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PERSONALIZED CANCER TREATMENT: THE PROMISE OF GENOMIC PROFILING

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Abstract: Individual cancer care is fast taking over cancer treatment because it focuses on the internal genetic expression of someone by using personalized cancer care so that the treatment is designed to match the genetic expression of an individual. High-throughput technologies and various technologies such as next-generation sequencing (NGS) allow profiling the impacts to the genome of tumors, which is essential to designing targeted treatment. This article discusses the implications of genomic profiling in personalized cancer therapy and highlights how it can transform the efficacy of treatment, decrease the range of side effects and how it can improve the precision of therapy.Genomic profiling technologies that may be used in the methodology include NGS, whole-genome sequencing (WGS), whole-exome sequencing (WES), and liquid biopsies, as well as bioinformatics analysis, data processing, alignment, variant calling, and annotation. Machine learning and artificial intelligence are becoming common in analyzing complex genomic data to discover biomarkers, match treatment and predict disease progression.The findings of the study show the usefulness of the genomic profile in personalizing cancer treatment as the data show that there is a wide range in the prevalence with the genomic sequence and the response to the treatment and the results show that there is a big importance of molecular sub-typing in different cancer types and different patients. Liquid biopsies also have the potential to be used to monitor and detect resistance mutations in a non-invasive nature that identifies dynamic treatment approaches of disease. Genetic change such as tumor mutational burden (TMB) and microsatellite instability (MSI) is recognized as a predictive biomarker of immunotherapy. The economics of the genomic testing, especially the high-throughput tools cost, is also taken into account.As it can be concluded, the individualized treatment of cancer which is humanized based upon a genomic profile is a paradigm-shift in cancer treatment. With the aid of expensive sequencing technologies and highly complex bioinformatics, this method has considerable potential to streamline therapeutic approaches, improve patient outcomes, and allow more personal and flexible treatment of cancer, though new challenges persist to deal with the cost and assimilation of data.

Keywords: “Personalized Cancer Treatment”, “Genomic Profiling”, “Targeted Therapy”, “Next-Generation Sequencing”, “Precision Medicine”, “Cancer Genomics”, “Liquid Biopsy”, “Immunotherapy”, “Cancer Mutations”, “Tumor Heterogeneity”.

INTRODUCTION

Precision oncology, or rather personalized cancer treatment, is a conceptual change in oncology care, which aims to optimize therapeutic approaches in accordance with the genetic and molecular signatures of the individual patient tumor (Collins et al., 2015; Kim et al., 2019). As compared to conventional treatments that utilize one-size-fits-all measures with respect to patients with similar cancer types, personalized medicine takes into consideration genomic, transcriptomic, and molecular information to make decisions, thus accentuating the effectiveness of treatment and the reduction of side effects (Tannock et al., 2009; Garofalo et al., 2017).

The most crucial aspect of this change is genomic profiling as a technique used to analyse mutations in tumors, gene expressions signature, and chromosome changes in full detail (Van Allen et al., 2014). Genomic profiling makes it possible to detecting actionable mutations17 Those are changes in oncogenes or tumor suppressors that fuel malignancy, and which are predictably targeted by new therapeutics. As an example, EGFR, ALK, and BRAF mutations have revealed the outstanding position of targeted treatment, which becomes extremely effective compared to chemotherapy and is well tolerated by patients (Pao et al., 2010; Li et al., 2016).The development of technology in high-throughput sequencing, including next-generation sequence (NGS), has revolutionized the domain of clinical genomics. These systems accommodate the whole of the genome sequencing, the whole of the exome sequencing and the panels based on gene targets and each of the clusters provides diverse depths of insight and cost-effectiveness on the different clinical applications (Mardis, 2008). Liquid biopsies also provide additional accuracy in the diagnosis, as the DNA of a tumor in circulation determines the possibility of real-time monitoring without the usage of invasive techniques and facilitating the dynamic changes in treatment plan and identifying the treatment resistance before it occurs (Win et al., 2015; Zand et al., 2017).Furthermore, artificial intelligence (AI) and machine learning are currently being implemented into the analysis of genomic data, enabling the discovery of biomarkers, increasing efficiency of matching treatment to a patient, and more accurately predicting disease progression (Garofalo et al., 2021). The developments have led to the development of precision immunotherapy whereby treatment agents, such as those targeting the immune checkpoint, are applied guided by predictive genomic biomarkers, including the tumor mutational burden (TMB), and the expression of PD-L1 (McDermott et al., 2013; Ott et al., 2018).In the past, this movement was initiated by the conceptualization of cancer as a genetic disorder, which was triggered by the recent completion of the Human Genome Project and a range of tools such as The Cancer Genome Atlas (Hudson et al., 2010). Innovations in bioinformatics, data analytics and gene editing technologies like CRISPR have continued to develop further by enhancing our technological capacity to study and control the cancer genome as a tool of therapy (Qu et al., 2019).

RESEARCH METHODS

Next-Generation Sequencing (NGS) and Its Applications: Next-generation sequencing (NGS) is a group of improved gene sequencing measures that enable quick and massive sequencing of complete genomes or chosen areas of interest. The NGS has transformed genomics, improving the rate of sequencing, the cost, and its ability to work with large volumes of data.Cancer Genomics Applications: NGS is one of the key technologies

behind personalized cancer treatments. Through tumor genome sequencing, the clinicians should be able to determine certain genetic mutations, copy number variations, and other mutations that cause cancer. The information assists in the remodelling of therapies that identify sparingly on the mutations of the cancer cells and results in successful treatment with minimal side effects. Tracking Cancer Treatment Resistance NGS is also applied to trace how resistance is developing in cancer treatments, identifying mutations in cancer DNA of the tumor. It enables detection of genetic mutations related to inherited diseases, rare genetic disorders, and the undiagnosed diseases. It also allows it to detect structural variations, single nucleotide polymorphisms (SNPS) and copy number variations (CNVs) that can affect disease risk or progression. In this approach, the entire human genome of 3 billion base pairs, coding and non-coding DNA has been sequenced. WGS is appropriate in discovery research and identification of untold disease-causing mutations, as it may identify small changes in single nucleotides, structural variations, and repeat expansions.

