

Effects of Microhydrin® Supplementation on Endurance Performance and Metabolism in Well-Trained Cyclists

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This study investigated whether the supplement Microhydrin® (MH) contains silica hydride bonds (Si-H) and if Microhydrin supplementation increased performance or altered metabolism compared to placebo (PL) during prolonged endurance cycling. Seven endurance-trained male cyclists consumed 9.6 g of MH or PL over 48 h in a randomized, double-blind, crossover design. Subjects cycled at ~70% of their $\dot{V}O_{2peak}$, coupled with 5 2-min bursts at 85% $\dot{V}O_{2peak}$ to simulate hill climbs over 2 h. Subjects then completed a time trial, which required them to complete 7 kJ/kg body mass as quickly as possible. Infrared spectrometry analysis showed a complete absence of Si-H bonds in MH. There was no difference in time trial performance between the 2 trials (PL: 2257 ± 120 s vs. MH: 2345 ± 152 s). Measured oxygen uptake, respiratory exchange ratio, carbohydrate (MH: 2.99 ± 0.13 g/min; PL: 2.83 ± 0.17 g/min avg. over 2 h) and fat (MH: 0.341 ± 0.06 g/min; PL: 0.361 ± 0.07 g/min) oxidation rates and all blood parameters (lactate, glucose, and free fatty acids) were all unaffected by MH supplementation. The volume of expired CO_2 and ventilation were significantly greater with MH supplementation ($P \leq 0.05$). The results indicate that oral Microhydrin supplementation does not enhance cycling time trial performance or alter metabolism during prolonged submaximal exercise in endurance-trained cyclists.

Key Words: ergogenic aid, silica hydride, lactate, cycling performance, time-trial

There are many factors that might limit exercise performance in an endurance athlete (for a review, see reference 8). During exercise onset, or during times of transition from a given submaximal intensity to a slightly higher one, the physiological factors underlying fatigue are already operating and their performance-detracting effects accumulate as time progresses. As submaximal exercise continues, the ability to work at a greater power output decreases over time as the result of the taxing nature of the preceding work (8).

During race situations, the majority of energy provision for endurance cyclists comes from aerobic energy production via oxidative phosphorylation. If the ability

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to provide oxidative ATP is less than adequate at any given time point, however, it forces the muscle to supplement energy provision from anaerobic processes, or substrate level phosphorylation (3, 7, 15). Anaerobic ATP can be produced via phosphocreatine (PCr) breakdown and the conversion of pyruvate to lactate in the glycolytic pathway. Unfortunately, there are associated ionic changes that accompany high glycolytic flux, which decrease intramuscular pH, creating a suboptimal environment for muscular function. With a sustained anaerobic contribution, such as an athlete pushing the pace in a racing situation, there is depletion of PCr and increases in muscle and blood lactate and subsequent ionic changes and acidosis which lead to fatigue and decreased performance (3, 7, 15).

It has been claimed that silica hydride (SH), the active ingredient in Microhydrin® (MH), delivers reduced hydrogen, or hydride ions (H^-), into the blood stream, therefore acting as a reducing agent or antioxidant when in solution (13). In buffering H^+ ions produced during exercise, MH could improve performance during cycling races in which there exist periods where anaerobic metabolism is called upon for energy provision.

To date, only 1 study has examined the effects of dietary Microhydrin on the performance, cardiovascular, metabolic, and central nervous system responses to exercise in humans (13). The only significant difference ($P < 0.03$) observed in this study was that MH lowered post-exercise blood lactate concentration (2.57 mmol/L) vs. placebo (3.37 mmol/L). Other than power output values recorded 6 times throughout exercise, the study failed to directly measure changes in performance that might have occurred as the result of supplementation because time trial times were not recorded. Therefore, the current study served 2 purposes. First, to determine whether Si-H bonds exist in the commercially available Microhydrin supplement via infrared spectrometry analysis. Second, to determine if supplementation with Microhydrin is effective in improving the performance of endurance-trained cyclists during a time trial following prolonged submaximal cycling exercise by altering any physiological responses related to prolonged cycling that are associated with performance.

