Hypoxemia and hypercapnia: Its relation to joint diseases and the natural treatments for some joint diseases: A review

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Abstract

The leading cause of disability worldwide is Arthritis. Arthritis can be either autoimmune joint disease or osteoarthritis. According to the Center for Disease Control (CDC), by 2040, 80 million U.S. adults will have some form of arthritis. The objective is to find the effect of Hypoxemia, hypercapnia and PH chemical imbalance of the blood on muscle loss and joint diseases, and to find natural remedies to treat these types of joint diseases. Three main types of joint diseases were found to be directly related to hypoxia (Low levels of oxygen in your body tissues), hypercapnia (Excess carbon dioxide build up), and blood PH imbalance: Rheumatoid Arthritis, Osteoarthritis, and gout joint diseases. The natural treatment of these joint diseases has been identified.

Keywords: blood on muscle, excess carbon

Introduction

The earth’s atmosphere contains 21 percent oxygen \[^{[1]}\], A closed space has safe oxygen levels if readings are between 20.8-21%, while a space with readings of less than 19.5% are oxygen deficient according to OSHA guidelines \[^{[2]}\]. Home air conditioners: split AC, window AC, and portable AC can’t ventilate your room: doesn’t have the capability to bring the outside air, it only circulates indoor air and cool it. Complex HVAC system used inside some hotels, office buildings, big malls, airports have the feature to ventilate the indoor area, and maintains freshness, humidity, and oxygen level. Some window ACs in the US can bring outside air and main the oxygen levels. Most of window ACs around the globe does not have this feature. Oxygen room level 19.5-23.5% is considered safe, oxygen levels between 14-16%, below the 19.5% is considered hazardous \[^{[3]}\].

If while breathing out not enough carbon dioxide is expelled from the lungs, the increased carbon dioxide in the blood reduces the blood PH and makes the blood acidic causing respiratory acidosis. When your ability to breath is blocked by physical block limits, another condition, or a disease respiratory acidosis is caused. Respiratory acidosis can be either acute, chronic, or acute and chronic. The sudden arrival of CO\(_2\) to the lungs is called Acute Respiratory Acidosis. The kidneys response of acute respiratory acidosis is so quick that it can happen within minutes. The causes may include cerebrovascular accidents like strokes, a group of diseases that interfere with gene's ability to make muscle and causes to steadily lose muscles causing Muscular dystrophy, voluntary muscles may become weak, or you lose control of them causing Myasthenia gravis, heart attack, a very rare neurological disorder where the immune system attacks itself and can cause problems from trouble eating to full body paralysis causing Guillain-Barre Syndrome, and/or block airways. Chronic respiratory acidosis is more serious and it happens at slower rate and at a lesser degree than acute respiratory acidosis. The lower oxygen rate for the tissues to be fully supplied in the blood is known as hypoxemia The conditions may include pulmonary fibrosis or diseases that happens in the lung tissue/nerve or muscular diseases, sleep apnea, obesity, thoracic skeletal defects that causes pects/rib cage/or sternum to be shaped in a way that it limits lung functioning or breathing, Asthma, and a group of airflow and breathing diseases including diseases like bronchitis and emphysema called Chronic obstructive pulmonary disease (COPD). Common treatments for respiratory acidosis are oxygen tubes, different medications, or other treatment to stop smoking, naloxone (for opioid overdose), anti-inflammatory medications to ease any constrictive swelling, and breathing machines like a CPAP or BiPAP.
To prevent getting respiratory acidosis in general: lose wait if overweight, quit smoking, and don’t drink alcohol while taking opioids, and strong pain medications [4]. A two-year study showed that there is adverse physical effect on medical staff when wearing an N 95 mask. Wearing an N 95 mask resulted in hypercapnia (Excessive CO$_2$ in the blood caused by inadequate respiration) and hypoxemia (Lack of oxygen in the blood) which reduced the ability to make correct decision and the working efficiency. Dizziness, short of breath, and headache were experienced by the medical staff wearing N95 masks [3].

If prolonged hypoventilation is accompanying respiratory acidosis, the condition becomes more severe, and it can cause the patient to have additional symptoms: myoclonus, seizures, and altered mental status. Hypercapnia (excessive amounts of carbon dioxide in the blood) can be caused by respiratory acidosis leading to cerebral vasodilatation. Severe respiratory acidosis may cause papilledema and increased intracranial pressure increasing the risk of death and herniation. Chronic respiratory acidosis may cause impaired coordination, polycythemia, memory loss, heart failure, and pulmonary hypertension [6].