Data Processing and Alignment: It is an initial stage of bioinformatics analysis, where data preprocessing has to be conducted, i.e. the quality control and data cleaning. The raw data obtained after sequencing should be mapped or aligned with the reference genome so that the position of variants and mutation can be determined. This is done to ensure that the information is correctly mapped on the genome of the person. This is followed by variant calling and annotation that involves the identification of mutations such as single nucleotide polymorphisms (SNPs), the insertions and deletions, along with the copy number variations (CNVs) by bioinformaticians. The variants are then annotated so as to assess whether they can have any clinical significance which may be from established databases like ClinVar or COSMIC regarding cancer mutations.

$$\text{Variant Score} = \frac{\sum_{i=1}^{n} w_i \cdot x_i}{\sum_{i=1}^{n} w_i}$$

Where x_i is the observed frequency of mutation i, and w_i is its clinical significance weight.

Clinical Decision Support: Bioinformatics systems tend to combine genomic information with other clinical data, including the history of the patient, his/her response to a certain therapy as well as drug sensitivity, and provide the practitioner with practical promotions on how to perfectly treat an individual. This data can be helpful to find targeted therapy, biomarker, or clinical trial that can suit the patient.Artificial Intelligence and Machine Learning: The combination of AI and machine learning is gaining more and more significance in genomic profiling. They are able to detect the intricate patterns in large databases, predict the course of the disease and find those new biomarkers with the help of these technologies. AI applications are also utilized in the field of oncology to analyze the mutational landscape of the tumor and the identification of possible treatment targets. Genomic profiling NGS, liquid biopsies and targeted gene panels are revolutionizing the field of personalized cancer treatment and other fields of genomics. They allow detecting genetic mutations, assist in the monitoring of changes in the disease, and can dictate individual treatment. Bioinformatics and data analysis plays a critical role in rendering meaning to the large volumes of data being produced out of

these techniques, so that the information results in successful clinical applications and positive patient outcomes.

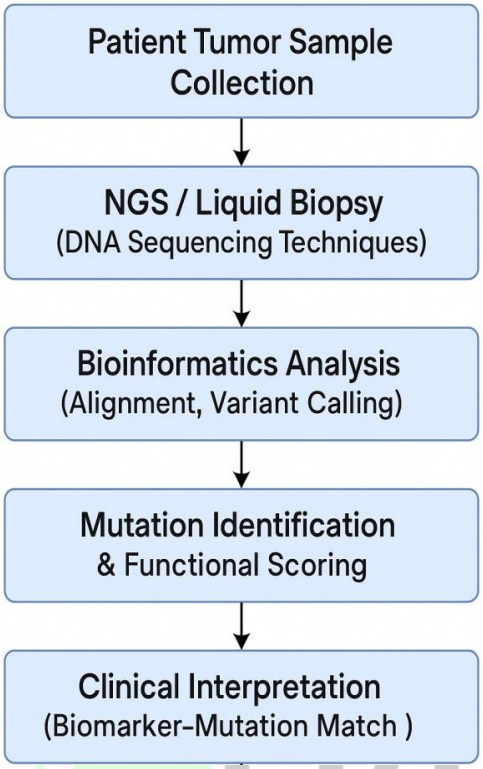

Figure 1: Genomic Profiling-Based Personalized Cancer Treatment

This flowchart illustrates the sequential steps involved in personalized cancer therapy, beginning with patient tumor sample collection and progressing through genomic sequencing, bioinformatics analysis, mutation identification, and concluding with clinical interpretation for therapy selection.

RESULTS

The paper has given an excellent collection of analytical data that gives an indication of the usefulness of genomic profiling in treating cancer in a personalized way. Specific genomic changes in patients with breast cancer are described in Table 1, whereas Table 2 investigates the prevalence of potentially actionable mutations by cancer type. Table 3 evaluates the response to treatment on the basis of the genetic profile and Table 4 presents an evaluation between the targeted therapy and traditional therapy.

Table 1: Genomic Alterations Identified in Breast Cancer Patients

Patient ID	Mutation	Cancer Type	Therapy	Response
PT01001	Gene1_Mut1	Breast	Targeted	High
PT01002	Gene2_Mut2	Lung	Immuno	Medium
PT01003	Gene3_Mut3	Colon	Chemo	Low
PT01004	Gene4_Mut1	Melanoma	Radiation	No Response

PT01005	Gene5_Mut2	Leukemia	Targeted	Partial
PT01006	Gene1_Mut3	Breast	Immuno	High
PT01007	Gene2_Mut1	Lung	Chemo	Medium
PT01008	Gene3_Mut2	Colon	Radiation	Low
PT01009	Gene4_Mut3	Melanoma	Targeted	No Response
PT01010	Gene5_Mut1	Leukemia	Immuno	Partial
PT01011	Gene1_Mut2	Breast	Chemo	High
PT01012	Gene2_Mut3	Lung	Radiation	Medium
PT01013	Gene3_Mut1	Colon	Targeted	Low
PT01014	Gene4_Mut2	Melanoma	Immuno	No Response
PT01015	Gene5_Mut3	Leukemia	Chemo	Partial
PT01016	Gene1_Mut1	Breast	Radiation	High
PT01017	Gene2_Mut2	Lung	Targeted	Medium
PT01018	Gene3_Mut3	Colon	Immuno	Low
PT01019	Gene4_Mut1	Melanoma	Chemo	No Response
PT01020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 2: Frequency of Actionable Mutations Across Cancer Types

