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INSR–PIK3CA Dysregulation as a Molecular Link Between PCOS-Related Insulin Resistance and Endometrial Cancer

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Abstract

Polycystic ovary syndrome (PCOS) is a common endocrine disorder strongly associated with insulin resistance and an elevated risk of endometrial cancer. To investigate whether shared molecular disruptions underlie this link, genomic, transcriptomic, and proteomic data from The Cancer Genome Atlas (TCGA) Uterine Corpus Endometrial Carcinoma cohort and related cBioPortal datasets were analyzed, focusing on the insulin signaling genes INSR and PIK3CA. Results showed that INSR alterations occurred in a subset of endometrial tumors, accompanied by significantly reduced transcript expression. Altered cases were enriched in low-grade endometrioid carcinomas and were associated with improved overall survival. In contrast, PIK3CA exhibited a high frequency of activating mutations, particularly the H1047R hotspot, driving hyperactivation of downstream PI3K/AKT/mTOR signaling despite lower transcript levels. Importantly, their oncogenic activity is mediated primarily through enhanced protein phosphorylation rather than increased mRNA abundance, explaining why transcript levels alone

underestimate their impact. Together, these findings indicate that dysregulation of the INSR–PIK3CA axis represents a potential molecular bridge between PCOS-related insulin resistance and endometrial carcinogenesis. Elucidating this connection may provide insight into cancer risk in women with PCOS and inform pathway-directed prevention and treatment strategies.

Keywords: Polycystic ovary syndrome (PCOS), Insulin resistance, INSR, PIK3CA, Endometrial cancer, PI3K/AKT/mTOR signaling

Introduction

Polycystic ovary syndrome (PCOS) is a multifactorial endocrine and metabolic disorder affecting approximately 6 to 21 percent of reproductive-aged women worldwide, depending on diagnostic criteria (Zhao et al., 2023). While PCOS is traditionally defined by hyperandrogenism, oligo/anovulation, and polycystic ovarian morphology, its clinical manifestations often extend to systemic metabolic disturbances, most notably insulin resistance (IR). Up to 95 percent of women with PCOS exhibit some form of IR, including lean women, indicating that IR is a core feature of the syndrome rather than a mere consequence of obesity (Zhao et al., 2023).

A key molecular player in insulin signaling is the insulin receptor gene (INSR), which encodes a heterotetrameric transmembrane tyrosine kinase receptor responsible for initiating downstream signaling cascades upon insulin binding. Variants in INSR, such as rs2059807, have been associated with PCOS susceptibility in certain populations (Feng et al., 2015). Functional studies have shown that impaired insulin receptor activity contributes to defective glucose uptake and hyperinsulinemia, which in turn enhances ovarian androgen synthesis and exacerbates

reproductive dysfunction. INSR dysregulation has been observed in multiple tissues, including skeletal muscle and adipose tissue, but also in ovarian granulosa cells where insulin signaling plays a direct role in follicular development (Throwba et al., 2022).

Downstream of INSR, the phosphatidylinositol 3-kinase (PI3K) pathway serves as a central axis for insulin-mediated metabolic and mitogenic signaling. The gene PIK3CA, which encodes the catalytic subunit p110α of class IA PI3K, is crucial for this pathway. In the context of PCOS, altered PI3K/AKT signaling has been documented in ovarian granulosa cells, leading to impaired glucose metabolism, reduced estradiol production, and follicular arrest (Throwba et al., 2022). This suggests that PIK3CA, like INSR, may be involved in both the metabolic and reproductive aspects of PCOS pathogenesis.

Importantly, PIK3CA is also one of the most frequently mutated oncogenes in endometrial cancer, particularly in type I (endometrioid) tumors. Aberrant activation of PI3K/AKT/mTOR signaling through PIK3CA mutations enhances cellular proliferation and survival, creating a pro-tumorigenic environment in endometrial tissue (Johnson et al., 2023). Beyond mutation prevalence, functional studies and proteogenomic analyses have shown that PIK3CA mutations, particularly the canonical H1047R hotspot, drive enhanced phosphorylation of downstream effectors such as AKT (Ser473, Thr308) and mTOR (Ser2448), even in the absence of transcript overexpression (Madsen et al., 2024; Weigelt et al., 2013). This indicates that the oncogenic activity of PIK3CA in endometrial cancer is mediated primarily by gain-of-function activation of the PI3K/AKT/mTOR signaling cascade at the phosphoprotein level, rather than elevated transcription alone. Given that hyperinsulinemia—common in PCOS—can

potentiate PI3K pathway activity, the PIK3CA-INSR axis may serve as a molecular bridge between metabolic dysregulation in PCOS and endometrial carcinogenesis.