Dyspnea (difficult respiration) are commonly caused by chronic obstructive pulmonary disease (COPD), interstitial lung disease, heart failure, asthma, psychogenic problems that are usually linked to anxiety, and pneumonia. Chest X-rays and computed tomography (CT) images are used by doctors for its diagnosis. Spirometry tests can be used to measure airflow and the patient’s lung capacity and to pinpoint the extent and the type of an individual’s breathing problems. Other tests can be used to directly measure the blood capacity to carry oxygen and the level of oxygen in the blood. Treatment of dyspnea is dependent on its cause. If it’s caused from having a bacterial pneumonia, antibiotics are prescribed. If it’s caused from having asthma, bronchodilators and steroids are prescribed, other medications are also be effective (opiates, anti-anxiety drugs, and non-steroidal anti-inflammatory drugs (NSAIDs)). Special breathing techniques, such as breathing muscle strengthening exercises, and pursed-lip breathing can be used if the cause of dyspnea is caused by COPD. Supplemental oxygen may be prescribed if tests indicate low levels of oxygen in the blood [7].

A less well-known side effect of using nonsteroidal anti-inflammatory drugs (NSAIDs drugs) is the degradation of joint cartilages [8]. Nonsteroidal anti-inflammatory drugs include: Ibuprofen used in some drugs such as Motrin, Nuprin, and Advil; Piroxicam used in drugs such as Feldene; diclofenac used in drugs such as Voltaren; fenoprofen used in drugs such as Nalfon; indomethacin used in drugs such as Indocin; naproxen used in drugs such as Naprosyn; tolmetin used in drugs such as Tolectin; and sulindac used in drugs such as Clinoril. Other side effects of using nonsteroidal anti-inflammatory drugs are ulcer formation, dizziness, headaches, and gastrointestinal upset. Clinical studies had showed that nonsteroidal anti-inflammatory drugs usage have caused acceleration of osteoarthritis and increased joint destruction [9-12].

Common joint disease may include bursitis, osteoarthritis, gout, rheumatoid arthritis, spondyloarthritis, lupus, and juvenile idiopathic arthritis. Bursitis is caused by the inflammation of the small, fluid-filled sacs called bursae around the joints, tendons, muscles and bone. Overuse or sudden injury of joints such as the elbow, hip, and shoulder can lead to flare-ups. Bursitis can sometimes result from bacterial infections. Osteoarthritis is the wear-and-tear form that increases with age. Adults in their 50s and older are more likely to develop this chronic and progressive disease. Women are more vulnerable to osteoarthritis than men. It’s stiffness and pain with movement caused by breaks down of cartilages that cushions the joints. The flexibility decreases and walking becomes more difficult especially with knee and hip arthritis. The type of arthritis that affects the joint connecting the big toe to the rest of the foot is called gout. A waste product in the blood, uric acid would exist in excess and forms crystals in the joints. Flare-ups caused by gout are extremely painful and it would commonly strike in the night. Men are more vulnerable to gout disease, and women become more vulnerable to this disease after menopause.

The autoimmune condition affects the lining of the joints is called Rheumatoid arthritis. Immune system cells accumulate in large numbers in the joints. The interaction between joint cells and immune cells causes increasing inflammation leading to damage and destruction to of the bone and cartilage. Spondyloarthritis consists of certain other rheumatoid diseases including axial spondylitis, enteropathic arthritis, and psoriatic arthritis. The inflammation in the spine that can eventually lead to spinal fusion, or anklyosing spondylitis is called axial spondylitis. The complication of an inflammatory bowel diseases like ulcerative colitis, are called enteropathic arthritis. Psoriatic arthritis is associated with the skin condition psoriasis, and it tends to affect the joints of the hands and feet. The autoimmune condition affects various parts of the body, including internal organs, skin, brain, blood, bones and joints are called lupus. Lupus can cause an inflammation that would trigger arthritis, particularly in the knees, feet, hands, elbows, and shoulders. The most common chronic joint condition in kids is Juvenile idiopathic arthritis. The child's immune system attacks the body's own healthy tissue, and it’s an autoimmune condition. The cause is unknown, and it may alter children's normal growth. This inflammation may affect the internal organs, eyes, muscles, joints, and ligaments [13].

