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# A Molecular Mechanism-Based Approach: Differences in the Roles of Berberine, Quercetin and Baicalin in the Treatment of Breast Cancer

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## ABSTRACT

Breast cancer is considered one of the most prevalent types of malignant tumours among women worldwide, and recent academic studies have revealed that natural products highlight a key role in the intervention and treatment of breast cancer. The present study focused on three typical natural active compounds, berberine, quercetin and baicalin. It investigates their molecular pathways and functional heterogeneity in the field of breast cancer therapy, further compares the characteristics of the molecular mechanisms of each of the three compounds and their differences in the treatment of breast cancer, and at the same time deeply discusses their potential application value in combination therapy strategies as well as their future development trends in the field of breast cancer therapy.

## KEYWORDS

Breast cancer, Berberine, Quercetin, Baicalin, Signalling pathway, Molecular mechanism

## 1. INTRODUCTION

Breast cancer ranks as one of the most common malignant cancers in women, characterized by a high death rate and a complex progression. According to statistics, about 2.3 million people worldwide will be diagnosed with breast cancer in 2020, and the number of people who died of the disease in the same year will be close to 700,000, making it the leading cause of cancer deaths among women, with the morbidity and mortality rates continuing to rise [1]. In recent years, traditional Chinese medicines and their active ingredients have opened up new research directions for the prevention and treatment of breast cancer due to their multi-target, multi-level and multi-pathway therapeutic mechanisms. Considerable attention has been focused on three active ingredients of traditional Chinese medicine: berberine, quercetin and baicalin for the past few years. A series of rigorous and exhaustive studies have been conducted on the prospective mechanisms of berberine, quercetin and baicalin in the treatment of breast cancer, as well as their possible synergistic effects, and comparison of the characteristics and differences in the effects of these three components, not only help to deepen our understanding of the mechanism of action of a single chemical substance of traditional Chinese medicine in the treatment of breast cancer, but also provide theoretical support for the optimisation of clinical therapeutic regimens.

## 2. PATHOPHYSIOLOGICAL FEATURES OF BREAST CANCER

The pathogenesis of breast cancer is complex and affected by a variety of factors such as heredity and endocrinology, and through research into the causes of breast cancer, it has been found that hereditary

factors play an important role. Data show that about 5 to 10 per cent of patients are genetically predisposed. Frequently, these instances correlate with alterations in cancer-inhibiting genes like BRCA1 and BRCA2, substantially elevating the likelihood of breast cancer in women. Women who carry a BRCA1 mutation have an approximately 72 per cent lifetime chance of developing breast cancer, while women with a BRCA2 gene variant have a nearly 69 per cent risk of developing the disease [2]. These genes are primarily responsible for DNA damage repair while maintaining genomic stability. After a mutation occurs, DNA damage accumulates in the cell. The process of cancer formation is accelerated by the accumulation of these damages. Studies have shown that the PI3K/Akt/mTOR signalling pathway, which is abnormally activated from time to time, is an important influence on the development of breast cancer, and that this pathway is important for the regulation of cell growth, differentiation and metabolism. In breast cancer, mutations in genes such as PIK3CA are more common, with an incidence of about 30%. Such mutations lead to overactivation of the PI3K enzyme [3].

The tumour microenvironment is crucial in the pathogenesis of breast cancer, with the presence of tumour cells in the environment, as well as paracrine factors. As an illustration, tumor cells emit growth factors, alongside stromal and immune cells. Tumour-associated macrophages (TAMs) have been found to infiltrate a high proportion of breast cancers and are significantly pro-tumourigenic. TAMs secrete factors such as IL-10, VEGF and MMP, which reduce anti-tumour immunity. They also promote neovascularisation and enhance the invasive capacity of tumour cells [4].

# 3. REFLECTING ON BERBERINE'S CONTRIBUTION TO BREAST CANCER THERAPY

## 3.1. Molecular Structure and Biological Activity of Berberine

In breast cancer therapy, berberine induces apoptosis, inhibits cell proliferation and alters the tumour microenvironment. Berberine concentration at 10-50 micromolar significantly reduces the survival of breast cancer MDA-MB-231 cells [5]. The growth of breast cancer cells is suppressed via the MAPK/ERK signaling route. At the same time, it comprehensively controls tumour growth, invasion as well as metastasis by adjusting STAT3, Wnt & β-catenin pathways. In recent years, it has been found that AMPK (activated protein kinase) is closely related to berberine. Berberine can strongly activate AMPK, improve cell metabolism and energy status, and then inhibit the growth of breast cancer cells. The effectiveness of berberine against breast cancer has been verified in animal studies. In a female nude mouse model, a tail vein injection of berberine into mice with breast cancer showed a significant reduction in tumour size.