Epidemiological data support this mechanistic link. A recent meta-analysis found that women with PCOS have an approximately fourfold increased risk of developing endometrial cancer compared to those without PCOS, with the risk being particularly elevated in premenopausal women (Hopkins et al., 2018). This elevated risk is attributed to a combination of factors including chronic anovulation, prolonged unopposed estrogen exposure, and metabolic abnormalities such as insulin resistance. Studies have also shown that endometrial tissues from women with PCOS exhibit dysregulated insulin signaling and altered expression of PI3K/AKT components (Throwba et al., 2022).

Despite these associations, few studies have directly examined whether the same molecular disruptions observed in PCOS-related insulin signaling are reflected in endometrial cancer, particularly at the level of INSR and PIK3CA. This research seeks to fill that gap by analyzing data from The Cancer Genome Atlas (TCGA) to assess the expression, mutation, and co-alteration patterns of INSR and PIK3CA in endometrial tumors. By drawing connections between these molecular features and existing PCOS-related literature, we aim to clarify whether shared insulin signaling disruptions may underlie the increased endometrial cancer risk in women with PCOS.

Methodology

To investigate the potential molecular connection between insulin resistance in polycystic ovary syndrome (PCOS) and endometrial cancer, publicly available bioinformatics tools and

databases were utilized to analyze the genomic and transcriptomic profiles of two key insulin signaling genes: INSR and PIK3CA.

Genomic Localization and Alteration Frequency

The cytogenetic locations of INSR and PIK3CA were retrieved using the UCSC Genome Browser. Genomic alterations, including mutations, copy number variations (CNVs), and structural variants, were examined using cBioPortal across five comprehensive endometrial cancer cohorts: Endometrial Carcinoma (CPTAC, Cell 2020), Endometrial Cancer (MSK, Cancer Discovery 2023), Endometrial Carcinoma cfDNA (MSK, Clin Cancer Res 2022), Endometrial Carcinoma MSI (MSK, Clin Cancer Res 2022), Uterine Corpus Endometrial Carcinoma (TCGA, PanCancer Atlas).

Oncoprint summaries, lollipop plots, and frequency bar charts were used to visualize the spectrum and distribution of alterations. Comparative analyses were conducted between altered and unaltered groups to identify co-occurring genomic events, and Kaplan–Meier survival analyses were performed to assess clinical impact.

Transcript and Protein Expression Analyses.

Transcriptomic and proteomic expression levels of INSR and PIK3CA were analyzed using the UALCAN portal. Data from The Cancer Genome Atlas (TCGA) and CPTAC proteomics projects were used to evaluate gene expression across various clinical and demographic parameters, including cancer stage, histological subtype, race, weight, age, menopause status, and TP53 mutation status. In addition, protein expression patterns were examined based on pathway and complex activity status within tumor samples.

