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# Sodium Bicarbonate: Potential Cancer Therapy

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**Abstract:** This article aims to describe the application of sodium bicarbonate for cancer therapy. Cancer is a leading cause of death worldwide. The extracellular tumor microenvironment (TME) acidity of cancer cells is a defining characteristic. It affects the behavior of cancer cells in several ways, including tumor growth, invasion, metastasis, and resistance to treatment. These changes trigger metabolic changes in cancer cells, enhancing their ability to proliferate and survive. In addition, scientists have stated that the TME generates acidity. Researchers even went so far as to claim that tumor invasion did not occur in less acidic zones. Experts propose to focus on this acidity as a potential therapeutic target. A few scientists proposed sodium bicarbonate to eliminate TME acidity. Few studies have conducted several in vivo tests to investigate sodium bicarbonate's potential as an anticancer agent. The effects of adding sodium bicarbonate to various treatments for different types of tumors were expressed without going into additional detail about the stages of occurrence. In this study, we focused on employing sodium bicarbonate to remove acid from the TME, thereby inhibiting tumor growth, blocking local invasion, and inhibiting tumor metastasis. A high hydrogen ion concentration ($H^+$) characterizes acidic solutions. When added to an aqueous solution, sodium bicarbonate breaks down into bicarbonate anions ($HCO_3^-$) and sodium ions ($Na^+$). The bicarbonate anions consume hydrogen ions ($H^+$) to produce carbonic acid ($H_2CO_3$). The carbonic acid then converts to carbon dioxide ($CO_2$) and water ($H_2O$). We propose conducting a clinical trial that focuses on pH as a measure of decreasing TME acidity and, as a result, inhibiting the development and proliferation of tumor cells.

**Keywords:** cancer, acidosis, tumor microenvironment, sodium bicarbonate, tumor growth.

# 碳酸氫鈉：潛在的癌症治療方法

**摘要**：本文旨在描述碳酸氫鈉在癌症治療上的應用。癌症是全世界死亡的主要原因。癌細胞的細胞外腫瘤微環境酸度是一個決定性特徵。它以多種方式影響癌細胞的行為，包括腫瘤生長、侵襲、轉移和對治療的抵抗。這些變化引發癌細胞的代謝變化，增強其增殖和生存的能力。此外，科學家指出細胞外腫瘤微環境會產生酸度。研究人員甚至聲稱腫瘤侵襲不會發生在酸性較低的區域。專家建議將這種酸度作為潛在的治療目標。一些科學家提出用碳酸氫鈉來消除細胞外腫瘤微環境酸性。很少有研究進行多次體內測試來研究碳酸氫鈉作為抗癌劑的潛力。表達了在不同類型腫瘤的各種治療中添加碳酸氫鈉的效果，但沒有詳細說明發生階段的更多細節。在本研究中，我們將重點放在碳酸氫鈉去除細胞外腫瘤微環境中的酸，進而抑制腫瘤生長，阻斷局部侵襲，抑制腫瘤轉移。高氫離子濃度(氢离子)是酸性溶液的特性。當添加到水溶液中時，碳酸氫鈉會分解成碳酸氫根陰離子(碳酸氢根)和鈉離子(钠)。碳酸氫根

陰離子消耗氫離子(氢离子)產生碳酸(碳酸氢)。然後碳酸轉化為二氧化碳(二氧化碳)和水(水)。我們建議進行一項臨床試驗，重點關注 pH 值作為降低細胞外腫瘤微環境酸度的指標，從而抑制腫瘤細胞的發育和增殖。

**关键词**：癌症，酸中毒，腫瘤微環境，碳酸氫鈉，腫瘤生長。

## 1. Introduction

According to the Global Burden of Disease (GBD) 2015 study, cancer is one of the primary causes of death for people worldwide [1]. Sung et al. [2] and Ferlay et al. [3] estimate that in 2020, 19.3 million new cases of cancer were worldwide, and over 10.0 million cancer-related deaths occurred, with lung cancer continuing to be the most common cause of cancer-related fatalities.

The International Agency for Research on Cancer (IARC), a Cancer Agency of the World Health Organization (WHO), presented its most recent estimates of the global cancer burden in 2022 on February 1, 2024. These estimates showed that there were an expected 20 million new cancer cases and 9.7 million deaths, and predicted 53.5 million people to still be alive five years after receiving a cancer diagnosis. One in five people will have cancer at some point in their lives; one in nine men and one in twelve women will die from the illness. According to these projections, there will be over 35 million new cases of cancer in 2050, representing a 77% increase from the 20 million cases expected in 2022 [4].

