

Article

Tumor microenvironment characteristics and prognosis differences based on genome map from a biomechanical perspective

Jiajing Yang¹, Chunxiang Shang^{1,2,*}

¹ College of Clinical Medicine, Qinghai University, Xining 810016, China
 ² The Fifth People's Hospital of Qinghai Province, Xining 810007, China
 * Corresponding author: Chunxiang Shang, 452344819@qq.com

CITATION

Yang J, Shang C. Tumor microenvironment characteristics and prognosis differences based on genome map from a biomechanical perspective. Molecular & Cellular Biomechanics. 2025; 22(4): 1439. https://doi.org/10.62617/mcb1439

ARTICLE INFO

Received: 23 January 2025 Accepted: 17 February 2024 Available online: 3 March 2025





Copyright © 2025 by author(s). *Molecular & Cellular Biomechanics* is published by Sin-Chn Scientific Press Pte. Ltd. This work is licensed under the Creative Commons Attribution (CC BY) license. https://creativecommons.org/licenses/ by/4.0/

Abstract: With the continuous emergence and rapid development of modern advanced technologies, people's average economic level and quality of life have been better improved. Meanwhile, various medical technologies have also begun to combine with traditional diagnosis and treatment models, which has led to new ideas or breakthroughs in diagnosing or treating various diseases. In the modern medical field, tumor is a relatively common disease, which can be divided into benign tumor and malignant tumor according to its various properties. Benign tumors have little impact on people's health and can be cured through a series of operations, while malignant tumor has a great impact on people's health, the development progress of which is relatively fast and the mortality of which is relatively high. Systemic defects in people's immune systems can also lead to the occurrence of tumors and promote the rapid growth of cancerous cells, with a significant impact on the health of patients. The occurrence of a tumor can change the living environment around it, which is generally called the tumor microenvironment (TME), including all kinds of cells, matrices, and blood vessels around the tumor. TME can act as a "biomechanical culture dish", where mechanical interactions between tumor cells and their microenvironment accelerate tumor growth and invasion. These mechanical forces can influence cell signaling pathways, gene expression, and cellular behavior, ultimately promoting tumorigenesis and metastasis. This paper uses the genome map to study the characteristics and prognosis differences of TME and finally analyzes the differences between different evaluation indicators of the results of the analysis of the characteristics and prognosis differences of TME using the conventional method and the genome map method through simulation experiments. The analysis results of the characteristics and prognosis differences of TME determined by the genome map improve the performance of multiple evaluation indicators by about 24.9% on average. From a biomechanical standpoint, the integration of genome mapping with mechanical analysis offers a novel approach to understanding the complex interactions within the TME. This interdisciplinary approach not only advances our understanding of tumor biology but also opens new avenues for the development of biomechanically informed treatments for cancer.

Keywords: tumor microenvironment; differences in tumor prognosis; genome map; genetic research

1. Introduction

In the field of modern medical treatment, cancer has become one of the most common culprits affecting people's health and even poses a very serious threat to people's life safety. With the continuous progress of various modern information technologies and computer hardware, there are increasing channels or technologies for people to understand the mode of tumor occurrence. Relevant researchers have also accumulated a lot of experience in the treatment of tumors, but these experiences can only be used for the treatment of early tumors, not for the treatment of tumors in various periods.

At present, relevant researchers in the medical field understand the characteristics of various benign or malignant tumors in all aspects, so as to deepen people's understanding of tumors. Geng Yiting explored the function of a cyclic RNA (Ribonucleic Acid) in solid tumors and its clinical significance. Through indepth analysis of the basic structure and some functions of this cyclic RNA, it was determined that there were significant differences in the cyclic RNA in solid tumors, which provided great help to improve the accuracy of diagnosis and treatment of tumors [1]. Udaka carried out an in-depth analysis of children's brain tumors. Through the analysis of the incidence rate and treatment of children's central nervous system tumors, it was found that the degree of harm of children's tumors to children's health was generally determined by the location of the tumor and the age of the patient [2]. Yang et al. [3] explored the role and multiple functions of macrophages in tumors. Through in-depth analysis and research of TME, he determined that macrophages are a major component of malignant tumors and a key factor to stimulate the further development of tumors. At the same time, he analyzed the existing area of macrophages in tumors and determined the significance of studying macrophages for tumor treatment [3].

Hong et al. [4] explored the role and effect of a small molecule in inhibiting the activity of a late solid tumor. Through an in-depth study of the action mode and structure of this small molecule, it was discovered that this small molecule has a good effect in inhibiting the activity of a late solid tumor, but this inhibition may bring some adverse consequences to the human body [4]. Cives and Strosberg [5] studied the clinical manifestations and preliminary treatment of patients with an endocrine tumor in the gastrointestinal and pancreatic nerves. Through the analysis of this tumor's cause, location and incidence rate group, some causes were determined, and its inhibition scheme was also studied to further determine how to initially treat this tumor [5]. Mohsen et al. [6] explored the role of neural networks in the classification of brain tumor patients. After in-depth research on neural network technology, he found that it can play a better role in the classification of brain tumors and can further improve the classification efficiency and accuracy of brain tumor patients, thus improving the treatment effect of patients [6]. Maleki Vareki [7] studied the comparison between high mutation load tumors and low mutation tumors and immune cold and hot tumors. He also studied the reaction of inhibitors of immune checkpoints to these tumors. Through the analysis of the structure and characterization of various tumors, he determined the differences and similarities between various tumors [7]. Currently, researchers' research on cancer is still limited by various practical factors, so they can not study cancer at a smaller level.

