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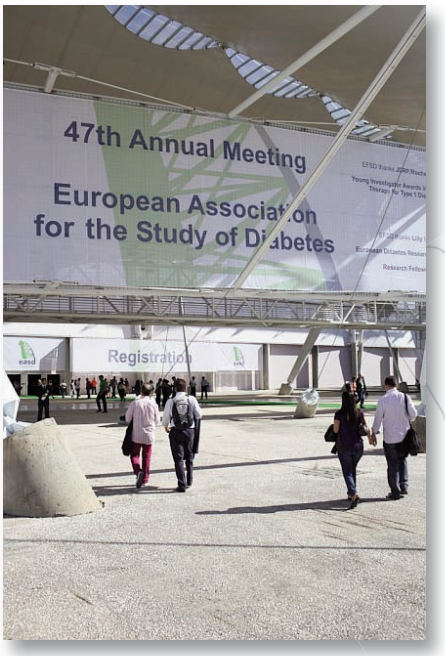
Ecos do "47th EASD Annual Meeting" – Parte II

Realizou-se, entre 12 e 16 de Setembro de 2011, no Centro de Congressos da Feira Internacional de Lisboa, no Parque das Nações, o "47th EASD Annual Meeting", maior congresso científico a nível mundial dedicado à diabetes, que reuniu, nesta edição, mais de 18.000 participantes de 120 países diferentes, na sua maioria profissionais de saúde ou investigadores biomédicos.

Dos 1.249 trabalhos científicos apresentados, 42 foram portugueses, número que corresponde a aproximadamente metade dos trabalhos científicos apresentados pelos norte-americanos, mas que foi superior ao dos trabalhos apresentado pela Suíça, o que mostra que a investigação nacional na área da diabetes se encontra em expansão.

Nesta parte II da Revista Internacional dedicada ao "47th EASD Annual Meeting", iniciamos a publicação dos "abstracts" dos trabalhos científicos apresentados por portugueses, por ordem de numeração no respectivo livro de "abstracts".



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Neurotensin downregulates the pro-inflammatory properties of skin-dendritic cells and increases epidermal growth factor expression

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Background and Aims: Diabetic foot ulcer is a painless and harmful disease that delays skin wound healing in diabetic patients. This illness is a consequence of differential causes, like neuropathy, impaired angiogenesis and decreased blood flow supply to the site of injury, cytokines deregulation, impaired expression of extracellular matrix (ECM) proteins and ECM remodeling by metalloproteinases.

In the last three decades, neuropeptides received much attention by the scientific community, as their impairment, for instance by neuropathy, may induce delayed WH. Neuropeptides modulate skin cells function, particularly skin inflammation. As recent reports have revealed the neuropeptide neurotensin (NT) as an immune mediator in the Central Nervous System and the gastrointestinal tract and its effects in the skin have not been identified, in this study we investigated the effects of NT on signal transduction and on pro/anti-inflammatory function of skin dendritic cells in the presence/absence of the inflammatory stimuli lipopolyssaccharide (LPS).

Materials and Methods: Fetal-skin dendritic cells (FSDC) were maintained in IMDM medium (control) or incubated with 10 nM of NT alone or simultaneously with LPS for 5 to 60 min to analyze the activation of signaling pathways by WB and during 6 to 30 h to determine neurotensin receptors (NTRs), cytokines and growth factors expression by real time RT-PCR. FSDC viability was determined by the MTT assay while the cytoskeleton and nuclei morphology of FSDC was determined by immunostaining for actin and nuclei.

