INTRODUCTION

Pregnancy is associated with dramatic changes in energy metabolism to meet the increased energy demands of fetal growth and maternal nutrition ***(Akdeniz et al.,2011)***. The developmentofinsulin resistance in the second-half of pregnancy is observed in normal pregnancy and is greatest during the third trimester ***(Nanda et al.,2012)***. The insulin changes over the course of pregnancy suggest that insulin resistance is mainly due to placental hormones, although the underlying mechanisms are not fully understood. Furthermore, insulin resistance has been proposed to be exaggerated in pre-eclampsia (PE) and to play a role in the pathogenesis of such a disorder ***(Lynch et al.,2010)***.

Pre-eclampsia, a pregnancy-specific complication of the second half of pregnancy is characterized by hypertension and proteinuria, and is one of the leading causes of perinatal mortality and morbidity ***(Lau and Muniandy,2011).*** Pre-eclampsia (PE) occurs in 6-8% of pregnanciesworldwide***(Kralisch et al., 2009)***. To the mother, it can cause multi-system dysfunction including renal failure, hepatic failure, coagulopathy and central nervous system disorders. To the fetus, it may lead to fetal growth restriction, prematurity and perinatal death***(Gül et al.,2012).***

The complete pathogenesis of this disease remains unclear. Deficient remodeling of the spiral arteries during the interaction between maternal and fetal sides at the time of trophoblast invasion has been postulated as a cause of placental insufficiency. This would lead to the release of inflammatory factors in the systemic maternal circulation ***(Young et al., 2010)***.Some studies suggested thatadipokines may play an important role in the pathogenesis of pre-eclampsia through their role in low-grade systemic inflammation, atherosclerosis, and insulin resistance. Therefore, it is reasonable to suppose that adipokines may directly or indirectly influence the function of placental endothelial cells ***(Arikan et al., 2009)***.

Insulin resistance is defined clinically as the inability of a known quantity of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as much as it does in a normal population ***(Jenum et al.,2012).***

HOMA (Homeostasis model assessment) was first developed in 1985 by Matthews. HOMA describesglucose–insulin homeostasis by means of a set of simple, mathematically derived nonlinear equations. The approximating equation for IR has been simplified and uses a fasting plasma sample in which glucose (fasting blood glucose; FBG) and insulin (fastinginsulin; FI) are measured, together with a constant. The product of FBG × FI is an index of IR. HOMA-IR = (glucose × insulin)/22.5. Insulin concentration is reported

in μU/L and glucose in mmol/L. The constant of 22.5 is a normalizing factor, i.e. normal FI of 5 μU/mL × the normal FBG of 4.5 mmol/L typical of a 'normal' healthy individual = 22.5 ***(Nanda et al.,2012).***

Adipokines, physiologically active polypeptide hormones derived from adipose tissue, are associated with the state of insulin resistance during pregnancy. Adiponectin, leptinand resistin are the most intensively investigated adipokines ***(Hivert et al., 2009)***. Resistin is an adipose-specific secreted hormone, which belongs to the family of cysteine-rich, c-terminal proteins.Itis a potent regulator of glucose homeostasis that is thought to oppose the action of insulin in peripheral tissues; it impairs glucose intake by adipocytes, increases plasma glucose concentration, and thus decreases insulin ***(D'Ippolito et al.,2012).*** Resistin is expressed in the human placenta and its serum level is enhanced in the third trimester of pregnancy; suggesting its pivotal role in the state of insulin resistance during pregnancy ***(Young et al., 2010).***