



INTRODUCTION

Pigment epithelium-derived factor (PEDF) is a glycoprotein that belongs to the super family of serine protease inhibitors. It was first purified from conditioned medium of human retinal pigment epithelial cells as a factor with potent neuronal differentiating activity (*Matsuyama et al., 2008*).

Recently, PEDF has been shown to be a highly effective inhibitor of angiogenesis in cell culture and animal models. PEDF inhibits the growth and migration of cultured endothelial cells (EC), and it potently suppresses ischemia-induced retinal neovascularisation. PEDF levels in aqueous or vitreous humors are decreased in patients with diabetes, especially those with proliferative retinopathy (PDR) (*Yamagishi et al., 2010*).

It was found recently that PEDF inhibits TNF- α -induced nuclear factor- β activation and subsequent IL-6 expression in endothelial cell by suppressing Nicotinamide Adenine Dinucleotide Phosphate oxides – mediated reactive oxygen species generation. Also prevented advanced glycation end products or angiotensin II-induced EC activation through its anti-oxidative properties (*Katakami et al., 2008*).

Metabolic syndrome is defined according to Adult Treatment Panel (ATP III). ATP III identified five components of metabolic syndrome (abdominal obesity, given as waist circumference (>101.6 cm for men and > 88.9 cm for women) ,triglycerides (≥ 130 mg// dl) ,HDL cholesterol (<50 mg/ dl for men and <40 mg/dl for women), BP($\geq 130/\geq 85$ mmHg) and fasting plasma glucose (≥ 110 mg/dl) (*Yamagishi et al.,2006*).



It is identified a novel role for PEDF as a negative regulator of insulin action in obesity. Given the observation that PEDF is increased in obese type 2 diabetic humans (*Yamagishi et al., 2006*) and (*Jenkins et al., 2008*) , therapeutic strategies to inhibit PEDF action in muscle and liver, or prevent adipocyte PEDF release, may prove a viable approach to ameliorate obesity-induced insulin resistance and its associated pathologies (*Akin et al. ,2012*).