INTRODUCTION

Preeclampsia, a syndrome unique to human pregnancy, is associated with increased maternal and fetal complications. It is the second leading cause of maternal death in the United States and the leading cause of maternal death in many parts of the world**.** ***(Dekker and Sibai, 1998)***. In addition, it is a major cause of preterm delivery, fetal growth retardation and perinatal mortality***(Odendal et al., 1990)***. The etiology of preeclampsia is unknown. During the past 100 years numerous clinical, biophysical and biochemical tests have been suggested to identify women who are at increased risk for future development of preeclampsia .***(Dekker and Kraayenbrink, 1999)***.

Leptin, the product of obesity gene, is a hormone mainly expressed in adipocytes(***Zahang et al., 1994)***. Through a negative feedback mechanism between adipose tissue and hypothalamic centers, leptin may contribute to regulation of obesity by inducing satiety and stimulate energy expenditure at the expense of storage (***Rohner, 1999).***

The regulation of leptin is not fully understood, but a covariation with insulin is documented. Circulating leptin levels reflect body fat contents , and association with insulin has linked leptin to the insulin ressistance syndrome, which includes obesity, glucose intolerance and dyslipoproteinemia (***Zimmet et al., 1999)***.

In addition to its relation to obesity, leptin may stimulate maturation of the reproductive axis. There is a gender difference in leptin, with higher concentrations in females than in males ***(Hassink et al., 1996)***.

In pregnancy, it has been shown that leptin is highly expressed in the placenta ***(Masuzaki et al., 1997).*** The protein has been detected in umbilical cord blood from week 18 of gestation, followed by increased level from the middle of the third trimester toward term ***(Jaquet et al., 1998)***.This increase coincides with the development of fetal adipose tissue and the results from some previous studies indicate that cord blood leptin is positively correlated with fetal adiposity at birth ***(Jaquet et al., 1998).***

The growth retarded infants exhibit wasting of subcutaneous fat and one would therefore expect lower leptin levels in umbilical cord blood from preeclamptic than from normotensives pregnancies.However one previous small study found no difference in cord leptin levels between cases of preeclampsia and controls with delivery at term***(McCarthy et al., 1999).***

Preeclampsia is associated with maternal obesity and maternal levels of circulating leptin appear to be increased in preeclamptic compared with normotensive pregnancies***(McCarthy et al., 1999 , Laivuori et al., 2000)***.

Because leptin may induce metabolic and circulatory changes that are characteristic of preeclampsia (***Haynes, 2000),*** it has been suggested that leptin may play a role in the pathogenesis of preeclampsia ***(McCarthy et al., 1999 , Laivuori et al., 2000).***

Since hyperleptinemia precedes established clinical symptoms of preeclampsia, measurements of plasma leptin have been proposed as a predictive non-invasive marker of preeclampsia. In addition, it may be

taken as an index of placental dysfunction ***(Lepercq et al., 2003).***

Several independent investigators have demonstrated through human and animal studies the association of androgens, especially testosterone, with hypertension ***(Baker et al., 1998).***

Interestingly accumulating evidence indicates that androgens have an important effects on vascular reactivity, the renin-angiotensin system, eicosanoids and platelet in preeclampsia ***(Acromite et al., 1999).***

Recently several studies on primigravid women have compared sex steroid concentrations between women with preeclampsia and those with normotensive pregnancies after controlling for body mass index (BMI) maternal age and gestational age ***(Acromite et al.,1999 and Serin et al., 2001).*** Testosterone (T) levels were reported to be significantly higher in preeclamptic patients than in nomotensives controls in the third trimester of pregnancy, suggesting the possible role of androgens in the pathogenesis of preeclampsia ***(Serin et al., 2001)***.

Total testosterone levels were significantly higher in pregnancies with either gender and significantly higher in male-bearing than in female-bearing pregnancies. This may indicate an androgen an androgen influence on the pathophysiologic mechanism of preeclampsia ***(Steier et al., 2002)***.

Atamer et al(2004) considered that elevated leptin and testosterone contribute to the endothelial dysfunction involved in the pathogenesis of preeclampsia.