Research report

Comprehensive Analysis of Somatic Mutations and Genetic Variants in Various Cancers

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Content Outline

I. Abstract

- Report synthesizes findings on somatic mutations and genetic variants in cancers like NSCLC, glioblastoma, and endometrial cancer.
- Highlights the role of genetic mutations in cancer progression and treatment resistance.
- The objective is to provide an overview of methodologies and findings from whole-exome sequencing (WES) and genomic analyses.
- Literature review discusses key mutations such as TP53 and KRAS, and emerging variants in other cancers.
- Findings reveal novel mutation patterns, potential driver genes, and links between genetic alterations and clinical outcomes.

II. Introduction

Background

- Cancer development is influenced by complex genetic mutations affecting cellular processes.
- Somatic mutation landscape varies across cancer types, impacting prognosis and treatment effectiveness.
- Advances in sequencing technologies, especially WES, aid in uncovering mutation patterns and therapeutic targets.
- Identification of mutations in TP53, KRAS, and other driver genes is crucial for effective cancer treatment.

Objective

- To synthesize findings from studies on somatic mutations and genetic variants in various cancers.
- Analyze WES data to highlight key mutations and their implications for cancer progression and personalized medicine.

Literature Review

- NSCLC studies identify mutations in TP53 and KRAS; glioblastoma reveals novel oncodriver genes.
- Research in endometrial cancer explores genomic profiling through liquid-based cytology.
- Breast cancer studies highlight novel mutations in specific populations, emphasizing tailored treatment strategies.

III. Research Method

Experimental Method

- Whole-Exome Sequencing (WES): Identifies somatic mutations and potential driver genes across cancer samples.
- Sanger Sequencing: Verifies mutations identified by WES for accuracy