According to [5, 6], the tumor microenvironment (TME) is a complex system that plays a crucial role in cancer progression. Tumor cells interact with the microenvironment constituents, such as fibroblasts, immune cells, macrophages, and extracellular matrix, to accelerate cancer development [6]. Preclinical and clinical research on cancer vaccines specifically targeting these elements has yielded encouraging results [7]. By balancing pro- and anti-malignancy factors, the TME also controls the growth of tumors [8]. So, a tumor's main characteristic is the acidity of the extracellular microenvironment surrounding cancer cells.

Our strategy is innovative in that it targets the acidity of the TME by administering sodium bicarbonate to patients with cancer eliminates the acidity of the TME, and subsequently reduces the primary source of cancer cell proliferation.

The remainder of this article is as follows: in section 2, we define acidic TME and discuss how it promotes tumor growth, invasion, metastasis, and treatment resistance. Section 3 shows that acidity is produced by the TME. Section 4 verifies that to construct tumor therapy, scientists should focus on the acidity of the TME. Section 5 presents the application of sodium bicarbonate as a buffer to lower the acidity of the TME, thereby impeding tumor growth, invasion, metastasis, and resistance. Section 6 recommends using current methodologies in clinical trials; section 7 closes the study.

## 2. Acidic Tumor Microenvironment

Cancer is a complex ecosystem involving a wide spectrum of non-cancerous cells and their numerous interactions within the tumor. It is not just a genetic disease. Microscopic analysis of solid tumors reveals the intricate complexity of cancer: the TME is a highly structured ecosystem in which cancer cells are encircled by a variety of nonmalignant cell types, all of which are embedded in a modified extracellular matrix that has undergone vascularization. The TME is composed of a wide variety of cell types that differ depending on the tissue, such as pericytes, endothelial cells, immunological cells, and cancer-associated fibroblasts [9]. The acidity of the TME influences the behavior of cancer cells in several ways, including tumor growth, invasion, metastasis, and treatment resistance. It causes cancer cells to undergo metabolic reprograming, which promotes their survival and growth.

Low extracellular pH, a result of anaerobic glycolysis and hypoxia, is its defining feature [10]. Early 1980s research on cancer revealed that the extracellular interstitial spaces of tumors have an acidic pH range of 6.3 to 7.0 [11].

The acidic nature of the TME, as shown by its lower pH than that of healthy tissues, is a crucial feature of tumors. This acidic extracellular microenvironment (AEM) strongly influences tumor growth, invasion, metastasis, and treatment resistance [12].

One of the main causes of tumor growth and treatment resistance in solid tumors is the tumor's acidic microenvironment [13], [14].

Because it enhances cancer cell function and interactions with the extracellular matrix, the acidic extracellular microenvironment has a substantial impact on tumor development [12]. In response to extracellular acidification, an extracellular pH activates SREBP2, which promotes cancer cell growth and survival [15].

This acidity enhances tumor cell resilience and promotes invasion, and it acts as a major regulator of neoplastic development and invasion [14]. Acidic

extracellular pH, which is mainly caused by lactate release, activating prometastatic proteins, facilitates tumor growth [16]. According to [17], acid-mediated tumor invasion occurs due to altered glucose metabolism and increased glucose uptake, leading to an acidic microenvironment that allows tumor cells to survive and proliferate in damaged normal tissue.

According to [16], [18], and [19], the acidic extracellular environment plays a vital role in tumor growth by promoting local invasiveness, metastatic spread, and resistance to chemotherapy and radiation therapy. The main cause of this acidity is the secretion of lactate from anaerobic glycolysis, which also triggers the release of exosome implicated in tumor growth and activates proteins that promote metastasis [20].

This acidity, which is frequently unaffected by hypoxia, may promote the development of metastases and local invasiveness. It can also contribute to resistance to radiation therapy and chemotherapeutic drugs [18], [19]. Lactate secreted from anaerobic glycolysis is the main cause of the acidity of the TME; it can activate lysosomal enzymes and activate genes that promote metastasis [16]. TME acidity influences genetic stability, epigenetics, cellular metabolism, proliferation, and survival, which in turn promotes the development of metastatic illness [12]. The acidic extracellular microenvironment in tumors significantly influences tumor invasion and metastasis [16], [18],[19], [21].

