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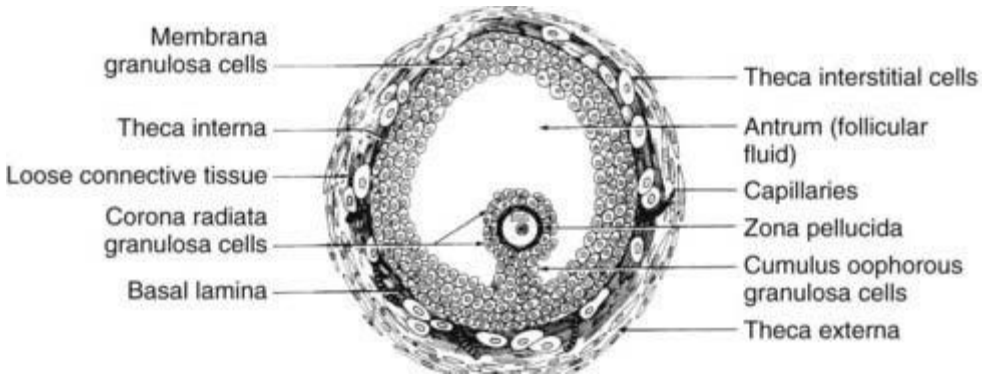
The corpus luteum (Latin, *corpus* = body, *luteum* = yellow) develops from the remains of Graffian follicle after ovulation. The corpus luteum (CL) is a transitory endocrine gland that forms on the ovary from the granulosal and thecal cells that remain in the postovulatory follicle. Its function is to secrete progesterone, preparing the uterus for implantation, as well as maintaining pregnancy by promoting uterine quiescence. If fertilization does not occur, or if embryonic signaling is insufficient, the CL will regress. Luteal regression (luteolysis) results in pregnancy failure, but also removes the negative feedback of progesterone on gonadotropin release, allowing for maturation and ovulation of a new follicle and another opportunity to establish a pregnancy. The ephemeral nature of the CL has intrigued scientists for decades and understanding the mechanisms responsible for luteolysis could shed light on the nature of cellular life or death in other tissues, including tumors. Although a viable CL is essential for reproduction in all mammals, the hormonal events that initiate luteolysis and the cellular mechanisms to sustain or overcome it remain at least partially elusive. This article will highlight the established pathways leading to luteal regression or rescue. For more information and specific citations of work to elucidate luteolytic mechanisms, the reader is referred to previous reviews listed in the Bibliography for this article.



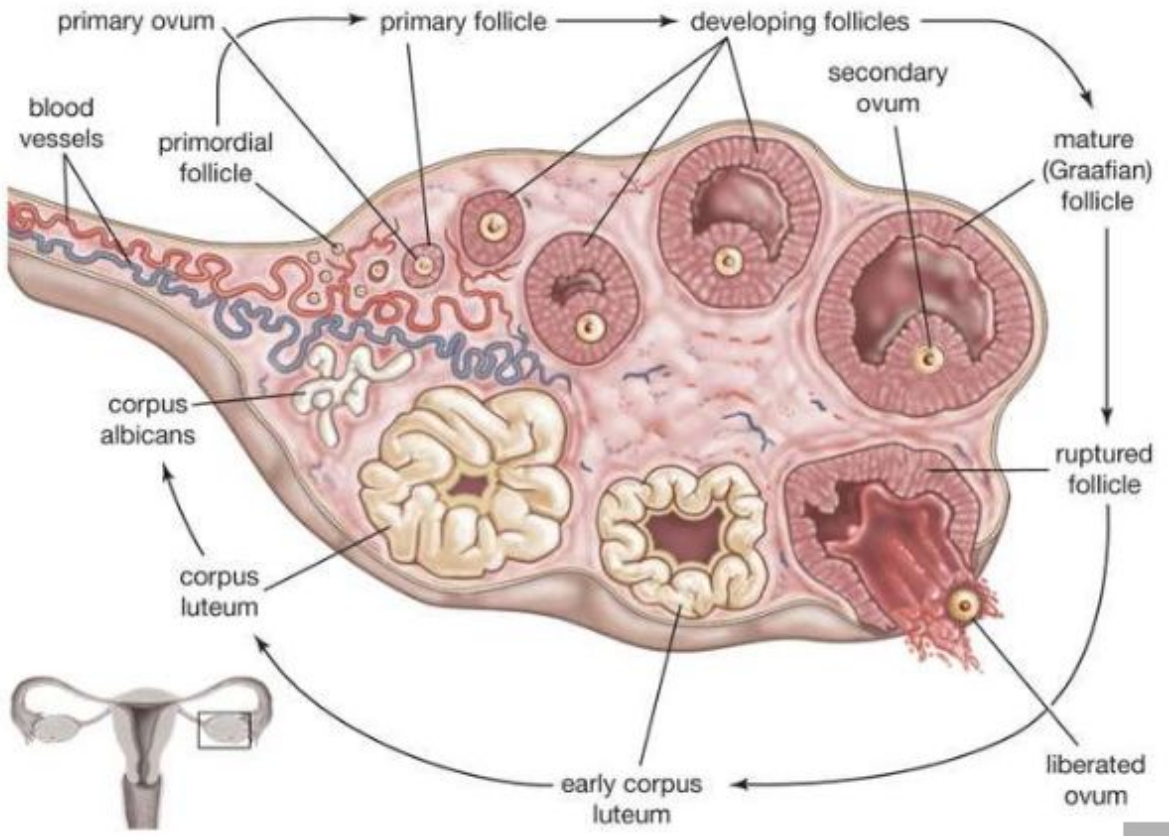
Granulosa Lutein Cells

The internal follicular granulosa cells, differentiate into the granulosal lutein cells (large luteal cells). This luteinization change occurs within 30 to 40 hours of the ovulatory LH surge and the cells become are terminally differentiated. The cells begin secreting increasing amounts of both progesterone and some estrogen that support the luteal phase of the menstrual cycle.

Progesterone secretion during non-pregnancy occurs for about 10 days. These cells initially proliferate, become terminally differentiated and stop dividing.

Theca Lutein Cells

The surrounding follicular theca interna cells, differentiate into the thecal lutein cells (small luteal cells). These cells express receptors for luteinizing hormone (LH) to produce androstenedione, that supplies the granulosa cells the precursor for estrogen synthesis.



hCG in assisted reproduction

The corpus luteum generally lives for 11–12 days in nonconception cycles; progesterone levels decrease, menses follows, and the next menstrual cycle ensues. In conception cycles, the placenta must eventually take over progesterone production from the corpus luteum to prevent menses and to permit the pregnancy to succeed. hCG is secreted exponentially and the long half-life extends the life span of the corpus luteum, maintaining progesterone secretion until the placenta begins to produce significant progesterone at approximately 7 weeks of gestation. hCG can be detected in the maternal circulation around the time of implantation; it peaks around 9–11 weeks of gestation. hCG then decreases to approximately one-tenth the peak level. The secretion of hCG is crucial for

maintenance of pregnancy until approximately the seventh week of gestation. Between 7 and 10 weeks of gestation, the corpus luteum is replaced by the placenta in terms of progesterone production. This phase of luteal function has been referred to as the “luteal−placental shift.” In conception cycles, the life of the corpus luteum is extended until the luteal−placental shift occurs.

The timing of the luteal−placental shift is crucial, particularly in IVF cycles. At approximately 8 weeks of gestation, most centers used either progesterone or hCG to supplement the luteal phase. Lower doses of hCG are usually administered (1250–2500 IU) daily after pregnancy has been confirmed. Some centers combine progesterone and hCG in the luteal phase.