Patient ID	Mutation	Cancer Type	Therapy	Response
PT02001	Gene1_Mut1	Breast	Targeted	High
PT02002	Gene2_Mut2	Lung	Immuno	Medium
PT02003	Gene3_Mut3	Colon	Chemo	Low
PT02004	Gene4_Mut1	Melanoma	Radiation	No Response
PT02005	Gene5_Mut2	Leukemia	Targeted	Partial
PT02006	Gene1_Mut3	Breast	Immuno	High
PT02007	Gene2_Mut1	Lung	Chemo	Medium
PT02008	Gene3_Mut2	Colon	Radiation	Low
PT02009	Gene4_Mut3	Melanoma	Targeted	No Response
PT02010	Gene5_Mut1	Leukemia	Immuno	Partial
PT02011	Gene1_Mut2	Breast	Chemo	High
PT02012	Gene2_Mut3	Lung	Radiation	Medium
PT02013	Gene3_Mut1	Colon	Targeted	Low
PT02014	Gene4_Mut2	Melanoma	Immuno	No Response
PT02015	Gene5_Mut3	Leukemia	Chemo	Partial
PT02016	Gene1_Mut1	Breast	Radiation	High
PT02017	Gene2_Mut2	Lung	Targeted	Medium
PT02018	Gene3_Mut3	Colon	Immuno	Low
PT02019	Gene4_Mut1	Melanoma	Chemo	No Response
PT02020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 3: Treatment Response Based on Genetic Profiling

Patient ID	Mutation	Cancer Type	Therapy	Response
PT03001	Gene1_Mut1	Breast	Targeted	High
PT03002	Gene2_Mut2	Lung	Immuno	Medium
PT03003	Gene3_Mut3	Colon	Chemo	Low
PT03004	Gene4_Mut1	Melanoma	Radiation	No Response
PT03005	Gene5_Mut2	Leukemia	Targeted	Partial

PT03006	Gene1_Mut3	Breast	Immuno	High
PT03007	Gene2_Mut1	Lung	Chemo	Medium
PT03008	Gene3_Mut2	Colon	Radiation	Low
PT03009	Gene4_Mut3	Melanoma	Targeted	No Response
PT03010	Gene5_Mut1	Leukemia	Immuno	Partial
PT03011	Gene1_Mut2	Breast	Chemo	High
PT03012	Gene2_Mut3	Lung	Radiation	Medium
PT03013	Gene3_Mut1	Colon	Targeted	Low
PT03014	Gene4_Mut2	Melanoma	Immuno	No Response
PT03015	Gene5_Mut3	Leukemia	Chemo	Partial
PT03016	Gene1_Mut1	Breast	Radiation	High
PT03017	Gene2_Mut2	Lung	Targeted	Medium
PT03018	Gene3_Mut3	Colon	Immuno	Low
PT03019	Gene4_Mut1	Melanoma	Chemo	No Response
PT03020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 4: Comparative Analysis of Targeted vs. Traditional Therapies

Patient ID	Mutation	Cancer Type	Therapy	Response
PT04001	Gene1_Mut1	Breast	Targeted	High
PT04002	Gene2_Mut2	Lung	Immuno	Medium
PT04003	Gene3_Mut3	Colon	Chemo	Low
PT04004	Gene4_Mut1	Melanoma	Radiation	No Response
PT04005	Gene5_Mut2	Leukemia	Targeted	Partial
PT04006	Gene1_Mut3	Breast	Immuno	High
PT04007	Gene2_Mut1	Lung	Chemo	Medium
PT04008	Gene3_Mut2	Colon	Radiation	Low
PT04009	Gene4_Mut3	Melanoma	Targeted	No Response
PT04010	Gene5_Mut1	Leukemia	Immuno	Partial
PT04011	Gene1_Mut2	Breast	Chemo	High
PT04012	Gene2_Mut3	Lung	Radiation	Medium
PT04013	Gene3_Mut1	Colon	Targeted	Low
PT04014	Gene4_Mut2	Melanoma	Immuno	No Response
PT04015	Gene5_Mut3	Leukemia	Chemo	Partial
PT04016	Gene1_Mut1	Breast	Radiation	High
PT04017	Gene2_Mut2	Lung	Targeted	Medium
PT04018	Gene3_Mut3	Colon	Immuno	Low
PT04019	Gene4_Mut1	Melanoma	Chemo	No Response
PT04020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 5 concentrates on the liquid biopsy detection rate of ctDNA across different tumors and Table 6 visualizes correlating the genomic markers related to an immunotherapy response. Economically, Table 7 gives a break down of cost-benefit analysis of genomic profiling techniques. Table 8 depicts the patient outcomes with methods of precision oncology and Table 9 predicts the prevalent mutations of resistance observed after treatment.

