SCIENTIFIC RESEARCH MONOGRAPH

by Rik J. Deitsch

Natural Cellular Defense

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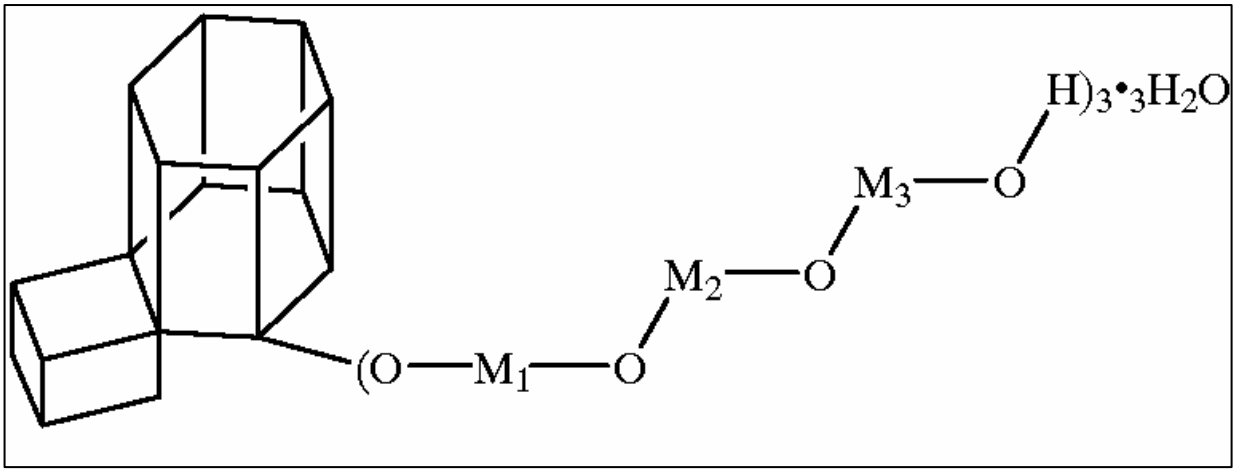

(where M_1 is silicon, M_2 is magnesium, M_3 is aluminum)

4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate

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ABOUT THE AUTHOR

Rik J Deitsch

Rik J Deitsch is the Chief Executive Officer of Nutra Pharma Corporation - a publicly traded Biotechnology holding company and incubator dedicated to researching neurological disorders and viral diseases. Mr. Deitsch served as the President of NDA Consulting Inc., a biotechnology research group that provided consulting services to the pharmaceutical industry. Rik J. Deitsch also serves as the Chairman of the Waiora Scientific Advisory Board. In this role, he is responsible for overseeing the direction and development of Waiora's products as they relate to the company's overall product strategy. Leading a team of knowledgeable, well-respected and highly credentialed physicians and healthcare professionals, the Board recommends formulations and ingredients to create efficacious products for Waiora.

Research conducted by Rik J Deitsch provided some of the beginning fundamentals for the development of some powerful new drugs. Mr. Deitsch has several papers and posters on rational drug design using computer simulations. He teaches several courses for Florida Atlantic University's Continuing Education Department and College of Business. Mr. Deitsch also teaches physician CME courses internationally, lecturing on lifestyle choices in the prevention and treatment of chronic disease states. He is also the co-author of *Are You Age-Wise*, a book that reviews current research in healthy aging as it relates to lifestyle choices and supplementation (*www.areyouagewise.com*).

About this Publication

The information presented in this monograph is intended for professional education and is obtained from published research, articles, and books. This monograph is not intended to replace the care of a licensed health professional in the diagnosis and treatment of illness.

Key Points

Natural Cellular Defense is derived from a naturally-occurring mineral zeolite. Zeolites are considered 100 percent safe and non-toxic by the FDA.

Natural Cellular Defense and its constituent zeolite have been shown to have the following functions:

- Retains the natural zeolite property of reducing cancer risk
- Acts as a potent anti-oxidant
- May aid in balancing bodily pH levels
- Acts to chelate and remove heavy metals from the system
- Reduces the incidence of diarrhea
- Improves nutrient absorption in the digestive tract
- Acts to balance digestive pH, thus reducing acid reflux
- Acts to stabilize immune system function (immunomodulatory)
- Acts as a broad spectrum anti-viral agent

1. Introduction

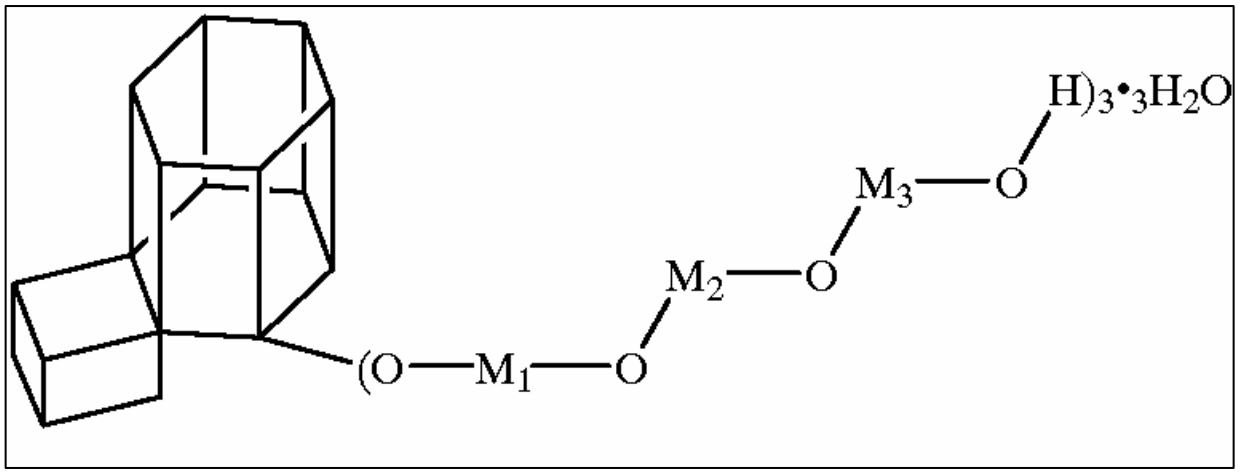
Zeolites are hydrated natural and synthetic microporous crystals with well-defined structures containing AlO_4 and SiO_4 tetrahedra linked through the common oxygen atoms. Most common natural zeolites are formed by alteration of glass-rich volcanic rocks (tuff) with fresh water in playa lakes or by seawater. The structures of zeolites consist of three-dimensional frameworks of SiO_4 and AlO_4 tetrahedra. The aluminum ion is small enough to occupy the position in the center of the tetrahedron of four oxygen atoms, and the isomorphous replacement of Si^{4+} by Al^{3+} produces a negative charge in the lattice. The net negative charge is balanced by the exchangeable cation (sodium, potassium, or calcium). Zeolites have been extensively used in various industrial applications based on their properties to act as catalysts, ion exchangers, adsorbents, and detergent builders. It is also known that silicates and aluminosilicates possess biological activity, either positive or negative. Talc and silica have been used in skin care for many decades, while well defined structures and catalytic activity make aluminosilicates an attractive model system for protein and enzyme mimetics. Recent results have also demonstrated that natural, biologically nontoxic zeolites are very effective as glucose adsorbents, and this has been suggested as a potential medication for individuals suffering from diabetes mellitus.

Natural zeolites are crystalline, hydrated alluminosilicates of alkali and alkaline earth cations, having three-dimensional structures. They are characterized by the ability to lose and gain water reversibly and to exchange constituent cations (positively-charged particles) without major change of structure. The basis of interest in the biological effects of zeolites concerns one or more of their physical and chemical properties, such as ion exchange capacity, adsorption and related molecular sieve properties.

For the purpose of this Review, only the properties of zeolites that address its effects as a supplement will be investigated. Although the mechanism of action of many of these claims is not well understood, the complex structure of the zeolite in question may provide clues to this mechanism. The zeolite acts as a 'cage' and allows the molecule to trap a variety of ions and compounds. By trapping charged particles, the zeolite can: act as an antioxidant, balance pH levels, lower incidence of heavy metal damage, improve nutrient absorption and reduce gastric reflux. The cage may also trap potential carcinogens, thereby reducing the risk of developing cancer. The anti-viral activity of zeolites may be founded in the molecule's ability to trap viral particles during assembly, thereby preventing viral assembly and proliferation.

This paper will endeavor to categorize the major claims made by Natural Cellular Defense.

2. Description

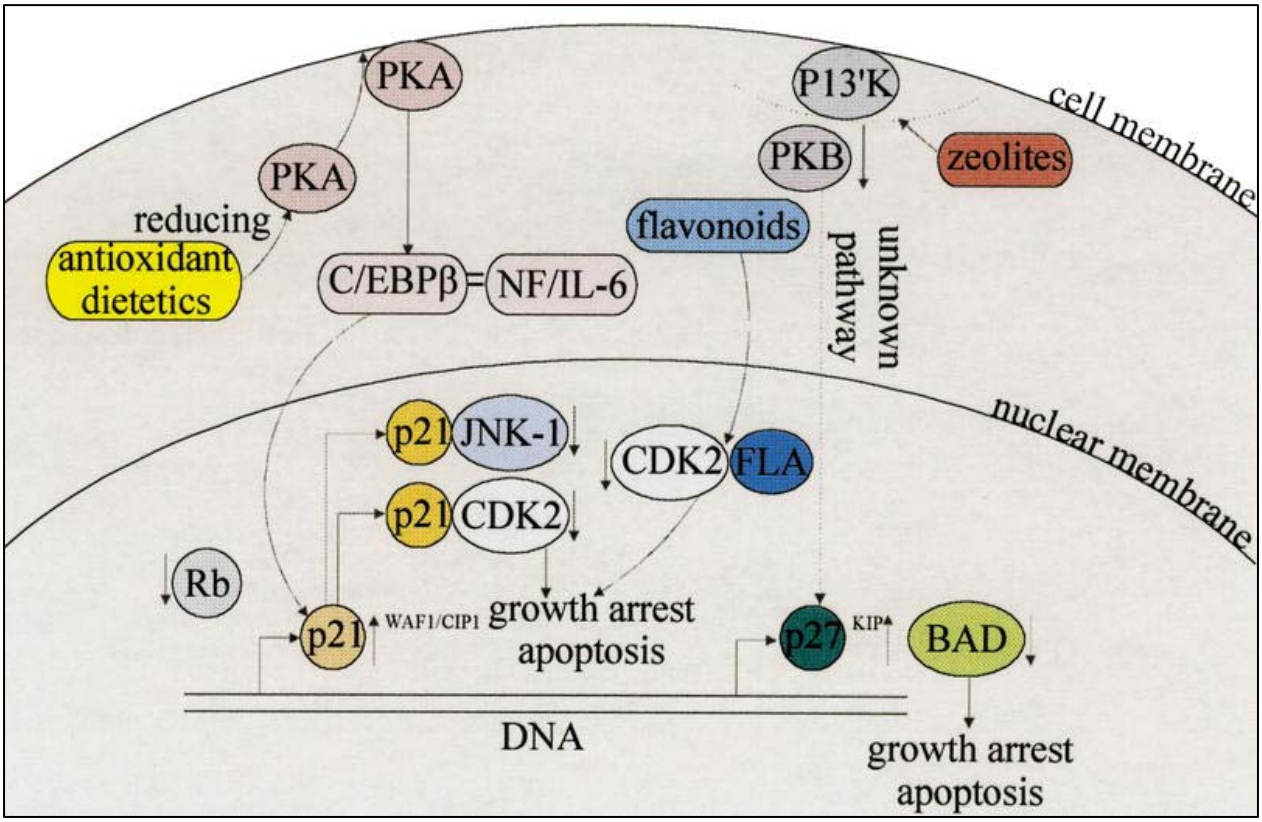


(where M_1 is silicon, M_2 is magnesium, M_3 is aluminum)

4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate

Natural Cellular Defense is identified generically as 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate ($3Mg^{2+}$ $3Al_2$ O_3 $3SiO_2$ $3H_2O$), which is a magnesium aluminosilicate. It is in the acetate, sulfate, chloride, or brominate form. Natural Cellular Defense is a slightly white, odorless, tasteless liquid. The zeolite particles are negatively charged in the entire pH range (pH 1–11). Electron microscopy shows an absence of fibers, and most particles are round with a very rough surface. The absence of fibrous, positively charged particles is encouraging since such particles are present in asbestos and erionite zeolites, which are highly carcinogenic and mutagenic. In addition, activated zeolite particles do not catalyze the production of hydroxyl radicals, unlike asbestos or erionite. It seems that absence of fibrous particles capable of producing hydroxyl radicals makes this zeolite sample nontoxic and noncarcinogenic, at least when applied orally.

3. Reducing Cancer Risk Factors



Mechanisms of action of potential anti-cancer supplements

The development of modern industry has caused increasingly serious pollution in the environment constituting a catastrophic health risk - including cancer. Cancer prevention is thus one of the challenges facing scientists in the 21st century and removal of carcinogens from the environment is an important step. Nitrosamines are probably the most widespread carcinogens, existing in the workplace, processed meats, cigarette smoke and beer. They are even produced in the stomach by the reaction of secondary amines and nitrite (NO_2^-) both taken from foods. A 1976 report of German scientists showed that the uptake of nitrosamines for a person was as much as 700ng daily. The occurrence of gastric cancer was related to the nitrosamines in the stomach. Many carcinogenic agents like nitrosamines or their precursors enter the human stomach through diet and drinking. No matter how carefully food is prepared; there is a level of nitrosamine content. Environmental pollution makes this hidden trouble more serious, because of the contaminated food and polluted atmosphere. However, although nitrosamines are well-known carcinogenic substances, they require metabolic activation before reaction with DNA to cause mutation and cancer. Therefore, it is

possible and necessary to trap the nitrosamines in the digestive tract provided a selective adsorbent material is employed. To seek this functional material, zeolites are considered as the best candidates. Zeolites and molecular sieves have been employed in slow release drugs, enzyme mimetic drugs, anti-tumor drugs and additives in cigarettes to remove carcinogenic agents like nitrosamines.

