Arginase is also found in the human placenta [21]. One study that evaluated the levels of serum hydrolases in human pregnancy found no increase in serum arginase activity in the first, second, or third trimester of pregnancy [21]. It is quite possible that arginase activity in pregnancy is increased significantly in the involved tissues, while does not increase in the serum [21]. One study on the arginase activities of various tissues in rats also found that while there was an increase in arginase activity during late pregnancy, it was not reflected in circulating urea levels [21]. Why arginase activity is increased during pregnancy is unknown. In rats, inhibiting uterine arginase activity had arrested the embryonic development [21]. This could be secondary to its effects on polyamine synthesis [21]. The timing of the increase in arginase activity at the end of pregnancy and the decrease in NO production at this time may reflect normal enzyme interaction. It is quite feasible that the increase in arginase activity is part of the trigger that normally decreases the myometrial NOS activity just prior to, and in preparation for parturition [21].

In studies on rats and mice, testosterone has been shown to stimulate arginase activity [21]. It was found that testosterone elicited a 50% decrease in the enzyme ornithine carbaomoyl transferase (OCT). Inhibiting OCT may cause a significant decrease in endogenous L-arginine production [21].

Patients with preeclampsia have been shown to have higher levels of testosterone than the level of testosterone typical of nonpreeclamptic pregnants. If testosterone stimulates the arginase in humans, then this could potentially decrease the L-arginine available to NOS and thus increase production of O_2^-; this was supported by the negative correlation between NO and testosterone serum levels found in preeclamptics, which was lost in normal gestation as seen in Figures:1.2,3 & 4

Biochemical changes in preeclampsia appear to be driven by over-production of testosterone (probably induced by placental dysfunction) which may lead to a reduction in nitric oxide synthesis (as evident by low serum nitrite). While measuring NO and testosterone before 20^{th} week gestation can be used as predictor of the disease.

Table 1: The mean serum testosterone, nitric oxide and nitric oxide synthase (NOS) in different preeclamptic and control groups (presented as mean ± SD).

Variable	G1	G2	G3	G4
testosterone (ng/ml)	1.89 ± 0.6**	2.9± 2.4**	0.85 ± 0.7	0.72 ± 0.3
Nitric oxide (µmol)	6 ± 0.9**	4.1± 2.4**	8.1 ± 3	8.8 ± 3.3
NOS (µmol/g/min)	0.08±0.01*	0.06±0.03*	0.1 ± 0.04	0.11 ± 0.04

NOS activity is expressed as nitrite / g protein / min.
** p< 0.05 , ** p < 0,01*