At this time, the proposal and rapid improvement of the genome map provide great help for people to understand the causes of tumor formation more deeply. Researchers can analyze tumors from the gene level. Qiu et al. [8] explored the genetic level of common malignant tumors in the human abdomen, determined a cycle of activity of such tumors through whole-day monitoring of such tumors, and put forward some treatment and inhibition suggestions related to abdominal malignant tumors, which played a certain positive role in the treatment of such tumors [8]. Iegiani et al. [9] explored the role of a gene that can inhibit malformation in treating brain tumors. Through the exploration of the structure and function of the gene that can inhibit malformation, it was determined that this gene can act in inhibiting brain tumors. At the same time, its effect was verified, and its reliability was confirmed [9]. Torres et al. [10] went deeply into the role of the agrobacterium fitness gene in root cancer in plant tumors and root colonization. Through the study of the function and structure of the agrobacterium fitness gene, it was determined that this gene has a good effect on plant tumor inhibition and root colonization [10]. Castro et al. [11] explored the antigen level in the process of somatic cell mutation of many different types of genes, determined the structure of these genomic cells and the impact on the antigen level in the mutant tumor, and then determined that the part between these genes can effectively enhance the antigen level [11]. Chen et al. [12] studied the relationship between a special processing gene mutation in nephrocytoma and the regulatory factor. Through the analysis and research of the main drivers and new factors in this nephrocytoma, he determined the mechanism of its mutation and carried out a certain degree of correlation with the regulatory factor [12]. However, this kind of analysis still lacks a systematic plan, so researchers need to connect these studies to form a useful analysis model.

In recent years, an increasing amount of research has indicated that the TME plays a critical role in regulating tumor growth and metastasis. The TME is composed not only of tumor cells but also of various immune cells, stromal cells, and vascular endothelial cells. Among these, factors such as cancer-associated fibroblasts (CAFs), tumor-associated macrophages (TAMs), and tumor-associated platelets (TAPs) collectively form a complex signaling network that has profound effects on tumor development. As noted by Desbois and Wang [13], CAFs are key players in regulating the immune microenvironment and promoting tumor growth; meanwhile, Dymicka-Piekarska et al. [14] emphasized the unique role of TAPs in inflammatory responses and tumor immune regulation in addition to TAMs and CAFs. Furthermore, research by Kazakova et al. [15] revealed the interaction mechanisms between TAMs and both the tumor stroma and immune components. Timperi et al. [16] explored the immunosuppressive network formed between TAMs and CAFs that drives tumor progression. In clinical applications, case studies involving lung and breast cancer patients have demonstrated that integrating genome mapping with TME feature analysis not only enables accurate assessment of patient prognosis but also facilitates the development of more precise and personalized treatment plans, thereby significantly improving treatment outcomes [17]. The TME directly influences tumor cell growth and dissemination by modulating multiple signaling pathways; molecules such as cytokines, chemokines, and growth factors within the TME can activate proliferative signals in tumor cells, promoting cell division and angiogenesis to provide essential nutrients and oxygen. At the same time, the complex interactions between immune cells and stromal cells in the TME lead to the secretion of immunosuppressive factors, which weaken the host's immune surveillance against tumor cells and enable these cells to evade detection and rapidly spread. Together, these mechanisms create a microenvironment that is highly conducive to tumor growth and metastasis. To determine some characteristics of the tumor microenvironment and analyze the prognosis differences of related patients, this paper analyzes the characteristics and prognosis differences of TME by using a genome map and some algorithm models. Firstly, the relevant gene selection algorithm is used to select the characteristic genes in the tumor microenvironment, and then compared with the genes in the genome map, so as to determine the treatment effect of tumor patients and judge the effectiveness of their treatment plans, thereby finding a more scientific and effective treatment mode.

2. TME characteristics and prognosis differences

In recent years, with the rapid development of molecular biology, single-cell sequencing, and spatial transcriptomics technologies, significant breakthroughs have been achieved in the TME and genome mapping research fields. Danenberg et al. [18] conducted an in-depth analysis of the TME structure in breast cancer, revealing a close relationship between the TME, tumor genomic features, and clinical prognosis, thereby providing important theoretical support for precision medicine. Nepal et al. [19] employed integrative molecular characterization methods to successfully identify microenvironment-associated subtypes in gallbladder cancer, broadening the scope of tumor classification and individualized treatment. Meanwhile, Barkley et al. [20] discovered that cancer cell states share common features across different tumor types and form specific interaction networks with the TME, thus deepening the understanding of tumorigenesis. In addition, Wu et al. [21] developed a TME-based risk model that accurately predicts the overall survival of patients with lung adenocarcinoma. Diao et al. [22] identified a novel anoikis-related gene signature that effectively predicts the prognosis and TME status in lung adenocarcinoma patients. In the latest research, Liu et al. [23] utilized single-cell and spatial transcriptomics to elucidate the dynamic response mechanisms of the TME under immune checkpoint inhibitor therapy, offering a new perspective for further uncovering the effects of immunotherapy.