Results: We observed that FSDC constitutively express NTR1 and NTR2 and that LPS treatment diminishes NTR1 (-1.14 ± 0.55, n=3), NTR2 (-1.88 ± 0.77, *p<0.05, n=3), while expression of NT was increased by 3.75 ± 0.50 (***p<0.0001, n=3) relatively to control. In LPS stimulated cells, NT downregulated significantly the activation of the inflammatory signaling pathways NF-κB and JNK, while the survival pathway ERK was upregulated. Neurotensin alone downregulated the expression of the cytokines IL-6 (-0.34 ± 0.26, n=3), TNF-α (-1.10 ±0.62, *p<0.05, n=3), IL-10 (-1.77 ± 0.91, *p<0.05, n=3) and the vascular endothelial growth factor (VEGF) (-0.29 ± 0.18, *p< 0.05, n=4) fold relatively to control, while the epidermal growth factor (EGF) (1.45 ± 1.18, *p<0.05, n=4) was significantly upregulated. Simultaneous cell exposure to LPS and NT induced a similar cytokine profile to that one induced by NT alone.

Conclusion: Overall, our results give new perspectives in the design of new therapies for skin diseases, like diabetic wound healing. Moreover, once NT levels are determined in skin pathologies and if NT is then defective, a treatment with NT may promote WH. Further similar studies should be performed both in other skin cells and in in vivo conditions, in order to disclose the potential benefic therapeutic role of NT on skin pathological conditions.

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Meal-induced insulin sensitisation depends on hepatic parasympathetic nerves and requires the presence of aminoacids and glucose in the gut

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Background and Aims: Importance of analyzing postprandial glucose homeostasis has been highlighted by several studies suggesting that loss of postprandial glycemic control can precede deterioration of fasting glycemia in the course towards diabetes. Our group had previously reported that peripheral hypoglycaemic action of insulin increases from the fasted to the fed state and that this meal-induced insulin sensitization (MIS) mechanism is not triggered by glucose alone. We tested the hypotheses that the intestine plays an essential role in activating MIS which is triggered by simultaneous presence of both carbohydrates and protein-based nutrients.

Materials and Methods: 24 h-fasted Sprague-Dawley rats were used.

Surgery involved gastric or enteric cannulation, and enteric band placement, to avoid intestine-stomach reflux. Three series of experiments were performed. 1) Insulin sensitivity (IS) was assessed by a rapid euglycaemic clamp both in fasting state (24 h-fast) and 120 min after administration of the liquid mixed-meal (carbohydrates, proteins and lipids, 10 ml/kg), either into the stomach (IG) or into the duodenum (IE). After hepatic parasympathetic inhibition (surgical ablation or atropine), IS was assess. 2) Hepatic parasympathetic denervation was done in the fasted state; IS was assessed in fasted+denervated state and after the mixed-meal (IE). 3) The effect of meal composition on IS was studied by determining IS in the fasted state and after IE administration of the following liquid meals: (i) glucose + aminoacids + lipids (GAL); (ii) glucose + aminoacids (GA); (iii) aminoacids + lipids (AL); (iv) glucose + lipids (GL); (v) aminoacids (A).

Results: 1) IG mixed-meal did not produce MIS, unlike IE mixed-meal, which induced 77.4±11.2 % increase in IS (from 117.8±12.6 to 200.9±11.7 mg glucose/kg bw; $p<0.001$ (fasting vs post-meal). Subsequent hepatic parasympathetic ablation reduced IS obtained after IE meal (denervation, 85.1±6.1 mg glucose/kg bw; atropine, 99.9±7.1 mg glucose/kg bw; $p<0.001$ vs post-meal), but did not affect post-IG-meal IS. 2) When parasympathetic denervation was performed in the fasted state, it prevented IS increment after IE meal (3.8± 13.3 %, ns). 3) The GAL meal induced significant insulin sensitization (MIS): IS increased from 97.9±6.2 mg/kg (fasted state) to 225.4 ± 18.3 mg/kg ($p< 0.001$), which corresponds to 133.7±23.5% potentiation of insulin action. The GA meal also produced MIS (IS increased from 115.3±15.3 to 241.6 ± 35.2 mg/kg, after GA; $p<0.05$), inducing 109.6 ± 9.1% potentiation of IS. None of the other meals tested produced a MIS close to the GAL or GA meals. Immediately before the post-meal IS assessement, insulinemias were similar in all groups. Furthermore, only GAL and GA meals induced sustained postprandial IS (higher than the fasting IS), as determined by a second post-meal IS test (data not shown).

