A high fructose–fed rat model of insulin-resistance syndrome has a metabolic profile very similar to that of the human X syndrome, which is associated with a high incidence of cardiovascular disease and hypertension. Insulin resistance, hyperglycemia, increased vascular resistance, sodium retention, and sympathetic overactivity have been linked to the development of hypertension. Taurine (2-aminoethanesulfonic acid) is the most abundant, nonessential, sulfur-containing amino acid found at high concentrations in skeletal muscle, heart, blood, nerve, brain, liver, and other organs. Many biological, physiological, and pharmacological functions of taurine have been reported in various tissues and species, including detoxification, osmoregulation, antioxidation, membrane stabilization, modulation of ion flux, and cardiovascular functions such as the regulation of hypertension. A previous study showed that taurine decreases the activation of mitogen-activated protein kinase and Bax, which prevents the generation of reactive oxygen species induced by lipopolysaccharides. Another study showed that taurine protects against DNA damage by oxidative injury by inhibiting quinone formation. Oral taurine supplementation decreases blood pressure without significant side effects as previously reported elsewhere. Exercise is frequently recommended as a useful way to lower blood pressure for the management of hypertension and increases exercise capacity by possibly antioxidation and maintaining NO concentrations.
and appropriate supplementation with antihypertensive agents is recommended for correction. To explore the potential of taurine as such a supplement, the present study used a high fructose–fed rat model to investigate the influence of oral tau-
rine supplementation on insulin resistance, development of hypertension, and decreased exercise capacity.

METHODS
Animal preparation. Male Sprague–Dawley rats (6 weeks of age and 120–140 g in weight) were purchased from the Korean Research Institute of Bioscience and Biotechnology (Daejeon, Korea). The rats were housed in a room with a temperature of 23 ± 5°C and alternating 12-h dark-light cycles. The animals were fed standard rat chow (20% crude protein, 4.5% crude fat, 6.0% crude fiber, 7% crude ash, 0.5% calcium, and 1% phosphorus) and were allowed free access to tap water during the adaptation period.

Experimental protocol. The rats were randomly allocated to five groups, each containing 15 rats. One group served as control. The remaining four groups were fed 35% fructose (Sigma-Aldrich St Louis, MO) in the chow and 5% fructose in drinking water, which collectively established an average 43.6 ± 0.1% fructose of the total food consumed. The daily intakes of the rats are presented in Table 1. The four high fructose–fed groups were designated as follows: high fructose–fed sedentary (fructose), high fructose–fed plus exercise (FE), high fructose–fed plus taurine supplementation (FT), and high fructose–fed plus exercise and taurine supplementation (FET). The FT and FET groups received 2% taurine dissolved in the drinking water. After 2 weeks, five representative rats from each group were selected and tagged for swimming capacity and fatigue tests. The investigation conformed to the Guide for the Care and Use of Laboratory Animals (NIH Publication No. 85–23, revised 1996).

Swimming. An adjustable current swimming pool (100 cm in length, 60 cm in width, and 70 cm in height, and 35-cm water depth) was used for the swimming test. The current in the pool was generated by four horizontally seated type BT-30F water pumps (Chuangxing Electrical, Chuangxing, China). The FE and FET groups were subjected to 1 h of swimming daily between 9:00 and 11:00 am 6 days per week, with 1 day off per week for rest. The strength of the current was adjusted by changing the water flow, and each pump was adjusted to a flow rate of 5.2 ± 0.6 l/min for swimming.

Swimming capacity and fatigue tests. After 4 weeks of regular exercise sessions, five rats from each group were selected for the swimming capacity test. The control and fructose groups had been trained every second day, 10 min per session, in the last 2 weeks before completion. Three fatigue tests were performed every other day as previously described.

Blood pressure measurement. SBP was measured noninvasively each week with a Rat Tail NIBP System manometer-tachometer (ADI Instruments, Heidelberg, Germany) using a B60-7/16” size tail-cuff (IITC Life Science, Woodland Hills, CA.) during the experimental period. Invasive arterial blood pressure (ABP) was measured at the end of the trial in rats anesthetized with ethyl carbamate (1.8 g/kg body weight, intraperitoneally). A catheter with an outer dimension of 0.96 mm and an inner dimension of 0.58 mm (Fisher Scientific, Pittsburgh, PA) was inserted into the carotid artery, and the ABP was recorded with a computerized transducer system (MP150; Biopack, Santa Barbara, CA) using Acknowledge 3.5 software (East Palo Alto, CA).