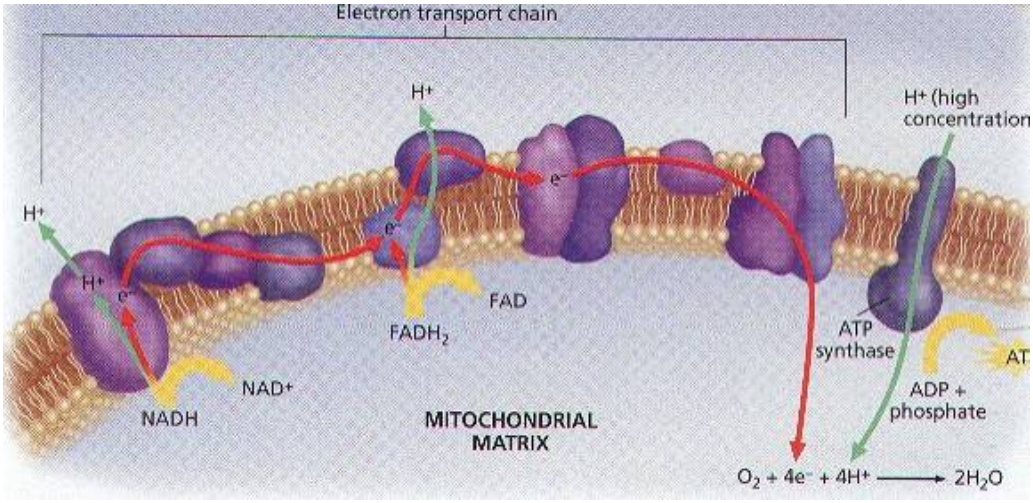
Numerous natural compounds are commonly used for the treatment of various diseases, including green tea and soybean extracts. Recent findings indicate that dietetic products and antioxidant compounds also have a beneficial effect particularly in cancer patients. In many cases the exact mechanism of their action is not fully understood. In the published literature the effect of natural zeolite particles on development of several cancer models in-vivo and in-vitro was studied. It has been found that zeolites may act as anticancer therapeutic agents in in-vivo animal studies and in tissue culture cell models. The constituent natural zeolite from Natural Cellular Defense applied orally in mice and dogs suffering from a variety of tumor types led to a significant shrinkage of some tumors and improvement in overall health status in some animals. The range of effects was diverse, ranging from negative antitumor response, to normalization of biochemical parameters, prolongation of life span, and decrease in tumor size. The best results in animal models were observed in the treatment of skin cancer in dogs, suggesting that adsorption of some active components is responsible for zeolite activity (direct contact action). Complementary studies performed in tissue culture indicated that zeolite treatment affects proliferation and survival of several cancer cell lines. Addition of zeolite inhibited cell proliferation in a concentration-dependent manner, in part due to induction of inhibitors of cycline dependent kinases and induction of programmed cell death. The data indicates that zeolite treatment might affect cancer growth by attenuating survival signals and inducing tumor suppressor genes in treated cells. In addition, toxicology studies on mice and rats demonstrated that the treatment does not have negative effects.

Finely ground natural zeolite powder has also been shown to induce activation of $p21^{WAF1/CIP1}$. As a universal inhibitor of cyclin-dependent kinases and one of the target genes of the tumor suppresser p53, $p21^{WAF1/CIP1}$ can act as a tumor suppresser through its ability to control cell cycle progression. Activation of $p21^{WAF1/CIP1}$ may halt the growth of tumors by directly suppressing growth signals. However, tissue culture experiments demonstrated that activated zeolite particles inhibit protein kinase B/akt, another kinase involved in antiapoptotic processes and cancer promotion. This happens only when growth of cells in tissue cultures is stimulated by the addition of growth factors. Zeolite particles might adsorb growth factors or prevent interaction of protein kinase B with membranes, where it is phosphorylated by phosphatidylinositol 3-kinase. It has recently been shown that inactivation of protein kinase B by, for example, the novel tumor suppressor molecule PTEN also results in induction of the tumor suppressor-CDK inhibitor $p27^{KIP1}$.

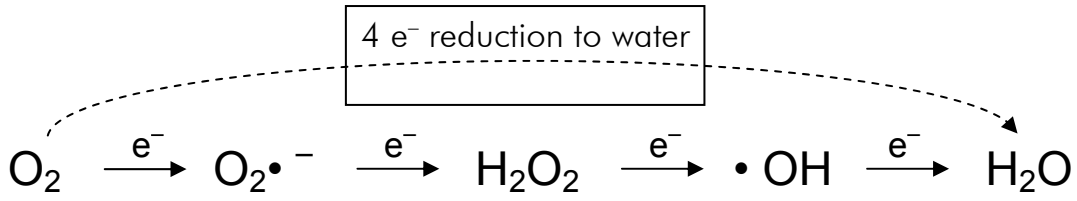
There also seems to be a relationship between $p21^{WAF1/CIP1}$ and another protein kinase involved in cell "decisions" about proliferation, arrest or apoptosis, namely c-Jun N-terminal kinase (JNK) 1. JNK-1 is a member of the recently discovered stress-activated protein kinases. Interestingly, while in reaction to stress such kinase activation results in apoptosis, its activation in some cancer cells actually promotes uncontrolled proliferation. This is particularly obvious in the human lung adenocarcinoma cell line A549. Research has shown that $p21^{WAF1/CIP1}$ inhibits JNK-1, and recent work indicates that those two molecules form a tight complex. Inactivation of JNK-1 may be part of the reason why antioxidants enhance cytotoxicity of chemotherapeutic agents towards cancer cells, while, on the other hand, they protect neurons from apoptosis caused by free radical damage.

4. Potent Antioxidant

Oxygen is actually a toxic byproduct of the metabolism of lower cell organisms, and the body uses it to produce energy. Oxygen free radicals are created through a biochemical process at the cellular level. Oxygen and glucose are used to produce adenosine triphosphate (ATP) through oxidative phosphorylation.

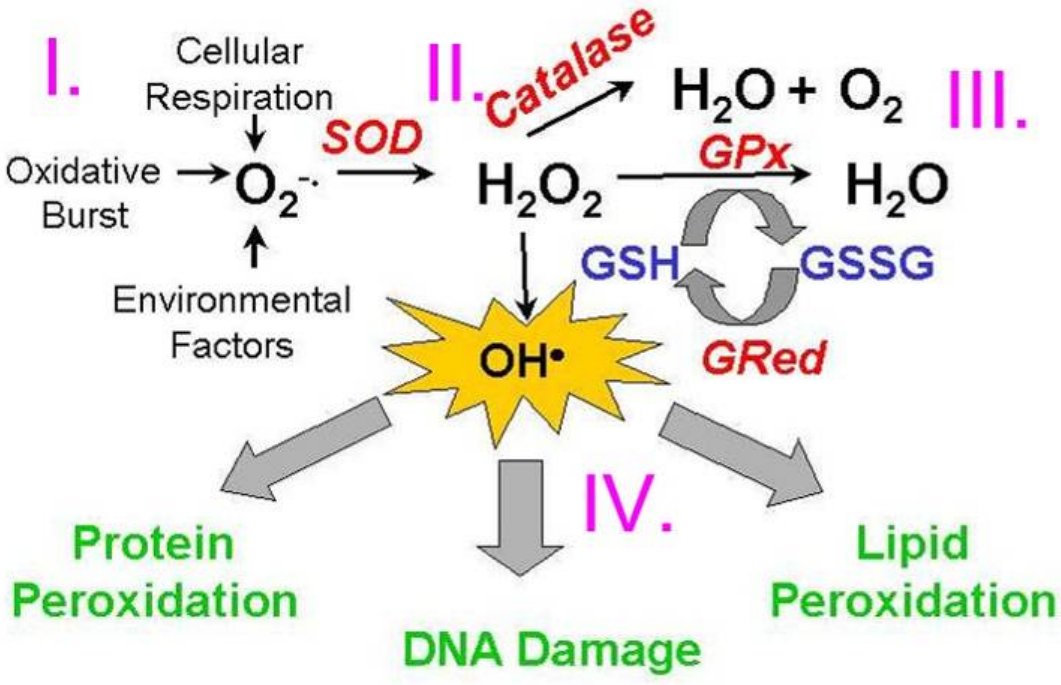


Oxidative phosphorylation is a process during which electrons not bound to an oxygen molecule move from one such molecule to another seeking to bind to other unpaired electrons. These oxygen molecules that contain unpaired electrons are called oxygen free radicals.



One percent to three percent of the oxygen we breathe creates oxygen free radicals, as do natural aging and chronic disease. In small amounts, some oxygen free radicals help to clean cells by taking part in phagocytosis, a function of a normal immune system, but as production of these radicals increases, they begin to attack and destroy cells. As their levels increase, oxygen free radicals attack and oxidize DNA, genetic molecular material that controls cell growth and development. Oxidation of DNA may lead to the adverse effects of aging, such as wrinkled skin, organ dysfunction, and cancer. As oxygen free radicals travel through the

blood, they set the stage for heart, blood vessel, cerebrovascular, and skeletal muscle disease by making it more difficult for cells to repair themselves.



Free radicals can damage the intima in blood vessels, causing inflammation and thereby leading to increased plaque formation, which can contribute to stroke and cardiac disease. Oxygen free radicals also free iron, which is usually tightly bound to protein molecules. Excessive levels of free iron are associated with Alzheimer disease, dementia, and Parkinson disease. In addition, oxygen free radicals cause an increase in neuronal loss by changing DNA structure. When a significant number of neurons are degraded, the central nervous system can no longer maintain homeostasis, and physiologic functions such as receptor-mediated signal transmission necessary to memory may become defective or be lost.

The cage structure of Natural Cellular Defense may help to trap free radicals, thereby inactivating them and preventing further damage to the surrounding tissue. This is completely unlike the action of the classic antioxidants. Compounds that have antioxidant properties include the vitamins A, C and E as well as the mineral selenium. There are also a host of other antioxidant compounds, including: flavanoids, catechins and specific enzymes. All of these antioxidants function by acting either as an electron donor or electron acceptor. This action stabilizes the free radical and halts the free radical cascade which would normally lead to further damage of the system and an eventual catastrophe (heart attack, stroke, cancer, etc..). All of these classic antioxidants have the ability to exist with one extra electron or one less electron without becoming a free radical in turn. Natural Cellular Defense may support

the action of these antioxidants through an entirely different mechanism. By literally trapping highly reactive free radicals, these dangerous compounds are inactivated and are easily eliminated from the system.

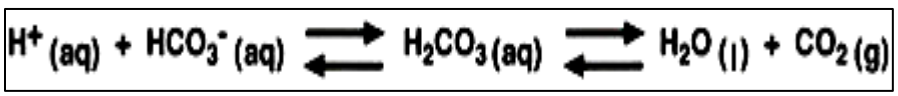
5. Systemic pH levels

The body has a wide array of mechanisms to maintain homeostasis in the blood and extracellular fluid. The most important way that the pH of the blood is kept relatively constant is by buffers dissolved in the blood. Other organs help enhance the homeostatic function of the buffers. The kidneys help remove excess chemicals from the blood. It is the kidneys that ultimately remove (from the body) H^+ ions and other components of the pH buffers that build up in excess. Acidosis that results from failure of the kidneys to perform this excretory function is known as metabolic acidosis. However, excretion by the kidneys is a relatively slow process, and may take too long to prevent acute acidosis resulting from a sudden decrease in pH (*e.g.*, during exercise). The lungs provide a faster way to help control the pH of the blood. The increased-breathing response to exercise helps to counteract the pH-lowering effects of exercise by removing CO_2, a component of the principal pH buffer in the blood. Acidosis that results from failure of the lungs to eliminate CO_2 as fast as it is produced is known as respiratory acidosis.

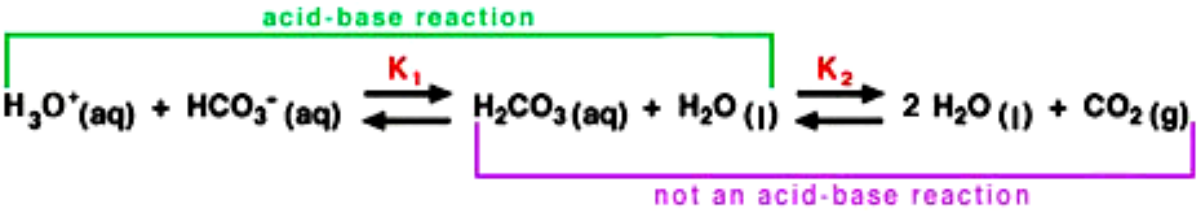
The kidneys and the lungs work together to help maintain a blood pH of 7.4 by affecting the components of the buffers in the blood. Therefore, to understand how these organs help control the pH of the blood, we must first discuss how buffers work in solution.

Acid-base buffers confer resistance to a change in the pH of a solution when hydrogen ions (protons) or hydroxide ions are added or removed. An acid-base buffer typically consists of a weak acid, and its conjugate base (salt). Buffers work because the concentrations of the weak acid and its salt are large compared to the amount of protons or hydroxide ions added or removed. When protons are added to the solution from an external source, some of the base component of the buffer is converted to the weak-acid component (thus using up most of the protons added); when hydroxide ions are added to the solution (or, equivalently, protons are removed from the solution), protons are dissociated from some of the weak-acid molecules of the buffer, converting them to the base of the buffer (and thus replenishing most of the protons removed). However, the change in acid and base concentrations is small relative to the amounts of these species present in solution. Hence, the ratio of acid to base changes only slightly. Thus, the effect on the pH of the solution is small, within certain limitations on the amount of H^+ or OH^- added or removed.

By far the most important buffer for maintaining acid-base balance in the blood is the carbonic-acid-bicarbonate buffer. The simultaneous equilibrium reactions of interest are:



To more clearly show the two equilibrium reactions in the carbonic-acid-bicarbonate buffer, the equation is rewritten to show the direct involvement of water:



The equilibrium on the left is an acid-base reaction. Carbonic acid (H_2CO_3) is the acid and water is the base. The conjugate base for H_2CO_3 is HCO_3^- (bicarbonate ion). Carbonic acid also dissociates rapidly to produce water and carbon dioxide, as shown in the equilibrium on the right. This second process is not an acid-base reaction, but it is important to the blood's buffering capacity. Note that as acid is added, the pH decreases and the buffer shifts toward greater H_2CO_3 and CO_2 concentration. Conversely, as base is added, the pH increases and the buffer shifts toward greater HCO_3^- concentration.