Conclusion: Our data suggests that the MIS mechanism is triggered at the intestine and requires activation of the hepatic parasympathetic nerves. Glucose and aminoacids are required in the intestine in order to fully trigger the MIS mechanism.

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Altered glucose excursions during a meal tolerance test in an impaired hepatic vagal model are mainly due to peripheral insulin resistance

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Background and Aims: In the last decades the hepatic parasympathetic system has emerged as a physiological modulator of insulin action having its main impact during the postprandial period. In the present work we investigated the hypothesis that the hepatic parasympathetic system is a determinant part in maintaining glucose and/or lipid homeostasis upon the ingestion of a meal. Moreover, the altered glucose excursions, as a consequence of the hepatic vagal impairment, are due to peripheral insulin resistance rather than to changes in insulin secretion/clearance or endogenous glucose production.

Materials and Methods: Male Wistar rats (12 weeks old) were divided into two experimental groups: sham and hepatic parasympathetic denervated (HPNden) animals. A MTT was performed. The MTT consists in the intra enteric administration of a complete mixed meal: 38.2 mg/mL lipids; 84 mg/mL aminoacids; 173mg/mL glucose. To evaluate endogenous glucose production (EGP) a tracer - [U-13C] Glucose, representing 0.5% of the EGP - was administered as a continuous perfusion throughout the experiment. To assess glucose appearance rate, glucose clearance and glucose disposal the mixed-meal was enriched with a 5% [6,6-H2] Glucose as a tracer. Samples were evaluated by LC-MS (Liquid Chromatography - Mass Spectrometry). Plasma samples were collected at different times (0, 2, 5, 10, 20, 30, 40, 50, 60, 90 and 120 minutes) of the study. Theses samples were used to measure glycaemia, plasma insulin, c-peptide and free fatty acids. Insulin secretion was determined based on plasma c-peptide; insulin clearance was assessed as the AUC of [c-peptide]/[insulin]. Finally, insulin sensitivity was determined by an hyperinsulinemic euglycemic clamp.

Results: The results showed an increase in glucose excursions from the sham animals to the HPNden animals (AUC: 15650 ± 621.9 n=8 vs. 17980 ± 876.2; n=8; $P < 0.05$).

Insulin and c-peptide levels obtained for both groups were indistinguishable during the MTT, excluding insulin secretion as a contributor for the differences observed in the glycemic profiles. Likewise, insulin clearance and endogenous glucose production did not differ between both groups. Insulin sensitivity was evaluated before and after administration of the meal. The HPNden animals showed decreased postprandial insulin sensitivity, when compared with the control (113.4 ± 26.48 mg glucose/kg, n=4 vs. 203.4 ± 31.14 mg glucose/kg, n=4; $P < 0.05$).

Conclusion: The results indicate that in the absence of a fully functional hepatic parasympathetic system whole-body glucose homeostasis cannot be maintained. This leads to increased glucose excursions as a result of increased peripheral insulin resistance. Furthermore, while evaluating the pathways through which this system influences glucose homeostasis it was observed that pancreatic insulin secretion, insulin clearance, endogenous glucose production and plasma free fatty acids were not affected by hepatic vagal impairment throughout the MTT. These results allow us to conclude that the hepatic parasympathetic system is a critical component of postprandial glucose homeostasis.

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Allelic variations of superoxide dismutase 1 (SOD1) gene and diabetic nephropathy in type 2 diabetic patients

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Background and Aims: Oxidative stress is involved in the pathophysiology of diabetic nephropathy (DN), and the enzyme superoxide dismutase 1 (SOD1) is essential for reactive oxygen species detoxification. Associations of SOD1 gene variants with DN have been reported in patients with type 1 diabetes. In this study, we assessed the impact of SOD1 allelic variation in the development and progression of DN in individuals with type 2 diabetes (T2DM) followed prospectively for renal events.

