

Studies show that precise regulation of canonical WNT signaling is crucial for ovarian follicle development and fertility. Disruptions, such as through *Rspo1* amplification or *Wnt5a* deletion, lead to ovarian subfertility. Similarly, aberrant WNT signaling is implicated in granulosa cell tumors (GCTs), with increased  $\beta$ -catenin levels observed in these tumors and epigenetic silencing of the WNT antagonist *DKK3*. Genetic models activating WNT signaling demonstrate a significant increase in GCT development. This study investigates the role of APC2, a known regulator of  $\beta$ -catenin/WNT signaling, in ovarian folliculogenesis, fertility, and GCT formation. While APC2's role in regulating WNT signaling is established in other tissues, its function in the adult ovary remains largely unexplored.