

# Role of Prostaglandin in Reproductive Physiology

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**Abstract:** *The role of prostaglandin in reproductive physiology is reviewed with particular emphasis on their possible importance in ovulation in humans. A possible interaction between gonadal steroids, biogenic amines and prostaglandin at hypothalamic-pituitary level, in relation to the release of luteinizing hormone releasing factor, and LH release are noted, and it is suggested that high oestrogen level may release prostaglandin from the uterus and centrally in humans in connection with the mid-cycle LH surge and ovulation. Prostaglandin are a group of lipid compound that are derived enzymatically from fatty acids and have important function in the body.*

**Keywords:** Anti ageing, chromosome

## 1. Introduction

Prostaglandin are closely related compound are a family of lipid substances derived from arachidonic acid which have potent and diverse pharmacological action. The role of prostaglandin in fertilization and their effect on the uterus are mentioned briefly. Their effect on the ovary in relation to progesterone synthesis, luteolysis and ovulation are discussed. Interaction between gonadal steroids, catecholamines and prostaglandin on the hypothalamus are discussed in relation to the release of luteinizing hormone releasing factor (LRF). LRF a polypeptide, is the hypothalamic hormone that controls that secretion of luteinizing hormone and follicle stimulating hormone from the pituitary.

## 2. Prostaglandin Biosynthesis

Prostaglandin are found in most tissue and organ. They are produced by almost all nucleated cells. They are autocrine and paracrine lipid mediators that act upon platelet, endothelium, uterine and mast cells. They are synthesized in the cell from the essential fatty acids. An intermediate arachidonic acid is created from diacylglycerol via phospholipids-A<sub>2</sub>, then brought to either the cyclo oxygenase pathway or the lipoxygenase pathway to form either prostaglandin and thromboxane, prostacyclin and prostaglandin D, E and F. Alternatively, the lipoxygenase enzyme pathway is active in leukocytes and in macrophages and synthesis leukotrienes.

## 3. Release of Prostaglandins from the Cell

Prostaglandin were originally believed to leave the cell via passive diffusion because of their high lipophilicity. The discovery of the prostaglandin transporter, which mediates the cellular uptake of prostaglandin.

## 4. Prostaglandin in Uterus

PGE<sub>1</sub>, PGE<sub>2</sub> and PGF uterine contraction in man, and the F prostaglandin contributes to uterine contraction at parturition. Intravenous infusion of PGF have been used to induce uterine contraction in pregnant women. The result show that: 1. The sensitivity of the myometrium to exogenous PG increases from day 19-22 of gestation. 2. The electrical response to PG, at maximally effective doses consists of slow depolarization which upon reaching

threshold initiates spike discharge. 3. These action are most pronounced 22 day and are due to a direct action on PG on the myometrical cells. 4. D-600 (a methoxy derivative of verapamil) abolish spike discharge and the phasic contraction induced by PG but has no effect on the slow depolarization and increased in tonic tension. 5. Slow depolarization depends on the presence of sodium in the external environment and is unaffected by the removal of calcium. 6. The spikes are dependent on the presence of calcium on the external environment.

## 5. Role of Prostaglandin in Ovary

Prostaglandin are involved in LH-induced progesterone synthesis and have a role in luteolysis. Luteolysis is the process by which the corpus luteum loses its capacity to synthesize and secrete progesterone. A luteolytic agent is any factor that can reduce luteal progesterone synthesis and prevent the action of luteotrophic hormone. A specific inhibitor of the prostaglandin contractile response, 7-oxa-13 prostenoic acid, blocked not only the effect of PGE<sub>1</sub> and PGE<sub>2</sub>, but also that of LH on cyclic AMP and progesterone formation. Although prostaglandin stimulate progesterone production, they may inhibit progesterone synthesis in vitro.

The factors controlling the release of LHR from the hypothalamus and of LH from the pituitary are complex and many facets remain uncertain. The process is influenced by change in circulating levels of gonadal steroids, by change in intracerebral catecholamines, and by prostaglandin and prostaglandin inhibitors.

## 6. Role of Prostaglandin in Ovulation

The position of the prostaglandin in the scheme of events leading to the ovulation. It is not assumed that all of the events depicted occur in a single cell type, although many of them are capable of occurring in isolated granulosa cells. The ovulatory surge of LH initially acts upon follicular cells by binding to its receptor, activating adenyl cyclase, and causing rise in intracellular cyclic AMP. FSH plays an important role in this process. Cyclic AMP appears to involve in protein kinase activation, protein synthesis, increased steroidogenesis and prostaglandin synthesis. The functions of the preovulatory prostaglandin increase in the ovulatory process. It has been suggested that follicular rupture involves an enzymatic weakening of the follicular wall and prostaglandin might be involved in the activation, release, or synthesis of an ovulatory enzyme.

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At the time of ovulation some women experience nausea and vomiting are the effect of prostaglandin. Changes in the serum level has not been show to correlate with the menstrual cycle in female. The prostraglandin E and F content of human semen is reduced by therapeutic doses of soluble aspirin, a less potent inhibitor of prostaglandin synthesis that indomethacin. If prostaglandin are shown to have a circular part to play in the release of LRF and LH in humans, it should be possible to find a prostaglandin antagonist that will specifically antagonize this rection and act as an effective contraceptive.

## **7. Adrenergic Mechanism in Ovulation**

Catecholamines have long been thought to have a role in the neural control of pituitary secretion. Adrenergic blocking drug delay ovulation in rat and block ovulation in rabbit. Catecholamine depleting drug, interrupts the normal oestrus cycle reduces LH contraction in the hypophysis. Injection of adrenaline or nor-adrenaline into the cerebral ventricles produces ovulation .

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