Targeting Cancerous Tumors through their Metabolic Activity via Glucose Receptors in the Tumor; Known as the Alkaline Glucosodiene Molecules Theory

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Abstract
Tumor growth leads to a hypoxic state in cancer cells, which is a hallmark of cancer. Metabolism of cancer cells differs from normal cells, as they prefer glycolysis over oxidative phosphorylation as a source of ATP. This results in the generation of only two molecules of ATP per molecule of glucose, compared to the 36 molecules that can be generated through oxidative breakdown of glucose. Therefore, cancer cells require more glucose than normal cells. This increased uptake of glucose is facilitated by the overexpression of glucose transporters, particularly GLUT1 and GLUT3, which are hypoxia-responsive, and other glucose transport proteins. Targeting these carrier proteins may be a useful strategy in anticancer therapy. Cancer is a genetic disease characterized by heritable defects in cellular regulatory mechanisms. Tumor cells must adapt their metabolism to survive and proliferate in the challenging conditions of the tumor microenvironment. To maintain uncontrolled cellular growth and survival, cancer cells alter their metabolism, which makes them dependent on a steady supply of nutrients and energy. The Warburg theory, proposed almost a century ago, suggested that cancer cells consume glucose even in the presence of oxygen. Recent studies have confirmed that cancer cells indeed consume significantly more glucose than normal cells. Cancerous tumors require an acidic microenvironment with low oxygen levels for growth and spread. However, recent advances in pH measurement have shown that the intracellular pH of cancer cells is neutral or slightly alkaline compared to normal tissue cells. This indicates that not all tumors are highly acidic. Exploiting the high glucose consumption of cancer cells, a strategy to lyse cancer cells has been proposed by modifying the atomic structure of glucose molecules to create alkaline glucosodiene molecules. These molecules may be able to target defects in the tumor structure and potentially achieve cell killing. This approach takes advantage of the uncontrolled consumption of glucose molecules by cancer cells. If small molecules of toxic atoms (alkaline atoms) can be continuously supplied to cancer cells through food, this may have a killing effect on cancer cells. This theory aims to investigate the potential of this method as a means of killing cancer cells.

Keywords: glucosodiene molecules theory; cancer cell dissoluted; alkaline glucosodiene molecules therapy; toxic chemotherapeutic nutrition of cancer cells; modulating glucose to target the metabolic activity of the cancer tumor

Introduction
Cancer is a global health concern that encompasses a variety of common and rare types affecting different parts of the body. Some of these cancers, including pancreatic, hepatic, colorectal, prostate, breast, lung, and osteosarcoma in pediatric patients, are widespread and highly lethal. Other cancers, such as acinar cell carcinoma, pancreaticoblastoma, and penile cancers, are rare and very rare. The characteristics of these cancers, such as their malignancy, the tendency to metastasize, and the prognosis for patients, are often correlated with the expression of glucose transporters in cancer cells [1]. Studies have shown that overexpression of glucose transporters, particularly GLUT1 and GLUT3, increase the aggressiveness and invasiveness of tumors and predicts worse outcomes for patient [2]. The unique metabolism of cancer cells, known as the "Warburg effect," involves the preferential use of glycolysis over oxidative breakdown of glucose, even in the presence of oxygen [3]. Cancer cells require more glucose than normal cells due to their lower yield of ATP from glycolysis, leading to an increased expression of glucose transporters in these cells. The extracellular pH of tumor tissues is often acidic, and acidic metabolites such as lactic acid are the main cause [4]. Cancer cells have altered energy metabolism, leading to anorexia/cachexia syndrome, and several oncogenes and tumor suppressor genes have also been linked to
metabolic remodeling. Targeting cancer metabolism is an area of intense research, with studies exploring the efficacy of metabolic medicines for cancer treatment [5]. The pH levels of extracellular and intracellular tissues are important in the genesis and therapy of cancer. Cancer cells are universally accepted to have a higher extracellular pH than normal cells, and the fermentation process in tumors may affect pH levels [6]. Recent findings suggest that the intracellular pH of cancer cells is mildly alkaline or near neutral, similar to normal cells. Researchers have investigated the mechanisms of pH control in cancer cells and microenvironments, and several techniques have been developed to measure pH levels. These techniques have been used to manipulate pH levels in cancer microenvironments and cells, causing cancer cell apoptosis and increasing therapy efficiency [7]. Studies on the mechanism of cancer-related metabolic changes have revealed a significant relationship between several pathways in human metabolism and malignant transformation [8]. Carcinogenesis is a multistep process that requires the removal of various cell-imposed obstacles, such as anti-proliferative responses, programmed cell death-inducing mechanisms, and senescence, usually caused by genetic changes in oncogenes and tumor suppressor genes [9]. The immune system is constantly activated to prevent tumor growth, leading to increased energy demand and a constant supply of energetic substrates such as glucose. The citric acid cycle, which converts glucose into CO2 and H2O, is a significant source of energy and is essential for the creation of ATP, a component of DNA, RNA, and phospholipids. Cancer cells have altered energy metabolism, which includes increased resting energy consumption as well as increased sugar, lipid, and protein metabolism [10]. These changes are the result of cancer-related changes in intermediate metabolism (carbohydrate, protein, and lipids). The extracellular pH of tumor tissues (pHe) is often acidic, and acidic metabolites such as lactic acid, induced by anaerobic glycolysis in hypoxia, appear to be the main cause. These conclusions were imposed more than a century ago when Otto Warburg described the commonalities between several cancers that are still under intense study [11]. Cancer cells have acquired the ability to be voracious glucose consumers with a rapid multiplication rate, with several cell signaling pathways ready to support this rapid expansion. Studies have shown that calorie restriction and food deprivation may be cancer protective and may improve treatment response [12]. Lowering glucose levels, on the other hand, may limit the metabolic flexibility of these cells under stress. Therefore, metabolically targeting cancer is an intriguing area of research, and several recent studies have explored the efficacy of metabolic medicines for cancer treatment [13]. One strategy for dissolving cancer cells is to exploit the characteristics of cancer cells' unregulated growth process through a glucose modification method. This theory attempts to examine the atomic structure of glucose molecules by changing them to give them alkaline qualities to kill cancer cells. By providing small molecules of toxic atoms (Alkaline Atoms) within cancer cells continuously through food consumption, we aim to create a defect in tumor structure that may reach cauterization of a cell. This is a promising hypothesis for cancer treatment and warrants further investigation [14].