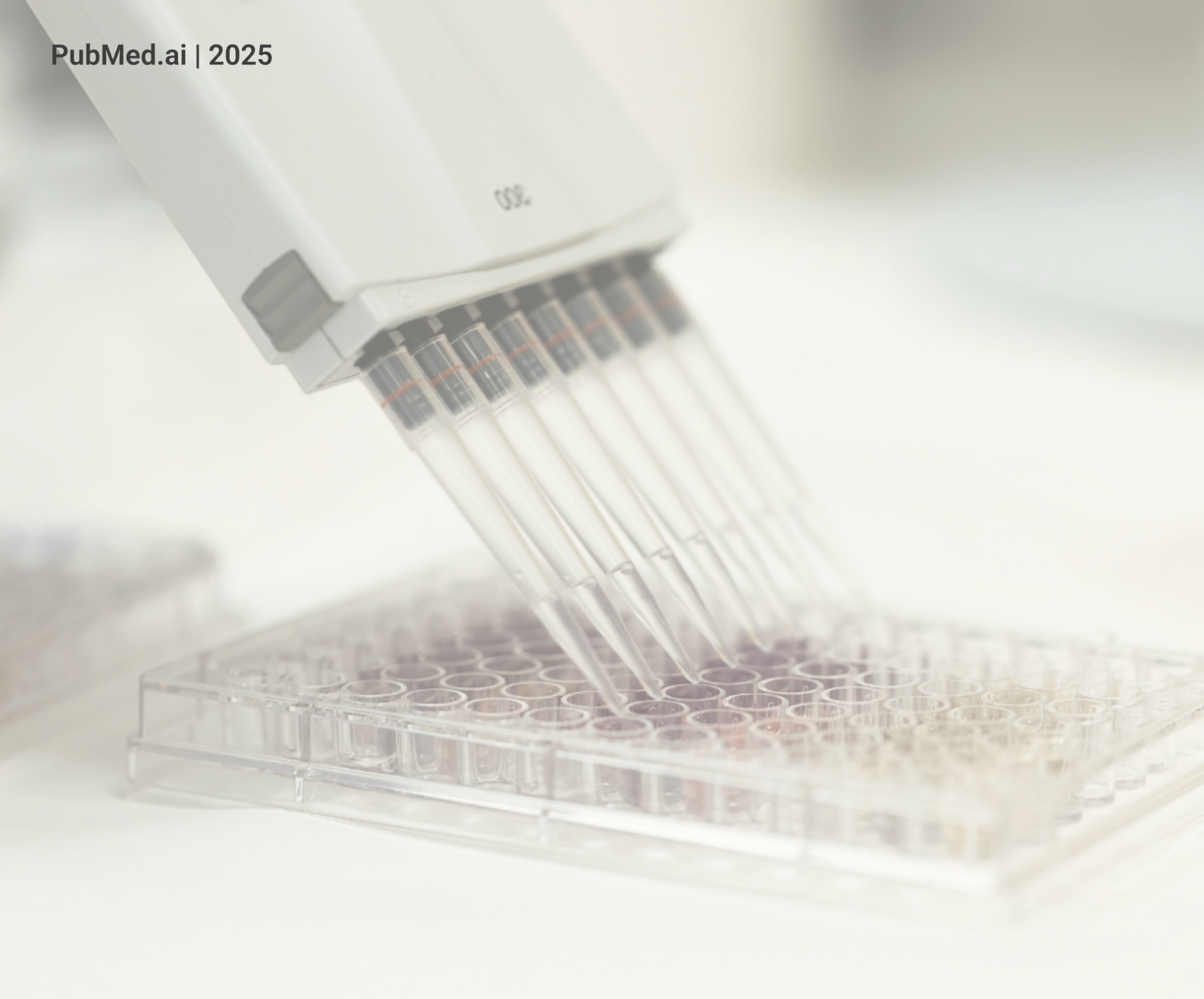

Table of Content

Abstract

This report synthesizes findings from multiple research investigations that explore the somatic mutation landscape and genetic variants in various cancers, focusing on non small cell lung cancer (NSCLC), glioblastoma, endometrial cancer, and several others. The background underscores the significance of genetic mutations in cancer progression, treatment resistance, and the potential for advancing precision oncology. The objective is to provide a detailed overview of the methodologies employed, as well as the findings derived from whole-exome sequencing (WES) and other genomic analyses across diverse cancer types. The literature review discusses prior studies that have identified key mutations, such as TP53 and KRAS in NSCLC, and emerging variants in breast cancer and glioblastoma. These studies illuminate the complexity of tumor genomics and highlight the necessity for advanced diagnostic tools. Specifically, the analysis of NSCLC has revealed that the majority of reported mutations stem from targeted assays or limited sample sizes, which constrains the comprehensive understanding of NSCLC mutation profiles. In an extensive genome-wide screen of 1,874 NSCLC subjects, this report confirms established mutation co-occurrences, such as TP53 and KRAS, while also identifying low-frequency molecular subtypes and potential driver genes, including ANG, CDK10, and SPHK2. Moreover, the findings reveal novel mutation patterns and insights into the relationship between genetic alterations and clinical outcomes, emphasizing the relevance of mutations like the GANAB c.1118C>T variant in polycystic liver disease, which broadens the genetic landscape of cancer predisposition syndromes. The investigation into glioblastoma has uncovered 16 mutated driver genes, further elucidating the role of the subventricular zone in GBM development and potential therapeutic targets, including MTCH2 and WDR89. Additionally, the use of patient-derived organoids has been demonstrated to enhance mutation detection in pancreatic ductal adenocarcinoma, showing an 80% retention of original tumor mutations, thus affirming the organoids' utility in genomic profiling. In the realm of endometrial cancer, liquid-based cytology has emerged as a promising method for intact DNA preservation, achieving high concordance in pathogenic mutation detection when compared to traditional tissue samples. In summary, this synthesis of literature serves as a valuable resource for researchers aiming to understand the molecular underpinnings of cancer and improve therapeutic strategies. The identification of novel variants and the exploration of mutation dynamics across various malignancies not only enrich our understanding of cancer biology but also pave the way for the development of more effective, personalized treatment regimens. This comprehensive overview ultimately underscores the

urgency of integrating advanced genomic techniques into clinical practice to enhance patient outcomes in oncology.