Methods

Subjects

Seven endurance-trained male cyclists volunteered to participate in the study. Subjects were engaged in regular endurance training (at least 9 cycling h/wk) prior to, and during, the testing period and none were using any conflicting medications. Their mean (\pm standard error) age, weight, and peak oxygen uptake (VO_{2peak}) were 25.0 ± 1.1 y, 72.0 ± 3.1 kg, and 65.3 ± 1.2 mL \cdot kg $^{-1}$ \cdot min $^{-1}$, respectively. All subjects were informed of the experimental protocol and associated risks of the study, both orally and in writing, before written informed consent was obtained. The ethics committee of the University of Guelph approved the study.

Pre-Experimental Protocol

Subjects initially underwent a continuous incremental cycle test to exhaustion to determine peak pulmonary oxygen uptake (VO_{2peak}) (Vmax 229c, SensorMedics, Yorba Linda, CA) on a cycle ergometer (LODE Instrument, Groningen, The

Netherlands). After the $\text{VO}_{2\text{peak}}$ tests, subjects visited the laboratory on 4 more occasions; twice for practice trials and 2 visits for the experimental protocol. Visits to the laboratory was separated by 1 wk, and during all trials subjects used their own bike seats and pedals for consistency between trials. Two practice trials were used to familiarize subjects with the exercise protocols and time trial for improved reliability, and to confirm the $\sim 70\%$ and $85\% \text{VO}_{2\text{peak}}$ during cycling. The mean (\pm standard error) absolute power output for the trials was 206 ± 9 W at $\sim 70\% \text{VO}_{2\text{peak}}$ and 253 ± 12 W at $\sim 85\% \text{VO}_{2\text{peak}}$.

During the 1st practice trial, subjects cycled for 18 min at $70\% \text{VO}_{2\text{peak}}$, 2 min at $\sim 85\% \text{VO}_{2\text{peak}}$ and a further 5 min at $70\% \text{VO}_{2\text{peak}}$. The subjects then immediately began the 1st practice time trial, which required them to complete 7 kJ of work/kg body mass as quickly as possible. The 2nd practice trial replicated the experimental protocol in full and is outlined in Figure 1. It consisted of a 10 min pre-trial rest period, a 2 h pre-time trial or steady state ride ($\sim 70\% \text{VO}_{2\text{peak}}$) and the time trial. During the 2 h pre-time trial segment, subjects rode the 1st 18 min at a baseline power output of $\sim 70\% \text{VO}_{2\text{max}}$. From 15 to 18 min, expired pulmonary gases were sampled and from 18 to 20 min the power output was increased to $85\% \text{VO}_{2\text{peak}}$. The power output increase to $85\% \text{VO}_{2\text{peak}}$ was designed to simulate a “real life” hill climb or mid-race surge that a cyclist would typically experience during a race. From 20 to 38 min the power output was returned to $70\% \text{VO}_{2\text{peak}}$. Heart rate (HR) and rating of perceived exertions (RPE) were taken just before gas sampling at 15 min and again after the hill climb at 22 min. A commercial sports drink (Gatorade®: 6.3% CHO, 18 mmol/L sodium) was provided at 22 min, and consumed within 10 min, to ensure proper fluid (5 mL/kg, ~ 350 mL) and carbohydrate (22 g CHO/350 mL) replacement. The total fluid and CHO consumption during the 2-h trial was 1.75 L and 110 g, respectively. This gas sampling, hill climb, and drinking protocol (from 15 to 35 min) was repeated 4 additional times at regular intervals (35, 55, 75, and 95 min) during the 120 min pre-time trial segment.