Material and Methods: We studied unrelated French type 2 diabetic patients from the DIABHYCAR (n=3137) and the DIABHYCAR_GENE (n=607) cohorts. DIABHYCAR was a 6-year clinical trial conducted in men and women with T2DM selected on the basis of persistent microalbuminuria (urinary albumin excretion, UAE=20-200 mg/l) or macroalbuminuria (UAE >200 mg/l) without renal failure at baseline. The trial tested whether a low dose of ramipril able to reduce UAE would also reduce cardiovascular and/or renal events. A renal event was defined as the doubling of the serum creatinine levels or the requirement of haemodialysis or renal transplantation during follow-up. It occurred in 77 cases (2.46%). Results were negative regarding the drug effect and were published previously. The DIABHYCAR_GENE cohort was recruited concomitantly to DIABHYCAR and included men and women with T2DM presenting with normal UAE (UAE<20 mg/l). Seven SNPs (rs2173962, rs9974610, rs10432782, rs2070424, rs1041740, rs17880135 and rs202449), giving information on ~90% of the allelic variation of the haplotypic block containing SOD1 gene were analyzed. Genotype associations with DN were assessed by logistic regression analyses and by Cox proportional hazards survival regression analyses. Adjustments for clinical and biological parameters were carried out by including these parameters as covariables in the regressive model. The power to detect associations of the SNPs with DN at baseline and with incidence of renal events during follow-up was 0.98 and 0.74, respectively, for odds ratio or hazard ratio equal or higher than 1.5 and alpha=0.05.

Results: In a first step, participants were divided into 3 groups according to UAE at baseline: normal UAE (DIABHYCAR_GENE cohort), microalbuminuria and macroalbuminuria (DIABHYCAR cohort, both groups). Allele and genotype frequencies of the seven SNPs were similar in the 3 groups. Next, we assessed the impact of allelic variations on the renal outcomes of the

original DIABHYCAR study. We have not observed any association of the SNPs with the incidence of renal events during follow-up neither in univariate analyses nor in complex models adjusted for sex, age, BMI, duration of diabetes, HbA1c levels, presence of arterial hypertension, history of myocardial infarction, treatment with ACE inhibitors or treatment group in the original DIABHYCAR study. In these analyses, only HbA1c, BMI, arterial hypertension and history of myocardial infarction at baseline were significantly and independently associated with the incidence of renal events at follow-up.

Conclusions: Allelic variations in the SOD1 gene were not associated with micro or macroalbuminuria nor with the deterioration of renal function in patients with T2DM followed prospectively for 6 years. These results suggest that SOD1 does not play a major role in the genetic determinants of DN in type 2 diabetes.

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Persistent lipid abnormalities in statin-treated patients: Portuguese diabetic sub-population of the Dyslipidemia International Study (DYSIS)

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Background and Aims: Diabetes mellitus (DM) is a well-established risk factor for cardiovascular disease (CVD). DM patients often experience multiple lipid abnormalities. This study aimed to evaluate the prevalence and type of persistent lipid abnormalities in Portuguese diabetic sub-population treated with lipid modifying therapies (predominantly statins).
Materials and Methods: The Dyslipidemia International Study (DYSIS) was a multicenter, epidemiologic cross-sectional study, conducted in 12 European countries and Canada. Entry criteria included patients≥ 45 years old, with a lipid profile assessment performed in the previous 6-12 months while on statin therapy for at least 3 months. In Portugal, along with the other countries, data concerning demographic characterization, cardiovascular risk factors and lipid modifying treatment were collected between April 2008 and February 2009 in primary care centres and private practice.
Results: In Portugal, 916 patients were recruited of which 348 (38.0%) had DM. Patients with DM had significantly lower LDL-c levels than patients without DM, however, there were no differences between the two populations regarding triglyceride and HDL-c values (Table 1). The percentage of diabetic patients that did not meet the ESC guidelines for total cholesterol and LDL goal levels was lower than the observed in patients without DM (61.8% vs 72.5%; p<0.001; 57.9% vs 66.3%, p<0.05, respectively). On the other hand, there were more DM patients with low HDL-c according to ESC guidelines (27.7% vs 18.8%, p<0.01). There were no differences in hypercholesterolemia treatment between patients with and without DM: the most frequently used statin was simvastatin in both groups (54.9% vs 56.5%, p=0.64).
Conclusion: The Portuguese results of DYSIS show that total cholesterol, triglyceride and HDL-c remain outside the recommended levels in more than 50% of statin-treated patients with DM. Although this is a high risk population for CVD events, lipid management strategies did not differ from the general population. This is in line with the international findings of this study. These patients remain at increased CVD risk and supplementary treatment may be indicated.