Berberine combination is quite promising. Research indicates that its combination with docetaxel markedly increases the rate of apoptosis in breast cancer. The rate of apoptosis increased by approximately 30%, demonstrating a good synergistic effect.

## 3.2. Mechanism of Action of Berberine in Anti-Breast Cancer

Recent years have seen a significant increase in the number of studies examining the mechanism of action of berberine in antitumour research. Berberine has the ability to control the cell cycle, trigger the apoptosis process in breast cancer cells, and markedly restrict their growth. Experimental data from Yi Yang Li showed that the apoptosis rate of human breast cancer cell lines MCF-7 and MDA-MB 231 increased significantly when treated with berberine concentrations in the range of 10 μM to 50 μM. The highest can be elevated to about 60% [6]. PI3K, AKT and mTOR constitute a pathway in breast cancer cells that is an important axis for regulating cell proliferation and migration. Zhao Jufen et al. demonstrated that berberine can inhibit breast cancer cell invasion and metastasis by blocking the PI3K/AKT/mTOR signalling pathway [7]. Berberine reduces the expression of key

proteins in breast cancer cells by down-regulating the level of AKT phosphorylation and inhibiting mTOR activity, resulting in the reduction of metallo-matrix proteases such as MMP-2 and MMP-9. The enzymes are responsible for degrading the basement membrane and extracellular matrix of tumour cells, and when inhibited, the tumour invasion ability is significantly reduced. Berberine was used in an in vivo experiment in MDA-MB-231 breast cancer xenograft mice, as shown by a preclinical model study by Yurong Zhao et al. The tumour volume was significantly reduced after a dose of 20 mg/kg was given to the mice via intraperitoneal injection. The tumour volume in the experimental group was much smaller compared to the control group with a reduction of about 47% [8].

Berberine exhibits a key role in regulating metabolic reprogramming of breast cancer cells. Breast cancer cells tend to rely on the phenomenon of enhanced aerobic glycolysis, known as the Warburg effect. This process supplies energy and metabolic intermediates for the rapid proliferation of cancer cells. Berberine reduces the uptake of glucose by breast cancer cells. It does this by regulating, and reducing, the level of expression of glucose transporter protein (GLUT1) - a protein that is an important enzyme in the process of glycolysis. Deng Xianguang et al. concluded that berberine inhibits the expression of hexokinase-2, which attenuates the efficiency of glycolysis in cancer cells, and when ATP production is reduced, breast cancer cells show impaired energy metabolism [9].

# 4. ROLE OF QUERCETIN IN BREAST CANCER TREATMENT

## 4.1. Molecular Structure and Biological Properties of Quercetin

Quercetin has attracted much attention for its anti-tumour properties, and its anti-tumour effect stems from its unique molecular structure. Research on quercetin reveals its role in hindering cell growth and encouraging cell death in breast cancer cells through the suppression of the EGFR/AKT/mTOR signaling route. Taking the human breast cancer cell line T47D cell line as an example, it was found that quercetin at a concentration of 50 μmol/L inhibited the proliferation of breast cancer T47D cells best [10]. Quercetin not only controls the balance between Bax and Bcl-2 but also liberates cytochrome C. Consequently, it markedly enhances Caspase-3's function, facilitating faster programmed cell death. Quercetin has low bioavailability and is often less than 10% absorbed after oral administration. Metabolic mechanism studies have shown that quercetin is excreted in the urine after undergoing a process of glucuronidation and sulfation mainly in the intestine [11]. Researchers have sought to improve the stability and tissue distribution of quercetin in vivo by nanoparticle delivery systems, liposomal carriers, or synergistic formulation with other ingredients to improve its bioavailability. Quercetin enhances the sensitivity of chemotherapeutic drugs. When quercetin was combined with paclitaxel, the experiment showed that quercetin could increase the sensitivity of PC-3 cells to paclitaxel [12].