Measurement of blood electrolytes and plasma glucose, and insulin. Blood electrolytes were measured immediately in a fresh blood sample (300 µl) with the use of a Nova stat profile M ion selective electrode (Nova Biomedical, Waltham, MA). Plasma glucose was measured with a Spotchem SP-4410 auto-
dry blood chemistry analyzer (Polymedco, Cortland Manor, NY) using a glucose measuring strip (Arkray, Kyoto, Japan). Plasma insulin was measured with a radioimmunooassay method (IVD Technologies, Santa Ana, CA) according to the manufacturer’s protocol.

Antioxidant component system measurement. Plasma malondi-
aldehyde (MDA) concentrations were measured with an MDA

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Body weight, food intake, fluid intake, fructose consumption, and taurine consumption during 4 weeks experiment period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>Initial body weight</td>
<td>129.48 ± 0.75</td>
</tr>
<tr>
<td>Final body weight</td>
<td>284.33 ± 1.44</td>
</tr>
<tr>
<td>Food intake (g/kg·b.w./day)</td>
<td>80.66 ± 2.58</td>
</tr>
<tr>
<td>Water intake (g/kg·b.w./day)</td>
<td>134.45 ± 2.51</td>
</tr>
<tr>
<td>Fructose intake (g/kg·b.w./day)</td>
<td>—</td>
</tr>
<tr>
<td>Fructose consumption %</td>
<td>—</td>
</tr>
<tr>
<td>Taurine intake (g/kg·b.w./day)</td>
<td>—</td>
</tr>
</tbody>
</table>

Body weight was measured every week; food and fluid intake are calculated from the weekly group total consumption. Fructose and taurine intake are calculated according to the supplementation used in food and water. Data are mean ± s.e.m. b.w., body weight; F, fructose; h.F, high fructose–fed group; FE, high fructose–fed with exercise group; FT, high fructose–fed with exercise and taurine supplement group; FET, high fructose–fed with taurine supplement group.

*P < 0.001 vs. control, **P < 0.01 vs. fructose, n = 10 each group.
assay kit (Biomol International, Plymouth Meeting, PA) at an absorbance of 586 nm according to the manufacturer’s protocol. Thiol concentrations were measured based on the reactivity of thiol groups with Ellman reagent (5,5′-dithiobis[2-nitrobenzoic acid]), which led to a colorimetric reaction modified as described previously. Reduced glutathione (GSH) and blood GSH concentrations were measured as previously described. Oxidized glutathione (GSSG) was measured using the previously described method. The samples were deproteinized with 20% trichloroacetic acid. For GSSG measurement, 50-μl acidic supernatant aliquots were treated with 2 μl 2-vinylpyridine and mixed continuously for 60 min for masking of GSH (2 μl added to 50 μl supernatant). During the preincubation period, the pH was adjusted to about 6 with triethylamine. Each sample was analyzed in duplicate, and the average value was used. The redox ratio (GSH/GSSG) was calculated by using the formula GSH/GSSG = (GSHt − 2GSSG)/GSSG, as described previously.

Plasma nitrite/nitrate measurement. Nitrite/nitrate, the major metabolite of nitric oxide (NO), was measured in plasma using a Nitrite/Nitrate Colorimetric Assay Kit (Fluka Analytical, Seelze, Germany). Nitrite accumulation in the plasma was determined by first reducing the nitrate using nitrate reductase. Nitrite was assayed colorimetrically after reaction with the Griess reagent (Sigma-Aldrich) according to the manufacturer’s protocol. The absorbance was determined using a SpectraMax M2 microplate reader (Molecular Devices, Sunnyvale, CA) at 540 nm. All measurements were performed in duplicate.

Data presentation and analysis of results. The data were analyzed by using GraphPad Prism program version 5.02 (GraphPad Software, La Jolla, CA), and the data are expressed as mean ± s.e.m. Differences were compared between the groups by using a Bonferroni post hoc test followed by a non-repeated one-way analysis of variance vs. the control or fructose group. In order to compare differences depending on time and treatment, a nonrepeated two-way analysis of variance was performed following a Bonferroni post hoc test for SBP data measured by tail cuff over 4 weeks. A P value <0.05 was considered to indicate a statistically significant difference.

