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Vinegar: Medicinal Uses and Antiglycemic Effect

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Abstract

Vinegar folklore is as colorful as it is practical. Legend states that a courtier in Babylonia (c. 5000 BC) “discovered” wine, formed from unattended grape juice, leading to the eventual discovery of vinegar and its use as a food preservative. Hippocrates (c. 420 BC) used vinegar medicinally to manage wounds. Hannibal of Carthage (c. 200 BC), the great military leader and strategist, used vinegar to dissolve boulders that blocked his army's path. Cleopatra (c. 50 BC) dissolved precious pearls in vinegar and offered her love potion to Anthony. Sung Tse, the 10th century creator of forensic medicine, advocated hand washing with sulfur and vinegar to avoid infection during autopsies. Based on the writings of US medical practitioners dating to the late 18th century, many ailments, from dropsy to poison ivy, croup, and stomachache, were treated with vinegar,[1] and, before the production and marketing of hypoglycemic agents, vinegar “teas” were commonly consumed by diabetics to help manage their chronic aliment. This review examines the scientific evidence for medicinal uses of vinegar, focusing particularly on the recent investigations supporting vinegar's role as an antiglycemic agent. Epidemiologic studies and clinical trials were identified by a MEDLINE title/abstract search with the following search terms: vinegar, glucose; vinegar, cancer; or vinegar, infection. All relevant randomized or case-control trials were included in this review.

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Vinegar Production

Vinegar, from the French *vin aigre*, meaning "sour wine," can be made from almost any fermentable carbohydrate source, including wine, molasses, dates, sorghum, apples, pears, grapes, berries, melons, coconut, honey, beer, maple syrup, potatoes, beets, malt, grains, and whey. Initially, yeasts ferment the natural food sugars to alcohol. Next, acetic acid bacteria (*Acetobacter*) convert the alcohol to acetic acid. Commercial vinegar is produced by either fast or slow fermentation processes. For the quick methods, the liquid is oxygenated by agitation and the bacteria culture is submerged permitting rapid fermentation. The slow methods are generally used for the production of the traditional wine vinegars, and the culture of acetic acid bacteria grows on the surface of the liquid and fermentation proceeds slowly over the course of weeks or months. The longer fermentation period allows for the accumulation of a nontoxic slime composed of yeast and acetic acid bacteria, known as the *mother* of vinegar. Vinegar eels (nematoda*Turbatrix aceti*) feed on these organisms and occur in naturally fermenting vinegar.[2] Most manufacturers filter and pasteurize their product before bottling to prevent these organisms from forming. After opening, *mother* may develop in stored vinegar; it is considered harmless and can be removed by filtering. Many people advocate retaining the *mother* for numerous, but unsubstantiated, health effects.

The chemical and organoleptic properties of vinegars are a function of the starting material and the fermentation method. Acetic acid, the volatile organic acid that identifies the product as vinegar, is responsible for the tart flavor and pungent, biting odor of vinegars. However, acetic acid should not be considered synonymous with vinegar. The US Food and Drug Administration (FDA) states that diluted acetic acid is not vinegar and should not be added to food products customarily expected to contain vinegar.[3] Other constituents of vinegar include vitamins, mineral salts, amino acids, polyphenolic compounds (eg, galic acid, catechin, caffeic acid, ferulic acid), and nonvolatile organic acids (eg, tartaric, citric, malic, lactic).[4,5]

In the United States, vinegar products must contain a minimum of 4% acidity.[6] European countries have regional standards for vinegar produced or sold in the area. White distilled vinegars are generally 4% to 7% acetic acid whereas cider and wine vinegars are 5% to 6% acetic acid. Specialty vinegars are grouped as herbal or fruit vinegars. Herbal vinegars consist of wine vinegars or white distilled vinegars, which may be seasoned with garlic, basil, tarragon, cinnamon, clove, or nutmeg. Fruit vinegars are wine and white vinegars sweetened with fruit or fruit juice to produce a characteristic sweet-sour taste. Traditional vinegars are produced from regional foods according to well-established customs. The balsamic vinegar of Modena, Italy, is made from the local white Trebbiano grapes, which are harvested as late as possible, fermented slowly, and concentrated by aging in casks of various woods. Traditional rice wine vinegars are produced in Asia, coconut and cane vinegars are common in India and the Philippines, and date vinegars are popular in the Middle East.