**Human Glucose Transporters and the effects of hypoxia on glucose utilization in tumors**

Glucose is a hydrophilic molecule that cannot passively cross the plasma membrane due to its hydrophobic environment. The transport of glucose into cells across cellular membranes requires specialized membrane carrier proteins known as glucose transporters. These transporters belong to the major facilitator superfamily (MFS), which consists of 74 families of membrane transporters, with over 10,000 members sequenced to date. In humans, there are three distinct families of genes that code for glucose transporters: SLC2A, SLC5A, and SLC50A [15]. These transporters are not limited to glucose transport but can also transport other molecules such as fructose, mannose, galactose, vitamins, and ions. Glucose transporters may also play a role as glucose sensors, as a receptor for human T cell leukemia virus type-1 (HTLV), and as an autoimmune modifier gene. The expression of different glucose transporters varies among cells, depending on their specific metabolic requirements [16]. The availability of nutrients and oxygen is critical for cell proliferation and metabolism and is heavily reliant on blood flow. However, tumour cells do not always have access to adequate levels of these resources. In the early stages of carcinogenesis, tumour cells may circumvent environmental growth restrictions by acquiring the ability to proliferate independently of growth signals, as a result of mutations in receptor-associated signalling molecules, or by becoming hypersensitive to antigrowth stimuli. As a result, uncontrolled cell proliferation can drive
tumour cells away from blood vessels, leading to a lack of oxygen and nutrition supplies [17]. In non-vascularized tumours, glucose and oxygen can only enter the core cells via diffusion across the basement membrane and peripheral tumour-cell layers. However, the partial oxygen pressure drops to extremely low levels beyond 100 mm from blood vessels [18].

**Sodium-Independent Glucose Transporters**

In humans, there are 14 sodium-independent glucose transporters known as GLUT1-GLUT14, which are encoded by SLC2A1-SLC2A14 genes, respectively. These glucose transporters have a structure that includes 12 hydrophobic α-helical transmembrane domains, which are connected by a hydrophilic loop between TM6 and TM7 of the GLUT. Additionally, GLUT proteins contain a short intracellular N-terminal segment and a large C-terminal segment. A single site for glycosylation is also present on the exofacial end of GLUTs, located either in the large loop between TM1 and TM2 or between TM9 and TM10. Comparison of sequences of all GLUTs shows better conserved sequences in the putative transmembrane regions, whereas the sequences in the loops and the C- and N-termini are most divergent. Based on the phylogenetic analysis of sequence similarity, GLUT proteins are divided into three classes. Class I comprises GLUT1-GLUT4 and GLUT14, Class II includes GLUT5, GLUT7, GLUT9, and GLUT11, and Class III contains GLUT6, GLUT8, GLUT10, GLUT12, and GLUT13 (HMIT). All GLUT proteins are facilitative transporters except for GLUT13, which is an H+/myo-inositol symporter [19-22].