RESULTS
Electrolyte status in the blood
Results of electrolyte measurements and acid-base status (pH) in the blood are shown in Table 2. Sodium ion concentration was significantly greater (P < 0.001) in the fructose and FE groups than in the control group. Na+ in the FT and FET groups was not significantly different from that in the control and fructose groups. The concentration of Cl− was significantly lower (P < 0.001) in the fructose group than in the control group and was significantly greater (P < 0.001) in the FE, FT, and FET groups than in the fructose group; hematocrit levels in the FT and FET groups were not significantly different from the level in the control group.

Effect of taurine and exercise on noninvasive SBP and invasive systolic ABP
The initial and final noninvasive SBP values in the control group were 114.5 ± 0.9 and 116.3 ± 1.4 mm Hg, respectively. A gradually increasing SBP was recorded in the fructose, FE, and FT groups. The initial and final SBP values were 115.1 ± 1.5 and 129.2 ± 0.9 mm Hg, respectively, for the fructose group; 114.3 ± 1.2 and 125.4 ± 1.5 mm Hg, respectively, for the FE group; and 114.5 ± 1.4 and 122.8 ± 1.2 mm Hg, respectively, for the FT group (Figure 1). After 4 weeks, SBP increased significantly (P < 0.001) in the fructose group, considering both time and treatment. The SBP was 118.2 ± 1.1 mm Hg in the FET group after 4 weeks, with no significant difference considering both time and treatment.

The invasive systolic ABP values in the control, fructose, FE, FT, and FET groups were 115.4 ± 1.6, 134.2 ± 2.8, 122.6 ± 2, 123.1 ± 1.7, and 115.2 ± 1.3 mm Hg, respectively, at the end of the 4-week trial (Figure 2a). Systolic ABP increased significantly in the fructose (P < 0.001) and FT (P < 0.05) groups, but the difference was not significant between the FE and FET groups and the control group. However, ABP was significantly lower (P < 0.001) in the FE, FT, and FET groups than in the fructose group. Diastolic ABP values in the control, fructose,

| Table 2 | Blood ionic status in different groups after the 4 weeks trial of taurine supplementation and exercise and their combined effect in high fructose-fed rats |
| Control | F | FE | FT | FET |
| pH | 7.37 ± 0.03 | 7.30 ± 0.06 | 7.36 ± 0.02 | 7.33 ± 0.04 | 7.37 ± 0.02 | NS |
| Na+, mmol/l | 135 ± 1.30 | 146 ± 1.39* | 144 ± 0.85* | 140 ± 1.75 | 142 ± 1.48 | <0.01 |
| K+, mmol/l | 4.38 ± 0.44 | 5.16 ± 0.38 | 4.63 ± 0.04 | 4.89 ± 0.28 | 4.36 ± 0.20 | NS |
| Cl−, mmol/l | 109 ± 1.79 | 101 ± 1.08* | 105 ± 0.76 | 103 ± 1.07* | 102 ± 1.03* | <0.001 |
| Hct (decimal) | 0.45 ± 0.80 | 0.41 ± 0.01* | 0.46 ± 0.01** | 0.44 ± 0.01*** | 0.45 ± 0.01** | <0.001 |
| Hb mg/dl | 12.66 ± 0.78 | 13.3 ± 0.47 | 14.9 ± 0.19* | 13.4 ± 0.37 | 14.1 ± 0.18 | <0.05 |

Values are represented as mean ± s.e.m. The P value in the last column is the overall P value for a one-way ANOVA performed across all of the groups.

CT, chloride ion; F, fructose; high-fructose-fed group; FE, high-fructose-fed exercise group; FT, high-fructose-fed for exercise and taurine supplementation group; FET, high-fructose-fed for exercise and taurine supplementation group; Hb, hemoglobin; Hct, hematocrit; K+, potassium ion; Na+, sodium ion; NS, not significant.