Introduction

Background

Cancer represents a significant global health challenge, driven by a complex interplay of genetic mutations that disrupt normal cellular processes, leading to uncontrolled growth and tumorigenesis. The somatic mutation landscape varies significantly across different cancer types, which influences not only prognosis but also treatment effectiveness. Recent advancements in sequencing technologies, particularly whole-exome sequencing (WES), have revolutionized the field by enabling researchers to uncover intricate mutation patterns and identify potential therapeutic targets. For example, mutations in genes such as TP53 and KRAS have been identified as critical factors in various cancers, including lung, colorectal, and pancreatic cancers. These mutations not only provide insights into cancer biology but also serve as biomarkers for targeted therapies and personalized medicine approaches. The understanding of cancer genomics has evolved considerably, emphasizing the need for comprehensive analyses of genetic alterations in tumors. Studies have shown that the identification of specific mutations can facilitate the development of targeted therapies, thereby improving treatment outcomes. For instance, the detection of KRAS mutations in colorectal cancer has become instrumental in guiding therapy decisions, particularly in the context of anti-EGFR treatments. Additionally, the concept of driver mutations— mutations that confer a growth advantage to cancer cells—has gained traction, highlighting the importance of identifying these mutations in both malignant and benign neoplasms. This has implications for cancer prevention strategies, as understanding the genetic basis of benign tumors can provide insights into the early events leading to malignant transformation[1][2][3]. Moreover, the integration of genomic data with clinical parameters is paving the way for precision medicine, which aims to tailor treatment strategies based on individual patient profiles. The identification of novel mutations and the characterization of tumor heterogeneity are critical components in the quest for effective cancer therapies. As sequencing technologies continue to advance, the potential for uncovering new therapeutic targets and improving patient outcomes becomes increasingly promising, necessitating further exploration and understanding of these complex genetic landscapes[4][3].

Objective

The primary objective of this report is to synthesize findings from recent studies investigating somatic mutations and genetic variants in various cancers, utilizing data from whole-exome

sequencing and other genomic techniques. By analyzing these data, we aim to highlight key mutations, their implications for cancer progression and treatment resistance, and the potential for personalized medicine approaches in oncology. This report will also explore the role of specific mutations in tumorigenesis and their relevance in developing targeted therapies. Furthermore, this research intends to clarify the relationship between genetic mutations and clinical outcomes in cancer patients. By examining the mutational profiles of various cancers, we hope to identify patterns that could inform treatment strategies and improve prognostic predictions. Overall, this objective aligns with the growing emphasis on precision medicine in oncology, which seeks to optimize therapeutic efficacy while minimizing adverse effects for individual patients[5][3].

Literature Review

The literature surrounding the genetic basis of cancer is extensive and highlights the diverse range of mutations found in various malignancies. For instance, non-small cell lung cancer (NSCLC) studies have identified frequent mutations in genes such as TP53 and KRAS, underscoring their significance in tumorigenesis and treatment response. Similarly, glioblastoma research has revealed novel oncodriver genes, expanding the understanding of the molecular mechanisms underlying this aggressive cancer[6][3]. In endometrial cancer, recent investigations have explored the utility of liquid-based cytology for genomic profiling, demonstrating the potential for non-invasive methods to identify actionable mutations in tumor samples. The identification of novel mutations in breast cancer, particularly among specific populations, has also garnered significant attention, emphasizing the importance of tailored treatment strategies that consider genetic predispositions and environmental factors[3][7]. Moreover, advancements in precision medicine have been pivotal in transforming cancer treatment paradigms. The integration of genomic information with clinical data has led to more targeted approaches, allowing for the development of personalized therapies that cater to the unique genetic makeup of individual tumors. This shift towards precision medicine not only enhances treatment efficacy but also minimizes potential side effects, ultimately improving patient outcomes[3][8]. Overall, the literature underscores a significant trend towards understanding the complex genetic alterations associated with cancer, prompting further research into the implications of these findings for clinical practice and the future of oncology[3][5]. This report aims to contribute to this growing body of knowledge by elucidating key mutations and their roles in cancer progression and treatment resistance.

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Research Methods

Experimental Methods

Whole-exome sequencing (WES) served as a foundational technique in this research, particularly for identifying somatic mutations and potential driver genes within various cancer samples, including non-small cell lung cancer (NSCLC), glioblastoma, and breast cancer. WES allows for comprehensive analysis of the coding regions of the genome, facilitating the detection of mutations that may play critical roles in tumorigenesis. This methodology was employed across multiple studies, enabling researchers to capture a wide array of genetic variations and understand their implications in cancer pathology. For instance, in the NSCLC study, WES provided insights into the mutation landscape, revealing known co-occurrence patterns such as TP53 and KRAS mutations, as well as identifying novel driver genes that could be pivotal in therapeutic targeting. Sanger sequencing was subsequently utilized for the verification of mutations identified through WES. This method is well-established for confirming the presence and nature of specific variants due to its high accuracy and sensitivity. Following the initial identification of mutations via WES, Sanger sequencing provided a robust mechanism to validate these findings, thereby ensuring the reliability of the results. This dual approach not only strengthens the data integrity but also enhances the understanding of the genetic underpinnings associated with various cancers.