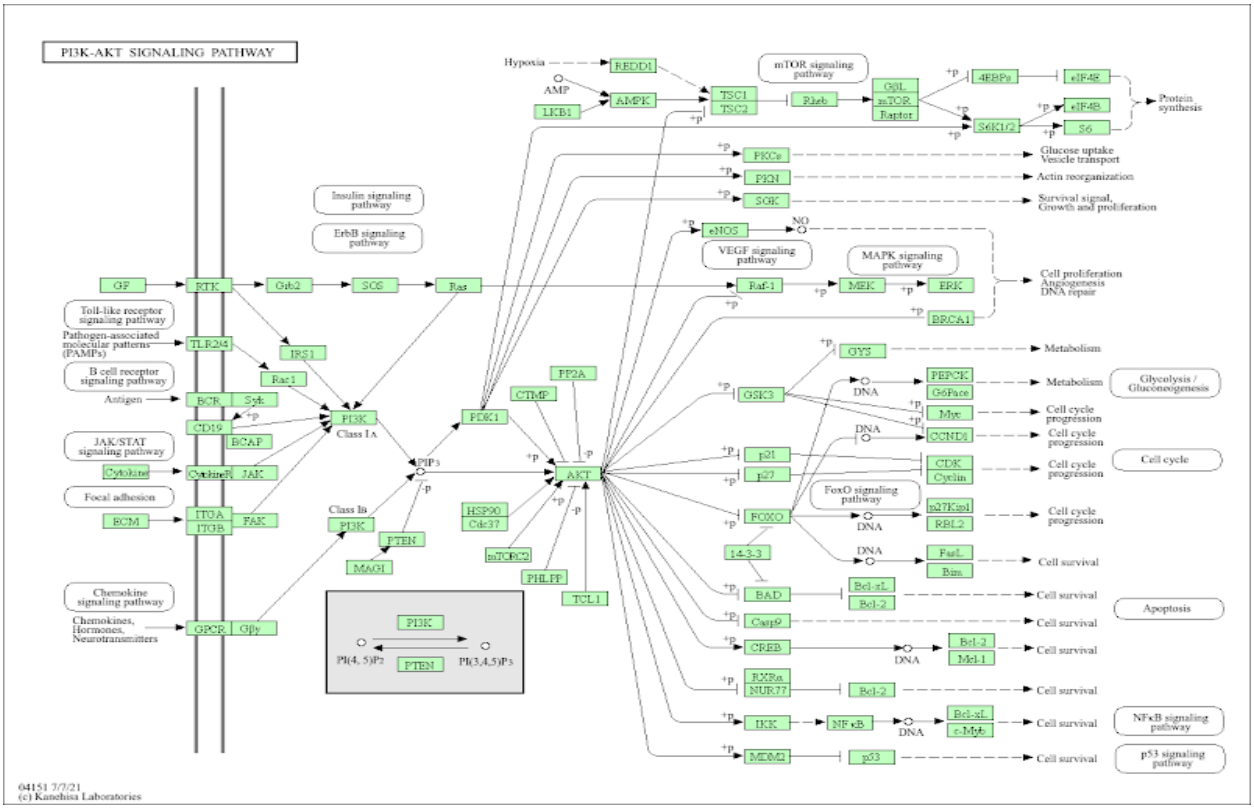
Protein Interaction and Pathway Analysis.

To explore the functional interaction between INSR and PIK3CA, STRING (v12.0) was used to generate a protein–protein interaction network, incorporating evidence from co-expression, co-occurrence, experimental validation, and text mining. MCL clustering (Markov Cluster Algorithm) with an inflation parameter of k = 3 was applied to identify functionally coherent subnetworks. Finally, the KEGG database was referenced to visualize the canonical PI3K-AKT signaling pathway (map04151), emphasizing the positions and roles of INSR and PIK3CA within insulin-mediated signaling cascades implicated in tumorigenesis.

The PI3K-AKT signaling pathway (Figure 1), retrieved from the KEGG database (map04151), highlights the central role of PIK3CA in mediating insulin signaling through IRS1. This pathway regulates essential cellular functions such as proliferation, metabolism, and survival—processes that are frequently dysregulated in endometrial cancer and associated with the insulin resistance characteristic of PCOS. In this cascade, insulin binds to its receptor (INSR), triggering phosphorylation of IRS1 and subsequent activation of PI3K, of which PIK3CA is the catalytic subunit. The downstream signaling through AKT affects critical effectors like mTOR, GSK3, and FOXO, which influence glucose uptake, cell cycle progression, and apoptosis inhibition. Because of their central role in insulin-related oncogenic signaling, any alteration in PIK3CA and INSR expression could significantly impact both PCOS pathogenesis and endometrial tumorigenesis. Therefore, based on this mechanistic framework, I chose to compare the expression levels of PIK3CA and INSR in tumor versus normal samples as a foundational step in assessing their relevance to cancer progression in the context of PCOS.

Figure 1

PI3K–AKT Signaling Pathway (KEGG map04151)



Note. This diagram illustrates how PIK3CA is activated via insulin signaling (IRS1) and regulates downstream pathways involved in cell survival, proliferation, and metabolism, key processes implicated in endometrial carcinogenesis associated with PCOS-related insulin resistance.

