

Numerous natural vinegar products have been used around the world for centuries, of which Apple Cider Vinegar (ACV) has gained the most attention in recent decades. The potential therapeutic effects of ACV discussed below may be attributed to the bioactive constituents of the organic acids generated in ACV production, including acetic, citric, formic, lactic, malic, and succinic acids,¹ which have demonstrated potential antimicrobial, antioxidative, antitumor, antiobesity, antihypertensive, and cholesterol-lowering properties in *in vitro* and animal studies, and antiglycemic properties in human studies².

Antimicrobial - The antimicrobial properties of ACV stem from the ability of organic acids (mainly acetic, citric, lactic, succinic and malic) to invade microorganism cell membranes and induce cell death. It should be noted that while this process may seem aggressive to otherwise normal cells, organic acids are naturally present in a variety of fresh fruits, vegetables and fermented foods for this purpose, as the acids specifically target the cell membrane of microorganisms, rather than healthy, functioning human cells^{2,3,4}.

Antioxidative - Oxidative stress presents in a variety of human diseases and conditions when reactive oxygen species (ROS) like superoxide, hydrogen peroxide and hydroxyl radicals contribute to damage at the cellular and tissue level⁵. However, oxidative stress can be balanced by antioxidant activity from bioactive substances such as polyphenolic compounds and vitamins, which are thought to inhibit the reactivity of ROS⁶. ACV has a number of phenolic compounds that contribute to its antioxidative capacity, including gallic acid, catechin, epicatechin, chlorogenic acid, caffein acid, and p-coumaric acid^{2,7}.

Antiglycemic - Insulin resistance and subsequent pre-diabetes or Type II Diabetes Mellitus plagues hundreds of millions of people worldwide, and in the majority of these individuals, both tissue insensitivity to insulin and insulin response to blood glucose is impaired. Perhaps the most established therapeutic effect of ACV is seen in its antiglycemic activity. In both animals and humans, acetic acid (80% of ACV), has been shown to significantly improve insulin sensitivity and suppress the rise in blood glucose after meals⁸. This effect is thought to be due to acetic acid's inhibition of digestive enzymes responsible for rapidly digesting and facilitating uptake of glucose, resulting in an aggressive rise in blood glucose, however this proposed mechanism is not yet well-established^{2,8}. Another proposed mechanism of action is vinegar's effect on slowing gastric emptying, resulting in a slower release of glucose into the blood stream¹⁴, however this too has yielded a variety of results in both human and animal studies. A number of individuals with controlled diabetes experience an increase in blood glucose in the early morning hours after a night-long fasting period, also known as the dawn phenomenon. A pilot study in humans found that two tablespoons of ACV at bedtime reduced morning blood glucose by 4-6%, results that may further indicate the antiglycemic capacity of ACV⁹. In another human study, a high carbohydrate meal was served with and without acetic acid to measure post-prandial blood glucose, insulin response and satiety. A dose-response relationship was seen 30 minutes post-prandial, with higher acetic acid consumption (28 mmol), resulting in the most significant lowering of blood glucose, highest stimulation of insulin response, and the highest level of satiety¹⁰.

Antitumor - Although little research has been performed on ACV or acetic acid and their involvement in the inhibition of tumor growth, ACV's potential antioxidative activity may play a role in early stages of tumor development. One animal study investigated the products of acetic acid fermentation present in the production of ACV and found a dose-response effect on the content of medium-sized alpha-glycans, active against tumors in experimental mice¹¹.

Antiobesity - The antiobesity effect of ACV may, from what we know, be attributed to its earlier induction of satiety, not lipid oxidation, which has been extensively studied to little avail. Variable results have been demonstrated in studies evaluating the rate of gastric emptying when preceding a meal with ACV, however some have shown a slower rate, which inherently induces satiety sooner, hence the plausibility of ACV's impact on weight loss².

Antihypertensive - *In vitro* and animal studies have suggested a multifactorial mechanism for acetic acid's potential hypotensive effect. A number of studies report the inhibition of angiotensin-converting enzyme, a key component of the renin-angiotensin system responsible for constriction of vascular muscle, and stimulation of aldosterone secretion and subsequent water and sodium retention^{2,12}. Acetic acid also has been demonstrated to have inhibitory activity on renin, one of the primary steps of the system responsible for hypertensive effects^{2,12}.