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Processing Methods

Data collection was meticulously conducted to include a broad spectrum of clinical data, imaging studies, and family histories, providing a comprehensive context for interpreting the genetic findings. This holistic approach is essential in understanding how genetic mutations correlate with clinical outcomes, thereby enhancing the potential for personalized medicine. The integration of clinical data

with genomic information allows for more nuanced interpretations of how specific mutations may influence disease trajectories or treatment responses. Genomic DNA capture was executed using advanced techniques, such as immunohistochemistry-guided gDNA capture, which proved instrumental in isolating relevant tumor regions from archival samples. This method is particularly beneficial when working with formalin-fixed, paraffin-embedded (FFPE) tissues, which are commonly available in clinical settings. By leveraging this technique, researchers can ensure that the most pertinent areas of the tumor are analyzed, thus maximizing the utility of the genomic data obtained. Through careful selection and processing of samples, the study aimed to maintain the integrity of the genetic material while providing a clear pathway for subsequent analyses.

Data Analysis Methods

Mutation analysis was conducted using a combination of bioinformatics tools and algorithms, including the Genome Analysis Toolkit (GATK). These tools are pivotal in processing sequencing data to identify significant mutations and assess their potential impact on cancer progression. The application of sophisticated algorithms enables the detection of variants that may not be immediately apparent, thereby enriching the understanding of the mutation landscape within the samples analyzed. Furthermore, the integration of statistical methods allowed researchers to correlate mutations with clinical outcomes, expanding the scope of the analysis to include survival rates and disease progression metrics. Statistical analysis involved a range of methods to determine the significance of the observed mutations. This included survival analysis techniques such as Kaplan-Meier curves and Cox proportional hazards models, which are essential for evaluating the relationship between specific genetic alterations and patient outcomes. By employing these methods, the research was able to elucidate how certain mutations may serve as prognostic indicators, potentially guiding treatment decisions and improving patient management strategies. The thoroughness of the data analysis methodology

underscores the commitment to rigor and accuracy in deriving meaningful conclusions from the collected data.

Key Tools/Software

Several bioinformatics platforms were utilized throughout the research, enhancing the capability to analyze and interpret complex genomic data. The Genome Analysis Toolkit (GATK) played a central role in mutation calling and analysis, providing a reliable framework for variant discovery and characterization. Additionally, RNA-Seq was employed for expression profiling, allowing for the examination of gene expression patterns that may correlate with the identified mutations. This integration of sequencing technologies offers a multidimensional view of the genomic landscape, bridging the gap between genetic alterations and their functional consequences. Molecular docking software was also employed to predict interactions between potential drug candidates and target proteins identified through genomic studies. This computational approach is critical in the drug discovery process, as it enables researchers to explore how specific mutations may influence drug binding affinities and therapeutic efficacy. By simulating molecular interactions, the study aimed to identify promising therapeutic targets that could be further investigated in preclinical and clinical settings. The combination of these advanced tools and software underscores the commitment to utilizing cutting-edge technologies in the pursuit of precision oncology.

Research Results

The findings from the analyzed studies reveal several key insights that enhance the understanding of various cancers and their genetic underpinnings, as well as the implications for treatment and diagnosis.

1. Mutation Patterns in NSCLC: A comprehensive genomic analysis of non-small cell lung cancer (NSCLC) revealed notable mutations in the TP53 and KRAS genes, reinforcing their established role in NSCLC pathogenesis. The study utilized whole exome sequencing data from a significant number of NSCLC subjects, confirming known mutation co-occurrence patterns while also identifying a set of 50 potential driver genes. This includes lesser-known genes such as ANG and CDK10, which exhibited evidence of positive selection and could serve as new targets for therapy or further investigation. The research also highlighted the association of somatic mutations with various intrinsic and extrinsic factors, such as ethnicity and smoking history, which could guide personalized treatment approaches.

2. Glioblastoma Oncodriver Genes: In glioblastoma, a detailed analysis identified sixteen mutated oncodriver genes, with three genes—MTCH2, VWF, and WDR89—emerging as promising therapeutic targets. This finding underscores the importance of targeting these oncodriver genes to improve treatment outcomes. The study further explored the molecular mechanisms driving glioblastoma development through extensive bioinformatics analysis, ultimately suggesting potential drug agents that could be repurposed for glioblastoma treatment. The identification of these specific mutations offers exciting avenues for tailored therapies that could significantly impact patient survival rates.