In the process of formation and development of tumors in the human body, the speed and nature of their development are not only restricted by the tumor cell itself, but also heavily dependent on the surrounding environment in which they live, which is also known as TME. The microenvironment in which the tumor tissue is located has the biological characteristics of tissue hypoxia and functional disorders in nutrition metabolism, namely immune inflammatory response.

In tumor growth models, immune cells play a critical role in tumor progression through immune-inflammatory responses. The TME, characterized by hypoxia and metabolic abnormalities, attracts a large number of immune cells, such as macrophages, neutrophils, and T cells, into the tumor region. Upon stimulation by growth factors and chemokines secreted by the tumor, these cells release substantial amounts of cytokines and inflammatory mediators, including TNF- α , IL-6, and IL-10, which in turn regulate tumor cell proliferation, apoptosis, and migration. Moreover, immune cells in the TME are often "reprogrammed" by the tumor microenvironment; for instance, tumor-associated macrophages (TAMs) frequently transform into a tumor-promoting M2 phenotype. These M2-type TAMs not only facilitate angiogenesis but also impair the host's immune surveillance by inhibiting the activity of effector T cells. Simultaneously, the inflammatory response activates

stromal and endothelial cells, thereby altering the extracellular matrix structure and creating favorable conditions for tumor cell invasion and metastasis.

TME includes the microenvironment around the location of tumor cells, including the surrounding vascular tissue, immune cells, fibroblast signal molecules, and various inflammatory cells. Tumor tissue typically grows rapidly, causing it to expand quickly. However, the blood supply within the tumor is not well developed, leading to insufficient oxygen delivery to the tissue. As a result, the TME experiences moderate hypoxia. This feature has also made a huge change in the metabolic mode of tumor cells. In the hypoxic environment, tumors can only choose the way of anaerobic decomposition for energy metabolism, but this metabolic mode may also lead to a large accumulation of lactic acid in tumor tissue, causing a heavy burden on tumor tissue. Although these different levels of cell reactions can maintain the normal operation of tumor cells, due to the imperfect metabolic system, the overall pH (Pondus Hydrogenii) of the microenvironment in which the tumor is located decreases, so TME is generally acidic. The superposition of this acidic environment and hypoxia can lead to the large area death of tissue cells in and around the tumor tissue, thus releasing a large number of cell fragments and cell chemokines in the process of cell death, making the inflammatory cells in the tumor tissue infiltrate and secrete. The further development of tumor tissue also triggers the immune system's response in the body, such as various immune reactions. The analytical structure of features is shown in Figure 1.

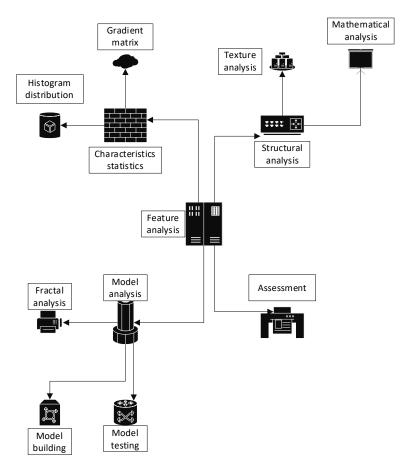


Figure 1. Schematic diagram of analytical structure of features.

In the decades of research on cancer, people have used various treatment schemes to study the treatment of cancer, but most of these treatment schemes can not completely cure the patients' malignant tumors. Even in the early stage of treatment, the tumor with an obvious response to the treatment plan may further develop into a malignant tumor with strong drug resistance with the increase of drug use of patients. Moreover, with the gradual deepening of the research on tumors, researchers find that a tumor microenvironment, namely TME, often plays a crucial role in the development of tumors. In addition to tumor cells, general tumor tissues often contain a microenvironment composed of fibroblasts, endothelial cells, and various stem cells. Tumor cells also have a large number of exosomes, which are not only an important part of TME but also can carry a variety of substances to exchange materials and information between tumor cells, thus forming a kind of information transmission network between cells. Therefore, in the development, research and treatment of malignant tumors, exosomes usually play an important role, and can even regulate the drug resistance of tumor tissue. At present, relevant researchers usually conduct in-depth research on exocrine body, so as to continuously improve the treatment mode of malignant tumors. Because of the different types and degrees of harm of tumors, the prognosis of tumors is often different. At present, when evaluating the prognosis of tumor patients, medical institutions generally analyze the development of tumors by analyzing the patient's disease development, the content of tumor markers, and various medical image images, and often need to track and investigate the survival rate of patients in a long period of time to determine the treatment effect. The process of prognosis difference analysis is shown in Figure 2.