During the pre-time trial cycling segment, the power output was controlled by the manually set, electronically braked, cycle ergometer. During the time trial segment the ergometer mode was switched from manual to linear, which allowed subjects to control power output by varying their pedal cadence. The linear mode was set to elicit a power output of $\sim 70\% \text{VO}_{2\text{peak}}$ at 90 rpm. Subjects volitionally worked at a higher power output by increasing their pedal cadence. During all time trials, subjects were aware of their rpm, power output (W), and total work completed (a running count of completed kJ), but were not aware of the elapsed time. The subjects competed for monetary prizes given to the 3 subjects with the lowest total performance time to complete both trials to encourage maximal effort in both trials.

Experimental Protocol

The 2 experimental trials were randomized and both the subjects and experimenters were blinded to the Microhydrin and placebo supplementation order. Post-trial questionnaires were given to test the subjects’ treatment blindness. Subjects were asked to maintain their normal training patterns but to refrain from intense exercise and caffeine and alcohol consumption 48 h before each trial. Subjects were allowed to eat as they would normally 24 h before a race and maintained 24-h diet records

over this period enabling the same diet to be followed on the subsequent trial (53% carbohydrate; 28% fat; 19% protein).

Before testing, subjects were weighed and an indwelling catheter was inserted into the antecubital vein for blood sampling; the line was kept patent by a saline drip (~ 500 mL infused during each trial). Subjects rested for 10 min before exercise, cycled for 120 min during a pre-time trial phase, and then immediately began the time trial (Figure 1). Blood samples (~ 4 mL) were drawn at -10 min, immediately before the start of exercise (0 min) and throughout exercise. HR was recorded using a Polar a1 heart rate monitor (Polar Electro Inc., Lake Success, NY) and subjective RPE was recorded using the modified Borg scale at rest and throughout exercise (2). Expired pulmonary gases were collected and measured for 3 min at 15, 35, 55, 75, and 95 min of the pre-time trial ride. Whole-body rates of CHO and fat oxidation (g/min) were calculated during the cycle rides from the rates of CO₂ production (VCO₂) and O₂ consumption (VO₂) by using the nonprotein respiratory exchange ratio (RER) values according to the following equations (12):

$$\text{CHO oxidation (g/min)} = [4.585 \times \text{VCO}_2 \text{ (L/min)}] - [3.226 \times \text{VO}_2 \text{ (l/min)}]$$

$$\text{Fat oxidation (g/min)} = [1.695 \times \text{VO}_2 \text{ (L/min)}] - [1.701 \times \text{VCO}_2 \text{ (l/min)}]$$

Microhydrin and Placebo Supplementation

Subjects ingested 6 capsules (600 mg/capsule, 3.6 g total) of either Microhydrin (Microhydrin dietary supplement, Royal BodyCare, Inc., Irving, TX) or D-glucose