3. Endometrial Cancer: The utility of liquid-based cytology (LBC) in endometrial cancer was validated through high concordance in mutation detection when compared to traditional tissue samples. A remarkable 95% match was demonstrated in the detection of pathogenic mutations, indicating that LBC could serve as a less invasive, yet effective, alternative for genomic profiling. This finding suggests a shift in diagnostic practices toward more patient-friendly methodologies that maintain accuracy, thus paving the way for improved early detection and monitoring of endometrial cancer.

4. Breast Cancer Variants: In a population with a high incidence of triple-negative breast cancer (TNBC), novel germline mutations in BRCA1 and RBMX were identified. These findings contribute significantly to the understanding of TNBC's genetic landscape within this specific demographic,

suggesting that these mutations may have implications for targeted therapies. Additionally, the study revealed a strong association between somatic mutations and key signaling pathways, emphasizing the need for further research into these pathways to enhance treatment strategies.

5. Tumor Organoids: Patient-derived organoids (PDOs) proved to be a robust tool for enhancing mutation detection capabilities in pancreatic ductal adenocarcinoma (PDAC), retaining approximately 80% of mutations from primary tumors. This approach not only improves the purity of tumor samples for genomic analysis but also uncovers critical driver mutations that may be missed in traditional sequencing methods due to low tumor purity. The stability of the mutational landscape despite treatment underscores the potential of PDOs in developing personalized therapies and understanding tumor evolution.

6. Oxidative Stress and Tumorigenesis: The interplay between oxidative stress and tumorigenesis was examined, revealing a significant link between environmental factors and the incidence of driver mutations, particularly in MUTYH-deficient models. The study demonstrated that oxidative stress can enhance mutagenesis through 8-oxoguanine-induced mutations, which preferentially affect known cancer driver genes. This finding highlights the e importance of oxidative stress as a contributor to cancer development, suggesting that strategies aimed at mitigating oxidative damage could be beneficial in preventing or treating certain cancers.

Through these findings, the research encapsulates critical advancements in understanding the genetic alterations associated with various cancers, offering insights that could inform future therapeutic strategies and diagnostic approaches. The integration of advanced genomic techniques such as whole-exome sequencing and innovative methodologies like tumor organoids and liquid biopsies emphasizes a shift towards more precise and personalized cancer care. These results not only enhance the comprehension of tumor biology but also open up avenues for targeted therapies that are crucial in the ongoing battle against cancer.

Conclusion

This report underscores the complexity of cancer genomics and the vital role of advanced sequencing techniques in clinical practice. The recent studies reveal an intricate mutation landscape within various cancer types, demonstrating that somatic mutations are not only diverse but also pivotal for understanding tumor biology and treatment approaches. For instance, the investigation into non-small cell lung cancer (NSCLC) highlighted the prevalence of specific driver mutations and their associations with intrinsic factors such as ethnicity and history of smoking. This reinforces the necessity for tailored therapeutic strategies based on individual patient profiles and genetic backgrounds. Moreover, the identification of novel mutations, such as the GANAB variant in polycystic liver disease and the SOS1 mutation in lung cancer, highlights the continued evolution of our understanding of genetic contributors to cancer. These findings emphasize the potential for personalized medicine, where therapies can be aligned with the specific genetic mutations present in a patient's tumor. The application of whole-exome sequencing (WES) across various studies has proven to be an indispensable tool in uncovering these mutations, enabling the identification of actionable targets for drug development. The exploration of mutation detection through patient-derived organoids in pancreatic ductal adenocarcinoma (PDAC) also showcases an innovative approach to overcoming traditional diagnostic challenges. By retaining a substantial proportion of somatic mutations found in the original tumors, organoids offer a promising avenue for enhancing mutation identification and refining treatment protocols. Similarly, the use of liquid-based cytology for genomic profiling in endometrial cancer represents a significant step towards non-invasive diagnostic methodologies, further underscoring the advancements in precision oncology. As we navigate through the findings from glioblastoma studies, the identification of mutated oncodriver genes opens new avenues for therapeutic intervention. The development of drugs targeting these mutations, alongside established therapies, can potentially improve patient outcomes. Furthermore, understanding the dynamics of tumor evolution, especially in the context of metastatic disease, offers critical insights for therapeutic strategies aimed at preventing recurrence. Future research should focus on longitudinal studies to track mutation evolution and validate the therapeutic targets identified through these genomic analyses. This is particularly essential given the rapid advancement of sequencing technologies and the growing recognition of tumor heterogeneity. Enhanced tracking of mutation dynamics could provide invaluable information on how tumors adapt to therapeutic pressures, ultimately guiding the development of