Other buffers perform a more minor role than the carbonic-acid-bicarbonate buffer in regulating the pH of the blood. The phosphate buffer consists of phosphoric acid (H_3PO_4) in equilibrium with dihydrogen phosphate ion ($H_2PO_4^-$) and H^+. The phosphate buffer only plays a minor role in the blood, however, because H_3PO_4 and $H_2PO_4^-$ are found in very low concentration in the blood. Hemoglobin also acts as a pH buffer in the blood. The hemoglobin protein can reversibly bind either H^+ (to the protein) or O_2 (to the Fe of the heme group). When one of these substances is bound, the other is released. During exercise, hemoglobin helps to control the pH of the blood by binding some of the excess protons that are generated in the muscles. At the same time, molecular oxygen is released for use by the muscles.

Being amphoteric, zeolites are partly soluble in acid or alkaline media, but within the physiological pH range the solubility is generally low. Minimal amounts of free aluminium or silicium from the ingested zeolites are resorbed from the gut. In view of the ion exchange properties, zeolites may be expected to change the ionic content, pH and buffering capacity of their surroundings. The zeolite cage system may exchange for varying excess ions found in the zeolite's immediate environment. This would have the overall effect of buffering the surrounding system and regulating pH to near-physiological levels.

6. Heavy Metals and Chelation

Many toxic heavy metals have been discharged into the environment as industrial wastes, causing serious soil and water pollution. Lead (Pb^{+2}), Copper (Cu^{+2}), Iron (Fe^{+3}) and Chromium (Cr^{+3}) are especially common metals that tend to accumulate in organisms, causing numerous diseases and disorders. They are also common groundwater contaminants at industrial and military installations. The main threats to human health from heavy metals are associated with exposure to lead, cadmium, mercury and arsenic. These metals have been extensively studied and their effects on human health regularly reviewed by international bodies such as the WHO. Heavy metals have been used by humans for thousands of years. Although several adverse health effects of heavy metals have been known for a long time, exposure to heavy metals continues, and is even increasing in some parts of the world, in particular in less developed countries, though emissions have declined in most developed countries over the last 100 years. Cadmium compounds are currently mainly used in re-chargeable nickel-cadmium batteries. Cadmium emissions have increased dramatically during the 20th century, one reason being that cadmium-containing products are rarely re-cycled, but often dumped together with household waste. Cigarette smoking is a major source of cadmium exposure. In non-smokers, food is the most important source of cadmium exposure. Recent data indicate that adverse health effects of cadmium exposure may occur at lower exposure levels than previously anticipated, primarily in the form of kidney damage but possibly also bone effects and fractures. Measures should be taken to reduce cadmium exposure in the general population in order to minimize the risk of adverse health effects. The general population is exposed to lead from air and food in roughly equal proportions. During the last century, lead emissions to ambient air have caused considerable pollution, mainly due to lead emissions from petroleum. Children are particularly susceptible to lead exposure due to high gastrointestinal uptake and the permeable blood-brain barrier. Blood levels in children should be reduced below the levels so far considered acceptable, recent data indicating that there may be neurotoxic effects of lead at lower levels of exposure than previously anticipated. Although lead in petroleum has dramatically decreased over the last decades, thereby reducing environmental exposure, phasing out any remaining uses of lead additives in motor fuels should be encouraged. The use of lead-based paints should be abandoned, and lead should not be used in food containers. In particular, the public should be aware of glazed food containers, which may leach lead into food. Numerous processes exist for removing dissolved heavy metals, including ion exchange, precipitation, phytoextraction, ultrafiltration, reverse osmosis, and electrodialysis. The use of alternative low-cost materials as potential sorbents for the removal of heavy metals has been emphasized recently.

The structures of zeolites consist of three-dimensional frameworks of SiO_4 and AlO_4 tetrahedra. The aluminum ion is small enough to occupy the position in the center of the tetrahedron of four oxygen atoms, and the isomorphous replacement of Si^{4+} by Al^{3+} produces a negative charge in the lattice. The net negative charge is balanced by the exchangeable cation (sodium, potassium, or calcium). These cations are exchangeable with certain cations in solutions such as lead, cadmium, zinc, and manganese. The fact that zeolite exchangeable

ions are relatively innocuous (sodium, calcium, and potassium ions) makes them particularly suitable for removing undesirable heavy metal ions from the environment and bodily systems. One of the earliest applications of a natural zeolite was in removal and purification of cesium and strontium radioisotopes.

Studies have shown that the constituent zeolite in Natural Cellular Defense has a high affinity for trapping lead, cadmium and other potentially harmful heavy metals. Through the process of cation exchange, Natural Cellular Defense may lower overall heavy metal exposure in individuals. This would have a dramatic effect in the risk reduction of certain cancers and heart disease – the two leading causes of death in the United States.

7. Digestive disorders

Natural silicate materials have been shown to exhibit diverse biological activities and have been used successfully as a vaccine adjuvant and for the treatment of diarrhea.

The potential growth promoting action of natural zeolites has been attributed to their high affinity for ammonium ions, resulting to the reduction in the uptake of ammonia produced from deamination of proteins during the digestive processes via the intestinal wall. Ammonia is recognized as a cell toxicant in higher animals and the reduction of the amount which the intestinal epithelial cells are exposed to, could lead to a reduction of epithelial turnover, a sparing of energy and a better nutrient utilization. In other words, the ability of Natural Cellular Defense to trap ammonium ions allows for a less toxic digestive tract and better overall nutrient uptake from food and supplements.

The best known positive biological activity of natural zeolite is its action as an anti-diarrheal drug. Zeolites lower the incidence of death and sickness (diarrheal syndrome) produced by intestinal diseases in swine, rats, and calves. Based on these results a comprehensive study was carried out on anti-diarrheal drugs based on natural zeolite as an active material, in the therapy of acute diarrheal diseases in humans. The research led to approval of the anti-diarrheal drug Enterex for use in humans

Peroral use of encapsulated zeolite powders enriched with vitamins, oligoelements or other ingredients has been claimed to exert beneficial medical effects. Ingestion of zeolites may be considered analogous to the clay eating (geophagia), considered in traditional medicine as a remedy for various illnesses. The bulk of ingested zeolite probably remains undissolved in the gut. In view of the ion exchange properties, zeolites may be expected to change the ionic content, pH and buffering capacity of the gastrointestinal secretions and to affect the transport through the intestinal epithelium. In addition, zeolites could affect the bacterial flora and the resorption of bacterial products, vitamins and oligoelements. The contact of zeolite particles with gastrointestinal mucosa may elicit the secretion of cytokines with local and systemic actions. This creates a healthier digestive tract with beneficial microflora and microfauna.

8. Immune system modulation

Accumulating evidence has indicated that zeolites play an important role in regulating the immune system. Several studies have reported that silica, silicates, and aluminosilicates act as nonspecific immunostimulators similarly to superantigens. Superantigens are a class of immunostimulatory and disease-causing proteins of bacterial and viral origin with the ability to activate relatively large fractions (5–20%) of the T cell population. Activation requires simultaneous interaction of the superantigens with V-domain of T cell receptor and with major histocompatibility complex class II molecules on the surface of antigen presenting cells. Proinflammatory macrophages, which belong to class II MHC antigen-presenting cells, are activated by fibrogenic silicate particulates. Indeed, experiments have shown that removal of MHC class II DP/DR positive cells results in a lack of macrophage stimulation by asbestos. Direct interaction of silicate particles with cells other than lymphocytes has also been identified and described. It seems that mineral particles can trigger alterations in gene expression by initiating signaling events upstream of gene transactivation. Exposure of cells to silicate particles has been shown to lead to activation of mitogen-activated protein kinases (MAPK), protein kinase C, and stress-activated protein kinases. Important transcription factors such as activator protein 1 and nuclear factor B are also activated, and expression of proinflammatory cytokines such as interleukin 1, interleukin 6, and tumor necrosis factor is enhanced. Modifications in receptor activation kinetics or activity of integrins may be responsible for the observed behavior

The aim of one study was to evaluate the antiviral properties of a natural non-toxic zeolite. Herein, a fine powder of micronized zeolite (MZ) was obtained. Different viral suspensions were treated with MZ in concentrations ranging from 0.5 to 50 mg/ml. The viral proliferation was evaluated by optical microscope as percentage of cytopathic effect (CPE). Human adenovirus 5, herpes simplex virus type 1 (HSV 1) and human enteroviruses (coxsackievirus B5 and echovirus 7) were used in the antiviral assay. Concentrations of 0.5 and 5 mg/ml of MZ induced a very low antiviral effect or the antiviral was not observed at all, while concentrations of 12, 25 and 50 mg/ml of MZ induced a significant inhibitory effect upon viral proliferation. MZ inhibited the viral proliferation of HSV 1, coxsackievirus B5 and echovirus 7 more efficiently than adenovirus 5. The antiviral effect of MZ seems to be non-specific and is more likely based on the incorporation of viral particles into pores of MZ aggregates than ion exchange properties of the zeolite. Preliminary results indicate a possibility of therapeutic application of MZ, either locally (skin) against herpesvirus infections or orally in cases of adenovirus or enterovirus infections.

The aim of a prospective, open, and controlled parallel-group study was to investigate the effects of supplementation with another zeolite on the cellular immune system in patients undergoing treatment for immunodeficiency disorder. A total of 61 patients were administered daily zeolite doses for 6 to 8 weeks, during which the patients' primary medical therapy was continued unchanged. Blood and lymphocyte counts were performed at baseline and at the end of the study. Blood count parameters were not relevantly affected in either of the two treatment groups. Zeolite administration resulted in significantly increased CD4+,

CD19+, and HLA-DR+ lymphocyte counts and a significantly decreased CD56+ cell count. No adverse reactions to the treatments were observed.

9. Safety and Tolerability

Food additives and supplements in the U.S.A. are approved by the Food and Drug Administration (FDA) under the Code of Federal Regulations (CFR) Title 21. Zeolites (CFR Title 21 (182.2727)) and Sodium Magnesium or Aluminum Silicates under CFR Title 21 (182.2227) are listed as GRAS (Generally Recognized as Safe), and are permitted as food additives (supplements) without FDA approval. Furthermore, all ingredients (zeolites) are found on the TSCA listing of GRAS chemicals used in every day commerce in the United States.

10. Appendix 1 - References:

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11. Appendix 2 - Patent

US006288045B1

(12) **United States Patent**
Kaufman

(10) **Patent No.: US 6,288,045 B1**
(45) **Date of Patent: Sep. 11, 2001**

(54) **EPITHELIAL CELL CANCER DRUG**

(75) Inventor: **Harvey Kaufman**, Hudson, OH (US)

(73) Assignee: **Lifelink Pharmaceuticals, Inc.**, Stow, OH (US)

(*) Notice: Subject to any disclaimer, the term of this patent is extended or adjusted under 35 U.S.C. 154(b) by 0 days.

(21) Appl. No.: **09/591,701**

(22) Filed: **Jun. 9, 2000**

(51) **Int. Cl.**[7] **C07F 7/02**; A61K 31/695
(52) **U.S. Cl.** **514/63**; 556/173
(58) **Field of Search** 514/63; 556/173

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Primary Examiner—Peter O'Sullivan
(74) *Attorney, Agent, or Firm*—George W. Moxon, II; Brouse McDowell

(57) **ABSTRACT**

A method of treating epithelial cell cancer comprising administering to a mammalian patient diagnosed as having an epithelial cell cancer a therapeutically effective amount of 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate, or its acetate, sulfate, hydrochlorate, or brominate salts. The composition is synthesized from a naturally occurring non-toxic zeolites, and has a 100% kill rate within 72 hours against buccal mucosa and ling squamous epithelial cell cancers. It is not cytoxic to healthy human cells.

19 Claims, 9 Drawing Sheets

(6 of 9 Drawing Sheet(s) Filed in Color)