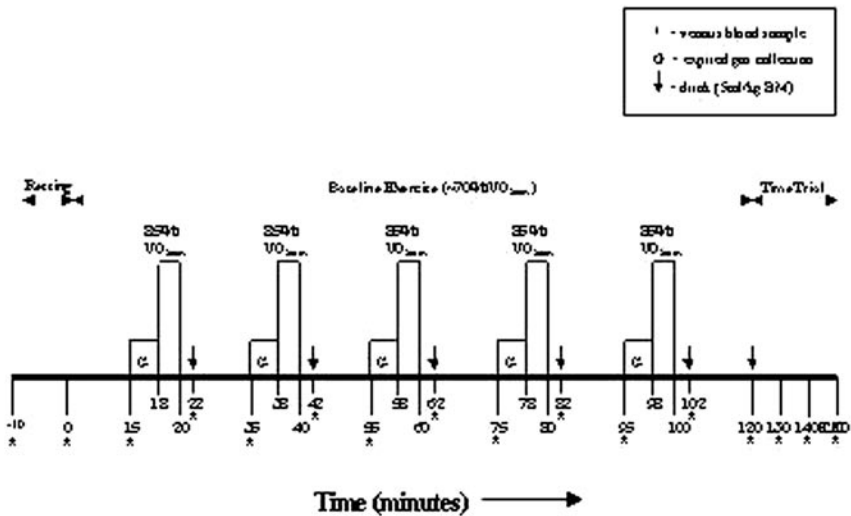


Figure 1 — Experimental design. Supplementation with Microhydrin or placebo occurred at 24 and 4 h prior to commencement of the exercise trial. During the initial 120 min of exercise (pre-time trial) the power output was ~70% VO_{2peak} with 2 min periods of increased power output to ~85% VO_{2peak} occurring 5 times as indicated. The time trial (TT) required the completion of 7 kJ of work/kg body mass (BM).

placebo (3.6 g total) with 1.2 L (200 mL/capsule) of water 24 h prior to each trial. Subjects also ingested 10 capsules of either Microhydrin (MH) or D-glucose (PL) (6.0 g total) with 2 L of water 4 h prior to each trial. The dosing regime was developed under the guidance of professional cyclists who are currently using MH before races, to mimic the supplementation protocol currently being used in actual race conditions.

In Vitro Analysis of Microhydrin

The anhydrous MH was analyzed for the presence of Si-H bonds by total internal reflectance infrared (IR) spectroscopy using a spectrometer (Nicolet 510 FT-IR, Thermo Electron Corp., Madison, WI) fitted with a ZnSe sample window (Figure 2). An absorption spectrum of a solid 600 mg sample was recorded displaying signature peaks of chemical bonds present in the sample. Si-H bonds, if present, absorb at $2100 \pm 50 \text{ cm}^{-1}$ wavenumbers. The presence of Si-H bonds in a confined standard was confirmed.

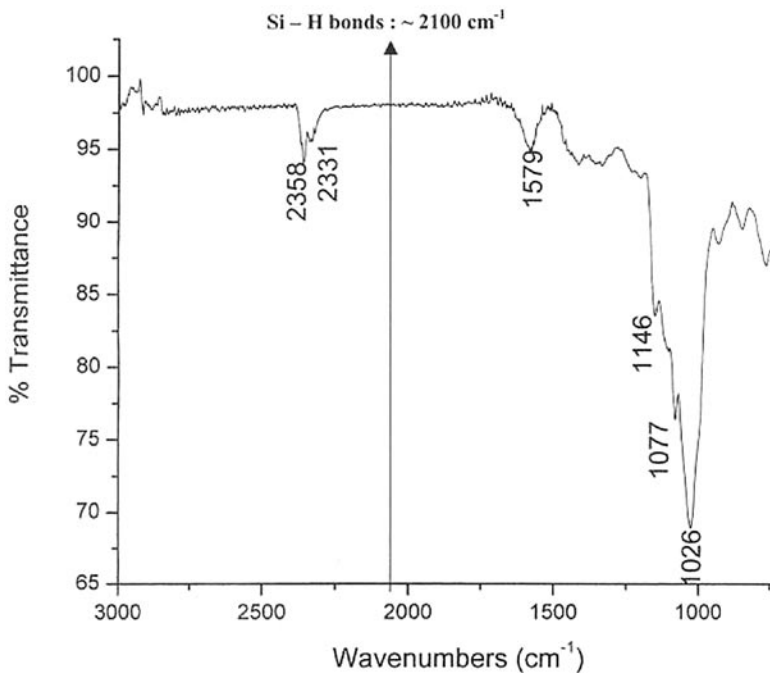


Figure 2 — Absorption spectrum of anhydrous Microhydrin using total internal reflectance infrared spectroscopy (IR). Silica hydride (Si-H) bonds, if present, absorb at $2100 \pm 50 \text{ cm}^{-1}$ wavenumbers.

