Investigation of FOXM1 Gene Expression in Breast Cancer and Its Relationship with Mir-216B-5P Expression Level

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Abstract : As a pressing public health concern, breast cancer stands as the predominant oncological diagnosis and principal cause of cancer-related mortality among women globally, accounting for 11.7% of new cancer incidences and 6.9% of cancerrelated deaths. The annual figures indicate that approximately 230,480 women are diagnosed with breast cancer in the United States alone, with 39,520 succumbing to the disease. While developed economies have reported a deceleration in both incidence and mortality rates across various forms of cancer, including breast cancer, emerging and low-income economies manifest a contrary escalation, largely attributable to lifestyle-mediated risk factors such as tobacco usage, physical inactivity, and high caloric intake. Breast cancer is distinctly characterized by molecular heterogeneity, manifesting in specific subtypes delineated by biomarkers-Estrogen Receptors (ER), Progesterone Receptors (PR), and Human Epidermal Growth Factor Receptor 2 (HER2). These subtypes, comprising Luminal A, Luminal B, HER2-enriched, triple-negative/basal-like, and normallike, necessitate nuanced, subtype-specific therapeutic regimens, thereby challenging the applicability of generalized treatment protocols. Within this molecular complexity, the transcription factor Forkhead Box M1 (FoxM1) has garnered attention as a significant driver of cellular proliferation, tumorigenesis, metastatic progression, and treatment resistance in a spectrum of human malignancies, including breast cancer. Concurrently, microRNAs (miRs), specifically miR-216b-5p, have been identified as post-transcriptional gene expression regulators and potential tumor suppressors. The overarching objective of this academic investigation is to explicate the multifaceted interrelationship between FoxM1 and miR-216b-5p across the disparate molecular subtypes of breast cancer. Employing a methodologically rigorous, interdisciplinary research design that incorporates cuttingedge molecular biology techniques, sophisticated bioinformatics analytics, and exhaustive meta-analyses of extant clinical data, this scholarly endeavor aims to unveil novel biomarker-specific therapeutic pathways. By doing so, this research is positioned to make a seminal contribution to the advancement of personalized, efficacious, and minimally toxic treatment paradigms, thus profoundly impacting the global efforts to ameliorate the burden of breast cancer.

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