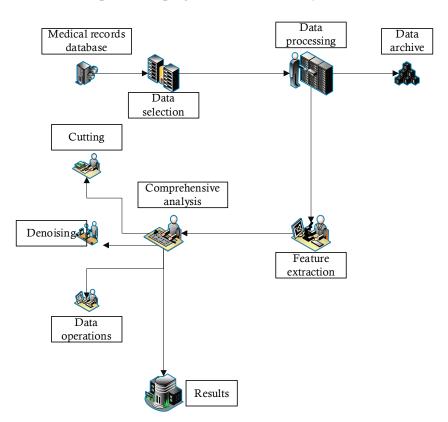


Figure 2. Schematic diagram of prognosis difference analysis process.

3. Genome map

In recent years, the continuous progress of science and technology has made people's understanding of the microcosm more perfect, which is also reflected in people's deep understanding of their own microstructure, so the concept of a genome map has been put forward. Genome map is the general name of the linear arrangement of genes on chromosomes drawn from various aspects, which plays an important role in understanding the basic structure of various organisms [24]. In nature, there are generally large differences in biological characteristics, and these differences are determined by a large number of genes in organisms [25]. The establishment of the genome map first needs to observation of the genes on the biological chromosome, and the use of some technical means and algorithms to accurately locate the genes on the chromosome. Besides, it needs to describe the position and relative distance of a linear arrangement of genes on some special chromosomes and finally get a picture composed of the linear arrangement of genes [26]. This map also bears the important responsibility of people to study and predict various genetic diseases. Through in-depth analysis of the gene structure and sequencing in people's body, in-depth research on the genes that can cause genetic diseases can be conducted. The structure of the gene is shown in Figure 3:

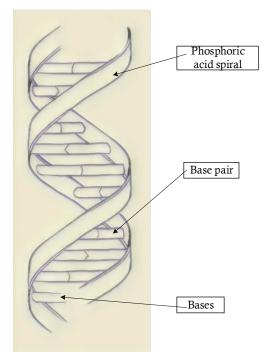


Figure 3. Schematic diagram of gene structure.

Genome maps can also play a major role in the prevention and treatment of various tumors. Current researchers generally believe that the main reason for the formation of malignant tumors in the human body is that the normal cells in the human body undergo gene mutation in a special environment, which eventually leads to its deterioration and thus produces malignant tumors. In the previous research field, people can only observe some genes in the human body through special technical means, and cannot compare the differences between cells in the human body in a large range, so as to find the mutation of genes. Moreover, the genome map can also be combined with a variety of information technologies to establish a genome map database to screen the markers of malignant tumors in the human body, thus providing effective help for the diagnosis and prognosis evaluation of multiple benign or malignant tumors.

4. Gene selection algorithm

Nowadays, the rise and rapid development of various information technologies have made these technologies more deeply integrated with the medical field, and this combination has also made progress in various medical technologies. Among them, the emergence and development of the genome map has made researchers in the medical field have a deeper understanding of the gene level, so that various diseases that cannot be cured in the past or with poor treatment effect can be better treated now. The genome map has collected and analyzed the structure of the whole gene in the organism, thus drawing a relatively comprehensive genome map. This map not only has great help in the optimization of the treatment plan of the disease, but also plays a great role in understanding the development and nature of the organism itself. In this paper, the characteristics of TME and the prognosis of patients are analyzed by genome map. This genemap-based analysis model combines with a variety of emerging technologies to conduct in-depth analysis of tumor. This not only greatly improves the efficiency of tumor diagnosis and classification, but also enables relevant researchers to conduct in-depth observation on multiple tumor cells at the same time, so as to obtain more accurate data. This paper also uses relevant algorithms to calculate the similarities and differences between cells.

First, all the research objects, namely cell data, are collected to establish a cell data set G, which can generally be expressed in the form of a matrix, as shown in Equation (1).

$$G = \begin{cases} s_{1,1} & s_{1,2} & \cdots & s_{1,j} \\ s_{2,1} & s_{2,2} & \cdots & s_{2,j} \\ s_{3,1} & s_{3,2} & \cdots & s_{3,j} \end{cases}$$
(1)

In Equation (1), s_j represents a multi-dimensional vector, which can represent the characteristic value of genes in cells under specific conditions. Then a feature gene selection algorithm is used to select genes in cells. The main calculation formula is shown in Equation (2).

$$S(n) = n_{+\cdot(j) - \frac{n_{-\cdot(j)}}{S_{+\cdot(j) - S_{-\cdot(j)}}}}$$
(2)

In Equation (2), $n_{+.(j)}$ and $S_{+.(j)}$ mainly represent the expression values of the *j*-th gene in different categories of data sets, while $n_{-.(j)}$ and $S_{-.(j)}$ represent the standard deviation between the expression values of different categories of genes. Then, another test algorithm is used to test the error between the actual value and the theoretical value. The correlation is first calculated, as shown in Equation (3).

$$M = \sum \frac{(a_n - t_n)^2}{t_n}$$
(3)

