Integrated Bioinformatics and Experimental Analysis of KIFs in Ovarian Cancer Reveals Mitotic Drivers and Germline Variant Associations

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Abstract

Background: Ovarian cancer is the deadliest gynecologic malignancy, yet its genomic drivers remain incompletely understood. Methods: We integrated multi-omics data from TCGA-OV (n = 379), GTEx normals (n = 88), and two GEO cohorts (GSE26712, GSE18520) to analyze differential expression, CNV, pathway enrichment, and protein protein interactions. Germline variants were assessed through eQTL mapping and functional annotation. KIF17 expression and rs13375609 genotypes were experimentally validated using qRT-PCR and T-ARMS PCR in an independent cohort (12 tumors, 10 normals). Results: We identified 3,200 dysregulated genes, with mitotic kinesins (KIF11, KIF17, KIF18A, KIF20A) markedly upregulated and frequently amplified. High KIF11 or KIF14 expression correlated with reduced overall survival. GSEA indicated strong enrichment of mitotic spindle and

G2/M checkpoint pathways, while PPI analysis identified KIF11 and KIF17 as central mitotic hubs. Two KIF17 variants (rs2297299, rs13375609) showed significant eQTL effects. Experimental validation confirmed elevated KIF17 expression and higher frequency of the rs13375609 A allele in ovarian cancer (27% vs. 11%; p = 0.012; OR = 2.8), with the TA genotype showing an even stronger association (p = 0.004; OR = 4.1). Conclusion: Multi-omics integration and experimental validation identify KIF11, KIF14, and KIF17 as key mitotic drivers and potential biomarkers in ovarian cancer, with rs13375609 emerging as a promising susceptibility variant.

Author keywords

bioinformatics; eQTL; genotyping; GSEA; KIF17; kinesin family proteins; Ovarian cancer

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Abstract

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