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 □-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 □-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.