Blood Analysis

Venous whole blood was drawn into heparinized tubes, and a portion was immediately deproteinized in a 1:2 ratio with 0.6% (wt/vol) perchloric acid (HClO_4). The extract was then stored at -80°C and later analyzed for blood lactate and glucose (1). A 2nd portion of blood was immediately centrifuged, and 400 μl of plasma was added to 100 μl NaCl and incubated at 56°C for 30 min to inactivated lipoprotein lipase. The plasma was stored at -80°C and analyzed for free fatty acids (FFA) via a colorimetric assay (Wako NEFA C test kit, Wako Chemicals, Richmond, VA).

Statistics

All data are presented as mean \pm standard error. Dependant variables were analyzed using 2-way repeated-measures ANOVA [treatment (PL, SH) \times time]. Statistical analysis was performed for all experimental time points and also individually between only the time trial points. A Student-Newman-Keuls post hoc test was used to test for significance when a significant F ratio was obtained. Performance time (MH vs. PL) was assessed using a paired t -test. Statistical significance was accepted at $P < 0.05$.

Results

Microhydrin Chemical Analysis

The absorption analysis of MH revealed a complete absence of Si-H bonds, which absorb at $2100 \pm 50\text{ cm}^{-1}$ wavenumbers (Figure 2).

Performance Time Trial

There was no difference in the average time to complete 7 kJ of work/kg body mass as quickly as possible between the MH or PL trials (2345 ± 152 vs. 2257 ± 120 s, respectively; Figure 3). Variability of performance times under different treatments was low with 4 subjects performing slightly faster on MH and 4 subjects performing slightly slower. The average time trial power output during MH supplementation (221 ± 15 W) was not significantly different when compared to PL trial power output (228 ± 14 W). The post-exercise questionnaire validated the subjects' complete blindness as to which supplement they were taking.

Cardiorespiratory Responses

During the 120 min pre-time trial ride, average HR (MH: 155 ± 5 beats/min; PL: 152 ± 4 beats/min) and RPE (MH: 13.0 ± 0.4 ; PL: 12.7 ± 0.5) were both unaffected by MH supplementation (Table 1). Time trial values for both HR (MH: 165 ± 5 beats/min; PL: 166 ± 4 beats/min) and RPE (MH: 16.5 ± 0.5 ; PL: 16.7 ± 0.5) were significantly greater than the 120 min values in both trials. Exercise VO_2 was not significantly affected by MH and increased ($P < 0.05$) over time in both trials (Table 1). V_E and VCO_2 were significantly higher in the MH compared to the PL trial ($P < 0.05$; trial effect, Table 1) and both increased over time. The RER was unaffected by MH, but decreased throughout exercise in both trials with all

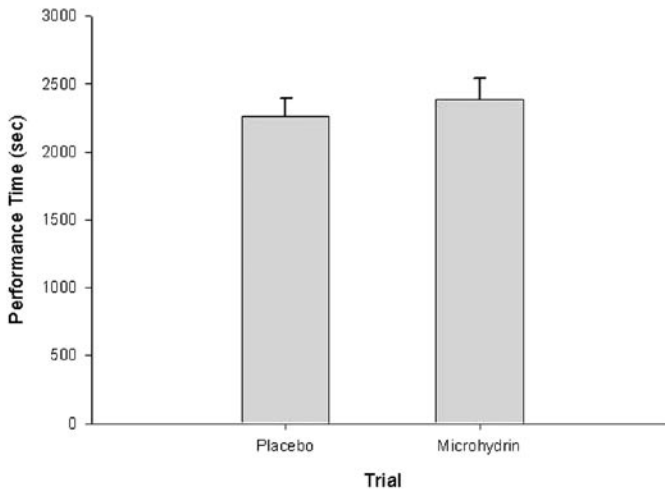


Figure 3 — Effect of Microhydrin (MH) or placebo (PL) ingestion on performance during a 7 kJ/kg time trial at the end of a 2-h pre-time trial cycle. Values are \pm standard error of the mean, $n = 7$. No significant difference between trials was observed.

