COMMENTARY

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Pyruvate: Beyond the Marketing Hype

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Pyruvate is the latest ergogenic supplement fad targeting athletic as well as sedentary populations. The most popular claims for pyruvate, currently being promoted by multilevel marketing (MLM) distributors, health food stores, and bodybuilding magazines, revolve around its purported ability to improve exercise endurance capacity (29, 30), augment weight and fat loss (23, 31, 32), serve as an antioxidant (4, 10), and lower plasma lipids (27, 28).

Pyruvate is a 3-carbon ketoacid produced from phosphoenolpyruvate in the end stages of glycolysis (1). Depending upon metabolic conditions, there are two major fates for pyruvate: It can be reduced to lactate by lactate dehydrogenase in the cytosol, or it can be oxidatively decarboxylated to acetyl CoA by the pyruvate dehydrogenase complex in the mitochondrion (34). Pyruvate and dihydroxyacetone will be referred to as trioses for the purpose of this review.

Research cited by marketers suggests that supplemental pyruvate may exhibit ergogenic properties; however, such claims should be considered, at best, preliminary. Promotional materials appear to highlight only positive findings of studies but consistently neglect to mention the limitations of the results. While the lay athlete may be impressed by the possibility of a true dietary panacea, a comprehensive critique of these claims against the original research from which they are derived is warranted.

Pyruvate As an Ergogenic Aid

Popular marketing literature maintains that pyruvate can enhance endurance performance by prolonging time to exhaustion, but this is only supported by two studies, both of which employed small numbers of untrained males. Stanko et al. (30) found that triose-supplemented (75 g dihydroxyacetone and 25 g pyruvate over 7 days) subjects ($N = 10$) exercising at 60% $\dot{V}O_2$peak during arm ergometry increased time to exhaustion by 20% over the placebo group. This difference may have been due to larger preexercise muscle glycogen stores and greater arteriovenous glucose extraction. It is well-established that increased muscle glycogen stores can enhance performance by providing additional substrate to working muscles (12, 35), but the specific mechanisms responsible for these augmentations after triose supplementation are not clearly understood.

Stanko et al. (29) reported a 20% increase in time to exhaustion during leg ergometry in triose-supplemented (75 g dihydroxyacetone and 25 g pyruvate) subjects ($N = 8$) exercising at 70% $\dot{V}O_2$peak after consuming a high-carbohydrate diet

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for 7 days. There was no difference in preexercise muscle glycogen concentration, so it appears that a high-carbohydrate diet negates the effect of improved glycogen storage previously seen with lower dietary carbohydrate content and triose feedings (30). It is unlikely that this improvement in endurance capacity was related to augmented substrate availability and should be eliminated as an ergogenic source. Stanko et al. (29) observed enhanced muscle glucose extraction and hypothesized that supplemental triose may not increase the rate of glucose oxidation during exercise but may instead prolong its oxidation, subsequently improving endurance time.

These studies appear valid, since appropriate controls (randomized, crossover, placebo-controlled, double-blind) were employed (8). However, for a number of reasons, it is imperative that these results not be arbitrarily ascribed to the general population.

First, these studies have not been reproduced by other researchers. Responsible science holds that no hypothesis shall be deemed true until there exists a substantive body of supporting evidence. If a hypothesis is demonstrated to be true, it is still considered tentative and subject to scrutiny by other researchers who may eventually prove it false (15).

Second, it is imperative to consider the experimental dosages of dihydroxyacetone and pyruvate employed in these studies, for this is the basis upon which supplemental pyruvate must be evaluated. Statistically significant differences in endurance time were observed when subjects consumed 100 g of triose, 25 g of which was pyruvate. This is in direct contrast to commercial pyruvate preparations, which can range from 500 mg to 1 g per capsule and may or may not contain dihydroxyacetone. Presently, there are no studies demonstrating the efficacy of pyruvate in such tiny doses (no dose–response relationship has been established for pyruvate). In addition, the Dietary Supplement and Health Education Act of 1994 allows products to be marketed without proof of safety, efficacy, or potency, so there is no guarantee the dosage will be consistent each time.

