**DIMINISHED ENDOTHELIAL FIBRINOLYTIC CAPACITY IN ADULTS WITH IMPAIRED FASTING GLUCOSE**

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Over 12 million Americans have impaired fasting blood glucose (IFG: 100-125 mg/dL). IFG is a risk factor for type 2 diabetes, hypertension and atherosclerosis. Endothelial cells are the principal site of synthesis and release of tissue-type plasminogen activator (t-PA), the main plasminogen activator in fibrinolysis. The capacity of the endothelium to release t-PA rapidly and acutely is a primary defense mechanism against thrombosis. We tested the hypothesis that the capacity of the endothelium to release t-PA is impaired in adults with IFG. Fifty sedentary, normotensive adults were studied: 30 with normal fasting glucose (NFG; 18M/12F; age: 58+2 yr; glucose: 90+1 mg/dL); and 20 with IFG (12M/8F; 59+2 yr; 105+1 mg/dL). Net endothelial release of t-PA was determined, in vivo, in response to intrabrachial infusions of bradykinin (BK: 12.5-50.0 ng/100 mL tissue/min) and sodium nitroprusside (SNP: 1.0-4.0 g/100 mL tissue/min). Rate of t-PA release was determined as the product of venoarterial concentration gradient and forearm plasma flow. t-PA release to BK was significantly blunted (~30%) in the IFG (1.0+0.6 to 42.6+4.7 ng/100 mL tissue/min) vs NFG (-0.2+0.7 to 58.8+4.3 ng/100 mL tissue/min) group. As a result, total t-PA release (area under the BK curve) was ~30% lower (p<0.05) in the IFG (222+24 ng/100 mL tissue) than NFG (307+23 ng/100 mL tissue) adults. There was a strong inverse relation (r=-0.35; p<0.05) between fasting blood glucose and total t-PA release. In summary, endothelial t-PA release is impaired in adults with IFG. Endothelial fibrinolytic dysfunction may contribute to the increased thrombotic risk with IFG.