time points significantly lower than the 15 min measurement ($P < 0.05$, Table 1). Because there was no difference between trials in RER, both average CHO (MH: 2.99 ± 0.13 g/min; PL: 2.83 ± 0.17 g/min) and FAT (MH: 0.34 ± 0.06 g/min; PL: 0.36 ± 0.07 g/min) oxidation rates were unaffected by MH supplementation (Table 1). As expected, however, CHO oxidation decreased and fat oxidation increased during the 2-h pre-time trial ride ($P < 0.05$).

Blood Measures

Blood lactate, glucose, and plasma FFA levels were unaffected by MH supplementation across all time points. During both trials, blood lactate increased significantly with exercise, and time trial values were significantly greater than the 120 min values in both trials (Figure 4). Blood lactate values also increased by ~ 0.5 mmol/L after the power output was increased to 85% $\text{VO}_{2\text{max}}$ for 2 min. During both trials, blood glucose decreased ($P < 0.05$) from rest at 15 and 22 min but subsequently increased after 22 min to higher values than at rest at 42 and 55 min (Table 2). ■ After 55 min, blood glucose values decreased ($P < 0.05$) steadily and were lower than resting values again at 140 min and at the END point of the time trial (Table 2). Plasma FFA were not affected by MH supplementation but did increase ($P < 0.05$) from rest and 15 min values, after 95 min of exercise (Table 2).

Discussion

The current study found no effect of Microhydrin on time to complete a performance time trial, blood FFA, glucose, and, unlike the only other human exercise study examining the effects of SH supplementation (13), found no differences in blood

Table 1 Respiratory responses during the pre-time trial (0 to 120 min) and time trial following the ingestion of Microhydrin (MH) or placebo (PL)

Measure	Trial	Time					
		Pre-Time Trial Ride at ~70% VO _{2max}				Time Trial	
		15-18 min	35-38 min	55-58 min	75-78 min	95-98 min	End of TT
HR (beats/min)	PL	146 ± 4	150 ± 5	152 ± 4	153 ± 4	154 ± 4	172 ± 4 ^{a,b}
	MH	147 ± 5	154 ± 5	154 ± 5	155 ± 5	157 ± 5	170 ± 5 ^{a,b}
RPE	PL	11.6 ± 0.7	12.0 ± 0.6	12.5 ± 0.4	12.8 ± 0.3	13.5 ± 0.4 ^a	18.3 ± 0.6 ^{a,b}
	MH	12.0 ± 0.5	12.5 ± 0.4	12.8 ± 0.3	13.0 ± 0.3	13.9 ± 0.5 ^a	18.8 ± 0.6 ^{a,b}
VO ₂ (L/min)	PL	2.75 ± 0.14	2.79 ± 0.14 ^a	2.81 ± 0.14 ^a	2.90 ± 0.14 ^a	2.91 ± 0.14 ^a	ND
	MH	2.77 ± 0.14	2.86 ± 0.14 ^a	2.91 ± 0.15 ^a	2.97 ± 0.15 ^a	2.98 ± 0.16 ^a	ND
VCO ₂ ^c (L/min)	PL	2.59 ± 0.13	2.58 ± 0.13	2.60 ± 0.13	2.66 ± 0.11	2.63 ± 0.11	ND
	MH	2.64 ± 0.13	2.65 ± 0.12	2.71 ± 0.12	2.73 ± 0.12	2.72 ± 0.13	ND
RER	PL	0.94 ± 0.01	0.93 ± 0.01 ^a	0.93 ± 0.01 ^a	0.92 ± 0.01 ^a	0.91 ± 0.01 ^a	ND
	MH	0.96 ± 0.01	0.93 ± 0.01 ^a	0.93 ± 0.01 ^a	0.92 ± 0.01 ^a	0.91 ± 0.01 ^a	ND
Ve ^c (L/min)	PL	67.8 ± 2.3	68.2 ± 2.5	70.2 ± 2.7	72.4 ± 2.5 ^a	72.9 ± 2.5 ^a	ND
	MH	70.2 ± 3.3	72.0 ± 3.0	73.1 ± 3.0	76.4 ± 3.4 ^a	77.2 ± 4.3 ^a	ND
CHO oxid. (g/min)	PL	3.00 ± 0.18	2.82 ± 0.20	2.85 ± 0.18	2.81 ± 0.15	2.67 ± 0.15 ^a	ND
	MH	3.19 ± 0.18	2.92 ± 0.12	3.05 ± 0.15	2.95 ± 0.08	2.84 ± 0.12 ^a	ND
FAT oxid. (g/min)	PL	0.26 ± 0.05	0.35 ± 0.06 ^a	0.34 ± 0.06 ^a	0.40 ± 0.07 ^a	0.46 ± 0.08 ^a	ND
	MH	0.19 ± 0.05	0.34 ± 0.05 ^a	0.32 ± 0.07 ^a	0.39 ± 0.05 ^a	0.43 ± 0.06 ^a	ND