Cholesterol-lowering effect - Atherosclerosis, a chief contributor to the development of cardiovascular disease and associated mortality, is an inflammatory process impacted by the formation of oxidized low-density lipoproteins (LDLs). The polyphenol content of vinegars, most notably the high content of chlorogenic acid in ACV, has suggested a potential inhibition of LDL oxidation in the blood stream, however further research is warranted in this area as few studies have been published that demonstrate this effect^{2,7}. In one animal study, acetic acid reduced serum triglyceride levels and accumulation of fat in adipose and liver tissue which the investigators credited to the inhibition of lipogenesis and promotion of triglyceride excretion in fecal bile acid, mechanisms which may further suggest acetic acid's potential protective effect on cardiovascular disease risk factors¹³.

Prepared by: Leanna Shea, MS, RD*

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Research has consistently demonstrated the poor absorptive capacity and bioavailability of curcumin, turmeric's bioactive constituent, and most established contributing component in the benefits of turmeric. In numerous animal studies, greater than 90-95% of oral doses of curcumin, is excreted¹. Studies measuring human serum levels of curcumin 1, 2, 3, and 4, hours after a large dose (500-8000mg) find minimal to no evidence of curcumin in the blood, which further demonstrate how poorly the gut absorbs curcumin¹. The limited absorption of turmeric stems from curcumin's ability to remain highly stable in acidic conditions, such as gastric acid (pH 1.5-3.0), leading to the inability to properly break down and prepare for absorption in the small intestine². However, the stability of curcumin in an acidic medium, such as in ACV (pH 2.8-3.0), will benefit its shelf-life and ability to stay intact in the drink during preparation and transportation.

Literature on the kinetics of curcumin metabolism once absorbed into the blood, show that curcumin is rapidly metabolized in the liver by a process called glucuronosylation, and subsequently eliminated in the bile^{3,4}. In short, of the small amount of curcumin that is successfully absorbed into the blood, most is excreted before any benefits can occur. It is therefore critical that turmeric administration is accompanied by one of the well-studied natural compounds known to enhance curcumin's bioavailability: piperine, quercetin, and genistein. The most established and widely used natural compound for enhanced digestion and absorption of curcumin is piperine, the bioactive constituent of black pepper. A 2016 study used advanced chemical and molecular technology to inform the mechanism by which piperine enhances curcumin availability. The mechanism of piperine in curcumin metabolism is two-fold; Piperine successfully competes with curcumin to bind UDP-glucuronosyltransferase (UGT), the liver enzyme responsible for the breakdown of curcumin into bile. The competitive bidding of piperine to UGT limits the availability of UGT to act on curcumin⁴. Piperine also intercalates with curcumin to form a structure that not only protects curcumin from UGT activity, but increases the binding affinity of piperine to UGT, further contributing to the inhibition of curcumin breakdown⁴. A clinical study using both animal and human subjects demonstrated the difference in bioavailability between administration of curcumin alone and concomitant administration of piperine, by measuring serum concentration and elimination of curcumin. In rats, bioavailability was increased by 154%, while humans demonstrated a 2000% increase in bioavailability between 2g (1tsp) curcumin alone, and 2g curcumin with 20mg piperine⁵.

Recommendation:

You could either use black pepper or black pepper extract, which would increase the piperine content in a given amount. Commercial black pepper contains 5-9% piperine. Because the study previously mentioned showed a remarkable impact on curcumin bioavailability with just 20mg piperine, black pepper would suffice. In terms of amount, at 5-9% piperine in black pepper, you would need at least 400mg (.4g) of black pepper to get 20mg piperine. Ground black pepper is about 2.2g per tsp., so I would say a 1/2 tsp. or 1.1g black pepper would be more than enough per drink.

*Addendum: Based on analyses performed by Krueger Food Laboratories, Inc., detecting piperine in the amount of 0.42g per 100g black pepper extract, if using black pepper extract in place of black pepper, I would recommend using 5g extract in order to procure the desired 20mg piperine per service as previously calculated.