*P < 0.001, Bonferroni post hoc test following one-way ANOVA vs. control. **P < 0.05, Bonferroni post hoc test following one-way ANOVA vs. F; n = 10 each group.
FE, FT, and FET groups were 78.7 ± 2.6, 103.4 ± 3.9, 81.5 ± 3.6, 91.4 ± 1.4, and 75.1 ± 3.1 mm Hg, respectively. Diastolic ABP was significantly greater (P < 0.001) in the fructose group than in the control group and was significantly lower (P < 0.001) in the FE, FT, and FET groups than in the fructose group. Mean ABP values were 90.8 ± 2.2, 113.7 ± 3.4, 95 ± 2.9, 101.9 ± 1.2, and 88.5 ± 2.3 mm Hg in the control, fructose, FE, FT, and FET groups, respectively. Mean ABP was significantly greater (P < 0.001) in the fructose group than in the control group and was significantly lower (P < 0.001) in the FE, FT, and FET groups than in the fructose group.

Swimming capacity
Average swimming time in the control group of animals was 65.9 ± 2.9 min, which was significantly greater (P < 0.001) than that (38.4 ± 1.5 min) in the fructose group (Figure 2b). The swimming time in the FE group was 59.3 ± 1.2 min, which was not significantly different from that in the control group but was significantly different (P < 0.001) from that in the fructose group. An extended swimming time of 71.7 ± 2.8 min was recorded in the FT group, but the difference was not significantly different from the control group. The maximum swimming time of 80.3 ± 1.1 min was recorded in the FET group, which differed significantly (P < 0.001) from the control and fructose groups.

Plasma glucose and insulin and insulin resistance
Plasma glucose concentrations in the control, fructose, FE, FT, and FET groups were 194 ± 10, 450 ± 4, 318 ± 33, 354 ± 16, and 189 ± 9 mg/dl, respectively (Figure 3a). Plasma glucose was significantly greater (P < 0.001) in the fructose, FE, and FT groups than in the control group, but the comparative difference in the FET group was not significant. Plasma insulin in the fructose group (32.3 ± 1.1 mU/l) was significantly greater (P < 0.001) than that in the control group (18.2 ± 1.2 mU/l). Insulin concentrations in the FE, FT, and FET groups were 27.1 ± 1.1, 23.9 ± 1.5, and 21.1 ± 1.4 mU/l, respectively, and concentrations in the FE and FT groups were significantly different from the concentration in the control group (P < 0.001 and P < 0.05, respectively; Figure 3b).

Insulin resistance was calculated as (glucose (mmol/l) × insulin (mU/l))/22.5. The greatest insulin resistance was calculated in the fructose group (35.9 ± 1.3), which differed significantly (P < 0.001) from that in the control group (8.7 ± 0.6). Insulin resistance was significantly greater (P < 0.001) in the FE and FT groups (21.2 ± 2.3 and 20.9 ± 1.8, respectively) than in the control group. Insulin resistance in the FET group (9.8 ± 0.7) was not significantly different from that in the control group (Figure 3c).

Creatine kinase in plasma
Plasma creatine kinase (CK) concentrations in the control, fructose, FE, FT, and FET groups were 446 ± 11, 811 ± 94,
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1,414 ± 156, 519 ± 48, and 423 ± 97 IU/l, respectively (Figure 4). CK was significantly greater in the fructose group \( (P < 0.05) \) and the FE group \( (P < 0.001) \) than in the control group but remained similar to the baseline level in the FET group.

**Antioxidant component system**

High fructose feeding significantly \( (P < 0.001) \) increased plasma MDA levels and decreased the concentration of plasma thiols in the fructose, FE, and FT groups. Both exercise and taurine supplementation were effective at improving the levels of MDA and thiols. The differences between the FET and control groups were not significant, but a significant decrease was found between the FET and fructose groups. Taurine supplementation along with exercise maintained plasma MDA and thiols near the control value (Table 3). High fructose consumption in the fructose group significantly \( (P < 0.001) \) decreased the blood level of GSH in the fructose and FE groups, but the values were not significantly different from the control group. A significant \( (P < 0.001) \) increase in GSSG was observed in the fructose group, but GSSG in the FE, FT, and FET groups was not significantly different from that in the control group. The GSH level was significantly \( (P < 0.001) \) restored in the FT and FET groups by taurine supplementation and by the combined effect of taurine supplementation and exercise (Table 3). Both exercise \( (P < 0.001) \) and taurine supplementation \( (P < 0.001) \) were significantly effective at decreasing the GSSG concentration. The combined effect of exercise and taurine supplementation successfully maintained the GSSG level in the FET group. The GSSG level was not significantly different between the FET and control groups, but was significantly \( (P < 0.001) \) different between the FE and taurine supplementation groups (Table 3). The redox ratio (GSH/GSSG) was significantly \( (P < 0.001) \) lower in the fructose group, by about half, than in the control group. GSH/GSSG in the FE and FT groups improved with the individual effects of exercise and taurine supplementation. GSH/GSSG in the FET group was maintained near the control value by the combined effect of exercise and taurine supplementation (Table 3).