According to [21], this acidity encourages the development of a population of tumor cells that are resistant to acid and have a higher propensity to invade and spread.

The TME, particularly its acidic extracellular component, has been demonstrated to have a substantial impact on both tumor progression and therapeutic resistance [22]-[24]. This effect is mediated by the interaction between the microenvironment and cancer cells; exosome containing pro-invasive molecules can be released and taken up by the microenvironment due to its acidic pH [24], and the acidic microenvironment has also been connected to the induction of epithelial-to-mesenchymal transition (EMT) in cancer cells, a process that is linked to resistance [25]. According to [26], the acidic microenvironment of tumors significantly influence the establishment and advancement of drug resistance. Acidity can also result from the pentose phosphate pathway's $CO_2$. Although acidic pH can be toxic to cells and promote tumor metastasis, cancers that adapt to this environment can become aggressive and drug resistant [16].

## 3. The Tumor Microenvironment Generates Acidity

Because of their low perfusion and elevated fermentation, solid tumors have an acidic pH. There is a theory that an acidic pH promotes metastatic and local invasions. Normal or almost normal pH zones did not exhibit tumor invasion. Cells in the invasive margins express the sodium-hydrogen exchanger NHE-1 and glucose transporter GLUT-1, both of which are linked to peritumoral acidosis, according to immunohistochemical analysis [27]. The TME generates an acidic pH, which drives local invasion [27].

Large amounts of lactate, extra protons, and carbon dioxide are produced by tumor cells, and these factors together lead to increased acidification of the extracellular TME, which typically has a pH in the range of 6.5–6.8 [28].

Compared with normal mammary cells, breast cancer cells are more capable of acidifying their extracellular environment, which may make it easier for released lysosomal cathepsins to be activated extracellularly [29].

## 4. Acidic Tumor Microenvironment as a Target for Therapy

This acidic environment can be targeted for cancer-specific imaging and therapy using pH-responsive nanoscale theognostic platforms [13]. It also plays a role in immune escape, making it a potential therapeutic target [30].

Tumor tissues exhibit an acidic extracellular environment, which is primarily caused by the release of lactate from anaerobic glycolysis. The proton pump ($H^+$-ATPase), which may be connected to tumor metastasis, and the $Na^+/H^+$ exchanger are two examples of the transporters and pumps that contribute to $H^+$ secretion. Lysosomal enzymes are activated, and prometastatic gene expression is induced in an acidic microenvironment. Effective treatment and avoidance of metastases depend on the determination of the pH of the tumor [16].

Estrella et al. [27], in a very intriguing finding, asserted that tumor invasion did not occur in normal or near-normal extracellular pH.

These results emphasize the significance of considering the TME, particularly its acidity, when developing effective cancer therapies. It appears possible to improve cancer patient outcomes and broaden the scope of cancer treatments by addressing the specific challenges posed by the acidic microenvironment.

## 5. Sodium Bicarbonate and Human Body Buffer System

One of the most important physiological processes in the human body is the maintenance of acid-base balance. A pH range of 7.35–7.45 is ideal for the human body, and maintaining this level is essential for normal function [31].

The body maintains pH balance through various

mechanisms. Short-term homeostasis involves acid and alkaline buffers in body fluids, whereas the respiratory and renal systems manage intermediate- and long-term homeostasis [32], [33]. The kidneys achieve this through various mechanisms such as $H^+$ secretion, bicarbonate reabsorption, and the excretion of acidic and basic equivalents like ammonia and phosphate [32].

The proper operation of metabolic processes depends on the body's capacity to maintain this pH range [34].

### 5.1. Metabolic Acidosis in Extracellular Fluid

Numerous factors, such as the buildup of endogenous acids, loss of bicarbonate, and drug-induced mechanisms, can result in metabolic acidosis [35]-[37].

Lactic acidosis, ketoacidosis, renal failure, and drug intoxication are conditions that can cause high anion gap metabolic acidosis [35]. Conversely, gastrointestinal bicarbonate loss, renal tubular acidosis, and drug-induced hyperkalemia can cause hyperchloremic metabolic acidosis [35]. Additionally, distal and proximal renal tubular acidosis can also contribute to metabolic acidosis [36]. Additionally, drug-induced metabolic acidosis can occur with either a normal or elevated anion gap, and various drugs are implicated in these conditions [37]. Finally, ethylene glycol poisoning is an uncommon but severe cause of metabolic acidosis and is marked by high anion and osmolal gaps [38].

