TACSM Abstract

Insulin Sensitivity Effects on Peripheral Vascular Responses to FMD in Metabolic Syndrome Women

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ABSTRACT

Sedentary populations with obesity and metabolic syndrome (MetSyn) have presented with impaired vascular dysfunction, including vasodilation reduced 40-50%. The mechanisms of vascular endothelial dysfunction has also been associated to the bioavailability of nitric oxide levels and in metabolic compromised individuals the diminished content of endothelial NO are a prime mechanistic target for study. **PURPOSE**: We hypothesize that decreased reactive hyperemia observed in MetSyn compared to age matched-control patients are primarily due to mechanistic dysfunction of the eNOS pathway and lower Insulin Sensitivity Index (ISI). **METHODS**: 30 participants (10 MetSyn and 20 Controls) completed brachial FMD testing and vascular changes were recorded using Doppler ultrasound with a linear vascular probe. A blood pressure cuff was placed on the upper forearm and upper calf for analysis of brachial and popliteal arteries sheer rate and reactive hyperemia. Images were analyzed with Brachial Analyzer software and sheer rate calculated by digital recordings of blood velocity with a digital auditory transducer recordings with a BIOPAC 150 system and AcqKnowlege software. Insulin sensitivity index was assessed by an oral glucose tolerance test with fasting and post-prandal glucose measured with a glucometer and insulin measured by a Human Insulin ELISA kit (Cayman Chemical). The resting bioavailability of Nitric Oxide assessed by Nitrate/Nitrite ELISA assay and NO EPR spectroscopy measurements. **RESULTS**: In the brachial arteries there is significant differences ($P<0.05$) in the time and rate to peak diameter from reperfusion but not within the % Dilation or sheer rate between the MetSyn and Control sedentary groups. In the popliteal arteries, there is a significant difference between the % dilation, time and rate to peak from reperfusion. We observed correlations among changes in arterial measurements, Insulin Sensitivity Index (ISI) and resting Nitric oxide concentration (NO). ISI is observed to be correlated to Peak Diameter ($p=0.0943$, $r= 0.59$) and Rate to Peak ($p=0.07$,$r=.0628$) measurements in the MetSyn group in the brachial arteries with no calculated relationship identified in the popliteal artery. **CONCLUSION**: We predict that the significant deficiencies observed between the control and MetSyn group may be explained by the vascular mechanisms of developing deficits associated with the metabolic deficiencies. A postulated mechanism of this endothelial dysfunction during insulin resistance begins with the decreased sensitivity of the insulin receptor preventing the effect of insulin and the AKT/PKB eNOS pathway. Research reported in this publication was supported by an Institutional Development Award (IDeA) from the National Institute of General Medicine Sciences of the National Institutes of Health under grant number P20GM103451.