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.

470
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