Results

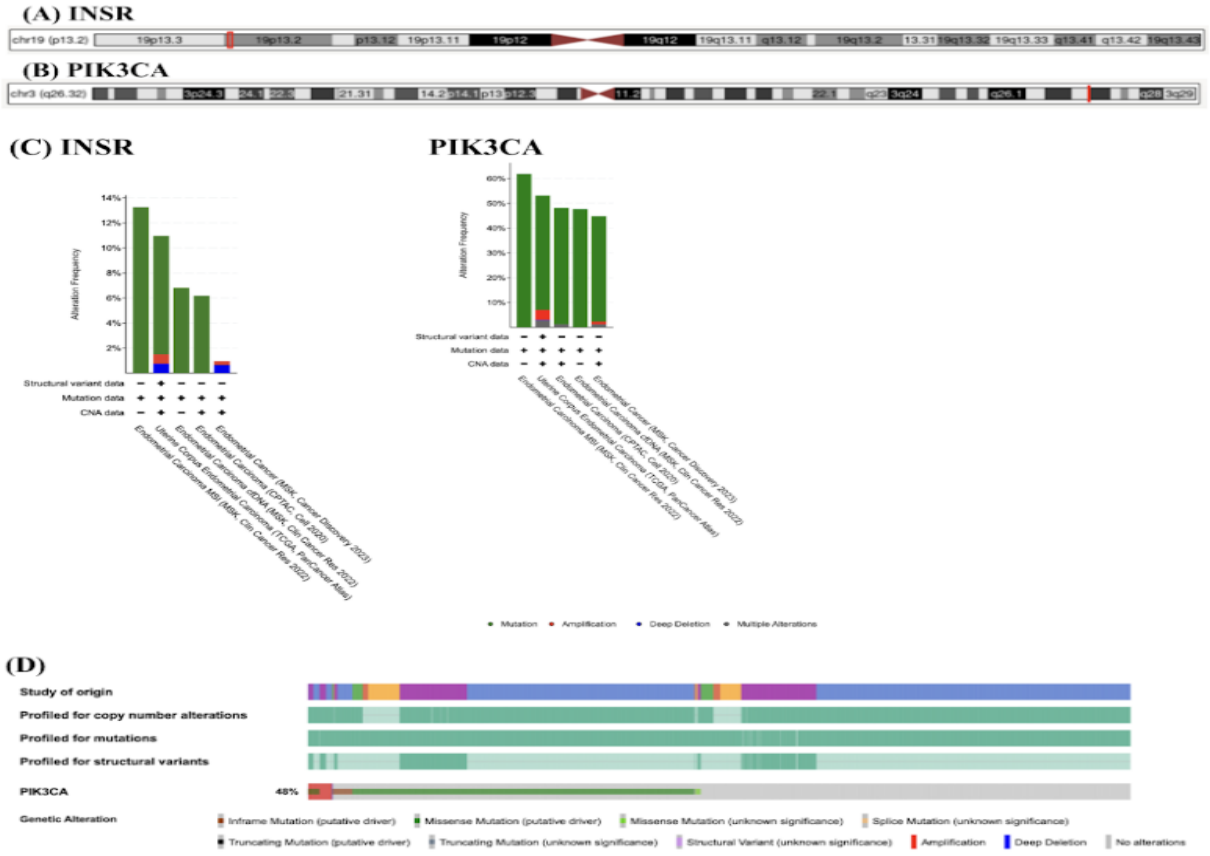
To examine whether disruptions in insulin signaling observed in PCOS are reflected in endometrial cancer, we first evaluated the genomic characteristics of INSR and PIK3CA across

multiple large-scale datasets. By mapping their chromosomal positions and comparing alteration frequencies among independent cohorts, we aimed to determine whether these genes exhibit consistent patterns of mutation or copy number change in endometrial tumors.

The INSR gene is located on chromosome 19p13.2, as shown in the cytogenetic banding pattern (Figure 2A). Analysis of five independent endometrial carcinoma datasets revealed that INSR is subject to genomic alterations in a subset of tumors, with alteration frequencies ranging from approximately 6% to 14% depending on the cohort (Figure 2C). The most frequent alterations included copy number amplifications, with additional contributions from mutations and structural variants. Notably, the MSK Cancer Discovery 2023 dataset showed the highest frequency of INSR alterations. PIK3CA is located on chromosome 3 at the q26.32 cytoband, as indicated by its chromosomal position (Figure 2B). This region has been associated with multiple cancers, suggesting its significance in genomic instability. In endometrial carcinoma, PIK3CA displays a high alteration frequency across datasets, with 48% of cases showing some form of genetic alteration (Figure 2C). These alterations include putative driver missense mutations, inframe mutations, and occasional amplifications. Moreover, PIK3CA exhibits a consistently high alteration frequency in endometrial cancer samples, ranging from approximately 45% to over 60% (Figure 2D). Mutations represent the predominant alteration type, with amplification and deep deletion observed less frequently. The variation in frequency can be attributed to differences in data availability for structural variants, copy number alterations, and mutations among the datasets. This high prevalence of PIK3CA mutations across diverse cohorts shows its potential role as a key oncogenic driver in endometrial tumorigenesis.