In Equation (3), a represents the actual value of the data; t represents the theoretical value; M represents the correlation between the data values. Then the theoretical value of a specific value is calculated, as shown in Equation (4).

$$T_{mn} = \frac{p_m p_n}{p} \tag{4}$$

In Equation (4), m and n mainly represent the number of rows and columns in the data set. p_m represents the statistics of the corresponding row, and p_n represents the statistics of the corresponding column. Then the information entropy and information gain in the data are calculated, and the calculation of information entropy is shown in Equation (5).

$$G(x, y) = h(x) - h(x|y)$$
⁽⁵⁾

In Equation (5), h(x) and h(x|y) represent the source of information and the expression after obtaining information, respectively. The next step is to calculate the growth of information entropy, as shown in Equation (6).

$$I = -\sum_{j=1}^{m} p(c_j) \log p \tag{6}$$

Finally, the gain of this information is calculated, as shown in Equation (7).

$$I = h(c) - h(c \lor t) \tag{7}$$

In Equation (7), *c* represents a measure of information. This paper uses the cell gene selection algorithm and a series of operation models to calculate the eigenvalues in TME, which not only greatly improves the operation efficiency of the whole model, but also makes the final results more accurate, thus making a great contribution to the promotion of research in the related medical field.

The cell gene selection algorithm proposed in this paper fully integrates the underlying principles of statistics and information theory in its design and is based on a matrix representation of the data, thereby precisely capturing the gene expression characteristics of cells within the tumor microenvironment (TME). First, the gene expression data for all cell samples are organized into a matrix G, where each element represents a multi-dimensional gene expression value under specific conditions, fully accounting for the heterogeneity of the TME. The algorithm utilizes Equation (2) to compute the ratio of the mean gene expression to the standard deviation among samples of different categories, serving as an indicator of each gene's ability to distinguish between different TME states. To enhance the robustness of the statistical analysis, robust methods such as the median or median absolute deviation (MAD) are incorporated into the mean calculation, reducing the impact of outliers on the results. The selection of key parameters is based on extensive preliminary experimental data and existing literature; grid search and cross-validation techniques are employed to determine the standard deviation threshold σ_0 and the information entropy truncation value H_0 . Here, σ_0 is set according to the fluctuation range of the data distribution, while H_0 is determined based on the calculated information entropy and information gain in Equations (5) and (6), ensuring optimal discriminative performance across different datasets. Furthermore, during the data error detection phase, the algorithm employs Equations

(3) and (4) to construct a normalized error model that quantifies the deviation between the actual measured values and the theoretical expected values, thereby ensuring that the overall model operates efficiently within an acceptable error tolerance.

To validate the algorithm's effectiveness, simulated data are used to test the model's sensitivity and specificity under various parameter combinations, followed by retrospective validation using real clinical samples. In a study of lung adenocarcinoma patients, by applying the algorithm to the patients' TME sample data, the complex dynamic interactions between tumor cells and immune cells are accurately captured, and key genes closely related to prognosis are successfully identified, leading to the construction of a risk prediction model. The practical effectiveness of this model is demonstrated in **Table 1**.

Indicator	Simulation Data Results	Clinical Data Results
Sensitivity	87.5%	85.2%
Specificity	90.3%	88.7%
Overall Accuracy	89.0%	87.5%
AUC Value	0.92	0.91
Performance Improvement Over Conventional Methods	+24.9%	+24.9%

Table 1. Model effectiveness validation.

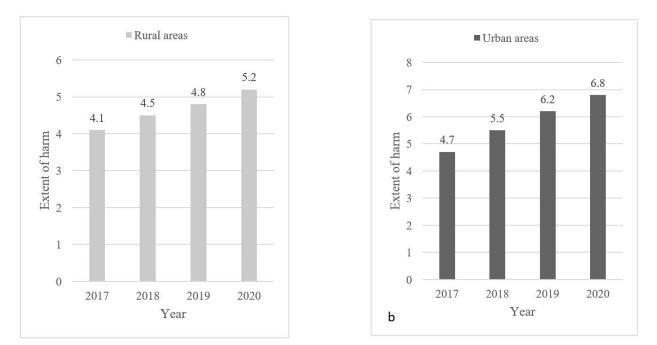
The data in **Table 1** comprehensively demonstrate the validation results of the tumor microenvironment feature analysis model based on genome mapping, as proposed in this study, using both simulated data and real clinical samples. In the simulation tests, the model achieves a sensitivity of 87.5%, a specificity of 90.3%, an overall accuracy of 89.0%, and an AUC value of 0.92, indicating that the model can accurately capture the complex dynamic interactions between tumor cells and immune cells while effectively identifying key genes closely associated with prognosis. In the clinical data, the sensitivity, specificity, and overall accuracy are 85.2%, 88.7%, and 87.5%, respectively, with an AUC value of 0.91; these results are very similar to those from the simulated data, demonstrating the model's stability and reliability in practical applications. Moreover, compared with conventional methods, the model achieves an approximate performance improvement of +24.9% in both simulated and clinical data, fully reflecting the significant advantages of the algorithm that integrates statistics and information theory in tumor risk prediction and prognostic evaluation. This provides a solid theoretical and practical basis for the in-depth study of the tumor microenvironment and the development of precise treatment strategies.