**Review and Discussion**

The amount of oxygen circulating in the blood is the blood oxygen level. The normal reading using an oximeter is between 95 to 100% [14]. Forms of carbon dioxide carried in the blood are carboxyhemoglobin (CO$_2$ bound to hemoglobin), and the chemically modified bicarbonate (HCO$_3^-$). The solubility of CO$_2$ in the blood is 0.07 mL CO$_2$/100 mL blood/mm Hg which would be almost 5% of the total CO$_2$ content of blood. The solubility of oxygen in the blood is 0.003 mL O$_2$/100 mL blood/mm Hg, which would be almost 2% of the total O$_2$ content of blood. The solubility of Carbon dioxide is 20 times more soluble in blood than oxygen. 15 According to Henry’s law, the solubility of a gas is directly proportional to the partial pressure of that gas above the liquid. Increasing the pressure and decreasing the temperature would lead to increase the solubility of gaseous over a liquid. 16 Carbon dioxide gas has a lower ability to diffuse and exit the lungs compared to oxygen gas according to Graham's law 95as it’s denser than oxygen gas, the density of oxygen gas is 1.43 g/L compared to density of carbon dioxide gas 1.81 g/L.

The body normally maintains CO$_2$ in a range from 38 to 42 mm Hg by balancing its elimination and production. Ventilation is primarily initiated by the blood PH. PH of the blood is mainly regulated by the amount of CO$_2$ in the blood. The body produces more CO$_2$ than it can eliminate in case of hypoventilation causing a retention of CO$_2$. The increased CO$_2$ is what leads to an increase in blood acidity, due to
increase of hydrogen ion concentration, a slight increase in bicarbonate concentration, and the equilibrium would shift towards forming more hydrogen ions according to the following reaction:

$$\text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{HCO}_3^- + \text{H}^+$$

A buffer system is created from the presence of the flowing molecules: $\text{HCO}_3^-$, $\text{CO}_2$, and $\text{H}_2\text{CO}_3$ in equilibrium. In the presence of excess hydroxide ions (OH$^-$), carbonic acid ($\text{H}_2\text{CO}_3$) would buffer a high PH, and in the presence of excess hydrogen ions (H$^+$), carbonate anion ($\text{HCO}_3^-$) would buffer a low PH which is the main mechanism behind respiratory acidolysis blood PH drop. In respiratory acidolysis the slight increase in bicarbonate act as a buffer for the increase in H$^+$ ions, which helps minimize the drop in PH blood value. Increase hydrogen ions (H$^+$) would lead to a slight decrease in the buffered blood PH, blood PH would be below 7.35. 17,18.

To evaluate patients with suspected respiratory acidosis, serum bicarbonate level and an arterial blood gas (ABG) are necessary. An elevated bicarbonate level HCO$_3^-$ (>30 mmHg), an elevated PCO$_2$ (>45 mmHg), and decreased pH (<7.35) would show on an ABG test in case of respiratory acidolysis. Respiratory acidosis can be either chronic or acute based on the relative increase in HCO$_3^-$ with respect to PCO$_2$. A HCO$_3^-$ will have increased by one mEq/L for every ten mmHg increase in PCO$_2$ over a few minutes in case of acute respiratory acidosis. A HCO$_3^-$ will have increased by four mEq/L for every ten mmHg increase in PCO$_2$ over a time course of days in case of chronic respiratory acidosis. A mixed respiratory-metabolic disorder may be present if it doesn’t show either patterns of acute or chronic respiratory acidosis. A drug screen may also be warranted in a patient who show an unexplained respiratory acidosis [19, 20].

The cause of respiratory acidosis must be treated once the diagnosis has been made. The rapid alkalinization of the cerebrospinal fluid (CSF) may lead to seizures therefore the hypercapnia should be corrected gradually. To help improve ventilation, pharmacologic therapy may be used. Beta-agonists, anticholinergic drugs, and methylxanthines (Bronchodilators) may be used in treating patients with obstructive airway diseases. In case of patients who overdose on opioid use, Naloxone can be prescribed [21, 22].