Figure 2

Genomic Alterations of INSR and PIK3CA Across Endometrial Cancer Datasets



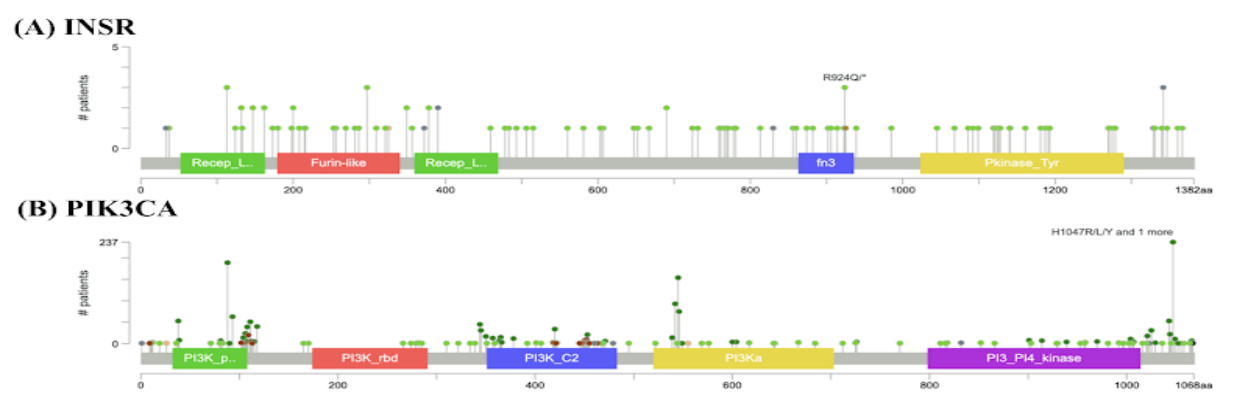
Note. (A) Cytogenetic location of the INSR gene on chromosome 19p13.2 (UCSC Genome Browser). (B) Cytogenetic location of the PIK3CA gene on chromosome 3q26.32 (UCSC Genome Browser). (C) Alteration frequencies of INSR (left) and PIK3CA (right) across five endometrial carcinoma cohorts from cBioPortal. Colors indicate copy number alterations (green), mutations (red), and structural variants (blue). (D) Frequency of PIK3CA alterations across cohorts, including missense, inframe, truncating, and structural variant mutations.

Mapping of INSR mutations across its protein structure revealed a dispersed distribution spanning multiple functional domains, including the Furin-like region, the fibronectin type III (fn3) domains, and the intracellular tyrosine kinase domain (Figure 3A). A recurrent alteration, R924Q/*, was identified in the fn3 domain rather than the kinase domain, highlighting the importance of this extracellular structural region in receptor activation and signaling. The presence of both missense and truncating mutations across these domains supports the idea that INSR dysfunction may contribute to impaired insulin signaling in endometrial cancer and help explain the reduced expression of INSR observed in tumor samples.

In contrast, PIK3CA exhibited a markedly higher frequency of mutations, with clear clustering within kinase-associated domains (Figure 3B). The most prominent hotspot, H1047R, located in the PI3_PI4_kinase domain, is a well-characterized activating mutation known to enhance PI3K pathway signaling. The concentration of alterations in these catalytic regions suggests a strong gain-of-function effect that likely drives oncogenic PI3K activity in endometrial cancer.