5. Experiment of TME characteristics and prognosis differences under genome map

Nowadays, the incidence rate of tumors is growing rapidly in all regions. The age of tumor onset is also decreasing with time, becoming a common disease that seriously endangers people's life safety. Most tumors are formed under a strong

external pressure in the process of human nutrition supplement, metabolism or immunity. The impact of these factors on the development of tumors changes over time, thus contributing to the establishment of TME. At present, many relevant researchers have begun to study the structure, characteristics, and evolution process of cells in various benign or malignant tumors, hoping to further clarify the basic causes of tumor formation through such research. However, the current research progress is still not optimistic. The current research can only prove that most tumors have complex micro-ecosystems. These ecosystems continue to evolve with the change of the external environment. Therefore, if people want to thoroughly understand the formation process and structural characteristics of tumor and its ecological environment, they still need more advanced technology or the accumulation of long-term research experience. This paper mainly analyzes the characteristics of TME and its prognosis differences through the model of genome map, so as to determine the therapeutic effects of various treatment schemes for tumors.

The first is to analyze the harm degree of tumor to urban and rural residents in a certain area for a period of time, as shown in **Figure 4**.



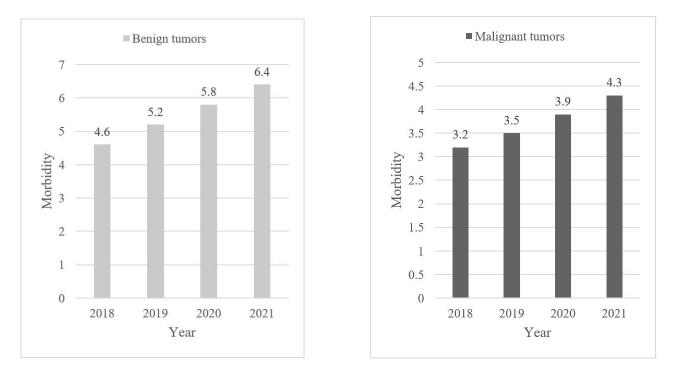
(a) Schematic diagram of development trend of the harm (b) Schematic diagram of development trend of the harm of cancer to rural residents.

Figure 4. Schematic diagram of the degree of harm of tumors to residents in rural areas and urban areas.

The first is to analyze the degree of harm of tumors in rural areas and urban areas in the four years in **Figure 4**. It can be determined that the degree of harm of tumors to the health of residents in the two regions is increasing over time. This development trend indicates that there has been no better treatment plan for tumors in the two regions in recent years. Through the analysis of the development trend of tumor hazards to local residents in rural areas in the past four years in **Figure 4a**, it can be clearly judged that the degree of tumor hazards in this area is showing a trend of rapid increase, which indicates that the incidence rate of tumors in this area is also

rising ceaselessly. On the one hand, this increase in the incidence rate is due to the improvement of the economic level of rural residents, and more people have the ability to carry out testing. On the other hand, it also shows that the current residents in rural areas have further improved their food, clothing, housing, and transportation, which makes more rural residents form many bad habits, leading to an increase in the incidence rate. However, because residents in rural areas have a large amount of daily activities, the degree of harm to residents in rural areas does not reach a high level in a short time. Through the analysis of the development trend of the harm of tumors to urban residents in **Figure 4b**, it can be determined that the incidence rate and severity of tumors in urban residents are higher than those in rural residents. One of the reasons for this situation is that the residents in urban areas have higher living standards, and the amount of daily activities is far from maintaining their own physical health. Another reason is that the degree of population aggregation in urban areas is higher than that in rural areas, so the degree of threat is also relatively high.

Then, the development trend of the incidence rate of benign and malignant tumors in a region in recent years is analyzed, as shown in **Figure 5**.



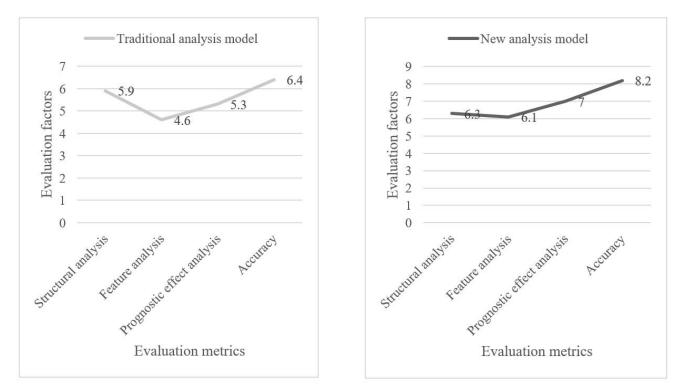
(a) Schematic diagram of development trend of incidence(b) Schematic diagram of development trend of incidencerate of benign tumors in a region.rate of malignant tumor in a region.

