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Miscellaneous

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The role of ATP-sensitive potassium channels in the vasodilatory effect of N-acetylcysteine

F. Söğüt[1], Ü. Çömelekoglu[2], Ö. Vezir[3], N. Sucu[4], A. E. Yalin[4], S. Yalin[5], N. Yilmaz[6], S. Yaman[7], M. I. Karagül[8], K. Kibar[6], M. Akkapulu[5], D. Seçer[5]

[1]Vocational School of Medical Services, Mersin University, Mersin, Turkey, [2]Department of Biophysics, Faculty of Medicine, Mersin University, Mersin, Turkey, [3]Mersin State Hospital, Mersin, Turkey, [4]Department of Cardiovascular Surgery, Faculty of Medicine, Mersin University, Mersin, Turkey, [5]Department of Biochemistry, Faculty of Pharmacy, Mersin University, Mersin, Turkey, [6]Department of Histology and Embriology, Faculty of Medicine, Mersin University, Mersin, Turkey, [7]Department of Biophysics, Faculty of Medicine, Kahramanmaras Sütçü Imam University, Kahramanmaras, Turkey

N-acetyl cysteine's (NAC) anti-inflammatory, antioxidant and vasodilator effects are well known. However, explaining the mechanism of the effects are not sufficient. In this study, on the vasodilatory effect of NAC, role of ATP-sensitive potassium (K_{ATP}) channel was investigated by using electrophysiological and molecular genetics methodologies.

In this study, aorta smooth muscle cell lines were used. One control and three dose groups were studied. The control group were not exposed any treatment. The dose groups were treated with 2, 5 and 10 mM NAC respectively, using the cellattached patch clamp K_{ATP} channel currents were measured for 10 min. Intracellular

calcium levels in the same groups has been monitored using confocal laser scanning microscope by taking images at 488 nm wavelength with 15 s intervals for 10 min. K_{ATP} channel gene expression levels were assayed using real-time quantitative reverse transcription polymerase chain reaction. K_{ATP} channel gene expression (Kcnj8, Kcnj11, Abcc8, Abcc9) levels for each group were determined.

K_{ATP} channel flow was significantly increased in all dose groups compared to the control group. However, significant differences were not found among dose groups. Although, intracel-

non-esterified fatty acids, glycerol, hormones, cytokines. The factors that are involved in the development of insulin resistance. Accumulating evidence suggests that endoplasmic reticulum (ER) stress is present in type 2 diabetes. Protein glycosylation enzymes controls polypeptide folding and many intracellular mechanisms in the ER and cytoplasm. Glycosylation is a necessary modification for determination of protein structure, function and stability. Cytoplasmic and nuclear proteins are modified by a single O-GlcNAc moiety at serine or threonine residues, termed O-GlcNAcylation. This modification has been demonstrated to play critical roles in numerous biological processes, including cell signaling, transcription, and disease etiology and regulated in response to nutrients, stress, and other extracellular stimuli. Altough the role of O-GlcNAcylation in insulin signaling and endoplasmic reticulum stress in liver, skeletal muscle are well established; the relationship between O-GlcNAcylation and adipose tissue is largely unkown.

We determined role of O-GlcNAcylation in genetically obese mice and wild type mice challenged with glucosamine (GlcN). Insulin signalling pathway, hexosamine biosynthetic pathway and endoplasmic reticulum stress markers were investigated in adipose tissues.

In increased insulin resistance conditions it has been shown that O-GlcNAcylation levels are decreased in adipose tissue of obese mice. Then we confirmed GlcN challenged group has a similar phenotype for insulin resistance and ER stress.

Our results suggest that O-GlcNAcylation of proteins in obese mice regulated by an end product of the hexosamine biosynthetic pathway uridine diphosphate N-acetylglucosamine (UDP-GlcNAc) inhibition. Our findings imply that O-GlcNAcylation has an important role for development of type 2 diabetes and metabolic syndrome in obesity.

Monday 5 September
17:30–19:30, Hall B

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