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Rationale for a novel nutraceutical complex 'K-water': Potassium taurine bicarbonate (PTB)

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Summary Potassium taurine bicarbonate (PTB), an equimolar blend of potassium bicarbonate and taurine, provides a convenient and feasible means of delivering physiologically significant doses of potassium, taurine, and organic base when dissolved in water ('K-water''). This brief essay reviews the versatile and complementary health benefits that likely would accrue in individuals making regular use of K-water; in particular, an adequate intake of PTB could be expected to aid blood pressure control, lessen risk for atherosclerosis and its thromboembolic complications (particularly stroke), promote maintenance of bone density, help to prevent calcium renal stones, and possibly reduce risk for weight gain and diabetes.

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Potassium taurine bicarbonate

Potassium taurine bicarbonate (PTB) is a mineral complex that provides three dietary compounds that are suboptimally supplied by most modern diets: potassium, taurine, and base (as bicarbonate ion). It consists of an equimolar blend of potassium bicarbonate and taurine. When added to water, these compounds form a soluble ionic complex which has a slightly lower pH than that of potassium bicarbonate alone, and that is less prone than potassium bicarbonate to evolve carbon dioxide when small amounts of acid are added to the solution. (J. Zielinski, personal communication). Most likely, this reflects formation of an ionic complex in which potassium is attracted to the negatively charged sulfonic acid group of the taurine zwitterion, the bicarbonate ion is attracted to the positively charged amine group of taurine, and potassium and bicarbonate are attracted to each other. When incorporated in this complex, bicarbonate is less prone to extract protons from solution, thus explaining the pH characteristics of this complex.

When added to a liter of water, 60 mmol of PTB (13.5 g) provides 2.35 g potassium and 7.5 g taurine. The first sip of the resulting solution can have a mildly acrid flavor, but this flavor rapidly down-regulates such that there is very little detectable flavor in subsequent sips: in other words, it tastes like ordinary water. Thus, an aqueous solution of PTB – designated "K-water" – represents a convenient, feasible means of administering PTB. K-water can be blended with neutral or mildly alkaline ingredients to produce soft drinks, teas, and modified juices. Acidic additives generate free

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potassium ion, ruining the flavor; thus, K-water cannot be used to make coffee.

Potassium, taurine, and organic base are each distinguished by the fact that daily multi-gram doses are required to provide optimal physiological benefit. Furthermore, consumption of potassium in a concentrated form — as in tablets or capsules — risks injury to the gastrointestinal mucosa. For these reasons, K-water represents a practical, convenient means of achieving physiologically significant intakes of each of the constituents of PTB.

Although clinical research with PTB remains to be accomplished, the health benefits conferred by this complex can likely be inferred from the known properties of its individual constituents:

Potassium

Owing to the promiscuous use of refined grains, sugars, oils, and fatty animal products in modern diets, as well as the relative paucity of fruits and vegetables in many of these diets, potassium intakes today are far lower than they were in Paleolithic hunter-gatherers who consumed no refined foods but had exceptionally high intakes of fruits and vegetables [1]. There is considerable evidence that increased intakes of potassium can lower elevated blood pressure to a worthwhile extent, especially in the context of high salt diets [2]. This effect has been demonstrated in numerous controlled studies, and validated in metaanalyses [3,4]; most of these studies assessed supplemental potassium intakes of 60 mmol daily or higher. Furthermore, epidemiology suggests that, independent of any impact on blood pressure, higher potassium intakes decrease risk for myocardial infarction and, more substantially, stroke [5– 11]. A portion of this protection reflects the fact that potassium has a natriuretic effect, thus offsetting some of the adverse health effects of salty diets [12-15]. In addition, the modest increase in serum potassium associated with potassium-rich diets acts to increase the membrane potential of vascular endothelium - thereby boosting activity of nitric oxide synthase while inhibiting that of the superoxide-generating NADPH oxidase [16-18].

Salt promotes loss of bone mineral by inducing renal calcium loss (calciuria); the natriuretic effect of potassium tends to counteract this effect, thus helping to preserve bone density [19–24]. Hence, improved potassium nutrition can be expected to benefit the healthful structure and function of the vascular system as well as of bone. And it is not unlikely that potassium, owing to its natriuretic activity, has a favorable impact on other pathologies linked to high-salt diets, such as cardiac hypertrophy and asthma [25].

Bicarbonate

Owing to a high intake of proteins rich in sulfhydryl amino acids - which generate sulfuric acid when catabolized in vivo - modern diets tend to generate metabolic acidosis, which is compensated in part by dissolution of bone mineral; bone phosphate acts as a buffer [1,26-28]. This problem is exacerbated by the fact that many diets are relatively low in organic anions - associated primarily with potassium, calcium, and magnesium in natural foods, especially fruits and vegetables - that are can be metabolized in vivo to yield bicarbonate ion, an effective buffer for metabolic acidosis that spares bone mineral [1]. In cross-sectional studies, high consumption of fruits and vegetables - rich in organic potassium salts - has been linked to increased bone density in men and women [29–31]. Researchers have repeatedly demonstrated that ample supplemental intakes (usually 60 mmol daily or more) of potassium bicarbonate or potassium citrate can substantially slow bone turnover and renal calcium loss in postmenopausal women, in all likelihood postponing the onset of osteoporosis and fracture [21,32-35]. The bicarbonate component of PTB is intended to provide this important benefit. The ability of potassium bicarbonate to decrease calciuria – which in part reflects the natriuretic action of the potassium - should have the added benefit of decreasing risk for calcium-based renal stones [33,36].