Medicinal Uses of Vinegar

Anti-infective Properties

The use of vinegar to fight infections and other acute conditions dates back to Hippocrates (460-377 BC; the father of modern medicine), who recommended a vinegar preparation for cleaning ulcerations and for the treatment of sores. Oxymel, a popular ancient medicine composed of

honey and vinegar, was prescribed for persistent coughs by Hippocrates and his contemporaries, and by physicians up to modern day.[7] The formulation of oxymel was detailed in the *British Pharmacopoeia* (1898) and the *German Pharmacopoeia* (1872), and, according to the *French Codex* (1898), the medicine was prepared by mixing virgin honey, 4 parts, with white wine vinegar, 1 part, concentrating and clarifying with paper pulp.[8]

Recent scientific investigations clearly demonstrate the antimicrobial properties of vinegar, but mainly in the context of food preparation.[9–12] Experts advise against using vinegar preparations for treating wounds.[13] At concentrations nontoxic to fibroblasts and keratinocytes (≤ 0.0025%), acetic acid solutions were ineffective at inhibiting the growth of *Escherichia coli*, group D *Enterococcus*, or *Bacteroides fragilis* bacteria, and only slightly effective at inhibiting the growth of *Staphylococcus aureus* and *Pseudomonas aeruginosa* bacteria.[13] Similarly, experts caution against using vinegar as a household disinfectant against human pathogens because chemical disinfectants are more effective.[14,15] However, undiluted vinegar may be used effectively for cleaning dentures, and, unlike bleach solutions, vinegar residues left on dentures were not associated with mucosal damage.[16]

Although investigations have demonstrated the effectiveness of diluted vinegar (2% acetic acid solution at pH 2) for the treatment of ear infections (otitis externa, otitis media, and granular myringitis),[17,18] the low pH of these solutions may irritate inflamed skin and damage cochlear outer hair cells.[19] Immediate vinegar application at the site of jellyfish stings is practiced at various coastal locations around the world[20,21] because vinegar deactivates the nematocysts. However, hot-water immersion is considered the most efficacious initial treatment for jellyfish envenomation because the venom is deactivated by heat.[22,23]

In the popular media, vinegar is commonly recommended for treating nail fungus, head lice, and warts, yet scientific support for these treatment strategies is lacking. Takano-Lee and colleagues[24] demonstrated that, of 7 home remedies tested, vinegar was the least effective for eliminating lice or inhibiting the hatching of eggs. Scattered reports suggest that the successive topical application of highly concentrated acetic acid solutions (up to 99%) alleviated warts, [25,26] presumably due to the mechanical destruction of wart tissue. One treatment protocol, however, required local anesthesia, excision, and rapid neutralization at the site of application, thus limiting its use by the lay public.

Although not a treatment modality, vinegar washes are used by midwives in remote, poorly resourced locations (eg, Zimbabwe and the Amazon jungle) to screen women for the human papilloma virus infection.[27,28] Contact with acetic acid causes visual alterations of the viral lesions permitting rapid detection of infection with 77% sensitivity[29] and the option of immediate treatment with cryotherapy.

Cardiovascular Effects

Kondo and colleagues[30] reported a significant reduction in systolic blood pressure (approximately 20 mm Hg) in spontaneously hypertensive (SHR) rats fed a standard laboratory diet mixed with either vinegar or an acetic acid solution (approximately 0.86 mmol acetic acid/day for 6

weeks) as compared with SHR rats fed the same diet mixed with deionized water. These observed reductions in systolic blood pressure were associated with reductions in both plasma renin activity and plasma aldosterone concentrations (35% to 40% and 15% to 25% reductions in renin activity and aldosterone concentrations, respectively, in the experimental vs control SHR rats). Others have reported that vinegar administration (approximately 0.57 mmol acetic acid, orally) inhibited the renin-angiotensin system in nonhypertensive Sprague-Dawley rats.[31]

Trials investigating the effects of vinegar ingestion on the renin-angiotensin system have not been conducted in humans, and there is no scientific evidence that vinegar ingestion alters blood pressure in humans. In their report, Kondo and colleagues[30] speculated that dietary acetic acid promoted calcium absorption and thereby downregulated the renin-angiotensin system.[32] In the rat model, acetic acid administration enhanced calcium absorption and retention[33]; moreover, in humans, calcium absorption in the distal colon was enhanced by acetate.[34] Clearly, much work is needed to establish whether vinegar ingestion alters calcium absorption and/or blood pressure regulation in humans.