**Plasma nitrite/nitrate**

The mean plasma concentration of nitrite/nitrate was 22.1 ± 1.5 µmol/l in the control group and, in comparison, was

---

**Table 3 | Antioxidant component system after the 4 weeks trial of taurine supplementation and exercise and their combined effect in high fructose fed rats**

<table>
<thead>
<tr>
<th>Control</th>
<th>F</th>
<th>FE</th>
<th>FT</th>
<th>FET</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma MDA (µmol/l)</td>
<td>2.31 ± 0.06</td>
<td>3.49 ± 0.23*</td>
<td>3.22 ± 0.13*</td>
<td>3.09 ± 0.16*</td>
<td>2.43 ± 0.15**,*** &lt;0.001</td>
</tr>
<tr>
<td>Plasma thiol (µmol/mg prot)</td>
<td>4.17 ± 0.03</td>
<td>2.36 ± 0.12*</td>
<td>3.23 ± 0.14*</td>
<td>3.65 ± 0.17*</td>
<td>4.18 ± 0.07**,† &lt;0.001</td>
</tr>
<tr>
<td>Blood GSH (µmol/l)</td>
<td>821 ± 25</td>
<td>667 ± 18*</td>
<td>714 ± 14*</td>
<td>778 ± 17**</td>
<td>813 ± 15**,† &lt;0.001</td>
</tr>
<tr>
<td>Blood GSSG (µmol/l)</td>
<td>7.50 ± 0.33</td>
<td>11.8 ± 0.84*</td>
<td>9.30 ± 0.69</td>
<td>8.80 ± 0.04**</td>
<td>7.60 ± 0.75** &lt;0.001</td>
</tr>
<tr>
<td>Blood GSH/GSSG</td>
<td>111 ± 4.55</td>
<td>59 ± 5.21*</td>
<td>80 ± 6.39*</td>
<td>91 ± 5.66**</td>
<td>109 ± 6.56**,*** &lt;0.001</td>
</tr>
</tbody>
</table>

Values are represented as mean ± s.e.m. The P value in the last column is the overall P value for a one-way ANOVA performed across all of the groups.  
F, fructose, high fructose-fed group; FE, high fructose–fed with exercise group; FT, high fructose–fed plus exercise group; FET, high fructose–fed plus exercise and taurine supplement group; FF, fructose–fed plus exercise and taurine supplement group; F, fructose; GSH, Reduced glutathione; GSSG, oxidized glutathione; MDA, malondialdehyde.  
*P < 0.001, Bonferroni post hoc test following one-way ANOVA vs. control. **P < 0.001, Bonferroni post hoc test following one-way ANOVA vs. fructose, n = 10 each group and ***P < 0.01.  
†P < 0.01 while FE vs. FET.

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![Figure 3](https://academic.oup.com/ajh/article-abstract/24/5/574/2282061/578)
significantly lower ($P < 0.001$) in the fructose, FE, and FT groups: $9.8 \pm 1.1 \mu mol/l$ (55.6%), $12.9 \pm 0.9 \mu mol/l$ (41.5%), and $15.2 \pm 1.1 \mu mol/l$ (31.2%), respectively. The combined effect of exercise and taurine supplementation maintained the plasma nitrite/nitrate concentration at $18.8 \pm 0.6 \mu mol/l$ in the FET group, which was 14.9% less than that in the control group (Figure 5).

**DISCUSSION**

The results of the present study in a rat model of insulin resistance indicate that high dietary fructose may be linked to the development of hypertension and a consequent decrease in exercise capacity in a manner that is possibly related to oxidative stress. The observed changes in markers of free radical damage are consistent with exercise-induced muscle injury. High reactivity of fructose and its metabolite may contribute substantially to the glycation of proteins.