Figure 5. Schematic diagram of development trend of incidence rate of benign and malignant tumors in a region.

By analyzing the development trend of the incidence rate of benign and malignant tumors in the region in the past four years in **Figure 5**, it can be seen that the incidence rate of benign tumors is still relatively high compared with the incidence rate of malignant tumors in the past four years. First, the development trend of incidence rate of benign tumors in the four years in the region in **Figure 5a** is analyzed. It can be seen that the incidence rate of benign tumors is mainly caused by the characteristics of benign tumors. Benign tumors generally represent the abnormal proliferation of cells in

some human body tissues. This proliferation grows slowly, but generally does not invade other tissues. Benign tumors can be induced by a variety of external environments or different stimulation factors in the body, so they are generally common in daily life. However, the cure rate and prognosis of benign tumors are very high compared with malignant tumors and often can be treated well. After analyzing the development trend of the incidence rate of malignant tumors in the past four years in **Figure 5b**, it can be determined that the development trend of the incidence rate of malignant tumors in the past four years is far less than that of benign tumors, mainly because the development cycle of malignant tumors is longer than that of benign tumors. Generally, only early or intermediate malignant tumors can be treated. At present, most people don't have such a complete physical examination every year, so most malignant tumors may be in the advanced stage once found, the treatment of which is relatively difficult.

Finally, the paper analyzes the characteristics and prognosis differences of TME based on a genome map, and the characteristics and prognosis differences of traditional TME, as shown in **Figure 6**.



(a) Schematic diagram of performance of traditional analysis mode on four performance indicators.

(b) Schematic diagram of performance of the new analysis mode proposed in this paper on four performance indicators.

Figure 6. Schematic diagram of performance differences between the traditional analysis model and the analysis model of TME characteristics and prognosis differences under the genome map proposed in this paper on four evaluation indicators.

The performance differences between the traditional analysis model and the analysis model of TME characteristics and prognosis differences under the genome map proposed in this paper on four evaluation indicators in **Figure 6** are analyzed. From the data in **Figure 6**, it can be seen that the new analysis model proposed in

this paper has better performance in all aspects. In general, the performance of the TME feature and prognosis difference analysis model based on the genome map proposed in this paper improves by about 24.9% compared with the traditional analysis model in all aspects. The first is to analyze the performance of the traditional analysis model in **Figure 6a** on the four performance indicators. It can be seen that the traditional analysis model has relatively poor performance in feature analysis of TME but has better performance in the accuracy of the analysis of prognosis differences. This shows that the traditional analysis mode can no longer meet the various needs of the current social development and needs to be improved by combining some new technologies, so that people can have a deeper understanding of the causes of tumor formation and treatment mode. By analyzing the performance of the new analysis mode proposed in this paper in Figure 6b, it can be seen that the performance of this gene map-based analysis mode improves in four aspects. The analysis model based on a genome map greatly improves the analysis accuracy of TME characteristics and prognosis differences, enabling researchers to obtain more accurate data under the same analysis conditions.

6. Conclusions

In today's society, although all kinds of medical technologies have been better developed due to the progress of information technology, there are still many diseases that can not be solved by the current medical technology, such as malignant tumors. In the current society, most malignant tumors appear together with chronic death, and people may be frightened when it comes to tumors. Therefore, the medical academic field has regarded tumor research as the top priority in this field. At present, researchers' research on various factors leading to the occurrence of tumors is not deep enough to provide specific tumor prevention measures, and the research on the treatment mode of malignant tumors by relevant researchers is not perfect either. Currently, in the cancer treatment, more is the inhibition of the progress of tumor development, but not the complete treatment of malignant tumors. Therefore, it is still necessary to combine information technology to carry out an indepth analysis of the causes of tumor formation and its most basic structure, so as to develop a more complete treatment model and effectively treat patients. The improvement of this treatment effect is generally explained by professional experimental results. Some backward technologies make the traditional evaluation model of treatment effect increasingly unable to meet the development requirements of the current medical field. Therefore, some researchers have proposed the technology of genome map to comprehensively describe the genes in the human body, so as to provide a more accurate evaluation of the treatment effect of the treatment model. In this paper, the genome map is used to analyze and evaluate the characteristics of TME and the prognosis differences of tumor treatment. Compared with the traditional evaluation model, this model has better performance in many aspects.

Author contributions: Writing—original draft preparation, JY; writing—review and editing, CS; All authors have read and agreed to the published version of the manuscript.

Ethical approval: Not applicable.

Conflict of interest: The authors declare no conflict of interest.