Whether chronic vinegar ingestion affects other risk factors for cardiovascular disease in humans is not known. Hu and colleagues[35] reported a significantly lower risk for fatal ischemic heart disease among participants in the Nurses' Health Study who consumed oil-and-vinegar salad dressings frequently (5-6 times or more per week) compared with those who rarely consumed them (multivariate RR: 0.46; CI: 0.27-0.76, *P* for trend = .001). Frequent consumption of mayonnaise or other creamy salad dressings was not significantly associated with risk for ischemic heart disease in this population (multivariate RR: 0.84; CI: 0.50-1.44, *P* for trend = .44). The study authors contend that because oil and vinegar dressings are a major dietary source of dietary alpha-linolenic acid, an antiarrhythmic agent, alpha-linolenic acid may potentially be the beneficial ingredient of this food.[35] Yet, creamy, mayonnaise-based salad dressings are also rich in alpha-linolenic acid and did not show the same risk benefit as the oil and vinegar dressings.

Antitumor Activity

In vitro, sugar cane vinegar (Kibizu) induced apoptosis in human leukemia cells,[36] and a traditional Japanese rice vinegar (Kurosu) inhibited the proliferation of human cancer cells in a dose-dependent manner.[37] An ethyl acetate extract of Kurosu added to drinking water (0.05% to 0.1% w/v) significantly inhibited the incidence (−60%) and multiplicity (−50%) of azoxymethane-induced colon carcinogenesis in male F344 rats when compared with the same markers in control animals.[38] In a separate trial, mice fed a rice-shochu vinegar-fortified feed (0.3% to 1.5% w/w) or control diet were inoculated with sarcoma 180 (group 1) or colon 38 (group 2) tumor cells (2×10^6 cells subcutaneously).[39] At 40 days post-inoculation, vinegar-fed mice in both experimental groups had significantly smaller tumor volumes when compared with their control counterparts. A prolonged life span due to tumor regression was also noted in the mice ingesting rice-shochu vinegar as compared with controls, and in vitro, the rice-shochu vinegar stimulated natural killer cell cytotoxic activity.[39]

The antitumor factors in vinegar have not been identified. In the human colonic adenocarcinoma cell line Caco-2, acetate treatment, as well as treatment with the other short-chain fatty acids (SCFA) n-butyrate and propionate, significantly prolonged cell doubling time, promoted cell differentiation, and inhibited cell motility.[40] Because bacterial fermentation of dietary fiber in the colon yields the SCFA, the investigators concluded that the antineoplastic effects of dietary fiber may relate in part to the formation of SCFA. Others have also documented the antineoplastic effects of the SCFA in the colon, particularly n-butyrate.[41] Thus, because acetic acid in vinegar deprotonates in the stomach to form acetate ions, it may possess antitumor effects.

Vinegars are also a dietary source of polyphenols,[6] compounds synthesized by plants to defend against oxidative stress. Ingestion of polyphenols in humans enhances in vivo antioxidant protection and reduces cancer risk.[42] Kurosu vinegar is particularly rich in phenolic compounds, and the in-vitro antioxidant activity of an ethyl acetate extract of Kurosu vinegar was similar to the antioxidant activity of alpha-tocopherol (vitamin E) and significantly greater than the antioxidant activities of other vinegar extracts, including wine and apple vinegars.[43] Kurosu vinegar extracts also suppressed lipid peroxidation in mice treated topically with H_2O_2-generating chemicals.[43] Currently, much interest surrounds the role of dietary polyphenols, particularly from fruits, vegetables, wine, coffee, and chocolate, in the prevention of cancers as well as other conditions including cardiovascular disease[44]; perhaps vinegar can be added to this list of foods and its consumption evaluated for disease risk.

Epidemiologic data, however, is scarce and unequivocal. A case-control study conducted in Linzhou, China, demonstrated that vinegar ingestion was associated with a decreased risk for esophageal cancer (OR: 0.37).[45] However, vinegar ingestion was associated with a 4.4-fold greater risk for bladder cancer in a case-control investigation in Serbia.[46]

Blood Glucose Control

The antiglycemic effect of vinegar was first reported by Ebihara and Nakajima[47] in 1988. In rats, the blood glucose response to a 10% corn starch load was significantly reduced when coadministered with a 2% acetic acid solution.[45] In healthy human subjects, although the glucose response curve was not significantly altered, the area under the insulin response curve following the ingestion of 50 g sucrose was reduced 20% when coadministered with 60 mL strawberry vinegar.[47] Several years later, Brighenti and colleagues[48] demonstrated in normoglycemic subjects that 20 mL white vinegar (5% acetic acid) as a salad dressing ingredient reduced the glycemic response to a mixed meal (lettuce salad and white bread containing 50 g carbohydrate) by over 30% ($P < .05$). Salad dressings made from neutralized vinegar, formulated by adding 1.5 g sodium bicarbonate to 20 mL white vinegar, or a salt solution (1.5 g sodium chloride in 20 mL water) did not significantly affect the glycemic response to the mixed meal.[48] Separate placebo-controlled trials have corroborated the meal-time, antiglycemic effects of 20 g vinegar in healthy adults.[49–51]