Table 1. Biochemical and historical results for DYSIS in Portugal.

	Patients with DM N=348 (38.0%)	Patients without DM N=568 (62.0%)
LDL-c (mmol/L) [Median and Quartiles]	2.7 (2.1-3.3)	3.2 (2.6-3.9)*
LDL-c <2.59mmol/L	42.1% (118/280)	24.5% (105/429)*
Triglycerides <1.69mmol/L	57.4% (193/336)	58.7% (315/537)**
HDL-c >1.03 (men)/1.29(women), mmol/L	65.8% (223/339)	71.8% (375/522)**
Systolic B.P. (mmHg) [Mean and S.D.]	137.4 (+/- 16.8)	136.3 (+/- 16.9) **
ACE Inhibitor treatment	41.4%	26.5%*
HbA1c (available in 304/348 - 87.4%)	7.0 (6.2-7.7)	-
Anti-diabetic therapy	67.5% (235/348)	-
Parental history DM	47.4% (165/348)	18.3% (104/567)*

*p<0.0001; **p=n.s.;
EASD/ESC Guidelines 2007: LDL-c target = 1.8 - 2.0mmol/L if DM and CVD; increased risk of CVD if triglycerides >1.7mmol/L, HDL<1 (men)/<1.2 (women)

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Risk factors in the Portuguese population with diabetes: diabetes prevalence study in Portugal

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Background and Aims: Background and aims: The objectives of this study were to determine the prevalence of risk factors in type 2 diabetes in the Portuguese population aged between 20 and 79 years
Materials and Methods: Taking into account the number and distribution of inhabitants (7,657,529 people aged between 20 and 79 years), 122 representative units were selected. The total sample participating in the study comprised 5,167 subjects. Data were standardized for the Portuguese population aged 20-79 years. Arterial hypertension (IDF/ESC/EASD) systolic values >=130 mm hg and diastolic >= 80 mm hg were considered hypertension. Excess weight and obesity - were defined using the BMI (body mass index), IDF/ESC/EASD criteria: Excess weight >= 28 kg/m2 and obesity >= 30 kg/ m^2. Abdominal perimeter >= 94 in men and 80 in women was considered abnormal (IDF criteria).
Results: Arterial hypertension - (TA >= 130/80) was found in 70.9% (95% CI 68.0% to 74.0%) of diabetic people in both sexes, 75.9% in males and 66.4% in females. Only 46.8% of the men and 58.4% of the women with arterial hypertension were taking anti-hypertensive medication. LDL cholesterol - Only 10.8% (95% CI 7.8% to 13.0%) of the women with diabetes had LDL Cholesterol <100 mg/dl and only 15.5% (95% CI: 12.4% to 19.1%) of the men with diabetes had LDL Cholesterol <100 mg/dl. Triglycerides - were < to 150 mg/dl in 57.7% (CI 95%: 54.4%-60.8%) of people with diabetes, with or without medication. HDL - Cholesterol - 39.7% (95% CI: 35.3% to 44.4%) of the female population had values < to 50 mg/dl and 21.2% (95% CI: 17,7% to 25.2%) of the males had values < to 40 mg/dl. Abdominal perimeter (AP) - The average abdominal perimeter in diabetic men was 102.4 cm (95% CI 101.3 to 103.4); median = 103. It was > 94 cm in 77.1% of the total. The average AP in non-diabetics was = 97 (95% CI 96.4 to 97.6) median 97, with AP> 94 cm in 59.6% of the total. In diabetic women, the average abdominal perimeter was 101.1(95% CI 99.9 to 102.2); median = 101. In 96.3% of all diabetic women it was > 80 cm. The average AP of non-diabetic women was = 92.8 (95% CI 92.3 to 93.3) median 92.2. Body Mass Index (BMI) - A significant difference was detected between the figures in diabetic people and non-diabetics and also between men and women. Non-diabetic men have an average BMI of 27 (95% CI 27.3 to 27.7), diabetic men - average BMI 29.3 (95% CI 29.0 to 29.7), variance analysis - F=65.247; p<0.0001; the figures for women are: average BMI in non-diabetic women 28.2 (95% CI 28.0 to 28.4) and in diabetic women 31 (30.5 to 31.5) - variance analysis - F=105.6253; p<0.0001.
Conclusion: Diabetes is a chronic disease with a high prevalence in Portugal, one of the highest in Europe. In addition to glycemic control there is a marked failure in the control of risk factors particularly blood pressure, dyslipidemia and obesity. A significant number of people take medication but remain uncontrolled. Strategies to underline these factors must be implemented not only among patients but also among health professionals.