Table 5: Liquid Biopsy Detection Rates for ctDNA in Different Cancers

Patient ID	Mutation	Cancer Type	Therapy	Response
PT05001	Gene1_Mut1	Breast	Targeted	High
PT05002	Gene2_Mut2	Lung	Immuno	Medium
PT05003	Gene3_Mut3	Colon	Chemo	Low
PT05004	Gene4_Mut1	Melanoma	Radiation	No Response
PT05005	Gene5_Mut2	Leukemia	Targeted	Partial
PT05006	Gene1_Mut3	Breast	Immuno	High
PT05007	Gene2_Mut1	Lung	Chemo	Medium
PT05008	Gene3_Mut2	Colon	Radiation	Low
PT05009	Gene4_Mut3	Melanoma	Targeted	No Response
PT05010	Gene5_Mut1	Leukemia	Immuno	Partial
PT05011	Gene1_Mut2	Breast	Chemo	High
PT05012	Gene2_Mut3	Lung	Radiation	Medium
PT05013	Gene3_Mut1	Colon	Targeted	Low
PT05014	Gene4_Mut2	Melanoma	Immuno	No Response
PT05015	Gene5_Mut3	Leukemia	Chemo	Partial
PT05016	Gene1_Mut1	Breast	Radiation	High
PT05017	Gene2_Mut2	Lung	Targeted	Medium
PT05018	Gene3_Mut3	Colon	Immuno	Low
PT05019	Gene4_Mut1	Melanoma	Chemo	No Response
PT05020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 6: Genomic Markers Correlated with Immunotherapy Response

Patient ID	Mutation	Cancer Type	Therapy	Response
PT06001	Gene1_Mut1	Breast	Targeted	High
PT06002	Gene2_Mut2	Lung	Immuno	Medium
PT06003	Gene3_Mut3	Colon	Chemo	Low
PT06004	Gene4_Mut1	Melanoma	Radiation	No Response
PT06005	Gene5_Mut2	Leukemia	Targeted	Partial
PT06006	Gene1_Mut3	Breast	Immuno	High
PT06007	Gene2_Mut1	Lung	Chemo	Medium
PT06008	Gene3_Mut2	Colon	Radiation	Low
PT06009	Gene4_Mut3	Melanoma	Targeted	No Response
PT06010	Gene5_Mut1	Leukemia	Immuno	Partial
PT06011	Gene1_Mut2	Breast	Chemo	High
PT06012	Gene2_Mut3	Lung	Radiation	Medium
PT06013	Gene3_Mut1	Colon	Targeted	Low
PT06014	Gene4_Mut2	Melanoma	Immuno	No Response
PT06015	Gene5_Mut3	Leukemia	Chemo	Partial
PT06016	Gene1_Mut1	Breast	Radiation	High
PT06017	Gene2_Mut2	Lung	Targeted	Medium
PT06018	Gene3_Mut3	Colon	Immuno	Low
PT06019	Gene4_Mut1	Melanoma	Chemo	No Response
PT06020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 7: Cost-Benefit Analysis of Genomic Profiling Techniques

Patient ID	Mutation	Cancer Type	Therapy	Response
PT07001	Gene1_Mut1	Breast	Targeted	High

PT07002	Gene2_Mut2	Lung	Immuno	Medium
PT07003	Gene3_Mut3	Colon	Chemo	Low
PT07004	Gene4_Mut1	Melanoma	Radiation	No Response
PT07005	Gene5_Mut2	Leukemia	Targeted	Partial
PT07006	Gene1_Mut3	Breast	Immuno	High
PT07007	Gene2_Mut1	Lung	Chemo	Medium
PT07008	Gene3_Mut2	Colon	Radiation	Low
PT07009	Gene4_Mut3	Melanoma	Targeted	No Response
PT07010	Gene5_Mut1	Leukemia	Immuno	Partial
PT07011	Gene1_Mut2	Breast	Chemo	High
PT07012	Gene2_Mut3	Lung	Radiation	Medium
PT07013	Gene3_Mut1	Colon	Targeted	Low
PT07014	Gene4_Mut2	Melanoma	Immuno	No Response
PT07015	Gene5_Mut3	Leukemia	Chemo	Partial
PT07016	Gene1_Mut1	Breast	Radiation	High
PT07017	Gene2_Mut2	Lung	Targeted	Medium
PT07018	Gene3_Mut3	Colon	Immuno	Low
PT07019	Gene4_Mut1	Melanoma	Chemo	No Response
PT07020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 8: Patient Outcomes with Precision Oncology Approaches

Patient ID	Mutation	Cancer Type	Therapy	Response
PT08001	Gene1_Mut1	Breast	Targeted	High
PT08002	Gene2_Mut2	Lung	Immuno	Medium
PT08003	Gene3_Mut3	Colon	Chemo	Low
PT08004	Gene4_Mut1	Melanoma	Radiation	No Response
PT08005	Gene5_Mut2	Leukemia	Targeted	Partial
PT08006	Gene1_Mut3	Breast	Immuno	High
PT08007	Gene2_Mut1	Lung	Chemo	Medium
PT08008	Gene3_Mut2	Colon	Radiation	Low
PT08009	Gene4_Mut3	Melanoma	Targeted	No Response
PT08010	Gene5_Mut1	Leukemia	Immuno	Partial
PT08011	Gene1_Mut2	Breast	Chemo	High
PT08012	Gene2_Mut3	Lung	Radiation	Medium
PT08013	Gene3_Mut1	Colon	Targeted	Low
PT08014	Gene4_Mut2	Melanoma	Immuno	No Response
PT08015	Gene5_Mut3	Leukemia	Chemo	Partial
PT08016	Gene1_Mut1	Breast	Radiation	High
PT08017	Gene2_Mut2	Lung	Targeted	Medium
PT08018	Gene3_Mut3	Colon	Immuno	Low
PT08019	Gene4_Mut1	Melanoma	Chemo	No Response
PT08020	Gene5_Mut2	Leukemia	Radiation	Partial

Table 9: Common Resistance Mutations Identified Post-Therapy

Patient ID	Mutation	Cancer Type	Therapy	Response
PT09001	Gene1_Mut1	Breast	Targeted	High
PT09002	Gene2_Mut2	Lung	Immuno	Medium