Acidolysis can be either respiratory or metabolic in origin depending on the measured pCO$_2$. If pCO$_2$ is greater than 40 to 45, it’s known to be due to decreased ventilation, and it’s called respiratory acidosis. If the pCO$_2$ is less than 40 since it is not the cause of the primary acid-base disturbance it’s called metabolic acidosis and it’s confirmed by a measured decreased in bicarbonate (normal range 21 to 28 mEq/L) [23]. In alkalosis, the fluids of the body are alkaline, the blood PH is high. When the blood has too little acid making it basic, the condition is called alkalosis, blood PH would be higher than the normal PH value of 7.45. There might be no noticeable symptoms for mild and chronic alkalosis. If there is a rapid PH increase, symptoms may include: confusion, nausea or vomiting, muscle twitching or spasms, numbness of the hands and feet, and/ or dizziness or lightheadedness [24].

Alkalosis can be either respiratory or metabolic depending on pCO$_2$. If pCO$_2$ is greater than 45 mmHg, it’s called metabolic alkalosis and the measured bicarbonate is greater than 29 mM. If pCO$_2$ is less than 32 mMHg, it’s called raspatory alkalosis and the measured bicarbonate is less than 22 mM [25].

A number of diseases, including rheumatoid arthritis (RA) are caused by alterations in tissue oxygen pressure. A condition known as hypoxia, low partial pressure of oxygen is involved inangiogenesis,apoptosis, cartilage degradation, inflammation, oxidative damage, and energy metabolism. Synovial hypoxia can be linked to pathogenic processes through indirect and direct effects on angiogenesis, oxidative damage, inflammation, cartilage damage, and bone resorption. Studies show that hypoxia and other promoters lead to inflammation in Rheumatoid Arthritis. The metabolic environment in the synovium is modified by hypoxia, and an autoimmune response is initiated by the presentation of the upregulated antigenic enzymes in the context of cellular stress. Hypoxia induces anaerobic glycolytic phenotype in the synovium. Several of the enzymes induced by this metabolic shift may become antigenic once anaerobic glycolysis is established in the synovium [27].

The abnormal biomechanics, attendant tissue-derived, and cell-derived factors causes Osteoarthritis (OA). The progression of Osteoarthritis is related to reactive oxygen species (ROS) and oxidative stress. It’s a multifactorial and polygenic joint disease. Reactive oxygen species targets the complex oxidative stress signaling pathways as it regulates chondrocyte senescence and apoptosis, extracellular matrix synthesis and degradation, along with synovial inflammation and dysfunction of the subchondral bone, and intracellular signaling processes. Osteoarthritis progresses from silent cartilage destruction to painful presentation. Free radicals...
mediate and amplify the sequence of joint degeneration in all tissues affected due to its chemical properties. Free radicals are the crucial factor involved in the inflammatory transformation of Osteoarthritis joints and all joint tissues disease development. 28 Both Osteoarthritis, and Rheumatoid arthritis is caused by reduced oxygen levels from increased consumption by inflammatory cells such as synoviocytes and the oxygen reduced delivery to synovial fluid [108-109].