Third, it has been widely promoted that supplemental pyruvate induces no side effects. Yet in both studies on exercise performance (29, 30), subjects did, in fact, experience gastrointestinal distress in the form of borborygmus, flatus, and diarrhea. It is likely that commercial pyruvate supplements do not induce similar changes, although this is likely due to their benign dosage rather than to manufacturers' formulations.

Finally, results from endurance studies are not applicable to all exercise modalities insofar as different activities exhibit different metabolic demands as well as different motor unit recruitment patterns. To illustrate, short-distance, high-intensity sprinting (i.e., a 40-yard dash) would activate the phosphagen energy pathway (33), thereby circumventing the need for blood glucose and liver and muscle glycogen as substrates. Gymnastics, unlike steady-state cycling, can be explosive or isometric in nature as evidenced by various competitions in this sport (parallel bars, springboard, floor routine, pommel horse, rings, or balance beam). Independent of metabolic demands, the motor unit recruitment patterns of gymnastics differ markedly from arm and leg ergometry protocols. Thus, the principle of sport specificity, which holds that specific demands induce specific adaptations, does not allow for direct comparisons between endurance and strength and power activities.

These endurance studies (29, 30) suggest the efficacy of pyruvate in a laboratory setting in a small number of untrained subjects. Currently, there are no published data on the effects of pyruvate in elite or trained populations over a broad

spectrum of activities. Studies using well-trained athletes will usually present less intra- and intersubject variability in performance, thus increasing the statistical power of the trial (7). The implication that pyruvate can improve performance in trained athletes remains in question and, therefore, must await corroboration by future studies.

Effect of Pyruvate on Weight and Fat Loss

Body weight and fatness are of key importance to athletes and sedentary individuals alike and are functions of one's genetics, diet, and training habits (6). Depending on the sport, excessive body fat can impair performance by hindering range of motion or by compromising an athlete's strength-to-mass ratio. The strength-to-mass ratio directly reflects an athlete's ability to accelerate his or her body (14), so for strength and power athletes, loss of body fat with concomitant increases in strength is very desirable. Given the important relationship between body fat and athletic performance, the potential role of pyruvate as a fat reductant may be enticing to athletes.

Original investigations into pyruvate (13, 20, 26) found that it helped mitigate the deleterious effects of ethanol-induced hepatic steatosis. These results led to subsequent rat (9, 21, 22, 25), swine (24), and human (23, 31, 32) studies that sought to elucidate the effects of trioses on lipid synthesis and weight and fat gain.

Rat (9, 21, 22, 25) and swine (24) studies demonstrated that partial substitution of hexoses with trioses (pyruvate and dihydroxyacetone) attenuates weight and fat gain without the attendant loss of lean body mass. It has been posited that increases in resting energy expenditure account, in part, for these changes (9). Stanko et al. (22) noted higher energy expenditure with a commensurate increase in CO_2 production in treated rats. In addition, depressed lipid synthesis occurred with lower blood insulin levels.

As described elsewhere (16, 19), there is speculation of a pyruvate-phosphoenolpyruvate (PEP) futile cycle in glycolysis in which heat is given off during the phosphorylation and dephosphorylation of pyruvate to PEP and back again. Some authors (9, 22) have suggested that this futile cycle can be augmented by addition of trioses to the diet, which may partly account for improved diet-induced thermogenesis.

With respect to previously mentioned animal studies (9, 21, 22, 24, 25), the data appear valid. However, animal metabolism differs from that of humans and, for this reason, must only serve as a preliminary foundation for future studies in humans. That is, animal studies can serve as models for purported biochemical and physiological mechanisms, yet it is imperative that results are not inappropriately extrapolated to humans (2).