Figure 3

Mutation Profiles of INSR and PIK3CA in Endometrial Cancer

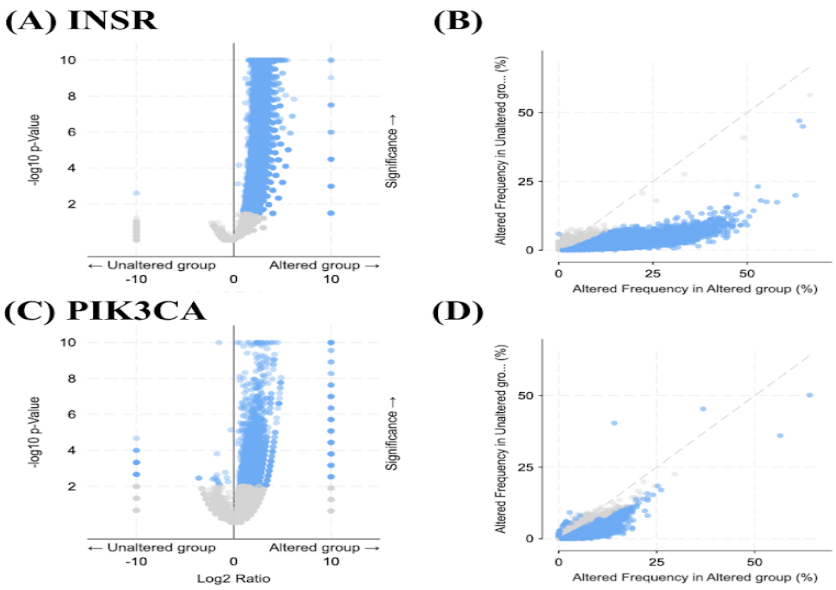


Note. (A) Lollipop plot of INSR mutations distributed across the Furin-like, fn3, and tyrosine kinase domains. (B) Lollipop plot of PIK3CA mutations highlighting the H1047R hotspot in the PI3-PI4 kinase domain.

To investigate the genomic context associated with these alterations, a comparative analysis was conducted between cases harboring alterations and those without. A volcano plot revealed numerous genes significantly enriched in the altered group, with PIK3CA showing one of the highest log2 ratios and statistical significance (Figure 4A). A scatterplot of gene-level alteration frequencies further highlighted the differences between the two groups (Figure 4B). Most genes displayed a trend of increased alteration frequency in the altered group compared to the unaltered group.

Figure 4

Differential Gene Alterations in INSR-Altered and PIK3CA-Altered Endometrial Tumors



Note. (A) Volcano plot showing genes with significantly different alteration frequencies between INSR-altered and unaltered cases. (B) Scatterplot comparing gene-level alteration frequencies between INSR-altered and unaltered tumors. (C) Volcano plot showing genes enriched in PIK3CA-altered cases. (D) Scatterplot comparing gene alteration frequencies between PIK3CA-altered and unaltered groups.

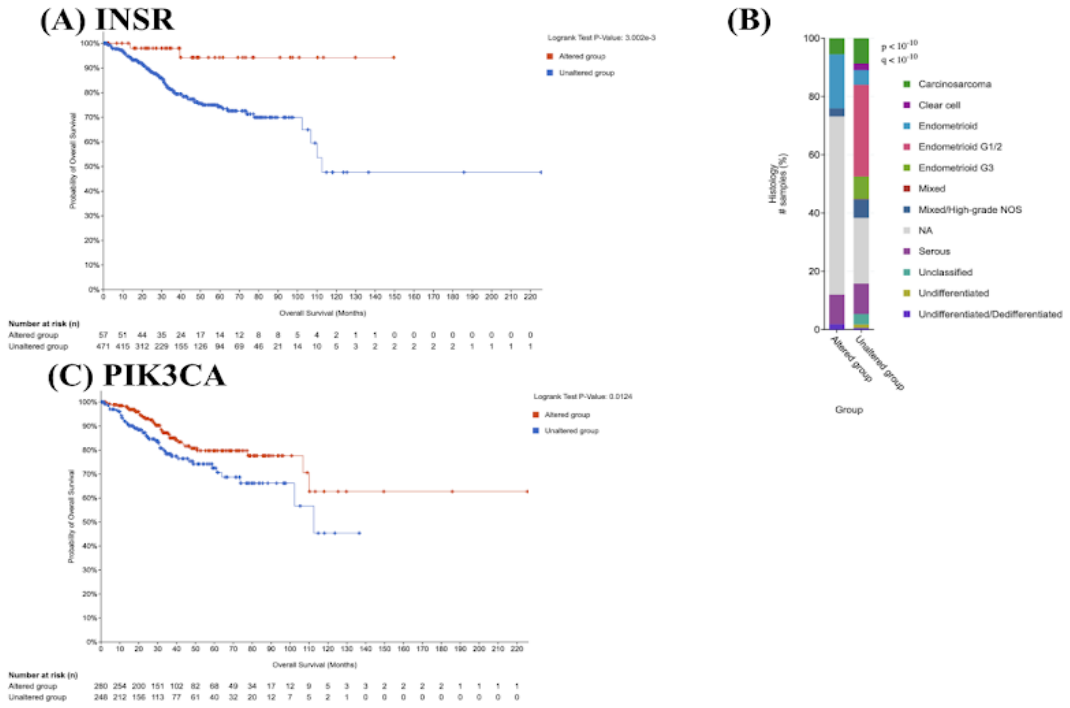
The volcano plot (Figure 4C) demonstrates a substantial number of genes upregulated in PIK3CA-altered endometrial cancer cases, many of which are statistically significant. This indicates that PIK3CA mutations are associated with broad transcriptional changes, potentially reflecting activation of downstream oncogenic signaling cascades. In parallel, the frequency comparison plot (Figure 4D) shows that alterations in other genes occur more frequently in the PIK3CA-altered group than in unaltered cases, suggesting a co-alteration landscape that may further amplify tumor progression. These findings support the idea that PIK3CA mutations act as potent oncogenic drivers in endometrial cancer, beyond being a recipient of upstream signals.