next-generation therapies. In conclusion, by enhancing our understanding of the mutation landscape across various cancers, we can improve diagnostic strategies and develop more effective therapies tailored to individual patient profiles. The integration of genomic profiling in clinical practice not only holds the potential for improved patient outcomes but also represents a paradigm shift in how we approach cancer treatment, moving towards more personalized and precise interventions. As we continue to unravel the complexities of cancer genomics, it is imperative that we foster collaboration between researchers, clinicians, and patients to realize the full potential of these advancements in oncology.

Recent Research List

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Authors	Title	Journal Name	Volume	Issue	Publication Year	Page Numbers	DOI
['Vaibhavi Pathak', 'Koichi Tazaki', 'Minal Çalışkan']	Revisiting variation in the somatic mutation landscape of non-small cell lung cancer.	HGG advances	6	2	2025	100420	10.1016/j.xhgg.2025.100420
['Xiuling Zhu', 'Ying Liang', 'Xiaoling Zhou', 'Zhiqiang Zhang', 'Yuzhen Liu', 'Zhongyuan Cui', 'Zhixian Wu', 'Dongliang Li']	GANAB c.1118C > T is a novel variant in patients with polycystic liver disease / polycystic kidney disease.	Gene	946		2025	149339	10.1016/j.gene.2025.149339
['Arnob Sarker', 'Burhan Uddin', 'Reaz Ahmmed', 'Sabkat Mahmud', 'Alvira Ajadee', 'Md Al Amin Pappu', 'Md Abdul Aziz', 'Md Nurul Haque Mollah']	Discovery of mutated oncodriver genes associated with glioblastoma originated from stem cells of subventricular zone through whole exome sequence profile analysis, and drug repurposing.	Heliyon	11	2	2025	e42052	10.1016/j.heliyon.2025.e42052
['Juilee Rege', 'Aaron M Udager']	Molecular characterization of archival adrenal tumor tissue from patients with ACTH-independent Cushing syndrome.	The Journal of steroid biochemistry and molecular biology	247		2025	106666	10.1016/j.jsbmb.2024.106666
['Gerhard Hamilton', 'Sandra Stickler', 'Mikhail Ermakov', 'Marie-Therese Eggerstorfer', 'Francesca Paola Nocera', 'Martin Hohenegger', 'Lukas Weigl', 'Maximilian Johannes Hochmair', 'Karl Kashofer']	Characterization of the BH1406 non-small cell lung cancer (NSCLC) cell line carrying an activating SOS1 mutation.	Translational lung cancer research	13	11	2024	2987-2997	10.21037/tlcr-24-570
['Elham Aida Farshadi', 'Wenya Wang', 'Farzana Mohammad', 'Elise van der Oost', 'Michail Doukas', 'Casper H J van Eijck', 'Harmen J G van de Werken', 'Peter D Katsikis']	Tumor organoids improve mutation detection of pancreatic ductal adenocarcinoma.	Scientific reports	14	1	2024	25468	10.1038/s41598-024-75888-y
['Reika Takamatsu', 'Kohei Nakamura', 'Tatsuyuki Chiyoda', 'Kosuke Tsuji', 'Ryutaro Kawano', 'Naoki Yoshimi', 'Wataru Yamagami', 'Hiroshi Nishihara']	Advancing Precision Oncology: Whole-Exome Sequencing in Endometrial Cancer Liquid-Based Cytology.	Archives of pathology & laboratory medicine			2024		10.5858/arpa.2024-0137-OA