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Ginger, one of the most frequently used ingredients in Eastern Medicine, has demonstrated a variety of therapeutic characteristics, most notably of analgesic, anti-inflammatory, and digestive nature, along with anti-microbial, tumor pathway-suppressing, and anti-oxidant activity¹. The root's active constituents, gingerols, shogaols, and lipophilic rhizome extracts may be responsible for the biologic activity reported upon *in vitro* and *in vivo* administration.

The therapeutic use of ginger for digestive health is perhaps the root's longest standing claim to fame. A number of pathways involved in gut function are thought to be directly activated by ginger's active constituents, particularly gingerols and shogaols. The TRPV1 pathway, responsible for initiating gastric motility and secretion of gastric and pancreatic digestive enzymes, is activated by the binding of gingerols and shogaols². Moreover, ginger extract (primarily gingerol), activates receptor TRPA1, additionally catalyzing activity such as gastric emptying, secretion of bile for fat digestion, sense of satiation, and reduced intestinal transit time by initiating secretion of gastric and pancreatic digestive enzymes, similar to the TRPV1 pathway². While the root's active components play a role in optimizing digestion, ginger also acts as an antagonist to both serotonin and acetylcholine receptors, which is thought to explain its antispasmodic, and therefore antiemetic, effect². These receptors, to which ginger's constituents bind, mediate the pathways involved in nausea and vomiting. A number of clinical studies have demonstrated the prokinetic and antiemetic effect of ginger. One study reported that consuming ginger prior to meals increased the number and frequency of contractions in the stomach and small intestine². Additionally, a systematic review of six clinical trials found that ginger was more effective than placebo in reducing post-operative nausea and vomiting².

In addition to ginger's therapeutic properties in digestive health, the root has been extensively studied for a variety of other health benefits. In mice, a gingerol dose of 25mg/kg (around 1.7g for a 150-pound individual) demonstrated a significant analgesic effect when compared to placebo, measured by writhing after induction of pain³. In the same study, a 50mg/kg gingerol dose (3.4g for a 150-pound individual) significantly reduced inflammation³. Ginger extract, containing the root's other active constituents in addition to gingerol, is reported to possess antioxidant characteristics by foraging radicals involved in the oxidative processes thought to contribute to the development and progression of a number of disease states⁴. Lastly, tumor-suppressing activity has been established in a number of *in vivo* and *in vitro* studies by up-regulating a tumor suppressing gene, catalyzing abnormal cell death, and inactivating the Vascular Endothelial Growth Factor (VEGF) pathway responsible for the angiogenesis and vascular penetration involved in tumor development and progression^{1,5}.

The safety and toxicity is well established in animal studies using mg/kg levels for extrapolation to human consumption. The following doses in amount, frequency, and duration have reported no adverse effects or effect on morbidity or mortality: 0.5-1.0g 2-3 times per day for 3 months to 2.5 years, single dose of 2.5g/kg body weight, and 500-2000 mg/kg body weight for 35 days^{1,6,7}. Also of importance, a 10-day dose of 100-1000 mg/kg ginger extract in pregnant rats during neonatal organ development showed no maternal or fetal effect⁸.

Recommendation:

I recommend using ginger juice, as opposed to isolating the beneficial constituents of ginger, in at least a 5g quantity. Juice or extract is preferred due to the additive or possible synergistic effect of the several biologically active constituents of ginger. An amount of 5g is recommended based on the current literature's demonstration of effects ranging from 25-100mg ginger extract per kilogram body weight (1.7-6.8g for a 150-pound individual). Given that ginger juice is often diluted in different amounts varying by manufacturer, I recommend using at least 5g ginger juice for highest likelihood of reported benefits.

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For centuries, Chinese and Ayurvedic medicine have supported the use of cinnamon in naturopathic practice. Several health benefits have been researched and documented relating to the spice's phenolic and oil content and their effect on hypoglycemic, lipid-lowering, anti-microbial activity, along with neurodegenerative and gastrointestinal conditions^{1,2}. These proposed benefits may be attributed mostly to cinnamon's main constituent, cinnamaldehyde, found at 65-80% of bark, which is then ground to make cinnamon spice².