Taurine, which contains a free amino group, may form a Schiff base with sugar carbonyls and, hence, spare proteins from glycation if they can be maintained at high concentrations in the tissues. Taurine is often classified as an antioxidant, and its physiological effects contribute to a reduction in oxidative stress and protect tissue damage through inhibition of reactive oxygen species formation. In the biosynthetic pathway for taurine, GSH, and hypotaurine and their associated enzyme systems are considered true antioxidative systems, similar to the superoxide dismutase enzyme system, for example. Taurine may prevent $Ca^{2+}$ overload and thereby minimize free radical generation, with the protective effect reflected in the degree of lipid peroxidation and NO production.

The currently used biomarkers of antioxidation, plasma chemistry measures (CK, glucose, insulin, and insulin resistance), NO status, and blood ions ($Na^+$, $K^+$, and $Cl^-$) might be useful in a clinical assessment of the benefits of exercise training and/or taurine supplementation programs for preventing hypertension and achieving better performance. A previous study reported that taurine has a cytoprotective role in exercise-induced muscle injury. Other studies showed that taurine increases force production in skeletal muscles with antifatigue properties. In agreement with these reports, the increased CK levels in this study might be an indicator of muscle and myocardial damage. High fructose feeding caused oxidative injury, which was further augmented by swimming, and taurine showed protective effects in this study.

Increased $Na^+$ concentrations in the circulation promote hypertension-induced hypertrophy of the vascular wall. Oral taurine supplementation can modify $Na^+$ homeostasis in high fructose–fed rats. Moreover, taurine has an insulin-like action on glucose metabolism that accelerates glucose uptake into the liver and muscle, and exercise additionally raises insulin activity, which reduces insulin resistance and improves glucose intolerance.

The increase in plasma MDA levels observed in the current study and the corresponding decrease in plasma thiols reflect the upregulation of protein oxidation with high fructose feeding. In most cases, taurine supplementation appeared to blunt the exercise-induced decrease in acid-soluble thiols. The available evidence suggests that physical exercise increases the activity of antioxidant enzymes and whereas acute exercise can induce oxidative stress in untrained individuals, regular exercise reduces oxidative stress. Exercise increases free radical production and attendant lipid peroxidation, with no effect of either isometric or dynamic exercise on plasma MDA levels. Hypoxic exposure increases plasma MDA levels in athletes, and increased oxidative damage provokes a marked reduction in physical performance. Exercise improves prognosis in hypertensive heart disease by increasing the efficiency of the antioxidant system and preserving mitochondrial energy metabolism. GSH is another fundamental defense...
mechanism during conditions of increased oxidative stress. Blood levels of GSH appear to decrease with acute exercise and are sometimes accompanied by an increase in GSSG. In addition to causing reactive oxygen species-mediated NO inactivation, oxidative stress can inhibit NO synthase activity. The observed elevation of NO in the plasma of exercised rats was probably due to changes in oxidative balance and/or to exercise-related increased endothelial shear stress, which is a potent stimulus of the activation of NO synthase activity.

Taurine supplementation might be a form of antioxidant protection against high fructose–induced detrimental effects on NO-mediated vasorelaxation.

Because of the high intracellular concentrations of taurine and the reactivity of the amino group toward carbonyl groups, taurine could be expected to react with the dicarbonyl intermediates and, thus, scavenge the intracellularly formed reactive carbonyl compounds and glycation intermediates. It is postulated that some of the protein crosslinking that occurs in vivo is fructose-induced, which causes fructosylation-dependent protein denaturation. Because the conversion of glycated proteins to the more complex denatured proteins involves oxygen free radicals, the experimental results indicate that the effects of taurine could be related to its possible oxyradical scavenging properties.

In conclusion, taurine supplementation in regularly exercising subjects has a twofold beneficial effect—prevention of hypertension and an increase in exercise capacity—is a proposal through antioxidant system management and systemic NO optimization. Thus, taurine may rebound in popularity as a fitness supplement in athletes’ intent on maximizing performance through antioxidant system management and systemic NO optimization. Therefore, taurine may reestablish its role as a fitness supplement in athletes’ intent on maximizing performance by virtue of its antioxidant, antifatigue, and anti–hypertensive properties.

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Disclosure: The authors declared no conflict of interest.

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