While compiling a glycemic index (GI) table for 32 common Japanese foods, Sugiyama and colleagues[52] documented that the addition of vinegar or pickled foods to rice (eg, sushi) decreased the GI of rice by 20% to 35%. In these trials, healthy fasted subjects ingested the reference and

test foods, each containing 50 g carbohydrate, on random days, and the food GI was calculated using the areas under the 2-hour blood glucose response curves. In the vinegar-containing foods, the amount of acetic acid was estimated to be 0.3-2.3 g, an amount similar to that found in 20 g vinegar (approximately 1 g). Ostman and colleagues[53] reported that substitution of a pickled cucumber (1.6 g acetic acid) for a fresh cucumber (0 g acetic acid) in a test meal (bread, butter, and yogurt) reduced meal GI by over 30%[53] in healthy subjects.

Recently, the antiglycemic property of vinegar was demonstrated to extend to individuals with marked insulin resistance or type 2 diabetes.[54] In this crossover trial, individuals with insulin resistance (n = 11, fasting insulin concentrations greater than 20 mU/mL) or with diagnosed type 2 diabetes (n = 10) consumed a vinegar test drink (20 g vinegar, 40 g water, 1 tsp saccharine) or placebo immediately before the consumption of a mixed meal (87 g total carbohydrate). In the insulin-resistant subjects, vinegar ingestion reduced postprandial glycemia 64% as compared with placebo values (P = .014) and improved postprandial insulin sensitivity by 34% (P= .01). In individuals with type 2 diabetes, vinegar ingestion was less effective at reducing mealtime glycemia (−17%, P = .149); however, vinegar ingestion was associated with a slight improvement in postprandial insulin sensitivity in these subjects (+19%, P = .07).[54] The lack of a significant effect of vinegar on mealtime glycemia in the type 2 diabetics may be related to the use of venous blood sampling in this trial. Greater within-subject variation in glucose concentrations are noted for venous blood as compared with capillary blood; moreover, the concentration of glucose in venous blood is lower than that in capillary blood. Thus, capillary blood sampling is preferred for determining the glycemic response to food.[55]

The marked antiglycemic effect of vinegar in insulin-resistant subjects is noteworthy and may have important implications. Multicenter trials have demonstrated that treatment with antiglycemic pharmaceuticals (metformin or acarbose) slowed the progression to diabetes in high-risk individuals[56,57]; moreover, because these drugs improved insulin sensitivity, the probability that individuals with impaired glucose tolerance would revert to a normal, glucose-tolerant state over time was increased.[57]

In healthy subjects, Ostman and colleagues[58] demonstrated that acetic acid had a dose-response effect on postprandial glycemia and insulinemia. Subjects consumed white bread (50 g carbohydrate) alone or with 3 portions of vinegar containing 1.1, 1.4, or 1.7 g acetic acid. At 30 minutes post-meal, blood glucose concentrations were significantly reduced by all concentrations of acetic acid as compared with the control value, and a negative linear relationship was calculated between blood glucose concentrations and the acetic acid content of the meal (r = −0.47, P = .001). Subjects were also asked to rate feelings of hunger/satiety on a scale ranging from extreme hunger (−10) to extreme satiety (+10) before meal consumption and at 15-minute intervals after the meal. Bread consumption alone scored the lowest rating of satiety (calculated as area under the curve from time 0-120 minutes). Feelings of satiety increased when vinegar was ingested with the bread, and a linear relationship was observed between satiety and the acetic acid content of the test meals (r = 0.41, P = .004).[58]

In a separate trial, healthy adult women consumed fewer total calories on days that vinegar was ingested at the morning meal.[50] In this trial, which used a blinded, randomized, placebo-controlled, crossover design, fasting participants consumed a test drink (placebo or vinegar) followed by the test meal composed of a buttered bagel and orange juice (87 g carbohydrate). Blood samples were collected for 1 hour after the meal. At the end of testing, participants were allowed to follow their normal activities and eating patterns the remainder of the day, but they were instructed to record food and beverage consumption until bedtime. Vinegar ingestion, as compared with placebo, reduced the 60-minute glucose response to the test meal (−54%, $P < .05$) and weakly affected later energy consumption (−200 kilocalories, $P = .111$). Regression analyses indicated that 60-minute glucose responses to test meals explained 11% to 16% of the variance in later energy consumption ($P < .05$).[50] Thus, vinegar may affect satiety by reducing the meal-time glycemic load. Of 20 studies published between 1977 and 1999, 16 demonstrated that low-glycemic index foods promoted postmeal satiety and/or reduced subsequent hunger.[59]