Studies conducted on morbidly obese women housed in a metabolic ward consuming restricted diets for 21 days found statistically significant differences in body weight and fat loss (31, 32) when subjects were fed 13–20% of energy from trioses. However, the actual differences were small. Thirteen women consuming a 500-kcal liquid diet (60% carbohydrate, 40% protein, <1 g fat) supplemented with 28 g dihydroxyacetone and pyruvate lost 0.9 kg (16%) more body weight and 0.8 kg (23%) more fat than the placebo group (32). Fourteen women consuming a 1,000-kcal liquid diet (68% carbohydrate, 22% protein, 10% fat) supplemented with 30 g pyruvate lost 1.6 kg (37%) more body weight and 1.3 kg (48%) more fat than the placebo group (31).

The effects of trioses on weight maintenance have been evaluated. A recent investigation employing 17 obese women found that subjects gained significantly less body weight and fat when fed 20% of energy (15 g pyruvate and 75 g dihydroxyacetone) from trioses (23); however, these changes were also small. Initial weight loss was induced by 21 days of hypoenergetic feeding (310 kcal/day as 60% carbohydrate, 40% protein, <1 g fat) followed by another 21 days of hyperenergetic refeeding (150% resting energy expenditure [55% CHO, 15% protein, 30% fat]). The treatment group gained 1.8 kg (36%) less weight and 1 kg (55%) less fat than the placebo group ($p < .05$).

While these studies offer evidence that pyruvate can influence weight and fat loss in morbidly obese women, the experimental protocols, sample populations, and dosages employed in these studies do not accurately reflect a true-life situation (2, 11), and, therefore, the practical utility of these data may be in question.

First, the experimental conditions and subject pool alone are so exclusive that the results cannot be accurately ascribed to the population at large. All subjects were morbidly obese women confined to bed while housed in a metabolic ward. Energy intake was very low, ranging from 310 to 1,000 kcal/day. Conversely, the average person is not confined to bed and may participate in regular physical activity. Energy intake for Americans is 1,742 kcal/day for women and 2,592 kcal/day for men (5), clearly larger than the energy-restricted diets employed in the studies. Considering the inapplicability of these results to the average population, they cannot be reliably conferred to the athletic population. To date, no studies have been performed on the effect of pyruvate on weight and fat loss in the active population.

Second, all studies examining the effects of trioses on weight and fat gain (31, 32) and regain (23) in humans employed dosages considerably larger (28–90 g/day) than those found in commercial supplements. Most commercial preparations provide a range of approximately 500 mg to 1 g pyruvate per capsule. There are no studies demonstrating the efficacy of pyruvate in such minuscule doses.

Finally, according to a National Institutes of Health Technology Assessment Conference Statement (3), bioelectrical impedance (BIA) is limited in its ability to assess adiposity in obese individuals. Furthermore, it is not useful in measuring short-term changes in body composition among individuals. Other literature (18) maintains that BIA has an error margin of 2–4% in predicting total body water and fat-free mass. Stanko and colleagues noted that BIA may not have been sensitive enough to detect the small changes (~1–2 kg) in body composition observed in their subjects (23, 31, 32). The authors acknowledged that their results must be considered preliminary and await corroboration of long-term, large-scale clinical evaluations. As of this writing, no long-term, large-scale trials have been performed. Unfortunately, this is always omitted from promotional literature and is therefore not available to consumers.

Pyruvate As an Antioxidant

Pyruvate has been widely promoted as a powerful antioxidant, yet there are no published studies examining the effect of oral pyruvate supplementation. Two studies (4, 10) illustrated the potential of pyruvate as an in-vitro antioxidant under tightly controlled experimental conditions. Tissue culture (4) and isolated postischemic heart model studies (10) demonstrated less free-radical production after perfusion with pyruvate. The effects of pyruvate may differ in the whole animal, since in the

intact animal immune responses and catecholamines provide additional sources of free radicals not found under experimental conditions (10). This is not to discount the relevance of these clinical findings; rather, the practical application of pyruvate as an oral antioxidant remains to be established.