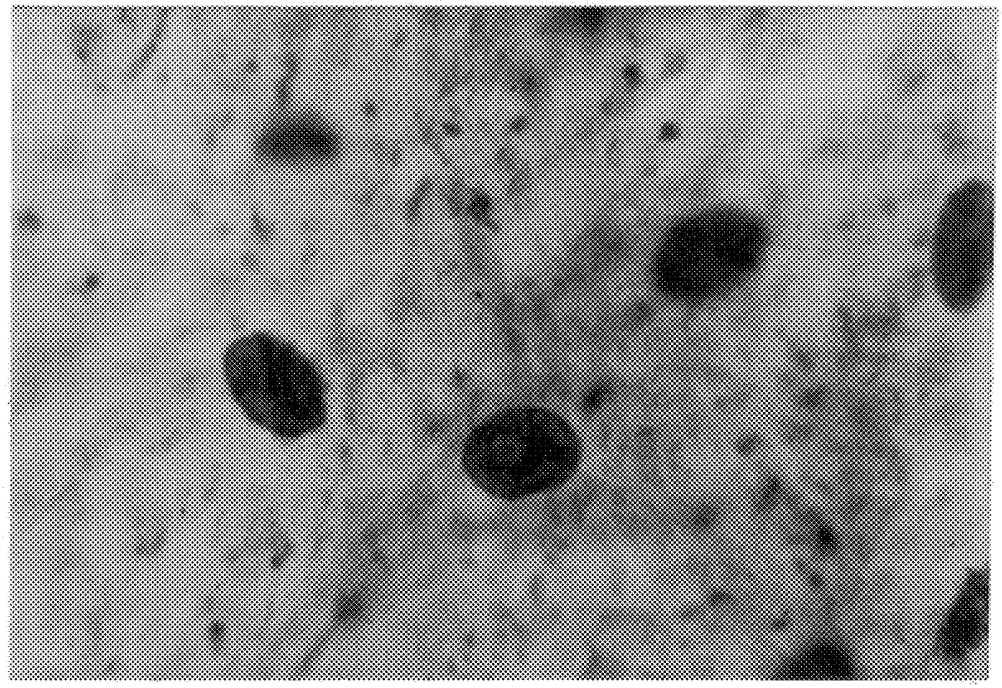


FIG. 1

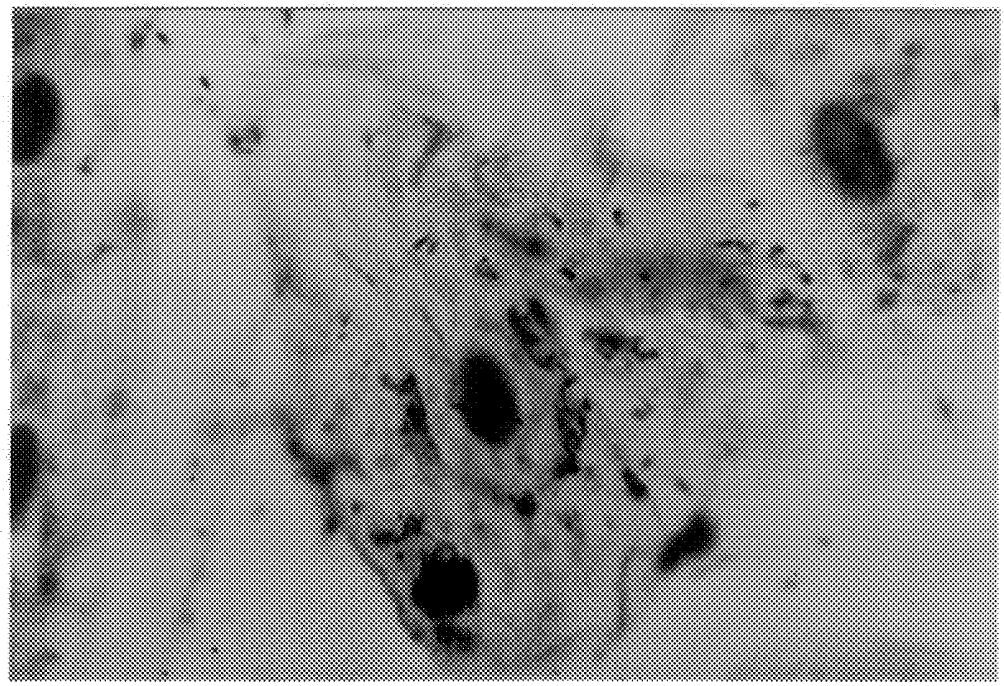


FIG. 2

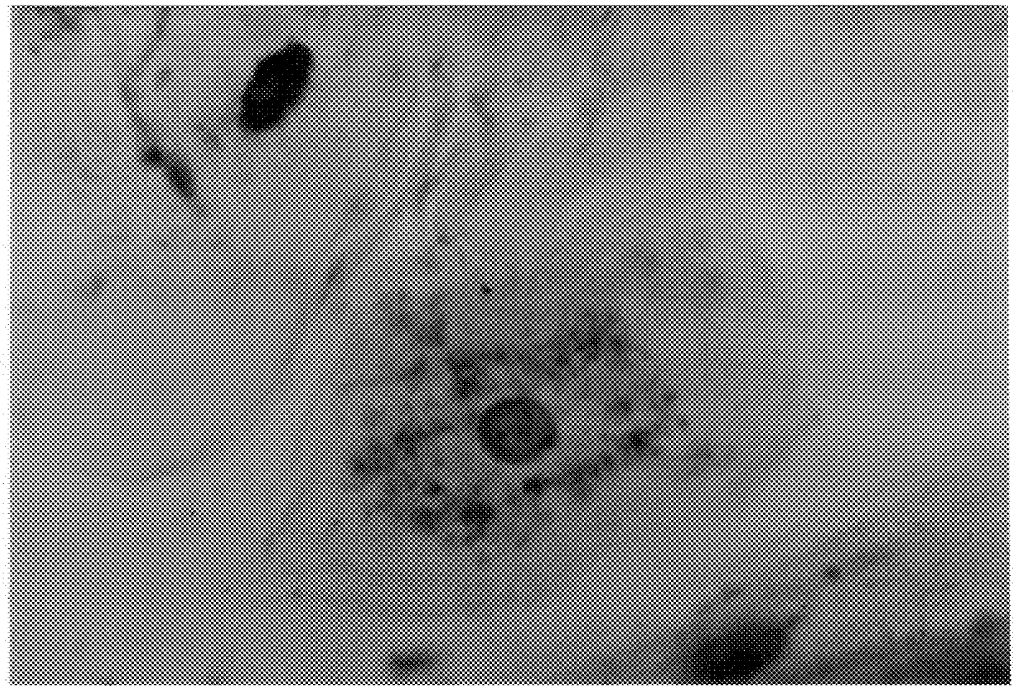


FIG. 3

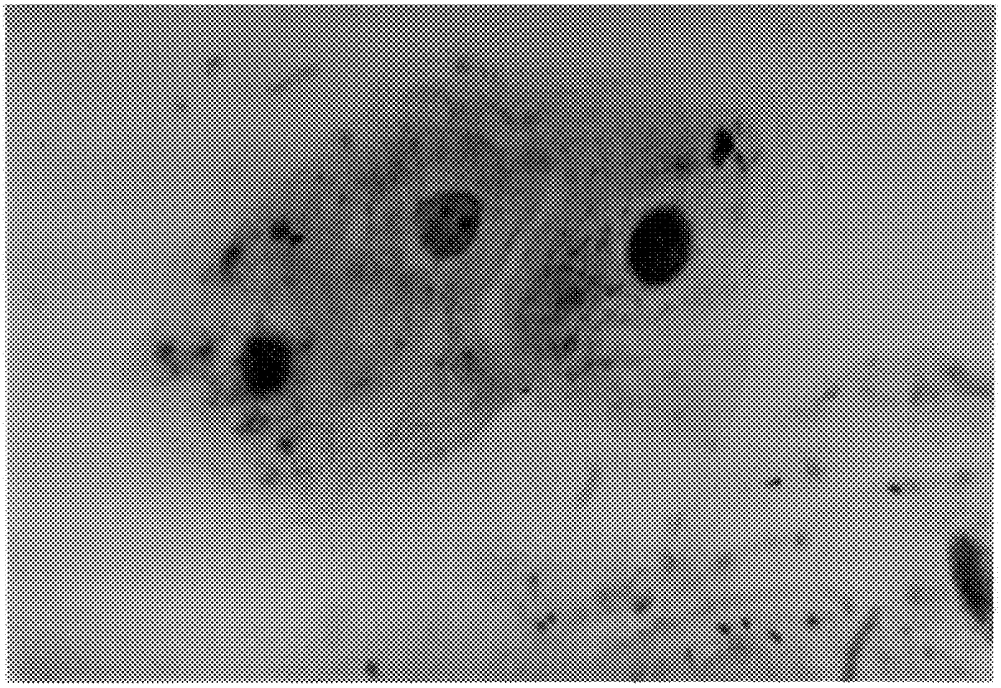


FIG. 4

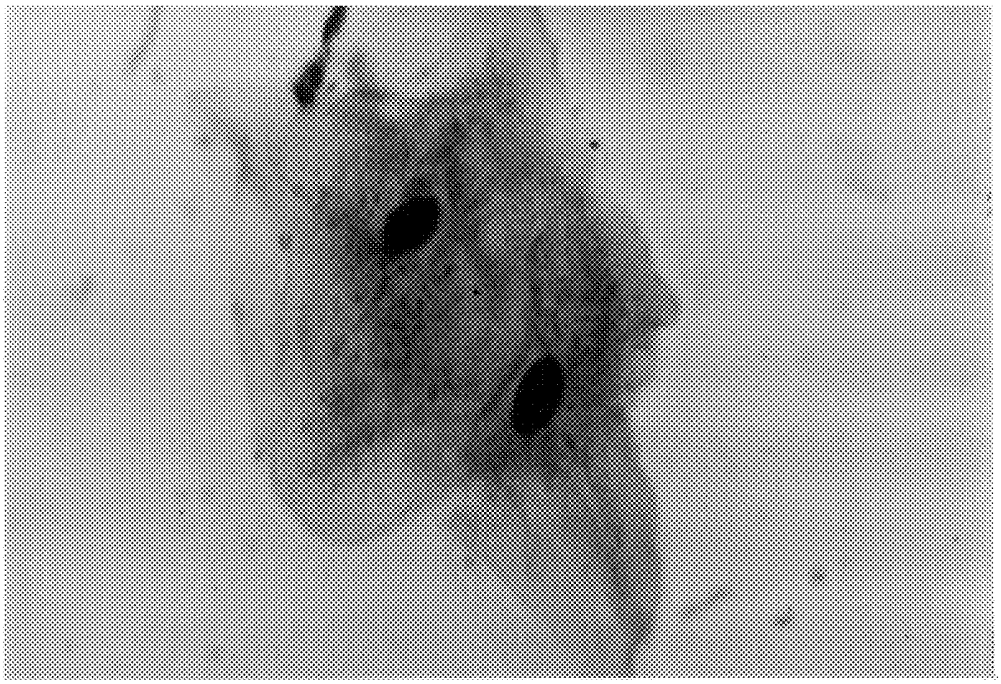
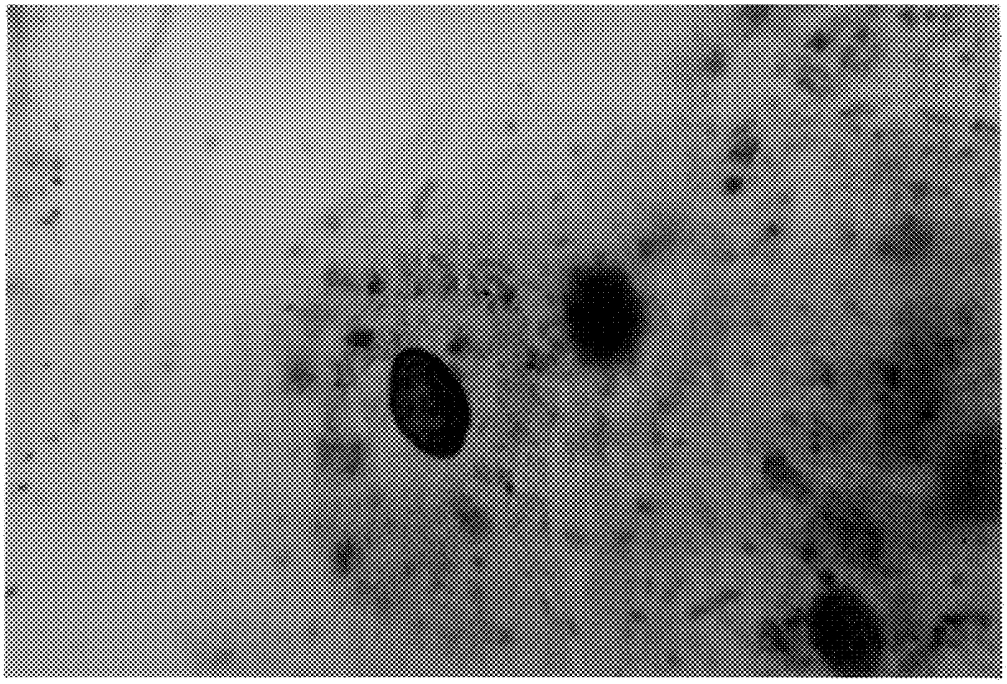


FIG. 5



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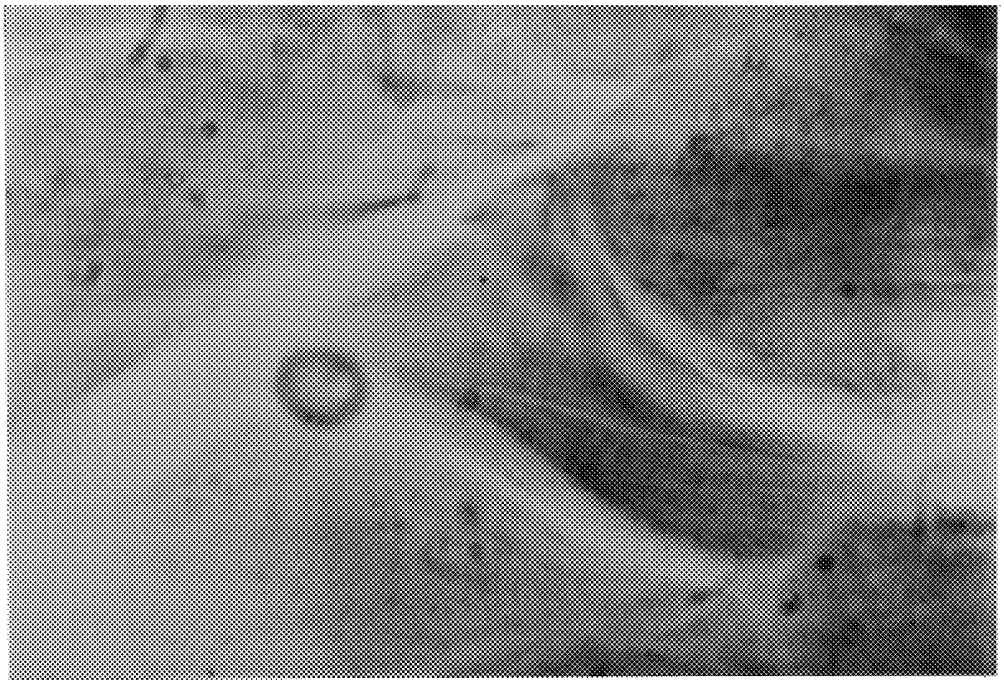

