENDOTELIAL ASSOCIADA À DIABETES TIPO 2
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A disfunção endotelial está intimamente relacionada com a aterosclerose, as doenças cardiovasculares e com a diabetes tipo 2. A berberina é um alcalóide natural existente em rizomas e raízes de várias plantas. Foi inicialmente utilizado como um agente destoxificante e anti-inflamatório na medicina chinesa. Tem sido frequentemente associada a efeitos benéficos no combate à diabetes tipo 2. Contudo, ainda não estão completamente esclarecidos os seus efeitos. Este trabalho teve como objetivo estudar o potencial terapêutico da berberina na disfunção endotelial associada à diabetes tipo 2. Foram estudados 4 grupos de animais com 12 meses de idade: ratos com berberina nos últimos 3 meses antes do sacrifício (W+B); ratos com diabetes tipo 2 (GK); ratos GK tratados com berberina nos últimos 3 meses antes do sacrifício (GK+B). A berberina foi administrada via oral numa concentração de 100 mg/Kg. Após o sacrifício foram avaliados diferentes parâmetros metabólicos e inflamatórios e foi ainda efetuada a caracterização funcional da artéria aorta dos diferentes grupos de animais em estudo. Verificámos que os animais tratados com berberina melhoraram o seu perfil metabólico nomeadamente