Pyruvate As a Cholesterol-Reducing Agent

Pyruvate's role as a cholesterol reductant is supported by meager evidence. Two 6-week studies were performed on obese, hyperlipidemic subjects consuming high-fat, high-cholesterol (28) or low-fat, low-cholesterol (27) diets. Decreased plasma cholesterol (4%) and low-density lipoprotein cholesterol (5%) levels were seen in pyruvate-treated (36–53 g/day) subjects consuming a high-fat diet as compared to the control group (28). No changes in plasma lipids were observed in pyruvate-supplemented (22–44 g/day) subjects consuming a low-fat, low-cholesterol diet (22–24 kcal/kg [60% carbohydrate, 16% protein, 24% fat]); however, the treatment group lost 0.6 kg more weight and 0.4 kg more body fat than the placebo group ($p < .05$) over the 6-week period (27). Subjects maintained their usual lifestyle habits but refrained from exhaustive exercise. Although statistically significant, the actual quantity of weight and fat loss was small.

Clearly, popular claims touting the cholesterol-reducing capabilities of pyruvate are not based on a solid foundation of research. A single study alone cannot possibly encompass the broad spectrum of variables that might affect plasma lipids. These investigations (27, 28) apply to obese, hyperlipidemic individuals consuming high-fat (28) or low-fat, low-cholesterol (27) diets for 6 weeks and do not represent what may occur in normal-weight, normolipidemic people.

Marketers' assertions that pyruvate is an attractive supplement for the typical American eating a high-fat, high-cholesterol diet are incongruent with today's public health messages, which recommend eating less fat and cholesterol. Pyruvate is not a cure for hyperlipidemia, nor is it an excuse to eat a high-fat, high-cholesterol diet.

False and Misleading Claims

The lure of selling a product seems to have spawned a new wave of false or misleading claims supported by nothing more than its promoters' wishful thinking or faulty extrapolations from legitimate research. The following can be found on internet websites, magazine advertisements, audio cassettes, and related promotional literature:

- *Pyruvate has been clinically proven as a more potent "fat-burner" than hydroxycitric acid (HCA) and chromium picolinate combined.*

This claim is false since no studies have ever compared pyruvate to HCA and chromium picolinate. Furthermore, there is no standard method to quantify the "potency" of a "fat-burner." As such, there is no basis in fact for this claim, yet popular marketing literature maintains that it has been clinically proven.

- *Pyruvate can improve cardiac function.*

This claim is ambiguous since the word *improve* is not clearly defined. However, one study (28) found that heart rate, diastolic blood pressure, and rate-pressure product decreased in the pyruvate-treated group. The protocol did not control for

physical activity, so there is no way to determine if these changes occurred as a result of the treatment (36–53 g/day for 6 weeks). Furthermore, these observed changes were not part of the research objectives and were merely incidental to changes in plasma lipids. Thus, these changes should not be considered conclusive but should prompt future studies.

In addition, the diet was not representative of the typical American diet; fat (45–47% of energy) and cholesterol (560–620 mg/day) intakes were much higher than in the general population. Also, the amount of pyruvate (36–53 g/day) used in this study (28) was considerably larger that the amounts recommended to consumers.

- *Pyruvate is backed by 27 years of clinical research on humans.*

A literature search of the Medline database shows that the earliest published research on pyruvate dates to 1978, not 1970 as advertisements would suggest. Furthermore, the 1978 study used animal subjects to investigate the role of pyruvate in preventing alcohol-induced hepatic steatosis (26).

- *Pyruvate is a "natural alternative" to Phentermine/Fenfluramine (Phen/Fen).*

In light of Phen/Fen's association with heart valve damage and its subsequent removal from the market, promoters have placed special emphasis on this claim. Nevertheless, pyruvate has never been clinically tested or proven to be more effective than Phen/Fen or any other diet drug. A reliable comparison cannot be made since one is a regulated drug with known pharmacological activity and the other is an unregulated supplement. The Dietary Supplement and Health Education Act of 1994 allows supplements to be marketed without proven efficacy, safety, or purity.