It is not known how vinegar alters meal-induced glycemia, but several mechanisms have been proposed. Ogawa and colleagues examined the effects of acetic acid and other organic acids on disaccharidase activity in Caco-2 cells.[60] Acetic acid (5 mmol/L) suppressed sucrase, lactase, and maltase activities in concentration- and time-dependent manners as compared with control values, but the other organic acids (eg, citric, succinic, L-maric, and L-lactic acids) did not suppress enzyme activities. Because acetic acid treatment did not affect the de-novo synthesis of the sucrase-isomaltase complex at either the transcriptional or translational levels, the investigators concluded that the suppressive effect of acetic acid likely occurs during the posttranslational processing of the enzyme complex.[60] Of note, the lay literature has long proclaimed that vinegar interferes with starch digestion and should be avoided at meal times.[61]

Several investigations examined whether delayed gastric emptying contributed to the antiglycemic effect of vinegar. Using noninvasive ultrasonography, Brighenti and colleagues[50] did not observe a difference in gastric emptying rates in healthy subjects consuming bread (50 g carbohydrate) in association with acetic acid (ie, vinegar) vs sodium acetate (ie, vinegar neutralized by the addition of sodium bicarbonate); however, a significant difference in post-meal glycemia was noted between treatments with the acetic acid treatment lowering glycemia by 31.4%. In a later study, Liljeberg and Bjorck[62] added paracetamol to the bread test meal to permit indirect measurement of the gastric emptying rate. Compared with reference values, postmeal serum glucose and paracetamol concentrations were reduced significantly when the test meal was consumed with vinegar. The results of this study should be carefully considered, however, because paracetamol levels in blood may be affected by food factors and other gastrointestinal events. In rats fed experimental diets containing the indigestible marker polyethylenglycol and varying concentrations of acetic acid (0, 4, 8, 16 g acetic acid/100 g diet), dietary acetic acid did not alter gastric emptying, the rate of food intake, or glucose absorption.[63]

Safety of Vinegar

Vinegar's use as a condiment and food ingredient spans thousands of years, and perhaps its use can be labeled safe by default. Yet there are rare reports in the literature regarding adverse reactions to vinegar ingestion. Inflammation of the oropharynx and second-degree caustic injury of

the esophagus and cardia were observed in a 39-year-old woman who drank 1 tablespoon of rice vinegar in the belief it would dislodge a piece of crab shell from her throat.[64] (The use of vinegar in these situations is a popular Chinese folk remedy.) Her symptoms resolved spontaneously after several days. Esophageal injury by vinegar is likely very rare but deserves notice. Chronic inflammation of the esophagus is a cancer risk; but, as reported previously,[45] vinegar use was inversely related to risk for cancer of the esophagus.

The unintentional aspiration of vinegar has been associated with laryngospasm and subsequent vasovagal syncope that resolved spontaneously.[65] Hypokalemia was observed in a 28-year-old woman who had reportedly consumed approximately 250 mL apple cider vinegar daily for 6 years.[66] Although speculative, the hypokalemia was attributed to elevated potassium excretion related to the bicarbonate load from acetate metabolism.

These complications attributed to vinegar ingestion are isolated occurrences, but with the increased interest in vinegar as adjunct therapy in diabetes, carefully controlled trials to examine potential adverse effects of regular vinegar ingestion are warranted.

Summary

For more than 2000 years, vinegar has been used to flavor and preserve foods, heal wounds, fight infections, clean surfaces, and manage diabetes. Although vinegar is highly valued as a culinary agent, some varieties costing $100 per bottle, much scrutiny surrounds its medicinal use. Scientific investigations do not support the use of vinegar as an anti-infective agent, either topically or orally. Evidence linking vinegar use to reduced risk for hypertension and cancer is equivocal. However, many recent scientific investigations have documented that vinegar ingestion reduces the glucose response to a carbohydrate load in healthy adults and in individuals with diabetes. There is also some evidence that vinegar ingestion increases short-term satiety. Future investigations are needed to delineate the mechanism by which vinegar alters postprandial glycemia and to determine whether regular vinegar ingestion favorably influences glycemic control as indicated by reductions in hemoglobin A1c. Vinegar is widely available; it is affordable; and, as a remedy, it is appealing. But whether vinegar is a useful adjunct therapy for individuals with diabetes or prediabetes has yet to be determined.

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