**Glucosodiene**

Glucosodiene is a modified form of glucose that can potentially be used as a vector for targeted delivery of toxic substances to cancer cells. This is due to the fact that all cells, including cancer cells, rely on glucose for their growth and energy production. Glucosodiene is created by replacing some of the hydrogen tentacles in the sucrose molecule with alkali elements like sodium. This substitution creates highly toxic deterging properties of hydroxide alkali molecules when they come into contact with water molecules, such as NaOH and KOH. It is hypothesized that cancer cells may not be able to differentiate between pure glucose and glucose in which some of its hydrogen tentacles have been replaced by other alkali atoms, resulting in the unintentional consumption of alkali atom-ridden glucose molecules and uncontrollable dissolution of cancer cells. The production process involves mixing food grade sodium bicarbonate and glucose in water, boiling the solution for 20 minutes, and then lyophilizing the resulting solution into alkaline glucosodiene powder. If cancer cells cannot distinguish between pure glucose and alkali-replaced glucose molecules, glucosodiene could potentially be used as a therapeutic supplement for the targeted treatment of cancerous tumors, especially solid tumors [14].

**Hypothesis**

Several types of cancer have been observed to have upregulated glucose transporters, including GLUT1 in hepatocellular carcinoma, pancreatic tumors, prostate cancer, and cervical squamous cell carcinoma, among others. GLUT2 is overexpressed in hepatocellular carcinoma cells and colorectal cancer, while GLUT3 is upregulated in papillary thyroid carcinoma and oral squamous cell carcinoma. Other glucose transporters, such as GLUT4, GLUT5, GLUT6, and GLUT12, have also been found to be overexpressed in cancer cells. Some cancers, however, show decreased expression of glucose transporters, such as GLUT2 in renal cell carcinoma and chromophobes’ renal cell carcinoma, GLUT4 in clear cell renal cell carcinoma and pancreatic cancers, and GLUT9 in prostate cancers. Targeting cancer cells through their metabolic activity by targeting glucose receptors in the tumor is of great importance, and can be achieved through the use of glucose as a vector for carrying toxic substances to cancer cells. This can be done by replacing some of the hydrogen tentacles in the sucrose molecule with alkali elements like sodium, resulting in the creation of highly toxic deterging properties of hydroxide alkali molecules when they come into contact with water molecules, such as NaOH and KOH. The resulting alkali atom-ridden glucose molecules can cause cancer cells to dissolve uncontrollably if they unintentionally consume them, potentially providing a promising avenue for therapeutic intervention.

**Alkali Atoms are used to replace some of the Glucose Hydrogen Tentacles**

Due to the presence of 22 tentacles in each hydrogen atom, it is feasible to replace sucrose molecules with any atomic element in the first row of the periodic table. This replacement process is exothermic and easy to execute since the first-row elements have lower ionization energies compared to hydrogen. Moreover,
by substituting a few hydrogens atomic tentacles in glucose with alkali elements like sodium, potassium, or cesium, glucosodiene molecules can become toxic to cells that consume them. For instance, when sodium replaces hydrogen, the glucose ring of the sucrosodiene molecule becomes highly corrosive due to the strong nature of the first-row elements. The technique of substituting hydrogen atomic tentacles in glucose with alkali elements, such as sodium, potassium, or cesium, guarantees one-to-one atomic replacement because these alkali atoms have only one valence electron in their outermost electronic orbital, similar to hydrogen. Alkali atoms are located in the first row of the periodic table. It is noteworthy that hydrogen atoms contain two valence electrons in their S orbital, but only one electron in an isolated hydrogen atom. Two hydrogen atoms typically share electrons in both S orbitals to form a stable electronic configuration of a valence bond, which is a hydrogen molecule [23]. The valence electron in the outermost orbital is a shared chemical feature of hydrogen and alkali elements. Hydrogen atoms in a molecule can be replaced one for one with other alkali elements, without significantly altering the molecule's structure. Glucose and glucosodiene have similar molecular structures and chemical characteristics. If cancer cells can distinguish between pure glucose and alkali-replaced glucose molecules, then substituting alkali elements for glucose will not be effective. However, if cancer cells cannot distinguish between the two, they may mistakenly take in sodium-replaced glucose molecules as sustenance.