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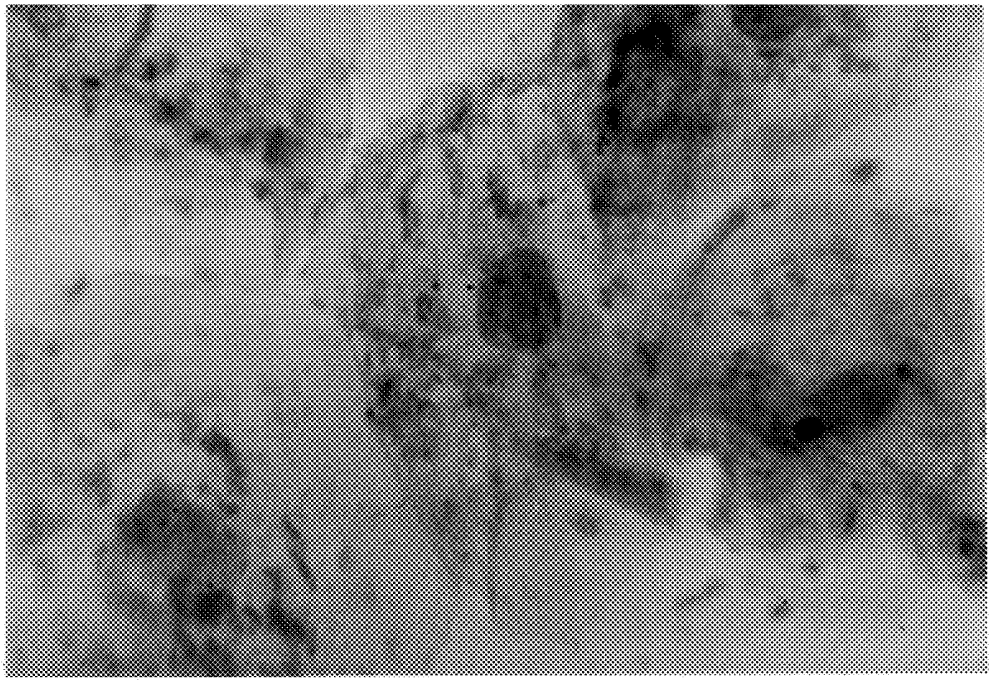