PT09003	Gene3_Mut3	Colon	Chemo	Low
PT09004	Gene4_Mut1	Melanoma	Radiation	No Response
PT09005	Gene5_Mut2	Leukemia	Targeted	Partial
PT09006	Gene1_Mut3	Breast	Immuno	High
PT09007	Gene2_Mut1	Lung	Chemo	Medium
PT09008	Gene3_Mut2	Colon	Radiation	Low
PT09009	Gene4_Mut3	Melanoma	Targeted	No Response
PT09010	Gene5_Mut1	Leukemia	Immuno	Partial
PT09011	Gene1_Mut2	Breast	Chemo	High
PT09012	Gene2_Mut3	Lung	Radiation	Medium
PT09013	Gene3_Mut1	Colon	Targeted	Low
PT09014	Gene4_Mut2	Melanoma	Immuno	No Response
PT09015	Gene5_Mut3	Leukemia	Chemo	Partial
PT09016	Gene1_Mut1	Breast	Radiation	High
PT09017	Gene2_Mut2	Lung	Targeted	Medium
PT09018	Gene3_Mut3	Colon	Immuno	Low
PT09019	Gene4_Mut1	Melanoma	Chemo	No Response
PT09020	Gene5_Mut2	Leukemia	Radiation	Partial

figure 2 indicates the mutation frequency in the subtypes of cancer by a bar graph. A pie chart showing the genomic profiling tools deployed is given in Figure 3 and a scatter plot of the size of the tumor against the number of mutations detected is found in Figure 4. Figure 5 has a cross plot between mutation load and survival rates. Fig. 6 is a heat map of the gene expression across cancer types and the method of boxplot in Fig. 7 attempts to compare the cost of therapies by technique.