An abnormal clinical process known as acidosis results in a net gain of hydrogen ions ($H^+$) in the extracellular fluid. When bicarbonate ions ($HCO_3^-$) are lost or accumulate $H^+$, metabolic acidosis results [39]. An organ's pH indicates how acidic it is; a lower pH indicates a more acidic organ [40]. The general expression $pH = -\log(H^+)$ describes the relationship between pH and hydrogen ion concentration ($H^+$) [41], [41]. Acidosis increases the number of $H^+$ ions in the blood, resulting in a decrease in pH [42].

The pH scale, which is typically used to measure the acidity and alkalinity of solutions, ranges from 0 to 14 [40]. With a pH of 7.35 and a plasma bicarbonate level of 22 mmol/L, metabolic acidosis is a common condition with various causes and implications [43]. A decrease in pH and $HCO_3^-$ indicates high-anion-gap metabolic acidosis, which is typically caused by lactic acidosis, diabetic ketoacidosis, and renal failure [44]. Acute metabolic acidosis increases morbidity and mortality because of its depressive effects on cardiovascular function, inflammation stimulation, and suppression of the immune response [45]. Metabolic syndrome is associated with increased cardiovascular morbidity and mortality in various population subsets [46].

Life on Earth depends on the presence of appropriate pH levels in and around living things and their cells.

The finding by [27] that no tumor invasion occurred in regions with normal or nearly normal extracellular pH was among the most significant findings in this context. Additionally, the authors noted that oral sodium bicarbonate administration was sufficient in a preclinical model to increase peritumoral pH, inhibit tumor growth, and stop local invasion, supporting the theory of acid-mediated invasion.

Modulating the pH of the TME with sodium bicarbonate can have a significant impact on tumor apoptosis because acidosis is more crucial for the survival of tumor cells [47].

According to [48], sodium bicarbonate, either alone or in conjunction with other therapies, may have anticancer effects based on a few in vivo studies.

### 5.2. How Does Sodium Bicarbonate Work?

Engelking [49] claims that sodium bicarbonate has been used to treat patients with metabolic acidosis who have both a low plasma concentration and a pH of 7.2 or less. Because $NaHCO_3$ is easily dissociated in aqueous solution, $Na^+$, $HCO_3^-$, and $H_2O$ are added to the extracellular fluid (ECF) compartment, as illustrated in Fig. 1. As a result, $HCO_3^-$ molecules will react with $H^+$ molecules, producing carbon dioxide and water and blood acidity is reduced.

In the ECF compartment, the weak base $HCO_3^-$ tends to associate with $Cl^-$, and because $Na^+$ molecules are being added without $Cl^-$, both effects contribute to an increase in the strong ion difference (SID), resulting in alkalinization. Furthermore, $HCO_3^-$ acts as a buffer, allowing $CO_2$ and $H_2O$ to be produced when combined with $H^+$. Although Pco rises by approximately 0.5 mmHg for every mEq/L increase in plasma $HCO_3^-$ concentration, this assumes that the lungs are in good health because these chemical interactions produce an excess of $CO_2$ [49].

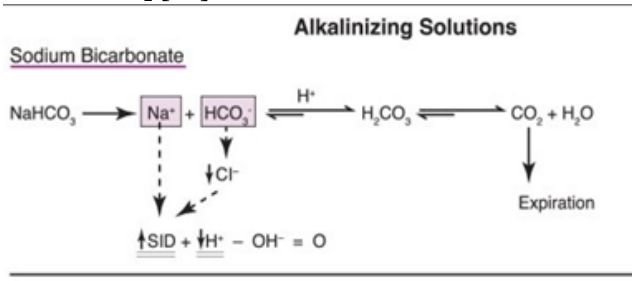


Fig. 1 Alkalinizing solutions. When dissolved into aqueous solution, sodium bicarbonate separates into bicarbonate anions and sodium cations. Bicarbonate anions consume hydrogen ions to produce carbon [49]

After administration, sodium bicarbonate ($NaHCO_3$) separates into two components: bicarbonate ($HCO_3^-$) and sodium ($Na^+$). Carbonic acid ($H_2CO_3$) is produced when bicarbonate anions $HCO_3^-$ consume hydrogen ions ($H^+$). Subsequently, carbonic acid is transformed into water ($H_2O$) and carbon dioxide ($CO_2$). The latter is excreted from the lungs. Plasma bicarbonate levels, which are known to buffer excess hydrogen ion

concentrations and raise solution pH to counteract the clinical manifestations of acidosis, are the primary therapeutic effects of administering sodium bicarbonate [50], [51].