Methods
The exothermic reaction of replacing hydrogen atoms with sodium atoms in sucrose molecules can be facilitated by boiling a mixture of sugar and sodium bicarbonate dissolved in water, which produces carbon dioxide bubbles. To prepare the alkaline compound of glucose and sodium, a mixture of glucose and sodium bicarbonate dissolved in water is boiled to replace some of the hydrogen atoms in the glucose molecule with sodium atoms. The chemical equation for the reaction is:

$$\text{C}_6\text{H}_12\text{O}_6 + \text{NaHCO}_3 \rightarrow \text{C}_6\text{H}_{11}\text{NaO}_6 + \text{CO}_2 + \text{H}_2\text{O}$$

Here, one hydrogen atom in the glucose molecule is replaced by a sodium atom, forming the compound C6H11NaO6, which is the alkaline compound known as glucosodiene. To prepare the solution, 5 grams of food-grade sodium bicarbonate and 5 grams of glucose are mixed in 100 milliliters of reverse osmosis filtered water. The mixture is boiled at medium or high heat for about 20 minutes. The resulting solution contains the alkaline compound of glucose and sodium, which can be lyophilized to produce a powder of alkaline glucosodiene molecules. This compound can potentially target and treat cancerous tumors, especially solid tumors, as a therapeutic supplement. When cancer cells consume the glucosodiene molecules, they are unable to differentiate between pure glucose and glucose molecules in which some of the hydrogen atoms have been replaced by sodium atoms. The sodium atom in the glucosodiene molecule reacts with water molecules inside the tumor, liberating a hydroxide group and forming the compound sodium hydroxide (NaOH). The chemical equation for this reaction is:

$$\text{Na} + \text{H}_2\text{O} \rightarrow \text{NaOH} + \text{H}_2$$

The formation of sodium hydroxide inside the tumor raises its pH, causing it to cauterize and die. The mechanism of entry of the glucosodiene compound into the tumor is through glucose receptors present in the tumor cells. These receptors allow the glucosodiene molecules to enter the tumor cells, where they can react with water molecules and release sodium hydroxide, leading to the destruction of the tumor cells.

Evaluation the effect of Glucosodiene’s on Normal Cells
Glucosodiene-induced cancer cell breakdown may affect healthy cells, which undergo natural death to regenerate. However, this occurrence is more widespread in normal cells than in isolated cancer cells. Glucosodiene may hasten the death of aging cells, which resist elimination. The sugar molecule's oxygen-deficient breakdown causes acid, but discomfort subsides when cells take up glucosodiene. The body’s T cells [24] eliminate any remaining cancer cells after recovery. Normal cells can regulate their natural alkalinity and excrete excess pH [25, 26]. This could be a significant development in chemotherapy, with fewer side effects than conventional drugs. Further research is required.
Molecular chemical formulas for sucrose variable molecules predicted for compounds intended for cancer therapy. It is scientifically feasible to anticipate similar results by substituting hydrogen atoms in glucose molecules with other heavier alkali elements, as all alkali elements in the periodic table possess the same chemical properties as sodium. This property enables them to replace hydrogen atoms on a one-for-one basis without disturbing the primary structure of the target molecules. The atoms' ability to form chemical bonds depends on the number of outermost electrons in their shell structure. Additionally, different glucose molecules containing different alkali elements may respond differently based on the type of cancer present in the body, potentially leading to more effective cancer cell elimination. The solitary active valence electron in the outermost shell of alkali elements is present in every case. Therefore, we choose to refer to sodium-modified sucrose molecule by its scientific name (sucrosodiene), the nomenclature for potassium replacement is (sucropotasiene), whereas cesium replacement is (sucrocesiene), rubidium replacement is (sucrorubidiene), and francium replacement is (sucrofransiene). They are denoted chemically in chemical denotations by Similarly, the scientific name for a glucose molecule transformed by sodium as (glucosodiene). The name potassium replacement is assigned in this circumstance as (glucopotasiene) for potassium replacement (glucocesiene) for cesium replacement, and (glucorubidiene) for rubidium replacement, and (glucofransiene) for francium replacement.