## 4.2. Quercetin Can Regul The Operational Methods of Quercetin in Treating Breast Cancer

Ate cell cycle-related proteins and thus inhibit the proliferation of breast cancer cells. Studies on quercetin have shown that it can bring the breast cancer cell cycle to a standstill by reducing Cyclin D1 and Cyclin B1 cell cycle proteins. Experiments by Gan Lin and other scholars have shown that after breast cancer cells such as MCF-7 cells were treated with 50 μmol/L quercetin, In the breast cancer cell lines MCF-7 and MDA-MB-435, there was a notable decrease in Bcl-2 protein levels compared to the control group, suggesting that quercetin significantly impeded the standard cell cycle [13].

Quercetin primarily induces cell death in breast cancer cells via internal and external routes. Changes in mitochondria-associated proteins are the main dependence of the endogenous pathway. Quercetin increases the expression of the pro-apoptotic protein Bax and decreases the expression of the anti-

apoptotic protein Bcl-2, resulting in a greater ratio of Bax to Bcl-2, followed by a loss of mitochondrial membrane potential and the release of cytochrome C (Cytochrome C).

Quercetin activates the caspase pathway, and the activity of Caspase-3 and Casapse-9 enzymes increased dramatically in the treated cells, which means that it effectively promotes apoptosis. The expression level of Fas receptor and death signalling related proteins such as FADD increased, which is an indication of the activation of the exogenous pathway. They enhance the sensitivity of cancer cells.

Quercetin has been identified as a significant component in the treatment of breast cancer, due to its ability to enhance the expression and distribution of PTEN proteins. PTEN proteins have been demonstrated to inhibit the PI3K/Akt signalling pathway, thereby contributing to the suppression of cancerous cell growth. The PI3K/Akt pathway is widely active in breast cancer cells and is a key pro-survival signal involved in the inhibition of apoptosis and promotes cell proliferation [14].

Quercetin can act in breast cancer cells by inhibiting the glycolytic pathway. The significant enhancement of glycolysis in breast cancer cells is the 'Warburg effect'. It gives cancer cells the energy and metabolites they need for rapid proliferation. Quercetin inhibits the activity of hexokinase and pyruvate kinase type M2, key glycolytic enzymes, giving breast cancer cells a significant reduction in glucose uptake, and lactate production.

Quercetin has significant advantages in the treatment of breast cancer, which acts through multiple pathways and on multiple lesion targets. It can regulate the cell cycle and induce apoptosis of cancer cells, as well as inhibit the process of glycolysis and regulate oxidative stress and inflammatory response to prevent the proliferation and survival of cancer cells. It also prevents the migration and invasion of cancer cells. These studies have laid a solid foundation for the development and application of quercetin as a natural anti-tumour drug. We want to continue to explore in depth the pharmacodynamic performance and bioavailability of quercetin, as well as its role in combination drug strategies. These studies will help to promote its wider application in the clinic.

# 5. ROLE OF BAICALIN IN THE TREATMENT OF BREAST CANCER

## 5.1. Molecular Structure and Biological Properties of Baicalin

Baicalin is biodiverse and has anti-inflammatory, antiviral and antitumour activities, which are of significant value in the field of breast cancer research. Baicalin affects cell cycle regulation, induces apoptosis, and inhibits cell migration and invasion by targeting multiple signalling pathways, such as PI3K/Akt, NF-κB, and MAPK, which are closely related to the proliferation and survival of tumour cells.

Lately, research into baicalein's role in breast cancer therapy has progressively revealed its ability to control oxidative stress in breast cancer cells and diminish their growth potential. In a study by Yi Yang Li in 2024, it was found that baicalin significantly reduced the production of hydrogen peroxide (H2O2) in breast cancer cells, and it helped to reduce gene mutations and DNA damage in breast cancer cells due to high levels of ROS [15]. Baicalin also has a regulatory effect on apoptosis in breast cancer cells. In a study by Hou Yong in 2023, it was noted that after treatment with baicalin, the apoptosis rate of MCF-7 cells in the experimental group rose from 5.6% (control group) to 21.8% (experimental group), with a significant difference between the two groups ($p$-value $< 0.01$) [16]. Baicalin inhibits migration and invasion of breast cancer cells. This has been verified in experiments with a variety of breast cancer cell models. Yetalloproteinase (MMP) -2 and -9 expression in breast cancer cells [17]. Urong Zhao's study demonstrated that baicalin effectively diminished the levels of matrix m.