Kaplan–Meier survival analysis demonstrated a significant difference in overall survival between INSR-altered and unaltered endometrial cancer patients (log-rank p = 3.002e-3). Patients in the altered group exhibited notably higher survival probabilities throughout the observed period, with survival remaining above 90% well beyond 100 months, while the unaltered group showed a steady decline over time, reaching approximately 50% survival by 130 months (Figure 5A). This survival advantage in the altered group may be linked to distinct histological compositions. In the histology distribution, the altered group was predominantly composed of low-grade endometrioid (G1/2) tumors, which are generally associated with favorable clinical outcomes. In contrast, the unaltered group encompassed a broader and more aggressive histologic spectrum, including serous, carcinosarcoma, and undifferentiated subtypes, all of which are typically correlated with poorer prognosis (Figure 5B). These findings suggest that INSR alterations may be enriched in less aggressive tumor phenotypes, potentially contributing to improved patient outcomes.

In the Kaplan–Meier survival analysis (Figure 5C), patients with PIK3CA alterations exhibit significantly improved overall survival compared to those without such alterations. While PIK3CA is a well-known oncogenic driver, this result may appear counterintuitive. However, it aligns with previous reports suggesting that tumors harboring PIK3CA mutations often have less aggressive clinical behavior or may be more responsive to certain targeted therapies.

Figure 5

Survival Outcomes and Histology Distribution in INSR- and PIK3CA-Altered Endometrial Cancer

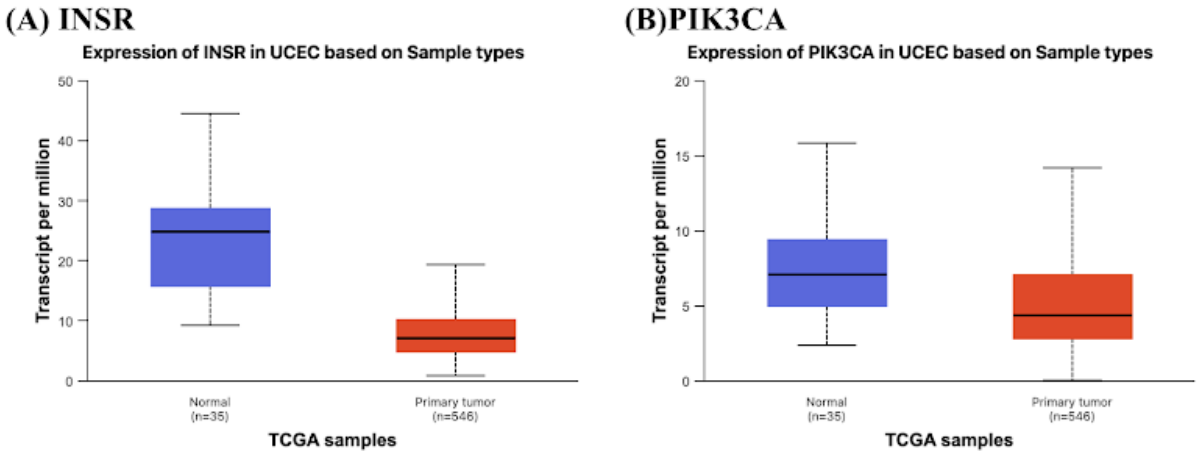


Note. (A) Kaplan–Meier overall survival curves for INSR-altered vs. unaltered patients (log-rank p = 3.002e-3). (B) Distribution of tumor histologic subtypes in altered vs. unaltered INSR cases. (C) Kaplan–Meier overall survival curves for PIK3CA-altered vs. unaltered patients (log-rank p = 0.0124).