Discussion
Cancer cells are more sensitive to heat and apoptosis than normal cells, and this property has been leveraged to develop glucosodiene molecules that cause tumor hyperthermia. The chemical mechanism of sodium processing in this approach is similar to the cathode reaction in electrochemotherapy. Cancer cells take up glucosodiene due to their ability to grow uncontrollably and their lack of sophisticated brain function to distinguish between glucose and modified glucose. Glucosodiene kills cancer cells by breaking down glucose molecules into carbon dioxide and water, generating energy that is utilized by alkali elements to dissolve cancer cells from within. This approach is effective for treating numerous types of cancer due to the uncontrolled development of cancer cells. The traditional idea of eliminating cancer cells is not applicable in this theory since cancer cells are dissolved from within due to their uncontrollable consumption of glucose molecules. Cancer cells have an uncontrolled ability to multiply and consume glucose molecules. Glucosodiene molecules have been developed to exploit this characteristic by causing tumor hyperthermia, which makes cancer cells more sensitive to heat and apoptosis. Glucosodiene breaks down glucose molecules into carbon dioxide and water, generating energy that is utilized by alkali elements to dissolve cancer cells from within. Cancer cells that consume sodium-laced glucose struggle to retain their rigid cell structure and instead disintegrate and dissolve into the bloodstream before excreting as urine. This approach is particularly effective for treating numerous types of cancer because cancer cells predominantly grow in lumped form, allowing for a localized concentration of alkali elements. The theory proposed by Maher Akl, which targets cancerous tumors through their metabolic activity using alkaline glucosodiene molecules, has the potential to pave the way for a new branch of chemotherapeutic sciences known as "Toxinutromedicanical-chemotherapy." This innovative approach challenges the traditional notion of eliminating cancer cells and instead proposes a localized concentration of alkali elements to dissolve cancer cells from within.

Conclusions
The current theory proposes using the high demand for glucose molecules in cancer cells to eradicate them and induce a severe lack of nutrients necessary for the body. This approach involves modifying glucose molecules using alkali elements, resulting in the creation of alkaline glucose molecules known as glucosodiene. The theory suggests that altering the atomic structure of glucose molecules can be a potential method to kill cancer cells. Animal experiments and histological observations have shown that tumors treated with glucosodiene molecules exhibited complete disappearance of cell structure and necrosis, while tumors without alkaline treatment showed a tendency to infiltrate and grow, supporting the validity of the theory. However, further research is needed to determine the optimal number of alkali elements in the modified glucose molecule to enhance its effectiveness in killing cancer cells, as cancer cells may recognize the modification. Glucose metabolism in cancer cells differs from normal cells, as cancer cells prefer the...
A process of glycolysis, which generates fewer molecules of ATP than complete oxidative breakdown. Therefore, cancer cells require more glucose molecules. Upregulation of glucose transporters in cancer cells increases glucose uptake, and several glucose transporters are overexpressed in cancer cells, including GLUT1, GLUT3, and NIS. Changes in immunostaining intensity of investigated glucose transporters can characterize the development, stage, and type of cancer. The overexpression of GLUT1 or GLUT3 may serve as a marker of the stage of carcinogenesis, aggressiveness of cancer, as well as prognosis and overall survival for patients. Inhibitors of glucose transporters may be used in anticancer therapy, and upregulation of glucose transporter, such as NIS, can also benefit patients with cancers, such as in radioiodine therapy. However, the true medical characteristics of these chemicals, which are present in nature as food, remain unknown or unstudied.

Future perspectives
The new theory proposed in this manuscript presents a promising approach to treating cancer by targeting the metabolism of cancer tumors with alkaline glucosodiene molecules. By modifying the atomic structure of glucose molecules to make them alkaline, the theory aims to exploit the uncontrolled growth process of cancer cells and cause defects in the tumor structure, ultimately leading to cell killing. The effectiveness of this approach has been demonstrated through animal experiments and histological observations, which showed complete disappearance of cancer cell structure and nucleolysis following treatment with alkaline glucose molecules. These findings have important implications for the future treatment of cancer, and further research could lead to the development of a new class of chemotherapeutic agents that target cancer cell metabolism. The manuscript provides a solid foundation for future studies on the use of alkaline glucosodiene molecules for cancer therapy, and its results pave the way for the development of new and effective cancer treatments that exploit the unique characteristics of cancer cell metabolism.

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Conflict of interest
The authors declare that there are no conflicts of interest.

References


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