## 5.2. The Operational Process of Baicalein in Treating Breast Cancer

Baicalin regulates breast cancer cells and affects their proliferation and apoptosis. The role of problematic cell cycle regulation as a critical pathway in the onset and development of breast cancer has been a subject of significant research interest. Research has demonstrated that baicalin can inhibit the expression of cell cycle proteins, such as Cyclin D1 and Cyclin A2, effectively. And by reducing CDK4/6 activity, it keeps the G1 phase of breast cancer cells from progressing to the S phase. Effectively inhibiting the growth of breast cancer cells, Yi Yang Li's experiments were found using the MCF-7 cell model of breast cancer. It was found that the proliferation rate of baicalein-treated cells decreased by 45.3% ($P<0.01$), and a significant cycle blocking phenomenon was observed [18]. It is mainly achieved by activating Caspase-3 and Caspase-9 of the cystatin family of proteases, while promoting the loss of mitochondrial membrane potential.

Baicalin plays an important role in inhibiting the ability of breast cancer cells to invade and metastasise. It targets proteins such as MMP-2 and MMP-9, which are degraded by the extracellular matrix of tumours, thus preventing the invasion of breast cancer cells. Baicalin inhibits the expression of MMP-9 and VEGF, among others, and inhibits tumour angiogenesis, therefore, the ability of tumour cells to spread is limited [19].

Baicalin is crucial in controlling the operations of immune cells. The microenvironment of breast cancer tumors often exhibits immunosuppressive characteristics, leading to a reduction in T-cell activity. Tumour-associated macrophages (TAMs), a class of immunosuppressive cells, appear in large numbers and infiltrate them. Studies on baicalein have shown that baicalein significantly reduces the distribution of TAMs as well as boosts the activity of helper T cells (CD4+) and cytotoxic T cells (CD8+), promoting anti-tumour effects. A study by Yurong Zhao in 2023 found that the baicalin-treated group gave a 35.8% increase in the proliferation rate of CD8+ T cells. Meanwhile, PD-L1 expression levels were reduced by 50.6% ($P<0.001$). This suggests that baicalin can improve the immunosuppressive status of breast cancer [20].

Baicalin regulates breast cancer cell behaviour, and the PI3K/Akt/mTOR signaling axis is one of the important regulatory targets. PI3K/Akt overactivation enhances tumour cell viability and leads to chemoresistance. Baicalin inhibits the PI3K/Akt/mTOR signalling axis and reduces the expression of the anti-apoptotic protein Bcl-2, which enhances therapeutic efficacy. Baicalin has attracted attention for regulating metabolic reprogramming in breast cancer cells. This substance impedes the Warburg effect, diminishing glucose absorption and lactate generation in breast cancer cells, thereby hindering their energy provision processes.

Baicalin has a potentiating effect on chemotherapy. Experimental results on baicalin show that it is effective in enhancing the uptake of doxorubicin in breast cancer cells. At the same time, baicalin also reduces the expression of the drug resistance-related gene ABCG2. Tang Yumei's study in 2023 showed that the apoptosis rate of breast cancer cells treated with baicalin in combination with chemotherapeutic drugs increased by 21.4% ($P< 0.01$) and side effects were significantly reduced [21].

Baicalin controls the proliferation, penetration, and immune milieu of breast cancer cells via distinct molecular processes and communication routes, showcasing its anti-cancer properties on various fronts. The findings not only outlined herein provide a robust molecular basis for the utilisation of baicalin in the treatment of breast cancer, but also open up a new path for subsequent drug development and clinical translation.

# 6. COMPARISON OF DIFFERENCES IN THE EFFECTS OF BERBERINE, QUERCETIN AND BAICALIN WITH REGARD TO THE TREATMENT OF BREAST CANCER

## 6.1. Exploring the Similarity of the Mechanism of Action of Berberine, Quercetin and Baicalin in the Treatment of Breast Cancer

Each of the trio of substances suppresses breast cancer cell proliferation and modifies the growth patterns of cancer cells. Studies have shown that berberine regulates the expression of such cell cycle-related genes as Cyclin D1 and CDK4. It stops the cell cycle progression of cancer cells in G1 or G2/M phase. Quercetin inhibits cell proliferation by decreasing the levels of Cyclin B1 and CDC2 proteins. Baicalin, on the other hand, exerts its inhibitory effect by regulating the expression of cell cycle factors such as p21 and p27, further confirming their similar effects in inhibiting breast cancer cell growth.