Thus, the question started to make much sense. A high hydrogen ion concentration ($H^+$) in blood indicates acidity. Sodium bicarbonate dissolves easily in aqueous solutions to form $Na^+$ and $HCO_3^-$. The anion $HCO_3^-$ is a weak base that absorbs hydrogen ions in the blood, leading to the extraction of acid from the bloodstream (i.e., extracellular microenvironment). It functions as a buffer and produces carbon dioxide ($CO_2$) and water ($H_2O$). Depletion of the resources available to cancer cells for survival and growth, consequently, prevents the tumor's primary processes, such as growth, invasion, metastasis, and resistance to treatment.

### 5.3. Does High Stomach Acidity Prevent Sodium Bicarbonate from Entering the Bloodstream?

Normal human individuals have a gastric pH of 1.0–2.5, which is very acidic and aids in digestion while acting as a barrier against ingested pathogens [52], [53].

Many researchers are curious as to how sodium bicarbonate manages to enter the intestines through the stomach's acid and survive the high acidity level. It might react with hydrochloric acid to be converted to carbon dioxide and water. Pinheiro et al. [54] trial, which comprised a randomized double-blind crossover trial with sixteen adult patients taking potassium citrate and sodium bicarbonate for 3 days each. Results showed that there was a comparable increase in urinary pH and urinary citrate excretion across the two supplements. This fact demonstrates that sodium bicarbonate penetrates the stomach and enters the blood. Additionally, [55] findings from a clinical trial involving five patients receiving potassium and sodium citrate demonstrated that both treatments caused an equal elevation in urine pH.

### 5.4. Safety of Sodium Bicarbonate

Studies like [51], [56]-59] suggest that sodium bicarbonate may be safe and even helpful for treating certain medical conditions and poisonings, as well as for correcting mild metabolic acidosis. However, other studies have pointed to risks like increased contrast nephropathy, major electrolyte abnormalities, and metabolic alkalosis, especially when used improperly or dosed incorrectly [60]-[62].

By monitoring the pH level to be in the range of 7.35–7.45 while administering sodium bicarbonate, an increase in metabolic alkalosis can be prevented.

Supplementing with sodium bicarbonate is generally safe for human consumption and can enhance several physical performance measures; however, tolerance to high dosages may be problematic, and individual reactions and potential side effects may differ.

### 5.5. Effects of Sodium Bicarbonate on Food Digestion

Food digestion requires an acidic environment of the stomach. As previously noted, stomach acidity ranges from 1.0 to 2.5. Conversely, sodium bicarbonate reduces acidity. Consequently, the medicine will interfere with the digestion process if taken while food is still in the stomach. Logically, it makes sense that taking sodium bicarbonate at least an hour before meals and two hours following is the appropriate time to do so. However, further investigations are required to fully understand the effects of sodium bicarbonate on food digestion.

## 6. Recommendations

Sodium bicarbonate is already prescribed for some clinical conditions, including heartburn. We recommend conducting a clinical trial to investigate its potential in reducing the acidity of the extracellular microenvironment surrounding malignancies. While some sodium bicarbonate is lost during oral administration due to the stomach's high acidity, intravenous injection guarantees that the entire dose reaches the intended location. Because sodium bicarbonate reacts with hydrogen ions to produce carbon dioxide and water, volunteer patients must have healthy kidneys and lungs.

The authors of the mini review [48] stated that their colleagues carried out several in vivo tests to investigate sodium bicarbonate's potential as an anticancer agent. As shown in Table 1, the effects of adding 200 mM sodium bicarbonate to various treatments for various types of tumors are expressed clearly without giving additional details about the stages of occurrence. The results obtained include immune system stimulation, tumor growth delay, and metastasis inhibition.