Studies on weight loss (31, 32) found significant, albeit small, changes in body weight (~1–2 kg) when subjects consumed energy-restricted diets (500–1,000 kcal/day) supplemented with 28–30 g/day of trioses. Only one investigation evaluated pyruvate (90 g/day) as an aid for keeping off lost weight after the termination of a 310-kcal/day diet (23). The treatment group gained significantly less weight (1.8 kg) than the placebo group after 3 weeks of hyperenergetic refeeding (150% resting energy expenditure). These results are not directly comparable to Phen/Fen users since most do not live in a metabolic ward, severely restrict energy intake, or avoid all physical activity. Furthermore, weight loss induced by Phen/Fen is much larger than that observed with triose supplementation.

- *Pyruvate is found naturally in the body and in a variety of foods.*

Pyruvate is a normal constituent of human and plant metabolism; however, the term *natural* is ambiguous and therefore confers a deceptive stamp of approval to the unsuspecting consumer. This tactic is grounded on the myth that if something is natural it must be safe and effective. Unfortunately, natural does not mean safe or effective: E. coli naturally occurs as part of the human intestinal flora but can be toxic if ingested.

- *Pyruvate has been clinically proven safe.*

Consumption of trioses up to 100 g/day for 1–6 weeks appears safe. However, some human subjects receiving such large doses of pyruvate and dihydroxyacetone did experience gastrointestinal distress. As previously discussed, the benign doses found

in commercially available supplements are probably not large enough to induce similar side effects, let alone a remarkable physiological effect.

Pyruvate Marketing Strategies

Nearly every possible media outlet has been accessed by pyruvate marketers, many of whom have no formal education in the nutritional sciences. As a result, this has created an environment very conducive to the promotion of nutrition misinformation.

Multilevel marketing (MLM) distributors are leading the massive marketing onslaught of pyruvate. Perhaps to absolve themselves of legal responsibility for making false claims, MLM firms appear reserved about what they print in their official promotional literature. But distributors are much more candid, armed with internet websites, electronic bulletin boards and newsgroups, audio cassettes, and photocopies of fitness magazine articles. The common denominator is an appeal to the emotions, not logic (17), of athletes looking for the elusive performance-enhancing agent or obese people looking to lose weight.

This appeal to emotion is evidenced by the widespread use of anecdotal testimonials. Because personal experiences do not separate cause and effect from coincidence, they cannot be considered scientifically valid. To illustrate, if a formerly sedentary individual begins taking pyruvate in conjunction with a healthful diet and exercise regimen, changes in body weight may actually reflect an energy deficit induced by the lifestyle change rather than the supplement. Unfortunately, many misguided users are also pyruvate distributors and consider this to be "scientific testing" of their product line.

Nearly all promotional material for pyruvate boldly states that its efficacy is supported by "clinical research," but this appears to be a gross distortion of the truth. While research has been conducted on pyruvate, these studies do not accurately reflect the situations for which pyruvate is recommended. The term *statistically significant* has no legitimate meaning outside the context of a research article. Thus, advertisements asserting that dieters can "significantly" improve exercise performance or weight loss are ambiguous and misleading—lose 48% more fat compared to what? To the scientifically uninitiated, the mere mention of clinical studies, irrespective of their content, validity, or practical relevance, is considered proof and overrides individuals' natural skepticism, thereby allowing them to fall prey to this fad.

Conclusion

Currently there is no substantive body of relevant research to support the purported role of pyruvate as an antioxidant, ergogenic aid for sport performance, and body weight, fat, and cholesterol reductant. It is possible that small doses of pyruvate may work in these areas. But until pertinent research is conducted on trained, normal-weight, normolipidemic individuals, promoters' claims for this supplement must be considered speculative.

While there is no immediate threat to public health, pyruvate appears to be an economic fraud. According to Young (36), education is the most effective way to warn and protect the public from economic frauds. With this fad clearly in its infancy, responsible health professionals should discourage pyruvate supplementation on the basis of inconclusive evidence.

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