The trio of compounds notably triggered cell death in breast cancer cells and demonstrated effective targeting in breast cancer therapy. Berberine triggers apoptosis, which is achieved through an endogenous pathway, a process in which the ratio of Bax to Bcl-2 rises. At the same time, Caspase-3 and Caspase-9 are activated [22]. Quercetin elevates ROS (reactive oxidative products) levels and opens the mitochondrial apoptotic pathway. Baicalin activates the p53 pathway to allow tumour cells to apoptose, and previous studies have found that baicalin also inhibits Survivin protein expression in the treatment of breast cancer cells.

Berberine, baicalin, and quercetin all reduce the ability of breast cancer cells to migrate and invade by similar mechanisms. When matrix metalloproteinase activity was inhibited, they were effective in curbing the invasive behaviour of tumour cells. Studies have shown that berberine can reduce the expression level of MMP-9 in breast cancer cells, making the cells less invasive. Similarly, quercetin has similar effect on inhibiting MMP-2 and MMP-9. Baicalin, on the other hand, blocked the EMT process - i.e., epithelial-to-mesenchymal transition - and controlled the expression of proteins involved in invasion, such as N-cadherin and Vimentin, thus curbing the migration and invasive ability of breast cancer cells. and invasion ability of breast cancer cells.

Berberine, quercetin and baicalin modulate inflammatory factors in the tumour microenvironment. Berberine was able to inhibit the NF-κB signalling pathway, thus reducing the secretion of pro-inflammatory factors such as IL-6 and TNF-α, so that the tumour microenvironment would be improved. And quercetin significantly inhibited the STAT3 signalling pathway, thus effectively reducing the secretion level of inflammatory factors in breast cancer cells. Baicalin can inhibit the TGF-β signalling pathway and improve the suppression of immune function.

Their antioxidant effects are an important feature of the similarity in mechanism of action. Berberine elevates the levels of superoxide dismutase and glutathione peroxidase, thereby reducing oxidative stress. Quercetin reduces ROS production and improves cellular redox status. Baicalin inhibits the production of free radicals and helps to reduce oxidative damage, thereby reducing the pro-proliferative effects on breast cancer cells.

## 6.2. Exploring the Differences in the Mechanism of Action of Berberine, Quercetin and Baicalin in the Treatment of Breast Cancer

Berberine primarily functions by suppressing the growth of cancer cells, triggering cell death, adjusting the cell cycle, and modifying the metabolic processes of cancer cells. Berberine induces apoptosis in breast cancer cells by reducing the levels of key molecules in the Akt/PI3K pathway. In vitro data show that berberine inhibits cell proliferation by up to 72% in the breast cancer MCF-7 cell line. It has an IC50 value of approximately 10 mol/L [23]. Berberine has significant antioxidant

properties and it reduces the level of oxidative stress in breast cancer cells. This is achieved by reducing the production of reactive oxygen ROS.

Quercetin can combat breast cancer by modulating multiple signalling pathways and also attenuates the inflammatory response of breast cancer cells by inhibiting the NF-κB pathway, which is important for the inflammatory microenvironment of tumours. Quercetin blocks MDA-MB-231 cells in cells in the G0 to G1 phase, preventing unlimited proliferation of breast cancer cells [24]. Study states that it has an IC50 value of about fifteen micromolar.

The anti-breast cancer effect of baicalin is reflected in its ability to inhibit invasiveness and modulate immunity. Baicalin inhibits the expression of VEGF and MMP-9, which reduces the formation of tumour blood vessels and the degradation of extracellular matrix. It thus significantly reduces the metastatic and infiltrative ability of breast cancer cells. Baicalin regulates T cell and macrophage functions, remodels the immune microenvironment and curbs tumour development.

Berberine has a unique molecular mechanism in the treatment of breast cancer, significantly acting on metabolic regulation and extensively modulating signalling pathways. As for quercetin, it has a prominent role in the modulation of tumour inflammatory response and sensitisation to chemotherapy. Baicalin, on the other hand, is more likely to inhibit tumour invasion and metastasis while modulating the tumour immune microenvironment. These differences suggest that the potential of all three in combination therapy or for different breast cancer subtypes is promising and deserves further investigation. Berberine is more challenging in terms of bioavailability, compared to quercetin and baicalin. Stability studies of pharmacokinetic parameters need to be intensified for maphyllin and baicalin. Further studies could optimise the formulation of each component and reveal a more complete mechanism of action. This will facilitate the process of clinical translation in breast cancer treatment.