FIG. 13

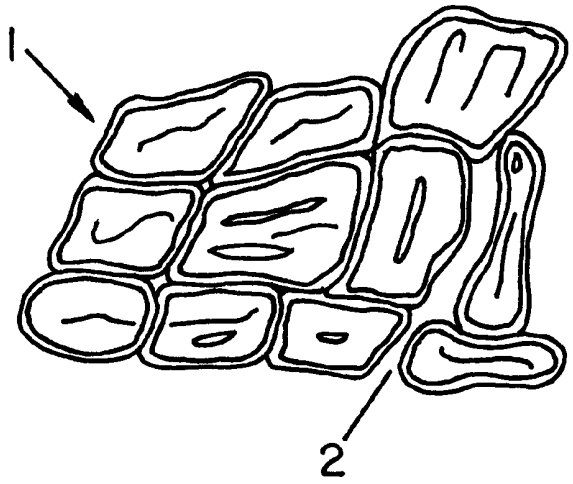


FIG. 8

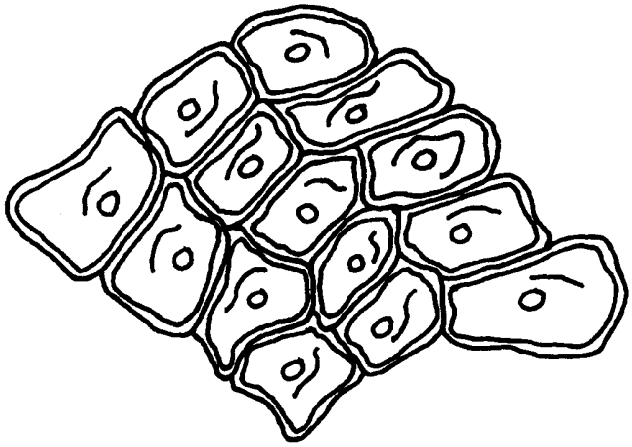


FIG. 9

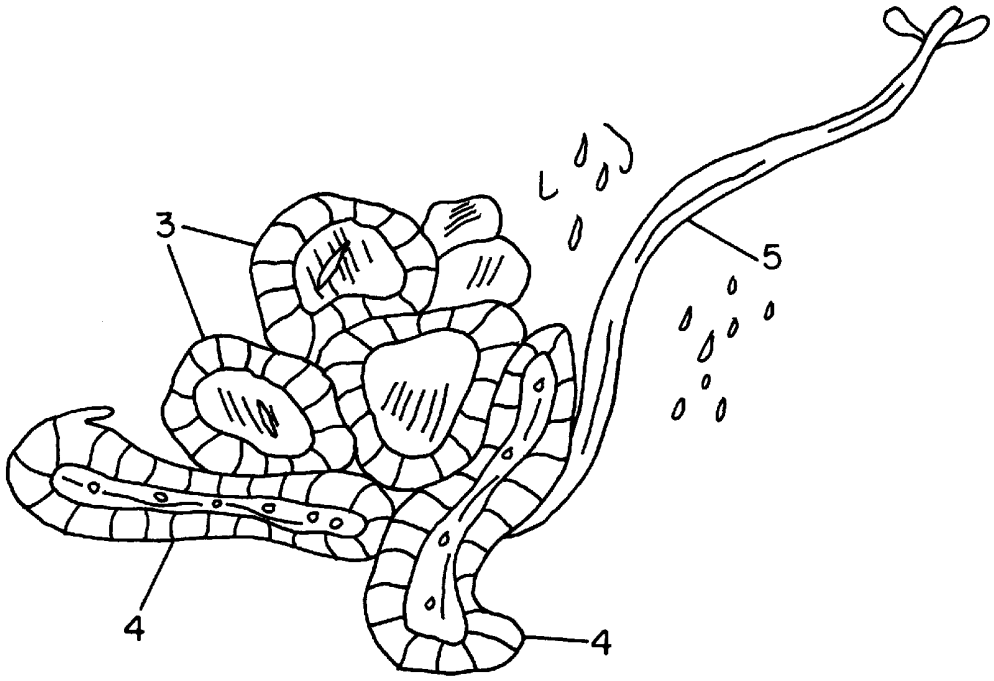


FIG. 10

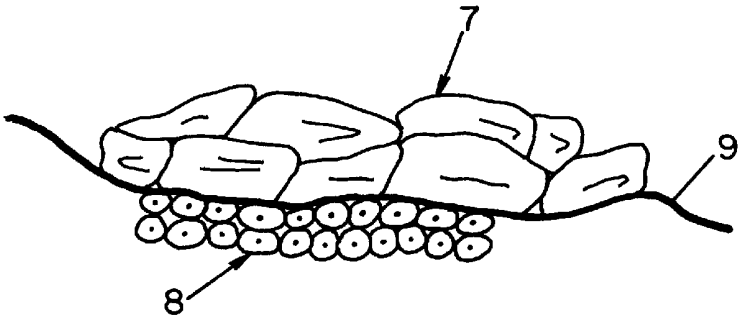


FIG. 12

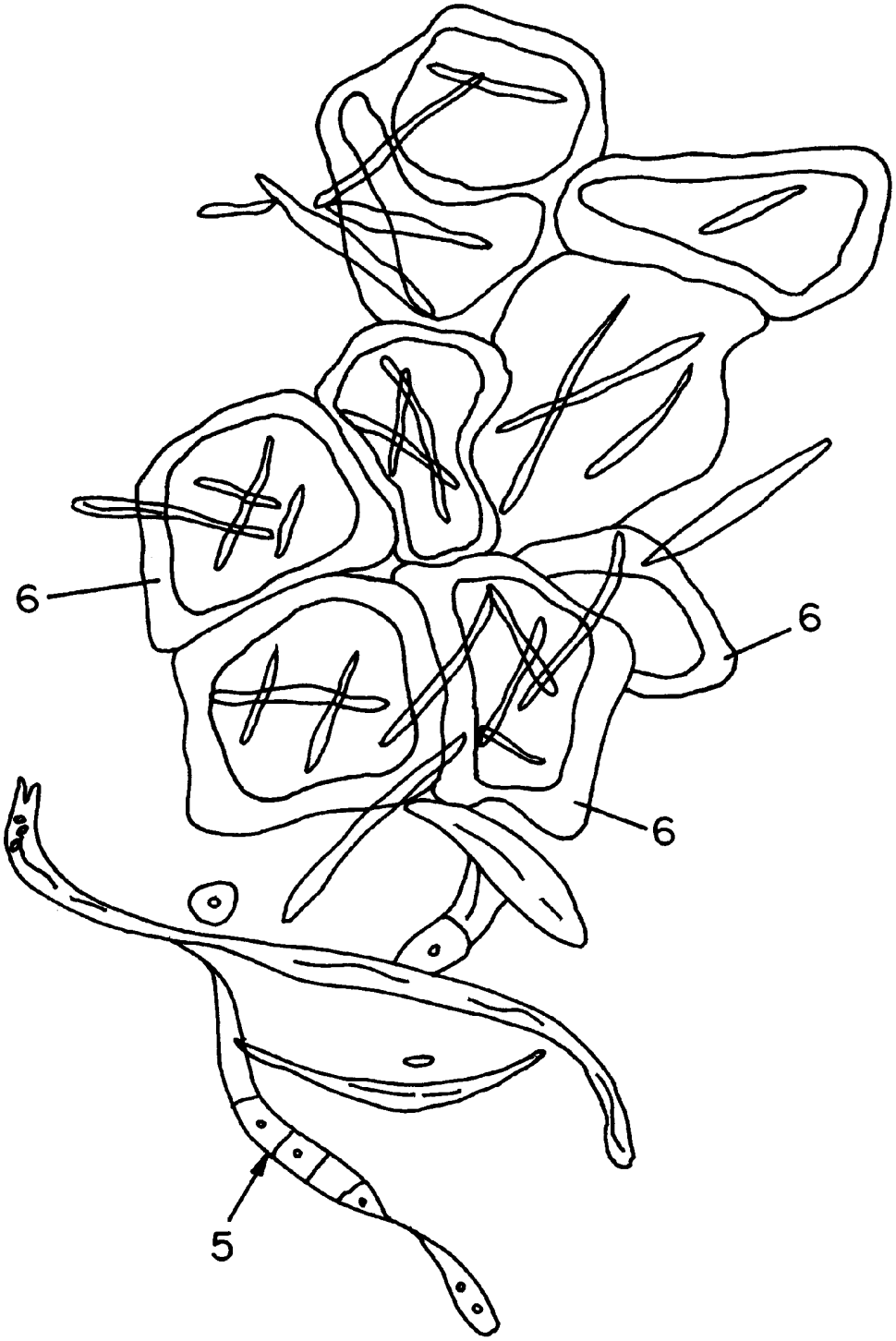


FIG. 11

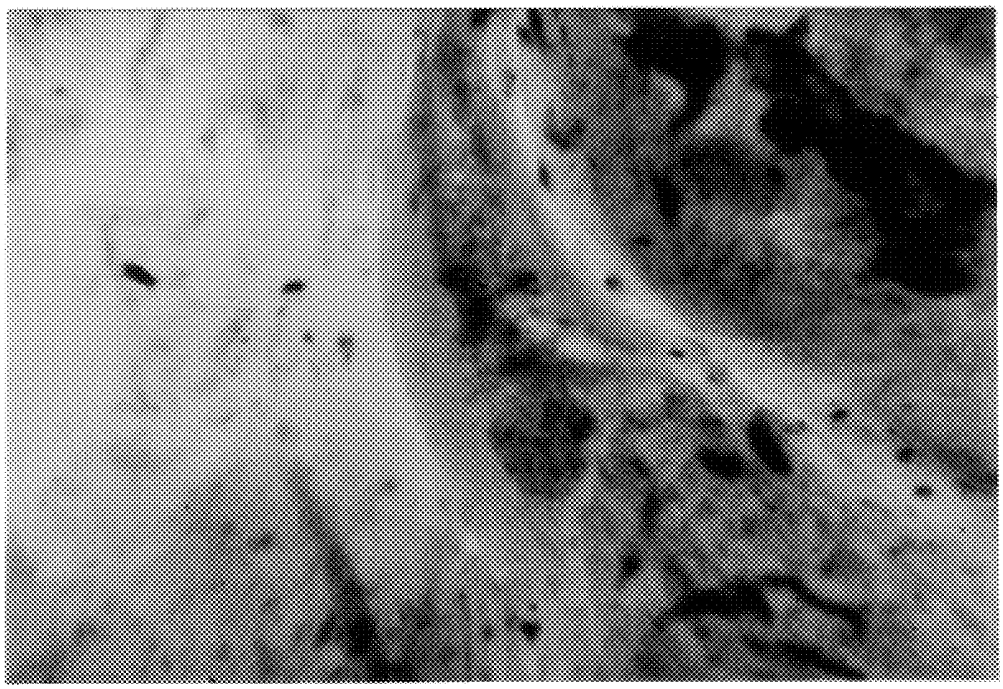


FIG. 14

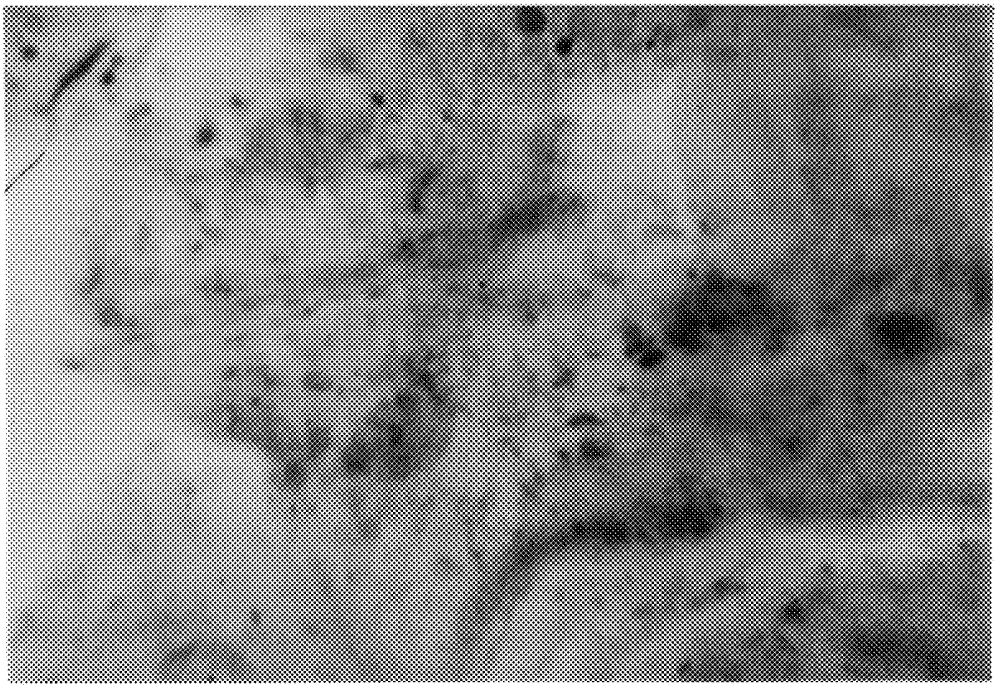


FIG. 15

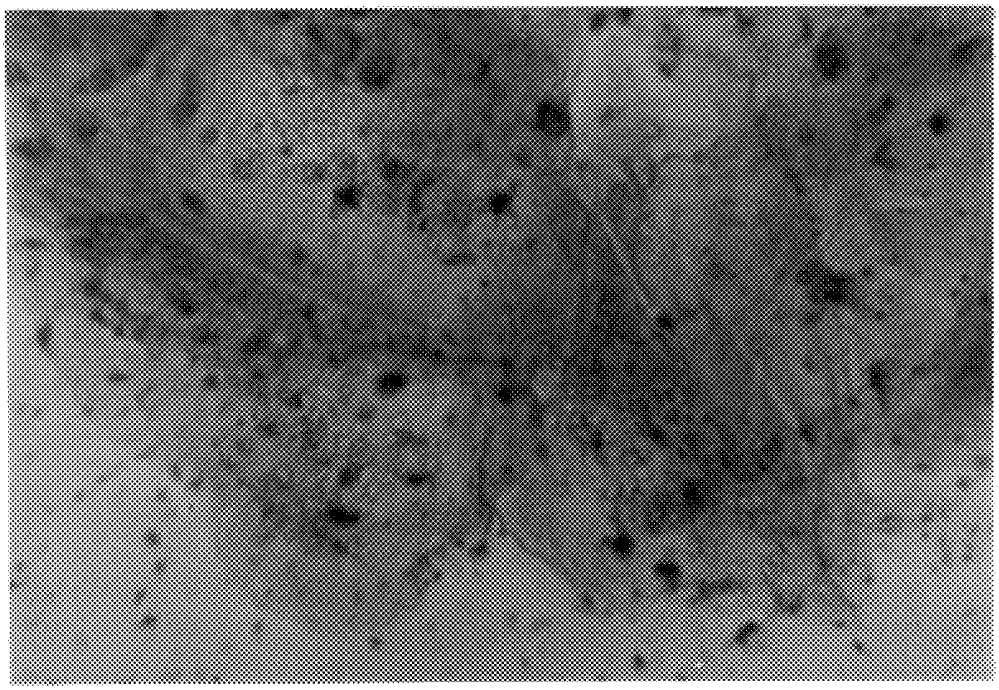


FIG. 16

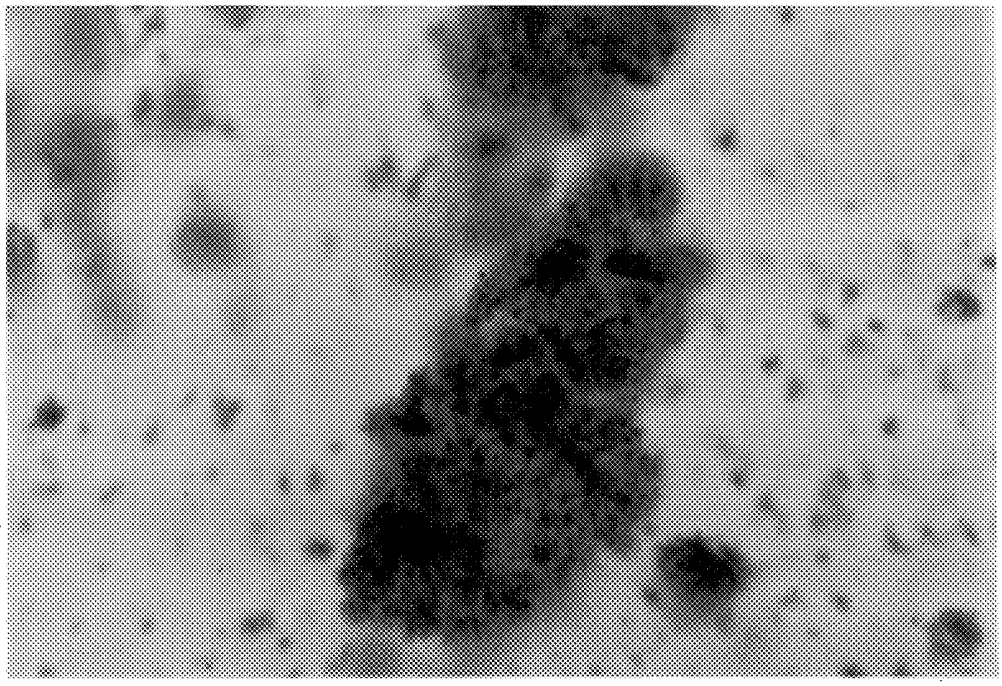


FIG. 17

EPITHELIAL CELL CANCER DRUG

BACKGROUND OF THE INVENTION

The present invention is directed to a new and unique anticancer drug, which is identified generically as 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate ($3Mg{+}{+}.3Al_2O_3.3SiO_2.3H_2O$), which is a magnesium aluminosilicate (referred to hereinafter as "MAS"), and which are in the acetate, sulfate, chloride, or brominate form. These compositions come from a class of inorganic aluminosilicate chemicals known as zeolites. The compounds of the present invention are particularly useful in treating epithelial cell cancers in mammals.

The involvement of cancerous epithelial cells, which lead to the formation of solid tumors in humans, in such organs as the lungs, breast, skin, mouth, and colon are known as carcinomas. Most of the epithelial cell cancers are treated using chemotherapeutic agents and these tend to be toxic, and have immunosuppressive side effects. When treated this way, the cancer patient must then wait up to 3 weeks for his next treatment, until his immune system has restored itself.

Cancers involving human epithelial cells come from solid tumors of the breast, lung, stomach, liver, uterus, colon, skin, mouth and uterine cervix can form. Adenocarcinomas from secretory tissue and squamous carcinomas from protective linings are the two basic categories of carcinomas. Epithelial cell based cancers proliferate rapidly respecting no cellular boundaries. To fully understand how to treat epithelial cell based cancer, one must start at the cellular level, this involving use of cell culturing techniques. Present day drugs used for chemotherapy do not directly attack the cancer cell with any great accuracy. Drugs such as Methotrexate and Vincristine are toxic to normal healthy cells and diminish immune system functions. These usually offer the cancer patient extremely disquieting side effects such as diarrhea, hair loss, vomiting and weakness. The toxicity of these drugs often shorten their use or require a very intermittent use. The average chemotherapy cannot be used more than once a month.

A large effort has been put forth by the medical research community to find new drugs for the treatment of epithelial cell based cancers. Carcinoma of the lung, breast, prostate, and colon all together account for more than half of the deaths from cancer in North America. Anticancer drugs have for the most part been categorized into alkylating agents such as cytoxan, antitumor antibiotics such as dactinomycin and antimetablite drugs such as methotrexate. Most, if not all, of these chemotherapies have major side effects and are toxic.

It is well known that chemicals cause 95% of all cancers contracted by humans. Some of the most potent carcinogens are aldehydes, ketones, pyrenes, benzpyrenes, benzene, and nitrosamines. Nitrosamines were looked at early on because they are carcinogenic agents found in cigarette smoke and in the causative agents of rubber polymers.

Zeolites are natural hydrated silicates of aluminum and, usually, either sodium or calcium or both. Zeolites such as sodium aluminosilicate have a unique multi-dimensional structure of cavities into which small to medium size molecules and cells can be trapped. They exist in natural and artificial forms and are used extensively for water softening, as detergent builders, and cracking catalysts. Natural zeolites include analcite, chabuzite, heulandite, natrolite, stilbite, and thomosonite.

Zeolites have been used in animal feed. For example, as reported in "World Food & Drink Report", Apr. 19, 1990,

hydrated sodium calcium aluminosilicate, an anti-caking agent used in animal feed, may reduce levels of aflatoxin in the milk of animals eating contaminated grain. Further, German patent DE19755921 teaches the use of zeolites or klinopitolites, that are used as food additives for human consumption as an aid to health, after they are treated with tribomechanical action to increase their surface area and destabilize their structure to release their chemical potential. These materials are thought to be a useful defense against cancers such as lung cancer, cancer of the colon, and skin cancer, and they are recommended for improving blood circulation.

SUMMARY OF THE INVENTION

The present invention has resulted from the discovery that epithelial cell cancer can be treated by administering to a mammalian patient having an epithelial cell cancer a therapeutically effective amount of 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate acetate, sulfate, hydrochlorate, or bromate. The composition of the present invention is synthesized from a naturally occurring non-toxic zeolites, and has a 100% kill rate within 72 hours against buccal mucosa and ling squamous epithelial cell cancers. It is not cytoxic to healthy human cells.

BRIEF DESCRIPTION OF THE DRAWINGS

The file of this patent contains at least one drawing executed in color. Copies of this patent with color drawing (s) will be provided by the Patent and Trademark Office upon request and payment of the necessary fee.

FIG. **1** is a photomicrograph of stained cells showing vacuoles containing the composition of the present invention prior to being brought into the cancer cells.

FIG. **2** is a photomicrograph showing the transition of vacuoles containing the new drug composition into the cancer cells.

FIGS. **3–6** are photomicrographs showing the transition of the new drug composition into the cancer cells.

FIG. **7** is a photomicrograph showing the destruction of the cancer cells.

FIGS. **8** and **9** show illustrations of non-cancerous crown gall pin oak leaf cells.

FIGS. **10** and **11** are illustrations showing cancerous gall pin oak leaf cells.

FIG. **12** is an illustration of crown gall pin oak leaf cells after treatment.

FIGS. **13–17** are photomicrographs of stained cells showing the progressive killing of the epithelial cancer by the drug composition of the present invention.

DETAILED DESCRIPTION OF THE INVENTION

The composition of the present invention is 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate ("MAS") salt synthesized from zeolites. It is used in its acetate, sulfate, hydrochlorate, or bromate salt forms. Zeolites are generally recognized as safe for humans. When these zeolites are converted and reacted with acetates, chlorides, sulfates, bromides, or gluconates the resultant product is a 4, 5, and 6-sided network of cavities holding anions and cations. The composition of the present invention has a molecular weight (weight average) of 1500 putting it into the polymer family.

The starting zeolite is sodium magnesium aluminosilicate. Preferably Hydrex R which is available from J. M. Huber

Corporation. Alternatively it could be a zeolite such as Thomsonite, where magnesium is substituted for the calcium. It is processed by reacting the zeolite starting material with an acid, such as a 5 molar glacial acetic, hydrochloric, sulfuric, hydro-bromic or gluconic acid, preferably at 1 milliliter per 250 milliliters of hypotonic saline solution, in the presence of a poloxy compound, such as iron oxide Fe_2O_3 being preferred), calcium oxide, or sodium oxide, with iron oxide being preferred, at an amount of between about 0.0001 and 0.005% by weight with 0.003% by weight being preferred. The ratio of the mixture of the zeolite and the acid will preferably be about 30% by weight of a zeolite with 70% by weight of the acid, at 5% concentration, although it could be in a range of about 20 to 40% by weight of zeolite and 80 to 60% of acid. The reaction takes about two hours at a temperature of about 200 to 250° F.

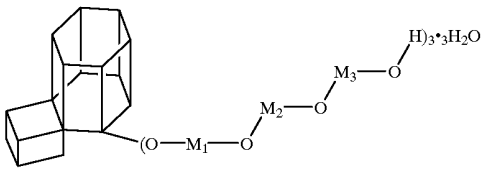
Synthesis of the MAS

The synthesis of the new drug was carried out in a 500-milliliter three neck reaction flask, fitted with a stirrer, thermometer and cold water condenser apparatus. The zeolite starting material was Hydrex R, a sodium magnesium alumino silicate in an amount of 150 grams, available from the J. M. Huber Corporation. The amount of a 5 molar acid was 350 grams, and the iron oxide compound was 1.5 grams. The starting ingredients were combined and were reacted at 250 C for 2 hours followed by cooling. The acid was sulfuric acid at 1 milliliter per 250 milliliters of hypotonic saline solution. The final product was then diluted by adding twice as much sterile water and filtered via sterile millipore filtration apparatus. Quality control, and characterization of the compound was done using derivatization techniques, ASTM 3526, paper chromatographic analysis, molecular weight studies, and various wet bench analytical techniques. The average concentration of the drug of the present invention was 98 MG per milliliter of water.

In order to see if the composition of the present invention had any biological activity or "kill power" against the harshest microorganisms known, including streptococcus, staphylococcus, bacillus, fungus, and mildews, the microorganisms were cultured in RPMI 1640 cell culture media and in a starch-based, solid matrix culture media. All of these microorganisms were destroyed and also prevented from growing when first initiated with the patent substance. Thus the new composition of the present invention has been found to be bacteriostatic and bacteriocidal, fungistatic, and fungicidal, and a mildewicide.

The composition has a structure as follows:

Formula I



wherein M_1 is silicon, M_2 is magnesium, and M_3 is aluminum.

The drug must be administered directly into the cancer tumor by injection or a 24-hour intravenous slow drip. The patent substance must be in close proximity to or infused into the cancer cells and/or in the blood stream supplying the tumor. The precise amount dosed will vary and does not appear to be critical as long as it is pharmaceutically effective. Usually, this will be in the range of from about 0.001 to 1000 milligrams, with between about 20 and 300 being preferred.

The method of the invention can be practiced on any mammal having a susceptible cancer, i.e., a malignant cell population or tumor. Compounds of the invention are effective on human tumors in vivo as well as on human cell lines in vitro. The present compounds may be particularly useful for the treatment of solid tumors for which relatively few treatments are available. Such tumors include epidermoid and myeloid tumors, acute or chronic. Such tumors also include, nonsmall cell, squamous, liver, cervical, renal, adrenal, stomach, esophageal, oral and mucosal tumors, as well as lung, ovarian, breast and colon carcinoma, and melanomas (including amelanotic subtypes). The present compounds can also be used against endometrial tumors, bladder cancer, pancreatic cancer, lymphoma, Hodgkin's disease, prostate cancer, sarcomas and testicular cancer as well as against tumors of the central nervous system, such as brain tumors, neuroblastomas and hematopoietic cell cancers such as B-cell leukemia/lymphomas, myelomas, T-cell leukemia/lymphomas, small cell leukemia/lymphomas, as well as null cell, sezary, monocytic, myelomonocytic and Hairy cell leukemia. These leukemia/lymphomas can be either acute or chronic.