INSR transcript expression was significantly reduced in primary tumor samples compared to normal tissue in the UCEC cohort (Figure 6A). Normal tissues had substantially higher median transcript levels, indicating that INSR is transcriptionally downregulated in tumorigenic states. This suppressed expression is likely a consequence of the observed genomic alterations in Figure 8, further supporting the notion that INSR plays a tumor-suppressive role in endometrial cancer.

Figure 6

Transcript Expression of INSR and PIK3CA in Normal and Tumor Endometrial Tissue



Note. (A) Boxplot comparing INSR transcript expression between normal and tumor samples in the TCGA-UCEC cohort. (B) Boxplot comparing PIK3CA transcript expression between normal and tumor samples. TPM = transcripts per million.

PIK3CA expression was found to be lower in tumor samples compared to normal tissues (Figure 6B). At first glance, this reduction in transcript abundance could suggest limited oncogenic impact. However, phosphoproteomic analyses from CPTAC datasets demonstrate that tumors harboring PIK3CA mutations display elevated phosphorylation of key downstream effectors, including AKT (pS473, pT308) and mTOR (pS2448), even in the absence of transcript overexpression. These data confirm that the oncogenic activity of PIK3CA in endometrial cancer arises primarily through gain-of-function mutations that enhance PI3K/AKT pathway signaling, rather than increased transcript or protein abundance.

Discussion

Our study suggests that shared insulin signaling abnormalities may provide a molecular explanation for the elevated cancer risk in women with PCOS. INSR expression was significantly reduced in tumors, consistent with impaired insulin signaling observed in PCOS. Genomic alterations in INSR were relatively infrequent but enriched in low-grade endometrioid tumors, which may explain the better survival seen in altered cases. These findings suggest that INSR loss may contribute to tumor initiation rather than aggressive progression.

By contrast, PIK3CA was one of the most frequently altered genes, with hotspot mutations such as H1047R driving constitutive PI3K/AKT activation. Although associated with oncogenic signaling, PIK3CA mutations correlated with improved survival, possibly reflecting less aggressive tumor behavior or therapeutic responsiveness. Importantly, reduced transcript expression alongside increased downstream phosphorylation highlights that oncogenic activity arises from gain-of-function mutations rather than transcriptional overexpression.

Taken together, these results suggest that the INSR–PIK3CA axis links PCOS-related insulin resistance with endometrial carcinogenesis. In PCOS, defective INSR signaling and compensatory hyperinsulinemia potentiate PI3K activity, while in cancer, PIK3CA mutations bypass upstream defects to sustain pathway activation.

Our findings underscore the potential of targeting the INSR–PIK3CA axis as both a preventive and therapeutic strategy. In the context of PCOS, interventions that improve insulin sensitivity (such as metformin or lifestyle modification) may reduce not only reproductive and metabolic complications but also long-term cancer risk. In endometrial cancer, where PIK3CA

mutations are highly prevalent, PI3K/AKT/mTOR inhibitors remain an attractive therapeutic avenue, particularly in patients whose tumors exhibit pathway hyperactivation. The observed survival advantage in PIK3CA-mutant cases may partially reflect responsiveness to such therapies, although this hypothesis requires direct clinical validation.

Conclusion

Several limitations should be noted. First, because the analyses relied on retrospective datasets from TCGA and cBioPortal, causal conclusions could not be drawn, and experimental validation was outside the scope of this study. Second, relying mainly on transcriptomic data likely underestimates the impact of PIK3CA alterations, since their oncogenic effects are driven more by protein phosphorylation than by changes in mRNA levels. Third, although we focused on INSR and PIK3CA, insulin signaling is a complex network that also involves IRS1, AKT isoforms, PTEN, and other regulators that may influence the PCOS–cancer connection.

Future studies should use integrative multi-omics approaches and experimental models based on PCOS-derived endometrial tissue to clarify how these disruptions are associated. It will also be important to examine racial and metabolic subgroups, as differences in PCOS prevalence and endometrial cancer outcomes suggest population-specific risks.

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