Table 1 In vivo experiments of sodium bicarbonate monotherapy in anticancer treatment [48] (Adopted by the authors)

| Tumor type | Model | Administration of $NaHCO_3$ |
|---|---|---|
| **Inhibition of metastases** | | |
| Breast cancer | MDA-MB-231 xenograft intrasplenic injection | 200 mM $NaHCO_3$ po ad libitum |
| Prostate cancer | PC3M xenograft tail vein injection | 200 mM $NaHCO_3$ po ad libitum |
| Melanoma | B16 allograft tail vein injection | 200 mM $NaHCO_3$ po ad libitum |
| **Induction of tumor growth delay** | | |
| Prostate cancer | TRAMP | 200 mM $NaHCO_3$ po ad libitum |
| Breast cancer | MDA-MB-231 cells mice dorsal window | 200 mM $NaHCO_3$ po ad libitum |

| | | |
|---|---|---|
| | chamber | |
| Colorectal cancer | HCT116 cells mice dorsal window chamber | 200 mM $NaHCO_3$ po ad libitum |
| Breast cancer | MDA-MB-231 xenograft | A single dose of 21 mg or 84 mg $NaHCO_3$ po 1 mL 1M $NaHCO_3$ ip injection |
| **Enhancement of immune system** | | |
| Melanoma | Yumm 1.1 allograft (CD8+ T-cell) | 200 mM $NaHCO_3$ po ad libitum |
| B-cell | lymphoma λ-myc mice (NK cells) | 200 mM $NaHCO_3$ po ad libitum |

Given the high acidity of the stomach, intravenous sodium bicarbonate administration appears to be more promising than oral sodium bicarbonate administration; however, we assume that both approaches be considered in future studies.

Various studies have verified that acidosis causes tumor growth, invasion, metastasis, and treatment resistance. Additionally, it enhances the survival and proliferation capacities of cancer cells. The experiments focused on TME acidity.

In technical terms, the most important indicator for us is the pH. Therefore, verifying that the pH of the TME was restored to acid-base equilibrium was the first step of the experiment.

The experiments begin by gathering images of tumor cells and measuring the pH of the patient's extracellular microenvironment. After that, the patient receives an injection of sodium bicarbonate. We continued to add sodium bicarbonate while keeping an eye on the pH level to make sure it has reached the acid-base equilibrium range (7.35 to 7.45). Next, while maintaining the pH value within the equilibrium range by adding more sodium bicarbonate as needed, we took fresh pictures of the tumor cells at the end of each week and compared them to the originals. For 2–3 months, monitor the pH level and take images of the tumor cells. Then, the findings are reported.

The primary objective of clinical studies is to validate the use of sodium bicarbonate as a tumor treatment. Afterwards, studies seek to determine the appropriate dosage, duration of therapy, and the comparative effectiveness of oral versus intravenous administration; these may vary according to the cancer type.

## 7. Conclusion

Cancer is one of the major causes of death worldwide. Scientists agree that the acidity of the tumor microenvironment (TME) influences the behavior of cancer cells in several ways, including tumor growth, invasion, metastasis, and treatment resistance. It causes cancer cells to undergo metabolic reprogramming, which promotes their survival and growth. For this reason, scientists argue that cancer treatment should focus on the acidity of the TME.

The presence of hydrogen ions ($H^+$) indicates liquid acidity. As the number of $H^+$ increases, acidity increases too. When added to an aqueous solution, sodium bicarbonate ($NaHCO_3$) dissolves readily to produce sodium ($Na^+$) and bicarbonate ($HCO_3^-$). Bicarbonate interacts with hydrogen ions to form carbonic acid ($H_2CO_3$). Ultimately, $H_2CO_3$ is converted to carbon dioxide ($CO_2$) and water ($H_2O$). As a result, the quantity of $H^+$ is reduced in the liquid, its acidity is subsequently reduced, and the liquid acidity is restored to equilibrium.

Combining these two results, we argue that alkalizing the tumor microenvironment with sodium bicarbonate can efficiently deplete tumor cell resources. Consequently, they prevent the growth, invasion, and spread of cancers.

To accomplish this, we suggested conducting a clinical experiment on patients with tumors, in which patients received sodium bicarbonate intravenously, and their pH levels were monitored until they reached the equilibrium range (7.35 to 7.45). Next, we took images of the tumor cells while maintaining the pH level within the equilibrium range. Continue doing this for a few months, and then evaluate the outcomes.

To examine sodium bicarbonate's impact on tumor therapy resistance, researchers combined it with different therapies and reported that the results were favorable. Instead, our method tested the scientific finding that sodium bicarbonate equilibrates the TME by removing acid, thus eliminating the primary source of cancer cells.

We encourage future researchers with access to medical facilities to employ our method to produce measurable results that reduce the number of deaths resulting from various forms of cancer.

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