Extensive literature review reported most health benefits using cinnamon extract (concentrated by ethanol extraction) or cinnamon oil (concentrated from oil extraction from bark), therefore it may not be appropriate to extrapolate findings suggesting extraordinary effects if using ground cinnamon, as the cinnamaldehyde and phenolic content would undoubtedly be compromised^{1,2}. One human study, however, used ground cinnamon-containing capsules in the amounts of 1g, 3g, and 6g per day and found that after 40-days, the participants in all groups experienced an 18-29% reduction in mean fasting serum glucose, 23-30% reduction in triglycerides, 7-27% reduction in LDL cholesterol and 12-26% reduction in total cholesterol³.

Recommendation:

Since it is unclear to what extent the active constituents are altered from cinnamon extract and oil to ground spice, I recommended using the form used in majority of research that demonstrates the most potential for therapeutic effect. Keep in mind that not only will using extract or oil concentrate cinnamon constituents, it will also concentrate its flavor as well, so tests on palatability would be necessary. If using ground cinnamon, it is difficult to determine whether 1g would be enough to produce any benefits because studies using ground cinnamon is so limited. However, if using oil or extract, amounts as low as 5 - 12.5mg/kg have elicited results⁴ (0.3, 0.8 and for a 150-pound individual, respectively), and given the concentrated flavor, minimal amount necessary may be used to maintain palatability.

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¹ Bandara T, Uluwaduge I, Jansz ER. Bioactivity of Cinnamon with Special Emphasis on Diabetes Mellitus: A Review. *International Journal of Food Sciences and Nutrition*. 2012;63(3):380-386.

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Turmeric, and its bioactive constituent, curcumin, have been extensively studied for their potential antioxidant, antimicrobial, wound-healing, hypoglycemic, and anti-inflammatory activity. Therapeutic use of turmeric extract, which typically contains 95% curcumin, and pure curcumin supplementation have yielded beneficial findings in clinical and laboratory studies relating to the development and management of cardiovascular disease, infections, rheumatoid arthritis, osteoarthritis and a number of other human diseases¹.

It is important to note that the safety and toleration of curcumin has been well-established in humans, and is approved by the United States Food and Drug Administration as being GRAS(generally recognized as safe)¹. A number of clinical studies administering regimens of relatively high doses between 6-12g per day for anywhere between 3 weeks and 6 months, reported minimal, if any, side effects, of which most pertained to the size of the curcumin capsule, which is irrelevant for the purpose of this drink²⁻⁶.

In an extensive review of the literature, curcumin intake has demonstrated clinical efficacy and impact on biomarkers related to the development and progression of disease. Albeit using small populations, peer-reviewed published studies in reputable scientific and medical journals suggest benefits of curcumin in the following conditions and diseases: Cancer of the colon, rectum, pancreas, lung, breast and prostate, multiple myeloma, inflammatory bowel disease, rheumatoid and osteoarthritis, postoperative inflammation, gastric and peptic ulcers, skin conditions vitiligo and psoriasis, atherosclerosis, diabetes, and metabolic syndrome¹.

The average dosage of turmeric extract or pure curcumin used to prompt significant benefits ranges from 0.5-1.5g/day¹⁻⁷. Conventional turmeric spice contains 2-6% curcuminoids, of which 80% is purely curcumin¹. Therefore, to obtain .5-1.5g curcumin, roughly 16-46g of turmeric spice would need to be used. At around 6.5g per tablespoon, the amount needed would equate to 2.5-7 tablespoons, which would undoubtedly compromise the palatability and texture of the drink. Turmeric extract, however, increases the curcuminoid content to about 95%, of which 80% is curcumin. Therefore, to obtain the beneficial curcumin amount of 0.5-1.5g, you would need anywhere between 0.65-2g (roughly 1/3-1 teaspoon) turmeric extract, a much more feasible and appropriate amount for this drink.

Recommendation:

Turmeric extract, as opposed to turmeric powder, in any amount ranging from 1-2g should be used in order to most effectively promote the benefits of curcumin, while maintaining palatability and desired consistency of the drink.

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¹ Gupta SC, Patchva S, Aggarwal BB. Therapeutic Roles of Curcumin: Lessons Learned from Clinical Trials. The American Association of Pharmaceutical Scientists Journal. 2013; 15(1):195-218.

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