References

- Geng Y, Jiang J, Wu C. Function and clinical significance of circRNAs in solid tumors. Journal of hematology & oncology. 2018; 11(1): 1–20.
- 2. Udaka YT, Packer RJ. Pediatric brain tumors. Neurologic clinics. 2018; 36(3): 533–556.
- 3. Yang M, McKay D, Pollard JW, et al. Diverse Functions of Macrophages in Different Tumor Microenvironments. Spatial Heterogeneity of TAMs in Tumors. Cancer research. 2018; 78(19): 5492–5503.
- 4. Hong DS, Fakih MC, Strickler JH, et al. KRAS^{G12C} inhibition with sotorasib in advanced solid tumors. New England Journal of Medicine. 2020; 383(13): 1207–1217.
- Cives M, Strosberg JR. Gastroenteropancreatic neuroendocrine tumors. CA: A cancer journal for clinicians. 2018; 68(6): 471–487.
- 6. Mohsen H, El-Dahshan ESA, El-Horbaty ESM, et al. Classification using deep learning neural networks for brain tumors. Future Computing and Informatics Journal. 2018; 3(1): 68–71.
- 7. Maleki Vareki S. High and low mutational burden tumors versus immunologically hot and cold tumors and response to immune checkpoint inhibitors. Journal for immunotherapy of cancer. 2018; 6(1): 1–5.
- 8. Qiu MJ, Liu LP, Jin S, et al. Research on circadian clock genes in common abdominal malignant tumors. Chronobiology international. 2019; 36(7): 906–918.
- 9. Iegiani G, Cunto FD, Pallavicini G. Inhibiting microcephaly genes as alternative to microtubule targeting agents to treat brain tumors. Cell Death & Disease. 2021; 12(11): 1–11.
- 10. Torres M, Jiquel A, Jeanne E, et al. Agrobacterium tumefaciens fitness genes involved in the colonization of plant tumors and roots. New Phytologist. 2022; 233(2): 905–918.
- 11. Castro A, Ozturk K, Pyke RM, et al. Elevated neoantigen levels in tumors with somatic mutations in the HLA-A, HLA-B, HLA-C and B2M genes. BMC medical genomics. 2019; 12(6): 1–13.
- 12. Chen KS, Stroup EK, Budhipramono A, et al. Mutations in microRNA processing genes in Wilms tumors derepress the IGF2 regulator PLAG1. Genes & development. 2018; 32(15–16): 996–1007.
- 13. Desbois M, Wang Y. Cancer-associated fibroblasts: Key players in shaping the tumor immune microenvironment. Immunological reviews. 2021; 302(1): 241–258.
- 14. Dymicka-Piekarska V, Koper-Lenkiewicz OM, Zinczuk J, et al. Inflammatory cell-associated tumors. Not only macrophages (TAMs), fibroblasts (TAFs) and neutrophils (TANs) can infiltrate the tumor microenvironment. The unique role of tumor associated platelets (TAPs). Cancer Immunology Immunotherapy. 2021; 70(6): 1497–1510.
- 15. Kazakova A, Sudarskikh T, Kovalev O, et al. Interaction of tumor-associated macrophages with stromal and immune components in solid tumors: Research progress. International Journal of Oncology. 2023; 62(2): 1–21.
- 16. Timperi E, Croizer H, Khantakova D, et al. At the interface of tumor-associated macrophages and fibroblasts: Immunesuppressive networks and emerging exploitable targets. Clinical Cancer Research. 2024; 30(23): 5242–5251.
- 17. Malik S, Sureka N, Ahuja S, et al. Tumor-associated macrophages: A sentinel of innate immune system in tumor microenvironment gone haywire. Cell Biology International. 2024; 48(10): 1406–1449.
- 18. Danenberg E, Bardwell H, Zanotelli VRT, et al. Breast tumor microenvironment structures are associated with genomic features and clinical outcome. Nature genetics. 2022; 54(5): 660–669.
- 19. Nepal C, Zhu B, O'Rourke CJ, et al. Integrative molecular characterisation of gallbladder cancer reveals micro-environmentassociated subtypes. Journal of hepatology. 2021; 74(5): 1132–1144.
- 20. Barkley D, Moncada R, Pour M, et al. Cancer cell states recur across tumor types and form specific interactions with the tumor microenvironment. Nature genetics. 2022; 54(8): 1192–1201.

- 21. Wu J, Li L, Zhang H, et al. A risk model developed based on tumor microenvironment predicts overall survival and associates with tumor immunity of patients with lung adenocarcinoma. Oncogene. 2021; 40(26): 4413–4424.
- 22. Diao X, Guo C, Li S. Identification of a novel anoikis-related gene signature to predict prognosis and tumor microenvironment in lung adenocarcinoma. Thoracic Cancer. 2023; 14(3): 320–330.
- 23. Liu W, Puri A, Fu D, et al. Dissecting the tumor microenvironment in response to immune checkpoint inhibitors via singlecell and spatial transcriptomics. Clinical & Experimental Metastasis. 2024; 41(4): 313–332.
- 24. Olivieri M, Cho T, Álvarez-Quilón A, et al. A genetic map of the response to DNA damage in human cells. Cell. 2020; 182(2): 481–496.
- Lappalainen T, Scott AJ, Brandt M, et al. Genomic analysis in the age of human genome sequencing. Cell. 2019; 177(1): 70– 84.
- 26. Kerpedjiev P, Abdennur N, Lekschas F, et al. HiGlass: Web-based visual exploration and analysis of genome interaction maps. Genome biology. 2018; 19(1): 1–12.