A further potential drawback of the mild metabolic acidosis associated with protein-rich modern diets is that it promotes increased cortisol production - a ''side effect'' of an up-regulation of adrenocortical activity that functions to boost the efficiency of renal acid secretion by stimulating renal production of ammonia [37-40]. Increased cortisol production would be expected to increase risk for visceral obesity and insulin resistance syndrome. Perhaps this explains why increased dietary potassium – typically associated with organic anions in foods - has been linked to decreased risk for diabetes in prospective epidemiology [41,42]. Furthermore, the modest improvement in nitrogen balance observed during supplementation with potassium bicarbonate [43] possibly reflects decreased cortisol production, as well as a decrease in renal ammonia production. The alkalinizing impact of PTB can be expected to have a down-regulatory impact on cortisol secretion in those eating acid-forming diets [44].

Taurine

This neglected nutrient appears to have extraordinarily versatile potential for promoting vascular health when administered in multi-gram daily doses. Clinical studies have evaluated taurine supplementation in doses up to 6 g daily, and no hint of adverse effects has emerged in these studies, presumably because excess taurine is rapidly cleared by the kidneys. Moreover, the body pool of taurine is very high, inasmuch as muscle, the heart, neurons, phagocytes and various other tissues contain taurine in millimolar concentrations. Although taurine contains a sulfonic acid group, it is not metabolized to yield sulfonic acid, and thus does not promote calciuria [45]. The potential vascular benefits of high-dose taurine include: a platelet-stabilizing effect that is complementary to that of aspirin [46–48]; an anti-hypertensive effect that, at least in part, appears to reflect a moderate down-regulation of elevated sympathetic activity [49–56]; an anti-inflammatory anti-atherosclerotic effect (independent of modulation of serum lipids) demonstrated in rodent models of atherogenesis [57–59]; a positive inotropic effect in patients with congestive heart failure that does not appear to entail the increased risk for arrhythmia associated with digitalis therapy [60-62]; and a possible beneficial impact on the symptoms of cardiac angina and intermittent claudication, as suggested by Italian clinical reports published in the 1960s [63].

The anti-atherogenic impact of taurine in rodents possibly reflects taurine's ability to detoxify hypochlorous acid [64], a potent oxidant that is the chief product of myeloperoxidase, an enzyme active in intimal macrophages. A high-expression polymorphism of myeloperoxidase has been linked to increased risk for coronary atherosclerosis in recent epidemiology [65], and hypochlorite-modified proteins are prominent in human atheromatous lesions [66,67]. Hypochlorous acid is capable of oxidizing LDL particles to high-uptake forms [68-70]; the fact that taurine is protective in certain rodent models of atherogenesis that fail to respond to vitamin E [71–73], suggests that hypochlorous acid may play a more important role than hydroxyl radical in promoting pathogenic modifications of LDL in vivo. (This in turn might explain why supplemental vitamin E has failed to confer cardiovascular protection in prospective supplementation trials) [74,75]. It has also been suggested that taurine's ability to quench hypochlorous acid might promote plaque stabilization, since hypochlorous acid functions to activate metalloproteases [76,77]. Furthermore, the product of taurine's interaction with hypochlorous acid, taurochloramine, can suppress activation of NF-kappaB [78,79] – an anti-inflammatory effect which would antagonize atherogenesis. The possible utility of taurine in other inflammatory disorders merits further investigation.

In diabetic rodents, taurine-enriched diets have been shown to ameliorate neuropathy and nephropathy [80-82]. There is a small amount of suggestive evidence that taurine may have a favorable impact on bone heath: it impedes the multiplication of osteoclasts in vitro, and, when administered orally to hamsters, is reported to slow loss of periodontal bone in a model of periodontal bone loss [83]. Taurine may also have neuroprotective potential, helping neurons to survive the excitotoxicity that accompanies ischemia and plays a pathogenic role in many neurodegenerative disorders. Under conditions that give rise to excitotoxicity, taurine is released from neurons into the extracellular space, where it can act as an agonist for GABA(A) receptors that promote neuron hyperpolarization by boosting chloride conductance; this in turn tends to protect neurons by moderating the excessive calcium influx that characterizes excitotoxicity [84-89]. Since supplemental taurine can increase brain taurine levels [90], it can be expected to potentiate this protective feedback mechanism. Supplemental taurine has in fact shown brain-protective activity in some [91,92] but not all [93] rodent models of excitotoxicity.

In light of the fact that high intakes of taurine may be required to optimize its health benefits, a solution of PTB represents a very practical means of achieving this high intake. Supplemental taurine may be of particular value to vegetarians, whose diets are virtually devoid of this nutrient; suboptimal taurine status may account for the platelet hyperaggregability that has been observed in vegetarians [94].

Clinical prospects

Drinking up to a liter of K-water daily, providing 60 mmol of PTB, can thus be expected to help preserve vascular health by a range of complementary mechanisms, help maintain bone density, prevent renal stones, and possibly reduce risk for weight gain and insulin resistance. Its practicality is enhanced by the fact that both potassium bicarbonate and taurine are quite inexpensive. It is contraindicated in patients experiencing renal failure, taking potassium sparing diuretics, or with other medical conditions in which increased potassium intakes would be considered inappropriate. Clinical studies examining the impact of K-water consumption in various clinical conditions — most notably essential hypertension — appear to be warranted.

Disclosure

The author owns stock in the company – Nutrition Company of America – to which the patent rights have been assigned.

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