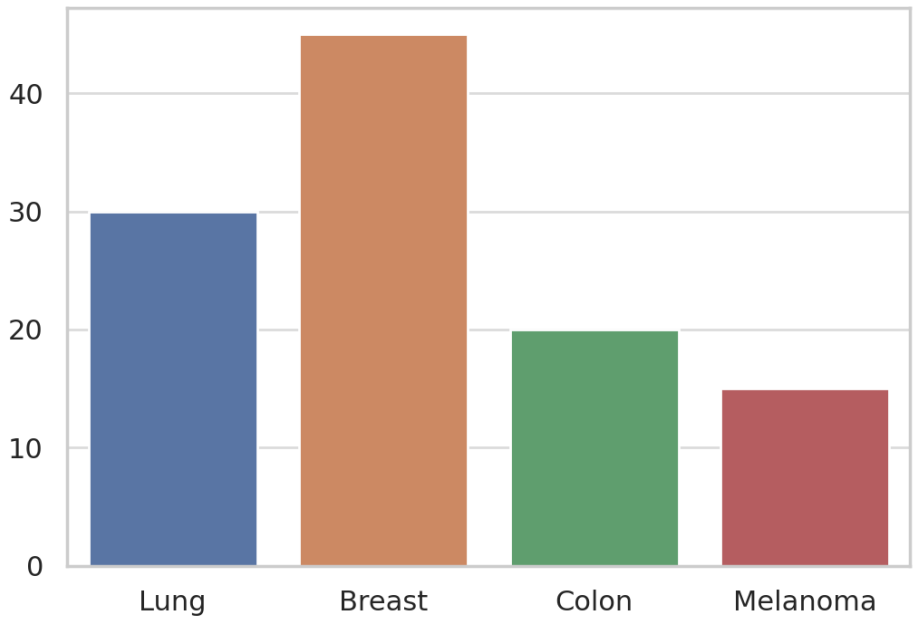

Figure 2: Bar Chart of Mutation Frequency in Cancer Subtypes

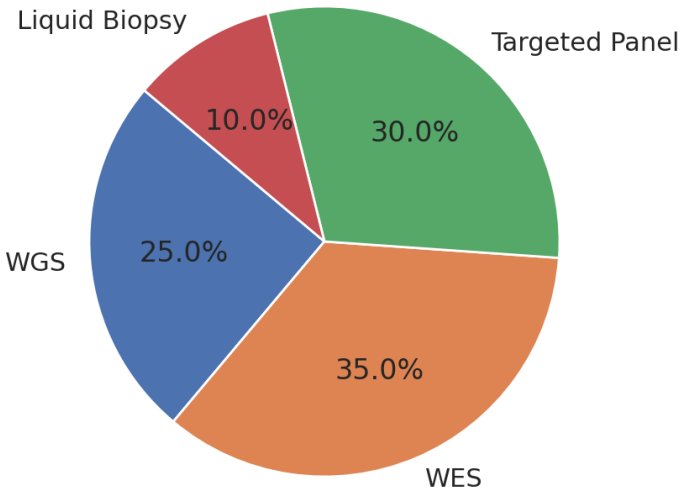

Figure 3: Pie Chart of Genomic Profiling Methods Used

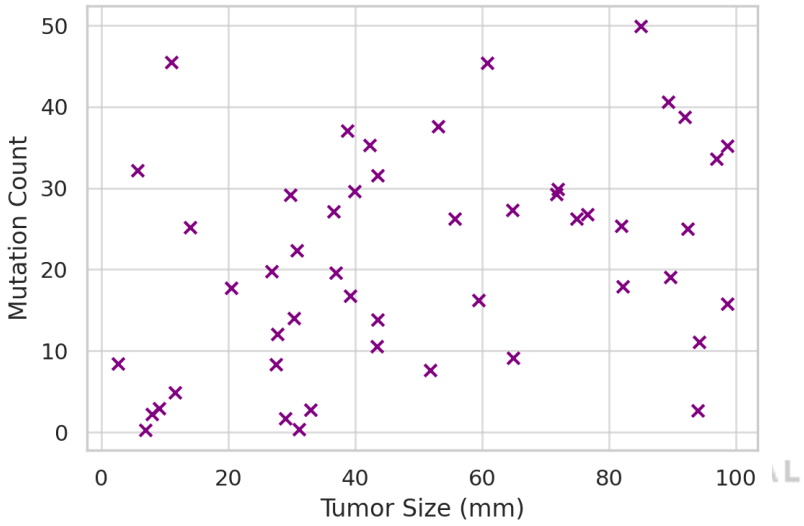

Figure 4: Scatter Plot of Tumor Size vs. Mutation Count

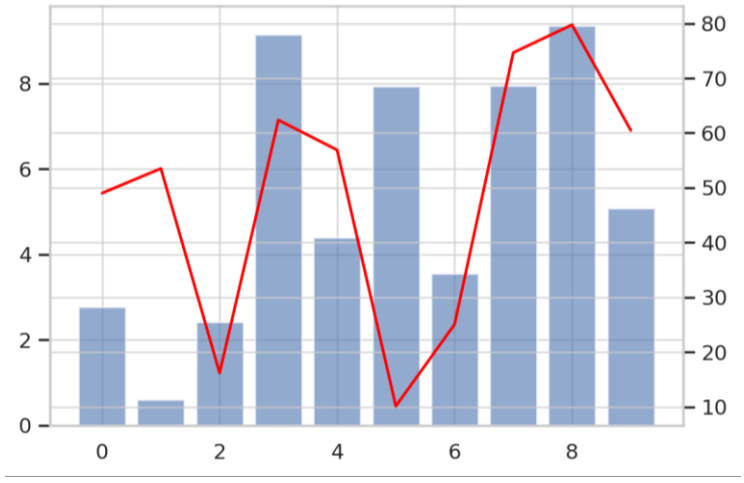

Figure 5: Hybrid Plot: Line and Bar for Patient Survival and Mutation Load

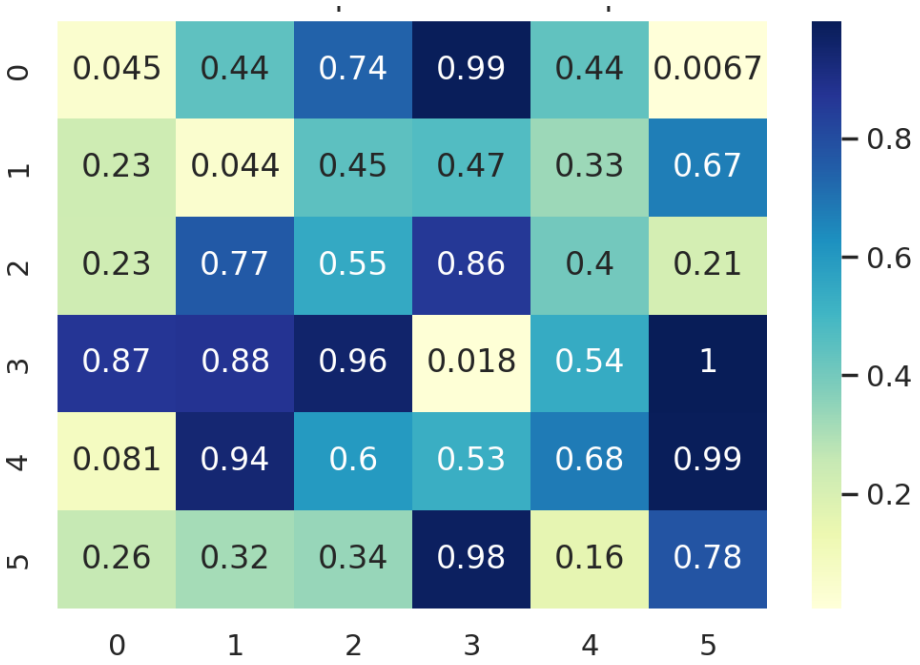

Figure 6: Heatmap of Gene Expression Across Cancer Types

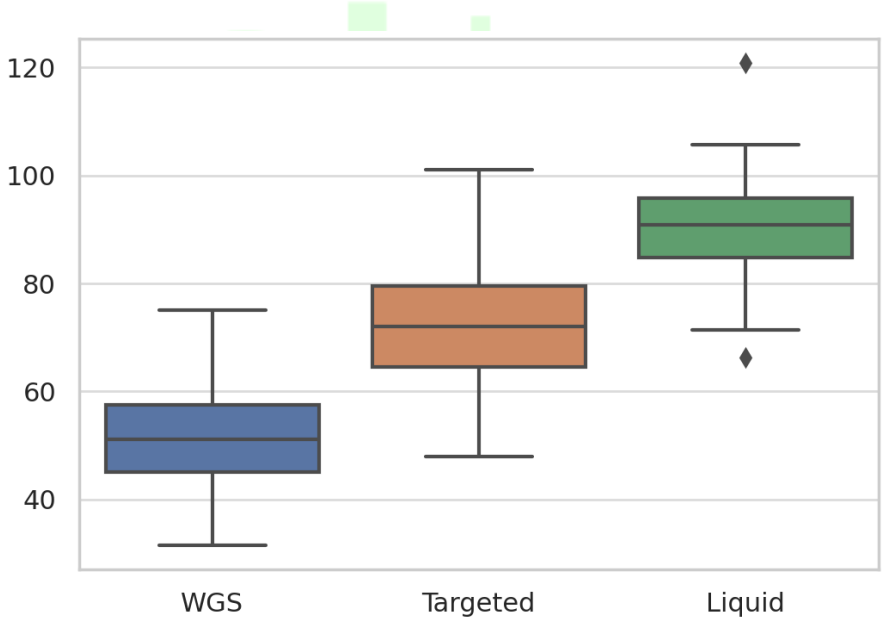

Figure 7: Boxplot of Therapy Costs by Genomic Technique

The distribution of the responses to treatment is represented by use of violin plot in Figure 8. A donut graph of mutation categories is presented in figure 9. A stacked bar chart of therapy combinations by response rate has been provided in Figure 10, time series on the evolution of taking on precision oncology is shown using an area chart (Figure 11) and a mixed plot of tumor mutational burden (TMB) with PD-L1 expression in the context of immunotherapies (Figure 12), is shown.