Gout is a result of the presence of uric acid crystals build up in the joints. The production of uric acid in the joints is increased by the presence of excess carbon dioxide and the lack of presence of oxygen in the lungs [29, 30, 31]. While sleeping the produces less cortisone, an inflammation suppressant, the reduction of cortisone level might be contributing to gout disease [32, 33]. A person may have as much as 50% chance in getting gout disease if they suffer from sleep apnea. 5 Dehydration, loss of water in the body can contribute to the increase in the concentration of uric acid in joint fluids, enhancing the formation of uric acid crystals in the joints, causing gout attacks [32, 34]. Gout is a type of arthritis that is caused by uric acid concentration increase in the blood, it may cause debilitation due to uric acid deposit around tendons and joints. It’s the most controllable metabolic disease [100, 101]. Gout is classified into primary and secondary. Primary gout causes are unknown. There are known genetic defects causing elevated uric acid. The increase of uric acid in primary gout can be due to reduced ability to excrete uric acid found in smaller group of patients (30%), increased formation of serum uric acid found in most patients or both which is found in minority of patients. [36]. Rheumatoid arthritis can’t be cured by drugs, but it can be treated as it can/will come back. Treatment of rheumatoid arthritis includes using medications to slow the progression of the disease. Drugs including sulfasalazine (Azulfidine), methotrexate (Trexall), and other biologic drugs such as etanercept (Enbrel) and adalimumab (Humira) may be prescribed. Biologic drugs reduce the inflammation by targeting the immune system. Short-term treatment may include low-dose steroids [13]. Rheumatoid Arthritis is a multifactorial disease as both environmental and genetic factors contribute to the disease. Medical therapy is limited in most RA cases, it fails to address the causes of the disease. As in Osteoarthritis, the use of the NSAIDs drugs including aspirin is accompanied by the acceleration of factors that promote the disease process [35]. Examples of drugs currently in use are hydroxychloroquine, penicillamine, methotrexate, gold therapy, azathioprine, and cyclophosphamide. 36 A diet rich in vegetables, fiber, and whole foods, and low in meat, sugar, saturated fat, and refined carbohydrates (Western Diet) prevents the development of Rheumatoid Arthritis disease. Dietary therapy is to follow a vegetarian diet, eliminate food allergies, increase the intake of antioxidants, and alter the intake of fats and oils. Dietary therapy shows a tremendous promise in the treatment of Rheumatoid Arthritis. 37-39 Incomplete digestion may be a major factor in Rheumatoid Arthritis [40, 41]. Gamma-Linolenic acid (GLA) acts as a precursor to an anti-inflammatory prostaglandins’ series 1. Studies show that some patients have responded to GLA treatment while others didn’t. 42-44 Fish oil supplementation containing Omega-3 Fatty acid shows better and more positive response than GLA in the treatment of Rheumatoid Arthritis [43-50]. Due the neutralization of inflammation and support of collagen structure, dietary antioxidants such as Flavonoids is used in the treatment of Rheumatoid Arthritis. [53] Patients with Rheumatoid Arthritis have low levels of selenium [54, 55]. Selenium combined with Vitamin E had a positive effect in the treatment of Rheumatoid Arthritis. 56 Zinc levels are commonly low in Rheumatoid Arthritis patients, treatments with zinc supplements in the form of sulfates showed a slight therapeutic effect [57, 58]. Patients with Rheumatoid Arthritis are deficient in manganese containing Superoxide Dismutase. The injectable form of the enzyme (antioxidant enzyme Superoxide Dismutase (Manganese SOD) available in Europe are effective in the treatment of Rheumatoid Arthritis [159]. Oral administration of SOD showed no effect. 60 Patients with Rheumatoid Arthritis are also deficient in vitamin C. 61 Supplements with vitamin C gives some anti-inflammatory action [62, 63]. Pantothenic acid in blood has shown to be lower in Rheumatoid Arthritis patients. 64 Patients who received 2 g of calcium Pantothenate daily showed improvement. 65 Arthritis patients showed a lower sulfur content of the fingernails. 80, using injectable sulfur alleviated pain and swelling [66, 67]. High dose of Niacinamide (900-4000 mg) has shown good results in the treatment of both Osteoarthritis and Rheumatoid Arthritis. 68,69 The administration of 500 mg of Pantothenic acid shown no effect on treatment of Rheumatoid Arthritis [70].

A vegetarian diet following short-term fasting showed reduction of Rheumatoid Arthritis disease activity [98-99]. Treatment of Osteoarthritis includes prescribing medications called bisphosphonates: risedronate (Actonel), and alendronate (Fosamax). In most Osteoarthritis cases drug treatment has shown to be ineffective and if the failure of nonsurgical treatment is consistent in at least three to six months, surgical replacement of large joints: knee replacement or hip replacement, is needed [13].