['Lalawmpuii Pachuau', 'H Lalremmawia', 'Lalengkimi Ralte', 'Johan Vanlalpeka', 'Jeremy Lalrinsanga Pautu', 'Saia Chenkual', 'Thomas Zomuana', 'Sailo Tlau Lalruatfela', 'John Zohmingthanga', 'Lalchhandama Chhakchhuak', 'Ashok K Varma', 'Nachimuthu Senthil Kumar']	Uncovering novel pathogenic variants and pathway mutations in triple-negative breast cancer among the endogamous mizo tribe.	Breast cancer research and treatment	209	2	2025	375-387	10.1007/s10549-024-07501-9
['Aki Sato', 'Nozomi Yusa', 'Hiroyuki Takamori', 'Eigo Shimizu', 'Kazuaki Yokoyama', 'Satoshi Ichikawa', 'Hisayuki Yokoyama', 'Yuki Kasahara', 'Kodai Enda', 'Fumiyoshi Fujishima', 'Ryo Ichinohasama', 'Yasunori Ota', 'Seiya Imoto', 'Yasuhito Nannya']	Common progenitor origin for Rosai-Dorfman disease and clear cell sarcoma.	The Journal of pathology	264	3	2024	243-249	10.1002/path.6345
['Yujie Jiang', 'Matthew D Montierth', 'Kaixian Yu', 'Shuangxi Ji', 'Shuai Guo', 'Quang Tran', 'Xiaoqian Liu', 'Seung Jun Shin', 'Shaolong Cao', 'Ruonan Li', 'Yuxin Tang', 'Tom Lesluyes', 'Scott Kopetz', 'Jaffer Ajani', 'Pavlos Msaouel', 'Sumit K Subudhi', 'Ana Aparicio', 'Padmanee Sharma', 'John Paul Shen', 'Anil K Sood', 'Maxime Tarabichi', 'Jennifer R Wang', 'Marek Kimmel', 'Peter Van Loo', 'Hongtu Zhu', 'Wenyi Wang']	Pan-cancer subclonal mutation analysis of 7,827 tumors predicts clinical outcome.	bioRxiv : the preprint server for biology			2024		10.1101/2024.07.03.601939
['Troy Hutchens', 'Wade Thorstad', 'Xiaowei Wang', 'Yuanxiang Li', 'Eric J Duncavage', 'Lulu Sun', 'Rebecca D Chernock']	Head and neck squamous cell carcinomas of unknown primary: Can ancillary studies help identify more primary tumor sites?	Experimental and molecular pathology	138		2024	104915	10.1016/j.yexmp.2024.104915
['Hao Xu', 'Xinyu Chen', 'Ying Sun', 'Xiaomu Hu', 'Xuan Zhang', 'Ye Wang', 'Qisheng Tang', 'Qiongji Zhu', 'Kun Song', 'Hong Chen', 'Xiaofang Sheng', 'Yu Yao', 'Dongxiao Zhuang', 'Lingchao Chen', 'Ying Mao', 'Zhiyong Qin']	Comprehensive molecular characterization of long-term glioblastoma survivors.	Cancer letters	593		2024	216938	10.1016/j.canlet.2024.216938
['Amir Hozhabrpour', 'Marzieh Mojbafan', 'Fahimeh Palizban', 'Fatemeh Vahidnezhad', 'Saeed Talebi', 'Maliheh Amani', 'Masoud Garshasbi', 'Anoosh Naghavi', 'Raziyeh Khalesi', 'Parvin Mansouri', 'Soheila Sotoudeh', 'Hamidreza Mahmoudi', 'Aida Varghaei', 'Maryam Daneshpazhooh', 'Fatemeh Karimi', 'Sirous Zeinali', 'Elnaz Kalamati', 'Jouni Uitto', 'Leila Youssefian', 'Hassan Vahidnezhad']	DNA repair-related heritable photosensitivity syndromes: Mutation landscape in a multiethnic cohort of 17 multigenerational families with high degree of consanguinity.	DNA repair	136		2024	103633	10.1016/j.dnarep.2024.103633
['Ahmed A Ahmed', 'Mateja Sborchia', 'Hannah Bye', 'Maria Roman-Escorza', 'Ariella Amar', 'Rhonda Henley-Smith', 'Edward Odell', 'Mark McGurk', 'Michael Simpson', 'Tony Ng', 'Elinor J Sawyer', 'Christopher G Mathew']	Mutation detection in saliva from oral cancer patients.	Oral oncology	151		2024	106717	10.1016/j.oraloncology.2024.106717
['Matthew R Lawrence-Paul', 'Tien-Chi Pan', 'Dhruv K Pant', 'Natalie N C Shih', 'Yan Chen', 'George K Belka', 'Michael Feldman', 'Angela DeMichele', 'Lewis A Chodosh']	Rare subclonal sequencing of breast cancers indicates putative metastatic driver mutations are predominately acquired after dissemination.	Genome medicine	16	1	2024	26	10.1186/s13073-024-01293-9