While not wishing to be bound by a particular theory, the composition of the present invention can react, or chelate into a complex, with carcinogenic agents. The basic chelation, or complexing ability of the Mg^{++}, (magnesium ions), O, (oxygen atoms) and aluminum ions which are inside the six (6) sided cavity of the zeolite composition of the present invention. This complexation now ties up the nitrogen to nitrogen to oxygen portion of the nitrosamine (—N—N—O) and prevents it from being metabolically activated by human liver enzymes inside cells to become the nitrous free radicals which then enter the healthy human cells nucleus, damaging the DNA of the chromosome and producing a rogue cell or cancer cell. The complexing of these carcinogenic nitrosamines was proven by using ASTM 3526 pager chromatography.

The compounds of the present invention target the cancerous epithelial cells, only, due to the different surface chemistry of the cancer cell itself Photomicrographs FIGS. **1** and **2**, under X1000 oil immersion, show the formation of vacuoles containing MAS, which are prior to being brought into the cancer cell by endocytosis. Once inside, lysosomes attach and chemical breakdown of the MAS. The trimetallic portion of the MAS molecule crosses the nucleus of the cancer cell intercalates with the cell mutated DNA and destroys the cancer cell from within.

The formulations disclosed within the terms of the present invention may also comprise any additives, carriers, or fillers commonly in usage with drugs to facilitate their delivery.

The following examples are illustrative of the present invention, and should not limit the scope of the invention.

EXAMPLE 1

The following experiment was for the purpose of demonstrating the functionality of the compounds of the present invention with respect to carcinogenic compounds.

Ten (10) grams of a cured natural rubber compound were cut up to act as a control. This natural rubber compound contained 2 rubber additives which are known to generate nitrosamines; N-t-Butyl-2-benzothiazyl sulfonamide and zinc dimethyldithiocarbonate. Another 10 grams of this natural rubber compound, the same compound, contained 0.25 parts per hundred of the composition of the present

invention. Both of the samples were extracted with warm acetone for 20 minutes, then cooled and the acetone carefully evaporated off. The remaining solids were dissolved in 0.5 ml of acetone and spotted in a paper chromatogram according to ASTM 3526. A 95% acetone, 5% ethyl acetate solvent system was employed. As can be seen in Table 1, the control generated nitrosamine at Rf (retention time) of 0.037. The composition of the present invention, at 0.25 pHR gave no nitrosamine fraction at 0.037, but the large, (high molecular weight) complex was at the origin or Rf=0, retention time 0. The nitrosamine was complexed by the patent substance and thus was made non-mutagenic, and non-carcinogenic.

TABLE 1

Sample	Rf	Fraction
Cured Natural Rubber Compound	0.037	Nitrosamine
"	.0448	Trace Santocure NS
"	0.761	Trace Methasan
"	0.970	Process Oil
"		
Cured Natural Rubber Compound plus 0.25 phr of MAS	0	Nitrosamine
Cured Natural Rubber Compound plus 0.25 phr of MAS	0.492	Trace Santocure NS
Cured Natural Rubber Compound plus 0.25 phr of MAS	0.716	Trace Methasan
Cured Natural Rubber Compound plus 0.25 phr of MAS	0.940	Process Oil

EXAMPLE 2

Over a three (3) year period of study, oral cavity epithelial cells were obtained from buccal mucosa harvests from a single source. The cells were immediately placed in RPMI 1640 medium, which is a standard medium developed at the Roswell Park Memorial Institute. The medium was supplemented with L-glutamine, penicillin and streptomycin in all cultures, and then incubated at 98.6 F. The study cultures were supplemented with a carcinogen and strong promoter, sodium or magnesium saccharin. The sodium form being preferred. The cultures were incubated for 120 hours with one medium change for the extended time cultures. The sterile solutions of sodium or magnesium saccharin were made to a strength of 250 milligrams per milliliter of hypotonic saline solution, the combination penicillin-streptomycin antibiotic system were used at 100 units per milliliter of hypotonic saline solution.

The carcinogen-promoter supplemented cell cultures, also incubated at 98.6 C, once producing cancerous epithelial cells were given 300 microliter additions of the 4,5 sided ring MAS sulfate having concentrations of 60 milligrams per milliliter of hypotonic saline solution. Slides were prepared at room temperature using giemsa stain and observed and analyzed using bright field microscopy up to X1000 magnification with photomicrographs taken by the Polaroid Microcam System.

Phase contract microscopy at X100, and X200 power was also used, with photographs taken by the Polaroid Microcam System. All solutions used were sterilized via filtration. All cell counts were done on grided 2 chamber, cell culture slide chambers.

The epithelial cells used were obtained from sterile "Buccal Smears" of human cheek cells and were placed in sterile RPMI 1640 cell cultures with and without the composition of the present invention. The concentration of sodium saccharin used to promote the epithelial cell human cancer was 300 microliters of a 5 gram per milliliter working solution. All in-vitro, cell cultures were analyzed over a 3 day period (72 hours). Cancerous-like changes were seen in these cell cultures within 8–16 hours, and all the cancer cells were killed before 72 hours had lapsed.

A microscopic view shows normal check cell morphology, nuclear size, cell wall and membrane capacity and growth clustering; no ghosting or other abnormal changes were seen. The results are dramatic cell changes due to the sodium saccharin introduction to the cell culture. Observation of these cell cultures at X400, and X100 showed very enlarged cell morphology, formation of enlarged, crescent shaped nuclei, cell wall membrane swelling, multi-nucleated cell areas, formation of elongated or hook type epithelial cells in large sheaths were observed, plus the very telling formation of abnormal protein growth "Blebs" seen on the outer cell wall of these cells. These are illustrated in photomicrographs, FIGS. **3** through **6**.

When the composition of the present invention is used as a non-toxic, anti-cancer agent in the cancerous epithelial cell cultures, at both 300 and 400 microliter dosages in these cell cultures, 100% epithelial cells induced to cancerous state were destroyed within 24 hours. This is shown in photomicrograph FIG. **7** by the "ghosting" or destruction of these cancer cells by the destruction of the cancer cell nucleus and cytoplasmic organelles. In some cases the cancer cells were destroyed in an outward burst of the cytoplasm by increased osmotic pressure. The composition of the present invention is totally amenable to the different receptor sites, cell wall membrane proteins, and surface chemistry of the cancer cell only. It does not effect ordinary, healthy cells as already shown and claimed above.

EXAMPLE 3

In this example, the plant cancer of study was the Pin Oak Tree Leaf Crown Gall plant cancer. The agrobacterium Tuma Faciens and its metabolites cause this well studied plant cancer. Normal leaf plant cells, and crown gall cancer cells were cultured in RPMI 1640 cell culture media, containing an antibiotic, and concentrations of the patent substance in its sulfate form. The average dosage of the patent substance was 300 microliters of a 98 milligram per milliliter concentration. Within 24–48 hours all cancer cells in the crown gall cell culture were dead as analyzed by Giensa stained microscope slides at X400, and X1000, power. The control cell culture of healthy pin oak leave section showed no cytotoxicity at all. The crown gall cells (cancer cells) were "ghosted" by the composition of the present invention.

The composition of the present invention crossed the cancer cell membrane, due to its different receptor site configuration and surface chemistry, destroyed the elongated nucleus and the cytoplasm bulged to one side of the cancer cell and the entire cellular contents dissipated into the surrounding environment. The membrane of most cells remained intact, but no cellular structures were left. Some cells were totally destroyed by cell wall and membrane collapse.

The healthy pin and cell cultures showed no ghosting, no elongated nuclear structure, or cell wall and membrane swelling. No extracellular growth or "Bleb" protein on the healthy cell wall, and membrane outer surface was noted. Usual cell counts were in the 300–500 cell range. FIGS. **8** and **9** show normal, non-cancerous (crown gall, non-cancerous) pin oak leaf cells. In FIG. **8**, leaf section **1** is depicted with stoma **2**. FIG. **9** shows normal non-cancerous crown gall pin oak leaf cells. FIGS. **10** and **11** show the pin

oak crown gall cells, which are cancerous. FIG. **10** shows a cancerous cell in which has gel like, "eye" cells, **3**, "hook" cells, **4**, which are elongated, "nucleii" gels, and "ghosted" filiments, **5**. FIG. **11** shows the enlarged cell membrane **6** as well as the filaments **5**. FIG. **12** depicts crown gall pin oak leaf cells after treatment with 400 microliters of the composition of the present invention at 98 MG per milliliter. The xylem cells, **7**, had some brownish color but no cellular damage. The lower cells were green, **8**, below the boundary layer **9**.

All cell culture preparations were done in sterile petri dishes with 25 milliters of RPMI 1640 cell culture media, and 100 microliters of antibiotic, cell counts ranged in member from 300–500, GIEMSA stained microscopic slides were prepared and observed at X400, and X1000. The concentration of composition of the present invention used was 300 and 400 microliters of a 98 MG per milliliter preparation.

EXAMPLE 4

Squamous cell human lung cancer cell cultures, which are commercially available as the A549 cell line from Bio-Whitaker Company, were cultured in RPMI 1640 Medium supplemented with L-glutamine, penicillin, and streptomycin. This culture was also incubated at 98.6° F., with cell culture times up to 96 hours, with at least one medium change. These cultures were also supplemented with doses of the drug of the present invention at 300 microliters of a concentration of 60 milligrams per milliliter of hypotonic saline solution. Fresh venous blood samples of 2 ml each were taken and placed in heparinized sterile test tubes for % hemolysis studies.

It has been shown that sodium saccharine or its magnesium counter is a weak carcinogen, but a strong promoter which increases the proliferation of epithelial cancer cells during cell cycling, over a three year period cell cultures of RPMI 1640 cells culture medium supplemented with L-glutamine and 100–200 microliters of penicillin and streptomycin each at 100 units per milliliter hypotonic saline solution, and dosed with 200–400 microliters of a concentrated solution of sodium saccharine (250 milligrams per milliliter of hyptotonic saline solution), incubated at 98.6° F., produced cancerous cells within 24–48 hours. Using phase contrast microscopy at X100, and X200 magnification and employing temporary slides for examination at X100, X400, and X1000 oil immersion magnification transitions from normal epithelial cells to well formed cancer cells could be cultured. In photomicrograph FIGS. **1**, **7** and **13–14**, giemsa stained cells and photographed at X1000 oil immersion magnification (using Polaroid microcam) we can easily see the transition of the healthy epithelial cells in photomicrograph FIGS. **1–3** to the transition of precancerous cells of photomicrographs FIGS. **4-6** where we observe a darkening of the cell cytoplasm, development of multinucleation, and a consistent swelling noted in the plasma membranes and large changes in cell morphology. Strikingly in photomicrographs FIGS. **7** and **13** we see the formation of oral cavity epithelial cells which have become cancerous in nature. We observe the proliferation of distorted cellular morphology to swollen, misshapen elongated, hook like cells, with thick fibrous cytoplasm and the growth of fibrous, abnormal proteins on the cells outer wall ('BLEBS'), multinucleation and nuclei that seem to elongate and become abnormal in shape, similar to the morphology changes of the cancer cell itself, are seen along a medial axis. A typical cell count of these cell cultures shows that the number of epithelial cells declines at the same rate as does the growth of the cancer cells, which result is shown by the data in Table 2. Normal control cultures lacking, the supplement of sodium saccharine, also incubated at 98.6° F. gave normal, healthy cell cultures of oral cavity epithelial cells. See photomicrograph FIGS. **1–3**.

TABLE 2

Typical Cell Count Statistics After Introduction of Sodium Saccharine

Time Hours	# of Normal Epithelial Cells	# of Transition Cells	# of Cancer Cells
T = 0	462	0	0
T = 24	200	60	260
T = 48	5	10	500

Over a three year period the cell cultures produced were treated with MAS Sulfate within 24–48 hours of the formation of epithelial cancer cells. Dosages of 100–400 microliters of MAS Sulfate at 60 milligrams per milliliter of drug were, and within 72 hours of addition, 100% of all epithelial cancer cells were destroyed, showing cell casts and/or entirely "ghosted" cells. In Table 3 we can see cell counts on a typical cell culture where MAS Sulfate was added. The photomicrographs were taken from giemsa stained slides at X100magnification, using oil immersion. Photomicrograph FIGS. **7** and **14–16** show the progressive killing of the epithelial cancer by MAS Sulfate. It will also be shown MAS Sulfate non-cytoxic to date.

TABLE 3

Data Obtained from a Hemolytic Titer of MAS Sulfate on Human Red Blood Cells

Dilution of MAS Sulfate in 1000 Microliters Hypotonic Saline	% Hemolysis of Human Red Cells
7.40 Microliters	0
8.00 Microliters	0
10.00 Microliters	0
50.00 Microliters	0
100.00 Microliters	0
200.00 Microliters	0
500.00 Microliters	0
700.00 Microliters	0
1000.00 Microliters	1.99
2000.00 Microliters	2.35

MAS Sulfate is not cytotoxic to these cells and preferentially target epithelial cancer cells only. A hemolytic titer study was done on the new drug to show non-cytotoxicity. Typical data obtained using human red cells shows the lack of cytotoxicity to cells. The concentration of MAS Sulfate used was 60 milligrams per milliliter of hypotonic saline solution. No hemolysis appears until 1 full milliliter of MAS Sulfate and then only 2.00% hemolysis. The next data point shows that at a concentration of 2.00 milliliters of MAS Sulfate only 2.50% hemolysis of red blood cells occurs. A concentration of 0.35 microliters of human red blood cells was used for each sample titer. Table 2 shows the titer data in graphic form exhibiting a flat line effect until the 1.00 milliliter sample of MAS Sulfate.

A close look at the cell count in Table 3 clearly shows the rapidity of MAS to selectively cross the epithelial cell (cancerous) plasma membrane, in cell culture experiments and provide 100% kill rates within relatively short time period (48 to 72 hours), in this case 1,090 cells counted at 48 hours after addition of a total of 300 microliters of MAS Sulfate at a concentration of 60 milligrams per milliliter

hypotonic saline solution. Also MAS Sulfate shows a strong inhibitory action in reducing cell culture proliferation of the cancer cells to 0.82%. MAS Sulfate and its family of conjugate drugs are totally water-soluble such that by % hemolysis studies concentrations up to 120 milligrams, where red blood cell hemolysis is barely perceptible, can be tolerated.