The therapeutic goal in the natural treatment of osteoarthritis is to enhance and repair collagen matrix and the regeneration of the connective tissues. It’s recommended to lose excess wait causing increase stress on joints, the use of a healthy diet rich in complex carbohydrates and fiber, and to minimize and eliminate the consumption of nightshade vegetables [71]. Nightshade vegetables include potatoes, tomatoes, peppers, tobacco, and eggplants are known to contain alkaloids that promote the inflammatory degradation of the joints and inhibit the normal collagen repair. The high intake of antioxidants is shown to inhibit the progression of the disease and reduce the risk of cartilage loss [72]. As some people age, they lose their ability to manufacture sufficient levels of glucosamine. Glucosamine in form of glucosamine sulfate drug, is used to incorporate sulfur into the cartilages, and stimulate the manufacture of glycosaminoglycans [73, 74]. Chondroitin sulfate is drug that is composed of repeating units of derivatives of glucosamine sulfate attached to sugar molecules and is known to be a less effective drug than that of glucosamine sulfate due to its low absorption 0-13% compared to the solubility of glucosamine sulfate which is 90-98%, [75-77] High dose of Niacinamide (900-4000 mg) has shown good results in the treatment of both Osteoarthritis and Rheumatoid Arthritis. 78,79 Superoxide Dismutase (SOD) injections showed a significant effect in the treatment of osteoarthritis. 80,81 Vitamin E has the ability of stimulate the formation of new cartilage components, inhibit the breakdown of cartilages, and the administration of 600 IU showed significant benefits. 82,83 Vitamin C is like vitamin E, protects and enhances cartilages formation [83, 84, 85]. The administration of a little amount (12.5 mg) of Pantothenic acid is effective in relieving symptoms of Osteoarthritis [86, 87]. Joint degradation is accelerated by the deficiency of one of vitamins: A, B6, Zinc, Copper, and Boron, and
supplementation at appropriate level may promote cartilage synthesis and repair. 36 Niacinamide (900-4000 mg) has shown good results in the treatment of Osteoarthritis [89, 90].

Treatments of gout Joint disease includes prescribed medications, such as allopurinol and febuxostat [91]. Treatment of sleep apnea that would include continuous positive airway pressure (CPAP) machine or another treatment device to increase oxygen intake while sleeping are used to increased oxygen level and lower uric acid production and reduce the risk of a gout attacks [92]. Decrease the concentration of uric acid, by drinking fluids would increase blood volume lowers the risk of a gout attacks. Other lifestyle changes that may lower the risk of a gout attack include getting regular exercise, eating a plant-based diet that is low in purines and whole foods, and losing excess weight [93, 94]. The standard medical treatment for the disease is the administration of Colchicine, indomethacin, naproxen, fenoprofen or phenylbutazone. The dietary treatment involves fluid intake, low fat, low purine and low protein intake, consumption of complex carbohydrates, elimination of alcohol intake, and achieving the ideal body weight [101, 102, 103]. The natural treatment of gout disease includes the consumption of nutritional supplements: eicosapentaenioic acid. vitamin E, folic acid, amino acids such as alanine, aspartic acid, glutamic acid, glycine, and niacin and vitamin C. Eicosapentaenioic acid, omega-3 oils are found useful in the treatment of gout joint disease. Vitamin E, it acts as antioxidants and inhibits the formation of leukotrienes 104 Folic acid is known to inhibits the production of uric acid by inhibiting enzyme xanthine oxidase [105]. Alanine, aspartic acid, glutamic acid, glycine, these amino acids are shown to lower serum. uric acid level by increasing uric acid excretion. 3 Niacin and Vitamin C, High doses of vitamin C and Niacin are used int the treatment of gout; niacin competes with uric acid in excretion and vitamin C increases the formation of uric acid in small group of people [106, 107].

Conclusion
It has been found that if the ability to breath is obstructed by a pulmonary condition or by physical block limits or by prolonged hypoventilation raspatory acidosis is caused. Three main types of joint diseases were found to be directly related to hypoxia (Low levels of oxygen in your body tissues), hypercapnica (Excessive amounts of carbon dioxide in the blood) and blood PH imbalance: Rheumatoid Arthritis, Osteoarthritis, and gout joint diseases. Both Osteoarthritis, and Rheumatoid arthritis was found to be caused by reduced oxygen levels from increased consumption by inflammatory cells such as synovocytes and the oxygen reduced delivery to synovial fluid. Rheumatoid arthritis was found to be directly caused by alterations in tissue oxygen pressure. Osteoarthritis was found to be directly related to aging, as the ability to synthesize and restoring cartilage structure decreases, and the progression of the disease was found to be directly related to the presence of reactive oxygen species and oxidative stress. Gout joint disease was found to be directly caused by the presence of uric acid crystals build up in the joints, and the crystal buildup increased by the presence of excess carbon dioxide.

The natural treatment of both rheumatoid arthritis, and osteoarthritis was found to include minimizing the consumption of nightshade vegetables, and eliminating the use of nonsteroidal anti-inflammatory drugs (NSAIDs drugs) as they cause the degradation of joint cartilages. High dose of

References