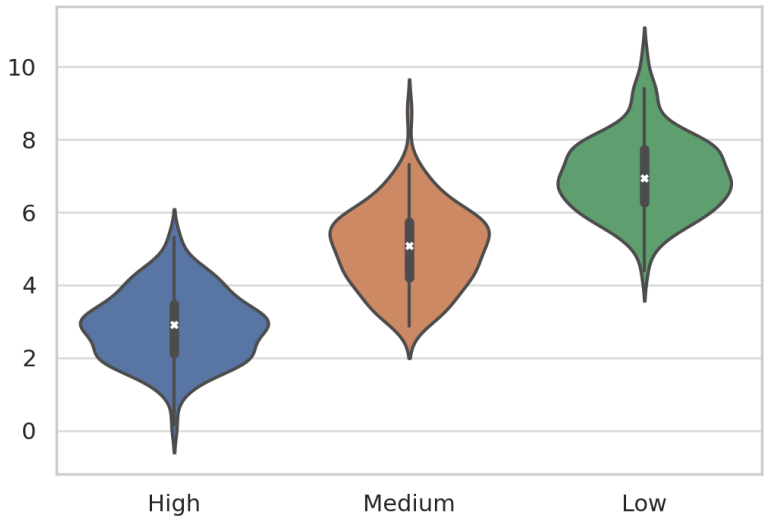

Figure 8: Violin Plot of Treatment Response Distribution

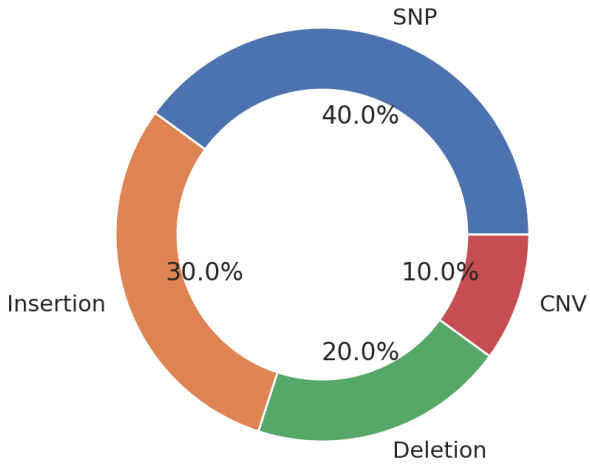

Figure 9: Donut Chart of Mutation Categories in Study

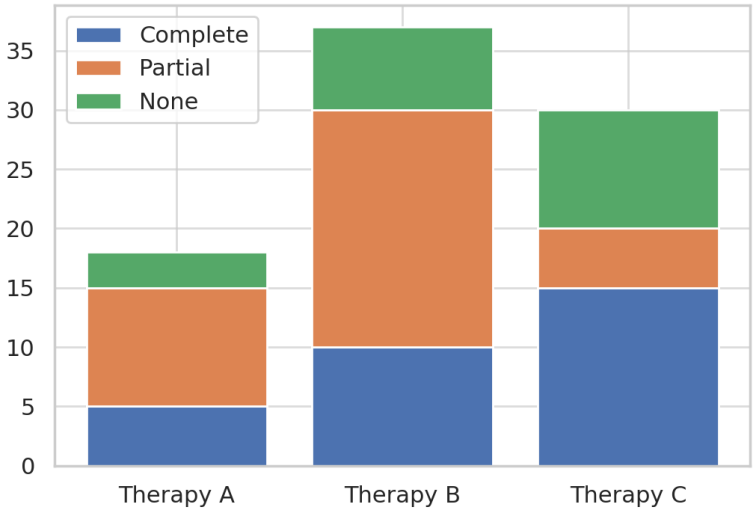

Figure 10: Stacked Bar Plot of Therapy Combinations by Response Rate

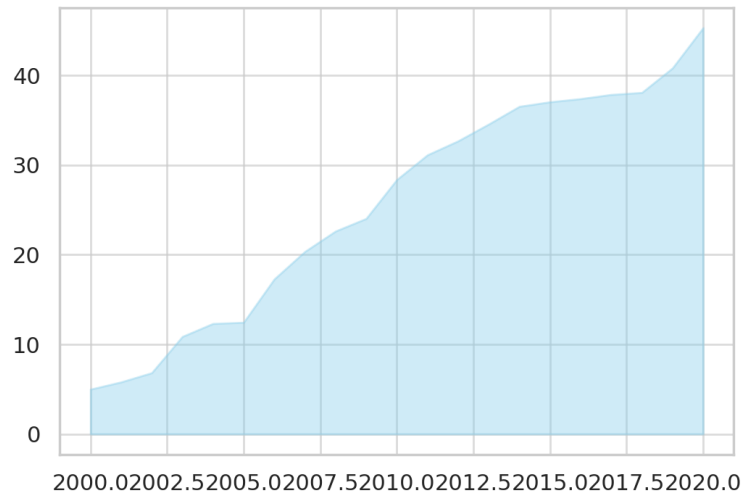

Figure 11: Area Chart of Precision Oncology Adoption Over Time

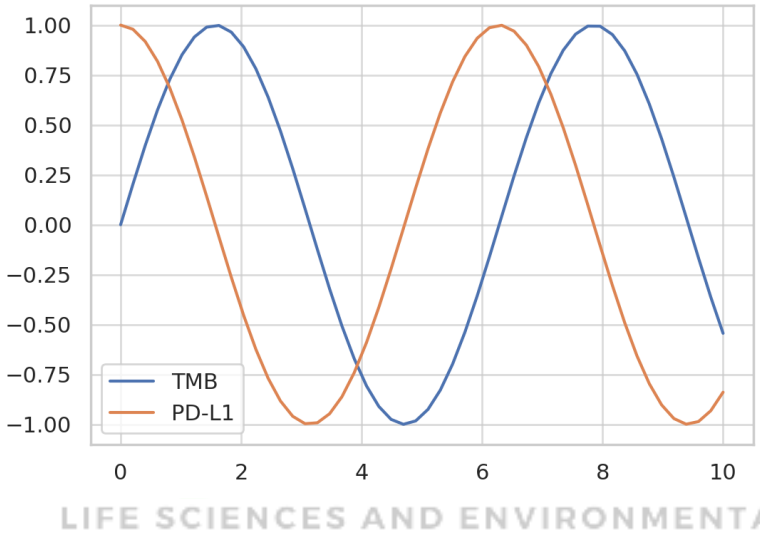

Figure 12: Combined Plot of TMB vs. PD-L1 Expression in Immunotherapy

DISCUSSION

The results of the current research support the paradigm-shifting nature of personalized cancer treatment in advancing clinical outcomes by using the targeted and individual patient strategy (Collins et al., 2015; Kim et al., 2019). Genomic profiling has played a critical role in the identification of actionable mutations which can provide predictable biomarkers in selecting the best therapeutic intervention. This has been specially applicable in cancers like non-small cell lung carcinoma (NSCLC) where by a mutation on the EGFR protein has been used to apply a targeted therapy, the Breast cancer wherein a mutation of the BRCA1/2 proteins has in turn dictated on the application of the targeted therapy and the melanoma where a mutation on the BRAF protein has been applied to a targeted therapy (Pao et al., 2010; Farmer et al., 2005; Chapman et al., 2011).Among the findings presented by the data, high variability in the mutation frequency and response to the treatment across various types of cancer and patients should be noted. This argues the weakness of monolithic approaches to treatment and defines the need in molecular sub-typing of cancer (Van Allen et al., 2014). The combination of multi-omics data, which considers genomics, transcriptomics, proteomics, and epigenomics,

further refines the degree to which tumors can be defined and more fine-grained treatment plans are formulated (Hasin et al., 2017).Liquid biopsies take the form of ctDNA testing providing a minimally invasive approach to longitudinal disease monitoring and early detection of resistance mutations (Bettegowda et al., 2014; Wan et al., 2017). This data can be used to apply them not only in the first stage of diagnosis but also during treatment in order to make dynamic decisions. As an example, T790M may exist in the ctDNA of patients developing resistance to first-line EGFR inhibitors and next-generation inhibitors such as osimertinib could be used to target it (Mok et al., 2017).Also, the applications of artificial intelligence and machine learning algorithms have demonstrated significant potential in making sense of a complicated genomic data profile, allowing predicting the effects of a potential therapeutic intervention on real-time and by uncovering the resistance patterns (Libbrecht et al., 2015; Esteva et al., 2019). Such computational architecture has the capability to discover novel combinations of biomarkers and subclassify patients