By repeating these same experiments with a cell line of squamous cell lung cancers (BIO-WHITTAKER A549), similar results are achieved as seen in Table 4.

TABLE 4

Quantitative Cell Culture of Human Buccal Mucosa Epithelial Cells

Time (AGG) of Culture	Time of DME Add	Vol. Sodium Saccharin	Vol. DME ML	Total Cells	# Normal Cells	# Transition Cells	# Cancer Cells	# Dead Cancer cells	# Cell Growth	% Increase Cell Growth
0	—	200 ML	—	463	463	0	0	0	0	0
24	—	—	—	661	561	68	32	0	198	42.76
48	—	100 ML	—	749	230	200	319	0	88	13.31
72	—	—	—	805	60	45	700	0	56	7.48
96	—	—	—	873	3	10	860	0	68	8.45
120	0	—	100	1020	0	0	1020	0	147	16.84
144	24	—	200	1081	0	0	250	831	61	5.98
168	48	—	—	1090	0	0	0	1090	9	0.82

It has been seen that pre-cancerous and cancerous cells have extremely different cellular characteristics and surface cell chemistry than normal cells. Predominantly amongst these characteristics are the gross morphological changes of the cancerous epithelial, lack of yet totally unknown plasma membrane protein growths familiarly known as 'BLEBS,' changes in numbers of and mutations in receptor sites such as the EFG, and VEG F receptor sites controlling cell cycle functions. Proteolytic enzymes and increased lysosomal activity are also important changes in the cytoplasm of these cells. It has been seen during our research studies that the MAS molecule itself is transported, selectively across the cancerous epithelial cell membrane and once in the environs, crosses the nuclear membrane and deactivates cancer cell replication by action on the mutated DNA. Then, with the collapse of the nucleus osmotic pressure within the cancer cell ruptures the plasma membrane outwardly. Photomicrograph FIGS. **1–7** and **13–16** show these cancer cell "casts" and "ghosted" cytoplasms of our treated epithelial cancer cells within the time of 48 to 72 hours as seen in Table 4. Referring to Table 4, the number of cancer cells here represent cells in G2 or late S phase at the time of introduction of the sodium saccharin, (all additions of sodium saccharin and MAS Sulfate were added after cell counts were taken). They were affected by the sodium saccharin to cause cancer as early as 24 hours after inoculation. The transition cell's morphology may represent affects of the sodium saccharin to cells in the early S phase of the cell cycle from the time of inoculation to the time of cell count at 24 hours. As seen in this data the number of normal cells seen after 24 hours of exposure to sodium saccharin represents cells safely in G2 phase of the cell cycle prior to the addition of the sodium saccharin. Referring to Table 4, normal cells seen after 48 hours in cell culture may represent cells that have survived in the culture unaffected by the sodium saccharin treatment the difference of the cell count 561–230 could possibly be cells experiencing normal cell death. In referring to Table 4, our cell cycle model, the transition cells increased in numbers after 48 hours, due to cells that were in the G1 cycle at the time of addition of the second dosage of sodium saccharin and only partially affected by the first dosage. As seen in Table 4, cancer cell counts reflect the total affect of the cells being exposed to the X2 dose of the sodium saccharin. The cells have transformed into transition (pre-cancer cells) and cancer cells within 24 to 48 hours after inoculation. The first sign of cancer cells after 24 hours is probably representative of cells in synthetic period of the cell cycle at the time of addition of the sodium saccharin. The rapid progression of cells to the transition state and consequently to the cancer cell state shows an aggressive division process that took place in the first 3–4 days in the cell culture. By the addition of the MAS Sulfate to the culture, there is a rapid demise of the cancer cell population after 24 hours and total cell death after 48 hours. The MAS Sulfate molecule, represented in Formula I selectively enters the cancer cell's plasma membrane.

It is the belief that this new pharmacological approach of selectively targeting cancer cells is a significant step in providing a new therapy, which is also non-cytoxic, for the treatment of carcinomas. The use of this drug will shorten the gap betweeen the number of patients getting cancer and a chemotherapeutic approach to saving lives. The ramifications of a substance which will target epithelial cancer cells selectively without harming normal tissue is overwhelming with regards to cancer treatment. Of ever greater significance, may be its ability to destroy transitional cells serving as a preventive agent in numerous clinical opportunities.

The drug of the present invention, in cell culture, effectively kills epithelial cell based cancers, at 100% "kill power" from the work presented. A patient could receive the new anticancer drug more than once every three weeks, perhaps several times a week.

The formation of cell wall membrane irregular proteins (Blebs) in these, nuclei as large as the cytoplasm, very differential cell morphology, some in shapes of hooks, others in large sheaths, and, formation of cell membrane swelling. It was also observed that the widespread deformation of these cancer cell nuclei are enlarged, crescent shaped, peanut shaped, and appear to be multi-nucleated in this study. The drug of the present invention was added to the known

human cancer cell cultures and within 24–48 hours as 100% lower cancer cell kill was established. The cancer cells have been "ghosted", their nuclei and organelles destroyed from within. This allows for stopping-cancer cell division, decreasing tumor size, stopping cancer metastasis, and not effecting healthy cells, like t-cells in the human immune system. The composition of the present invention can be used as a cancer chemotherapy systemically via IV or directly infused into the tumor and made available to the cancer patient several times weekly.

EXAMPLE 5

A study was conducted using standard immunodeficient (SCID) mice. Some of the mice were injected with the standard A549 squamous cancer cell line, with the C.B-17/IcrCrl-scidBR strain being selected, while some of the mice were injected with the A549 cell line and MAS sulfate drug prepared in accordance with the present invention. The mice were divided into three groups:

Group I: Two mice were a control and were not treated with either the A549 cell line nor the MAS sulfate drug.

Group II: Four mice were treated with the A549 cell line, but not the MAS sulfate drug.

Group III: Four mice were treated with the A549 cell line and the MAS sulfate drug.

The A549 cell line dose was at 2.5×10^6 cells per milliliter. The MAS sulfate was dosed at 30 miligrams per injection, three times per week. The injections were in dorsal thorax area.

The animals were about six to seven weeks of age at the start of the test and their weight variation did not exceed ±20 percent of mean weight. The animals underwent a quarantine/acclimation period of five days prior to implantation with the A549 cells. The animals were handled in accordance with the "Guide for the Care and Use of Laboratory Animals", published by the Institute of Laboratory Animal Resources, Commission on Life Sciences, National Research Counsel, including environmental conditions, handling, storage and feeding.

The observations of the changes in the body weights are shown in Table 5, which shows expected weight gains.

TABLE 5

	Body Weights in Grams				
Animals	Pretest Weight in Grams	Week 1 Weight in Grams	Week 2 Weight in Grams	Week 3 Weight in Grams	Week 4 Weight in Grams
Group I					
A	21.4	22.8	23.7	25.1	25.8
B	21.5	22.8	23.8	25.4	26.2
Mean Wt.	21.5	22.8	23.8	25.3	26.0
Group II					
A	20.0	21.6	21.6	22.6	22.8
B	19.9	20.5	20.9	22.3	23.5
C	20.9	22.5	22.7	24.3	25.0
D	22.3	23.8	24.6	26.0	26.8
Mean Wt.	20.8	22.1	22.5	23.8	24.5
Group III					
A	20.7	22.4	22.1	23.3	24.5
B	21.9	23.4	23.5	24.5	25.6
C	21.8	23.3	23.2	24.7	25.2
D	21.0	22.5	22.5	24.0	24.3
Mean Wt.	21.4	22.9	22.8	24.1	24.9

At the end of the observation period of the study, which was approximately five weeks, the mice were humanely terminated using a standard procedure of carbon dioxide asphyxiation and opening the thoracic cavity or exsanguination. Necropsies were performed and tissues, namely liver, kidney, spleen, lung, and any other abnormal appearing tissue, were examined via histopathology. The results are shown as follows in Tables 6 and 7.

TABLE 6

	Tumor Mass Measurements							
	Week 2		Week 3		Week 4		Week 5	
Animal	L × W × H	Vol.	L × W × H	Vol.	L × W × H	Vol.	L × W × H	Vol.
Group I								
A	No Mass		No Mass		No Mass		No Mass	
B	No Mass		No Mass		No Mass		No Mass	
Group II								
A	9 × 6 × 3	162	8 × 8 × 3	192	10 × 6 × 6	360	12 × 11 × 8	1056
B	9 × 7 × 3	189	10 × 5 × 3	150	14 × 8 × 4	448	15 × 10 × 9	1350
C	5 × 5 × 3	75	12 × 3 × 10	360	15 × 12 × 8	1440	20 × 14 × 5	1400
D	2 × 2 × 1	4	10 × 7 × 3	210	12 × 9 × 4	432	16 × 10 × 8	1280
Mean		107.5		228.0		670.0		1271.5
Std Dev		84.4		91.5		514.8		151.9
Group III								
A	1 × 3 × 2	6	10 × 7 × 3	210	13 × 7 × 4	364	18 × 9 × 6	972
B	1 × 3 × 2	6	10 × 4 × 3	120	14 × 7 × 4	392	17 × 8 × 6	816

TABLE 6-continued

	Tumor Mass Measurements							
	Week 2		Week 3		Week 4		Week 5	
Animal	L × W × H	Vol.	L × W × H	Vol.	L × W × H	Vol.	L × W × H	Vol.
C	5 × 3 × 2	30	10 × 7 × 3	210	11 × 8 × 5	440	13 × 10 × 6	780
D	3 × 4 × 2	24	10 × 5 × 3	150	11 × 6 × 4	264	14 × 9 × 5	630
Mean		16.5		172.5		365.0		799.5
Std Dev		12.4		45.0		74.3		140.4

TABLE 7

Histopathology Incidence Table

	Group I Animal		Group II Animal				Group III Animal			
	A	B	A	B	C	D	A	B	C	D
HEART										
Epicardium, Mineralization							2	3	3	
KIDNEY	X			X	X	X		X	X	C
Cortex, Tubules, Regeneration		1	1				1			
LACRIMAL GLAND							X			
LIVER	X	X	X	X		X		X	X	X
Hematopoiesis					1		1			
LUNG										
Alveolus, Hemorrhage	1		2	1	1	1	2	1		
Alveolus, Histiocytosis			2							
Peribronchial Lymphoid Depletion	5	5	5	5	5	5	5	5	5	5
LYMPH NODE, NOS							N			
SKIN/SUBCUTIS										
Carcinoma, NOS			P	P	P	P	P	P	P	P
Adnexal Atrophy			1	1		1		2		
Dermis, Fibrosis			1				1	2	1	
Dermis, Inflammation, Chronic Active			1	1				2		
Epidermis, Erosion/Ulceration			1	1		3	1	2	1	
Epidermis, Hyperplasia			2	2		2	2	2	2	1
Epidermis, Inflammation, Suppurative							2	2	1	
SPLEEN										
Hematopoiesis	4	4	4	4	5	4	4	4	4	4
Lymphoid Depletion	5	5	4	4	5	4	5	5	5	5
TESTIS					N					

X = Not Remarkable
N = No Section
P = Present
Scale:
1 = Minimal
2 = Slight/Mild
3 = Moderate
4 = Moderately Severe
5 = Severe/High

As seen in Table 6, the tumor mass in the mice that were not treated with the drug of the present invention was significantly higher than those that were treated. Further, the results in Table 7 show that for the spleen, the liver and the kidneys, no significant change occurred from the use of the drug of the present invention. Further, since mice are immunodeficient, the results were consistent with not dosing the mice with the cancer or the drug of the present invention.

The foregoing embodiments of the present invention have been presented for the purposes of illustration and description. These descriptions and embodiments are not intended to be exhaustive or to limit the invention to the precise form disclosed, and obviously many modifications and variations are possible in light of the above disclosure. The embodiments were chosen and described in order to best explain the principle of the invention and its practical applications to thereby enable others skilled in the art to best utilize the invention in its various embodiments and with various modifications as are suited to the particular use contemplated. It is intended that the invention be defined by the following claims.

What I claim is:

1. 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate acetate.

2. 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate sulfate.

3. 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate hydrochlorate.

4. 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate brominate.

5. A composition consisting essentially of 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate acetate, sulfate, chlorate, or brominate.

6. A pharmaceutical composition comprising a drug selected from the group consisting essentially of 4,5 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate acetate, 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate sulfate, 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate chlorate, and 4,6 cyclo, trisilico, trimagnesium, trialumino, oxyo, trihydrate brominate, and a pharmaceutically acceptable carrier.

7. The composition of claim **6** wherein said drug is present in an amount of between about 0.001 and about 1000 milligrams.

8. The composition of claim **6** wherein said drug is present in an amount of between about 20 and about 300 milligrams.

9. A cancer drug comprising the reaction product of a sodium magnesium aluminosilicate and an acid selected from the group consisting of glacial acetic, hydrochloric, sulfuric, hydro-bromic, and gluconic.

10. The composition of claim **9** wherein the reaction is conducted in the presence of a poloxy compound.

11. The composition of claim **10** wherein the poloxy compound is selected from the group consisting essentially of iron oxide, calcium oxide, and sodium oxide.

12. The composition of claim **9** wherein the reaction is conducted at a temperature of between about 200 and 250° F.

13. The composition of claim **9** wherein the silicate is present in an amount of 20 to 40% by weight of the mixture and the acid is present in an amount of about 80 to about 60% by weight of the mixture of the silicate and the acid.

14. The composition of claim **10** wherein the poloxy compound is present in an amount of between about 0.0001 and 0.005% by weight.

15. A method of treating epithelial cell cancer comprising administering to a mamalian patient diagnosed as having an epithelial cell cancer a therapeutically effective amount of a drug selected from the group consisting of 4,5 di-cyclo, disilico, dimagnesium, dialumino, oxyo, trihydrate acetate, sulfate, chlorate, and brominate.

16. The method of claim **15** wherein the drug is dosed is about 0.001 to about 1000 milligrams.

17. The method of claim **15** wherein the drug is dosed is about 20 to about 300 milligrams.

18. The method of claim **15** wherein said compound is administered to a human.

19. The method of claim **15** wherein said compound is in combination with a pharmaceutically acceptable dilutent or carrier.

* * * * *