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Risk determinants for type 2 diabetes

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Background and Aims: Diabetes is a growing worldwide problem. In 1995 the prevalence of diabetes in Portugal was estimated to be about 5.1% of the population. In 2010 the first national study on the prevalence of diabetes (PREVADIAB) revealed a prevalence of 12,3% of the adult population aged between 20 and 79 years old. With this subsequent analysis we aimed to find risk determinants for type 2 diabetes.
Materials and Methods: Using the 2001 Portuguese Census, a random sample of people aged between 20 and 79 years was selected from 122 locations representative of the distribution of the Portuguese population. Demographic characteristics were registered and an OGTT was performed. Diabetes WHO criteria were used for the diagnosis of diabetes.
Results: The total sample consisted of 5167 people that corresponds to an 83.8% response rate. A total prevalence of diabetes (diagnosed and undiagnosed) of 12,3% (CI 10.8-12.6%) was found with 43% of undiagnosed cases. The highest prevalence was in male gender (14,6% vs 10,2% in females) and in older groups (2,0% in the group 20-39 years, 12,8% in the group 40-59 years old and 27,1% in the 60-79 years old). People with the lowest level of literacy (not finishing first level of education) had a prevalence of 30,3%, different from the ones with the first level (19,4%) and the secondary level (7,9%) and university level (6,6%). In the group with BMI >30 the diabetes prevalence was 19,5%, also different from the group with BMI between 25-30 (12,1%) and the group with normal BMI (5,5%).
Conclusion: The diabetes growing prevalence raises governments' and health systems' concerns. In this study we showed an average prevalence of 12,3% in an adult population. As in other countries gender differences were found, higher in men than women. According to the known physiopathology of type 2 diabetes, we also found that the prevalence of diabetes has a strong correlation with BMI. However, the greatest difference between groups was the literacy level, showing that the lowest level of literacy was related to the highest prevalence of type 2 diabetes. This could be one of the greatest risk determinants for type 2 diabetes.

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Key role of AMPK in glucose-evoked Na,K-ATPase modulation