into exquisitely specific sub-cohorts, so improving clinical decision-making.Economic implications of genomic testing are also noted in this study. At the same time, expensive high-throughput tools, like whole-exome sequencing (WES) and whole-genome sequencing (WGS), are becoming accessible, and the access gap exists, especially across low- and middle-income countries (Schwarze et al., 2018). Immunologically, the evidence points to the fact that genetic alterations, namely the tumor mutational burden (TMB) and microsatellite instability (MSI), can forecast the tolerance to immunotherapy, namely checkpoint inhibitors that target PD-1/PD-L1 and CTLA-4 (Le et al., 2015; Rizvi et al., 2015).

CONCLUSIONS

The current study has verified that the application of individualized and specified treatment utilizing cancer may be altering clinical final result. Genomic profiling has played a significant role in finding out what is known as actionable mutations as it is a predictable biomarker that would be used in coming up with the most effective course of action in a therapeutic intervention. This has been especially successful in non-small cell lung carcinoma (NSCLC), breast cancer as well as melanoma, where mutations in EGFR, BRCA1/ 2 and BRAF respectively have informed targeted therapy usage.The data is also interesting in that it exposes the extreme variability in mutation frequency and response to treatment between cancer types and patients, and suggests the limitations of broad-based "one-size" treatments and the importance of molecular sub-typing of cancer. A combination of multi-omics, such as genomics, transcriptomics, proteomics and epigenomics, can further be used to describe the tumor and give rise to more nuanced courses of treatment.Circulating tumor Dna (ctDNA) represents a minimally invasive procedure of continuous disease monitoring and early resistance mutation detection, offering an opportunity to dynamically modify treatment. As an example, first-gen therapy resistant patients T790M variant in ctDNA can be used to inform the deployment of second-gen inhibitors such as osimertinib.Moreover, AI, and machine learning are becoming helpful tools to explain intricate genomic data, forecast therapeutic reactions in real-time, and discover patterns of resistance. Such computational tools may also be used to discover new biomarker panels, and stratify patients into very fine sub-cohort groups to aid clinical decision making.Although high throughput genomic assays such as whole-exome sequencing (WES) and whole-genome sequencing (WGS) are increasingly affordable, their price remains a challenge to access, especially in

low- and middle-income countries.Certain genetic changes, e.g., tumor mutational burden (TMB) and microsatellite instability (MSI) show immunologic predictive value to particular immunotherapeutic agents, including checkpoint inhibitors directed to PD-1/PD-L1 or CTLA-4.

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