### 6.3. Exploring the Potential Synergistic Therapeutic Potential of the Three

Berberine, baicalin and quercetin have been confirmed by numerous studies to have significant pharmacological effects amid the stream of breast cancer, and the study of the synergistic therapeutic effects of these three compounds is important for optimising breast cancer treatment strategies, enhancing efficacy and reducing side effects. Combined use of quercetin and baicalin can jointly enhance the expression levels of apoptotic proteins (e.g., Caspase-3, Caspase-9) and effectively block the growth of breast cancer cells [25]. As berberine, quercetin and baicalin each act on different signalling pathways, it was found that this feature can bring about a combined effect for tumour cells, showing action on a synergistic mechanism. Berberine regulates the AMPK/mTOR signalling pathway, while quercetin inhibits the PI3K/Akt signalling pathway. The combination of the two may be able to dually inhibit breast cancer cell proliferation. Baicalin modulates the NF-κB signalling pathway and attenuates the inflammatory response in the tumour microenvironment. In view of this, the therapeutic effect of the other two ingredients would also be enhanced.

From a pharmacokinetic point of view, these three ingredients might complement each other synergistically. Berberine has low bioavailability but a stable drug half-life. It is suitable for long-term action, quercetin can improve the state of cellular oxidative stress, and baicalin, with its advantageous and obvious characteristics in tissue penetration and distribution, can well solve the problem of uneven distribution of berberine as well as quercetin in breast cancer tissues.

However, it should be noted that the drug-drug interactions and the optimal dosage ratio should be taken into consideration when applying the drugs, which is very important to exert the synergistic effect of the three. We need to quickly tease out the appropriate combination drug regimen through methods such as network pharmacology. Its feasibility is further investigated in the clinical treatment of breast cancer.

In the clinic, the combined use of the three substances cracks the limitations of breast cancer monotherapy. For example, their natural origin and good safety can cope with cellular resistance and reduce toxic side effects, which has a broad application prospect in the field of adjuvant therapy. In devising upcoming treatment plans, it's crucial to consider the patient's unique genetic makeup, tumor classification, and medical history, necessitating additional studies to explore the combined effects of these elements in tailored dosing.

# 7. CONCLUSION AND OUTLOOK

## 7.1. Summary of Findings

This article comprehensively analyses the molecular mechanisms, signaling pathways and anti-breast cancer effects of three active ingredients of traditional Chinese medicines, and summarizes the key advances in the field of breast cancer treatment in recent years. The article can bring a new scientific basis for the treatment of breast cancer, and at the same time lays a solid foundation for the in-depth investigation of multi-targeted anti-breast cancer effects of active ingredients of traditional Chinese medicines. The article provides theoretical support for clinical application and reveals the necessity and prospect of multi-target combination therapy strategy.

## 7.2. Looking to the Future Regarding the Therapy of Breast Cancer

Breast cancer treatment holds great potential for the future, with plans to delve into novel treatments, thoroughly investigate gene mutation mechanisms to enhance drug targeting, and bolster cross-disciplinary collaboration to consistently elevate patient survival rates and life quality. With the in-depth study of genomics and proteomics, we will gradually unveil the molecular pathological mechanism of breast cancer. It is often difficult to meet the needs of patients with a single treatment plan, and we can predict the risk, diagnose the typing and discover efficient targets with the help of genomics and proteomics research. Genomic analysis has revealed that mutations in genes such as HER2 and BRCA1/2 are associated with breast cancer progression. This points the way to developing targeted therapeutic drugs. Extensive use of immunotherapy. In recent years, there has been a notable increase in the use of immune checkpoint inhibitors, such as PD-1 and PD-L1 inhibitors, in the treatment of breast cancer. In the future, we will study how to better combine immunotherapy and reduce side effects. We are also working to develop personalised immunotherapies that are better suited to breast cancer patients. Using artificial intelligence and big data to help breast cancer treatment and assist doctors to develop individualised treatment plans. Breast cancer treatment needs to emphasise interdisciplinary cooperation and follow international research trends. The future of breast cancer treatment is moving towards more accurate, efficient and less toxic, and there is more hope for humanity to overcome breast cancer.

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