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Hormonal and molecular mechanisms involved in the formation of ovarian cysts in sows - review

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Abstract: *Ovarian cysts are a common reproductive disorder in sows, characterized by the presence of fluid-filled structures on the ovaries that can disrupt normal estrous cycles and fertility. Their formation is associated with dysregulation of follicular development, hormonal signaling, and molecular pathways within ovarian cells. Follicular activation and maturation are controlled by interactions between oocytes, granulosa, and theca cells, as well as by hormonal input via the hypothalamic-pituitary-ovarian axis. Dysfunctions in luteinizing hormone (LH) secretion, prostaglandin production, and signaling pathways such as PI3K/AKT/FOXO3, WNT/β-catenin, insulin, Notch, and Hedgehog can lead to abnormal follicular growth and cyst formation. Additional risk factors include endocrine disorders, age, exogenous hormone treatments, nutritional imbalances, and stress-induced glucocorticoid elevation. Molecular studies indicate that altered expression of genes such as FGF7, FGF10, LDHB, IBSP, and PRLR may contribute to the pathogenesis of ovarian cysts. Understanding these hormonal and molecular mechanisms is essential for improving reproductive management and reducing the incidence of cystic ovarian disease in swine herds.*

The ovary is an essential organ of the female reproductive system, responsible for producing oocytes and synthesizing steroid hormones such as estrogens and progesterone. These hormones regulate the estrous cycle, ovulation, and the maintenance of pregnancy. Disruptions in follicular development or hormonal signaling can lead to the formation of ovarian cysts, a common condition in sows that significantly affects reproductive performance.

Ovarian cysts in sows

Ovarian cysts are fluid-filled structures with a minimum diameter of 11 mm, although they can sometimes exceed 60 mm. Depending on their origin and structure, they are classified into three main types: follicular, luteal, and corpus albicans. Most large cysts originate from luteinized follicles. Cystic formations can be found not only in sterile sows but also in fertile or even pregnant animals, and their etiology is associated with endocrine system dysfunctions.

The role of hormones in the development of ovarian cysts

The development of ovarian cysts appears to be primarily associated with dysfunctions in the secretory activity of the hypothalamic–pituitary–ovarian axis and/or a luteinizing hormone (LH) deficiency, rather than an intrinsic ovarian abnormality. Under these conditions, follicles fail to ovulate and may develop into cysts. In addition, inadequate prostaglandin production, particularly $PGF_2\alpha$, may contribute to this process.

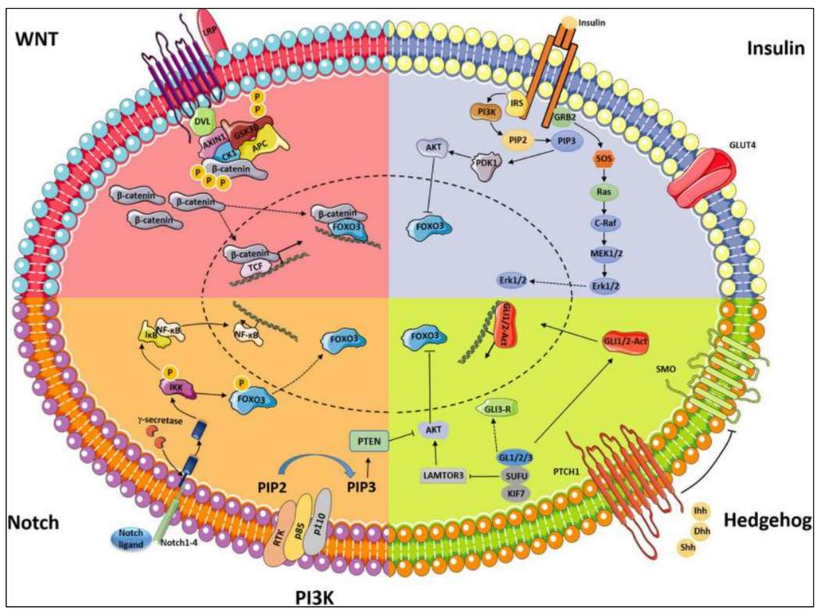
Molecular mechanisms involved in the pathogenesis of ovarian cysts

Jiage Dai et al. (2024) conducted a transcriptomic analysis and found that the mRNAs of FGF7 and FGF10 were overexpressed in TIMG cells derived from follicular cysts compared to normal mature follicles. Genetic analysis of follicular cysts revealed the presence of nonsynonymous mutations in the IBSP, LDHB, and PRLR genes, which may be involved in the pathogenesis of these cysts. These results indicate that LDHB, IBSP, and PRLR may serve as molecular markers for the early detection of follicular cysts.

Signaling pathways involved in ovarian follicle development

The PI3K/AKT/FOXO3 pathway controls the activation of primordial follicles and granulosa cell survival.

The WNT/β-catenin pathway regulates granulosa cell proliferation and differentiation. The insulin signaling pathway activates MAPK and AKT, increases sensitivity to FSH and LH, and stimulates proliferation and differentiation of granulosa and theca cells. The Notch pathway regulates granulosa cell proliferation, theca cell differentiation, and follicular vascularization. The Hedgehog pathway influences theca cell differentiation and communication between granulosa and theca cells.



Schematic representation of classical signaling pathways (WNT, insulin, Notch, and Hedgehog) and their interaction with the PI3K/AKT–FOXO3 pathway during ovarian follicle development

- Conclusions

Ovarian cysts in sows arise from a complex interplay of hormonal imbalances, disrupted intra-ovarian signaling, and environmental or exogenous influences. Molecular evidence points to key roles for genes such as FGF7, FGF10, IBSP, LDHB, and PRLR in regulating follicular cell proliferation, differentiation, and steroidogenesis, with their dysregulation contributing to cyst formation.

Environmental and management factors, such as stress, thyroid dysfunction, exogenous gonadotropin administration, and feed contaminants like micotoxins, goitrogens, and phytoestrogens, can exacerbate cyst development.