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Background and Aims: Na,K-ATPase is an integral plasma membrane protein responsible for generating and maintaining transmembrane ionic gradients. In pancreatic β-cells, Na,K-ATPase is regulated by glucose and this regulation is impaired in glucose intolerant subjects, however, the underlying mechanism is still unclear. Since glucose has marked effects on intracellular ATP and AMP levels and AMP-activated protein kinase (AMPK), a key player in energy homeostasis providing exquisite sensitivity to small changes in AMP levels, the involvement of AMPK in the cascade of events regulating Na,K-ATPase in pancreatic β-cell was postulated. The aim of this work was to evaluate the putative role of AMPK in the glucose-evoked regulation of Na,K-ATPase activity in the pancreatic β-cell.
Materials and Methods: Pancreatic β-cells from normal (control) or glucose-intolerant Wistar rats (GIR) were isolated and cultured (48h). After a pre-incubation (30min) with 2.1mM glucose (G2), cell batches were challenged with G2 or G8 (8.4mM glucose) for 20min, in the presence or absence of AMPK agonist (AICAR 1mM) and antagonist (compound C (CC), 10μM). Na,K-ATPase activity was assessed in intact cells by quantification of Pi, in the absence and presence of 1mM ouabain. Immunocytochemistry (ICC) of β-cells treated as previously described was performed using anti-α1-Na,K-ATPase and anti-phospho(ser-23)-α1-Na,K-ATPase antibodies. Western blots (WB) were performed in lysates of islets incubated in similar conditions plus AICAR or CC to evaluate α1-Na,K-ATPase (ser-23) phosphorylation.
Results: In G2 the activity of Na,K-ATPase from control and GIR pancreatic β-cells was similar (0.184±0.030 and 0.186±0.020 μmolPi/min/mgProt, respectively). Challenging the GIR β-cells with G8 evoked a significantly lower inhibition (40%) of Na,K-ATPase activity compared to a 62% inhibition observed in control β-cells. In control β-cell, the addition of AICAR abolished glucose-induced Na,K-ATPase inhibition (0.166±0.011 μmolPi/min/mg) whereas CC had no effect (0.063±0.003 μmolPi/min/mg). In the contrary, in GIR β-cells CC significantly potentiated glucose-evoked inhibition of Na,K-ATPase to values similar to those observed in the controls (66%). WB analysis revealed that Na,K-ATPase-α1 (ser-23) phosphorylation was increased by G8 (28±6% over basal) and abolished by AICAR. Additionally, CC induced an increase in phosphorylation equivalent to that observed in G8 (22±5% over basal). ICC showed an equivalent immunostaining intensity for α1-Na,K-ATPase despite glucose concentration. However, for the phosphorylated (ser-23) α1-Na,K-ATPase, a higher intensity was observed in cells exposed to G8 compared to G2.
Conclusions: The AMPK agonist AICAR counteracted the glucose inhibitory action on Na,K-ATPase from control β-cells whereas CC amplified the glucose-induced inhibition of Na,K-ATPase from GIR β-cells. These results suggest that AMPK plays a key role in the cascade of events regulating Na,K-ATPase and that the defect in GIR β-cells must be upstream of AMPK. AMPK inhibition by glucose metabolism and subsequent activation of PKC, phosphorylating Na,K-ATPase in ser23, may constitute steps of the mechanism underlying glucose-induced inhibition of Na,K-ATPase that might be uncoupled in GIR. Occurring prior to overt type 2 diabetes, this might be a feature of the disease development.

Página da SPD
SPD Page

AGENDA DE CONGRESSOS

2012

XVIII Curso Pós-Graduado de Endocrinologia, Diabetes e Metabolismo
29 a 31 de Março
Local: Centro de Congressos do Hotel Porto Palácio, Porto
Organização: Serviço de Endocrinologia, Diabetes e Metabolismo do Hospital S. João/Faculdade de Medicina do Porto

II International Advanced Course on Endocrinology, Diabetes and Nutrition
17 a 19 de Maio
Local: Hotel Porto Palácio, Porto

72th Scientific Sessions of the American Diabetes Association (ADA)
72ª Reunião Científica Anual da Associação Americana de Diabetes (ADA)
8 a 12 de Junho
Local: Filadélfia, Pensilvânia, EUA
Informações: www.diabetes.org

48th EASD Annual Meeting
48ª Reunião Científica Anual da Associação Europeia para o Estudo da Diabetes
1 a 5 de Outubro
Local: Berlim, Alemanha
Informações: www.easd.org