Note. Values are means ± standard error of the mean, $n = 7$. ND, no determination. ^aindicates significantly different than the 15 to 18 min measurement of same trial, in both MH and PL ($P < 0.05$); ^bindicates significantly different than the pre-time trial measurements of same trial, in both MH and PL ($P < 0.05$); ^cindicates significant main effect for trials of MH being greater than PL ($P < 0.05$).

lactate concentrations. The current study also found no effect of MH on RPE, VO₂, heart rate, and performance power output values. Infrared spectrometry analysis also showed a complete absence of Si-H bonds in MH. Taken together, the results demonstrated that oral MH supplementation did not enhance time trial performance or alter metabolism during prolonged submaximal cycling in endurance-trained cyclists.

Microhydrin Chemistry, Commercial Viability, and Practicality of Si-H Bonds

Recent in vitro data suggests that Microhydrin is a potential antioxidant against reactive oxygen species (ROS)(16-18). These studies suggest the primary antioxi-

Table 2 Blood glucose and plasma free fatty acid (FFA) concentrations at rest and during rest (-10 to 0 min), pre-time trial (0 to 120 min) and time trial (120 min to END) cycling following the ingestion of Microhydrin (MH) or placebo (PL)

Measure	Trial	Time (min)										
		-10	0	15	35	55	75	95	120	130	140	END
Glucose (mmol/L)	PL	4.15±0.38	4.43±0.21	3.58±0.10 ^a	4.62±0.16	4.73±0.22 ^a	4.62±0.16	4.27±0.16	4.54±0.26	4.32±0.10	3.94±0.11 ^a	3.45±0.34 ^a
	MH	4.15±0.25	4.81±0.18	3.70±0.28 ^a	4.34±0.22	4.78±0.15 ^a	4.72±0.07	4.50±0.11	4.50±0.14	4.14±0.20	3.78±0.21 ^a	3.69±0.21 ^a
FFA (mmol/L)	PL	0.14±0.03	0.14±0.02	0.10±0.01	0.17±0.01	0.18±0.02	0.24±0.04	0.40±0.08 ^a	0.38±0.06 ^a	0.38±0.07 ^a	0.43±0.09 ^a	0.53±0.10 ^a
	MH	0.26±0.07	0.26±0.07	0.17±0.05	0.19±0.05	0.21±0.06	0.27±0.07	0.32±0.07 ^a	0.43±0.09 ^a	0.45±0.11 ^a	0.51±0.11 ^a	0.66±0.16 ^a

Note. Values are means ± standard error of the mean, $n = 7$. ^aindicates significantly different from rest (-10 and 0 min) of same trial, in both MH and PL ($P < 0.05$).

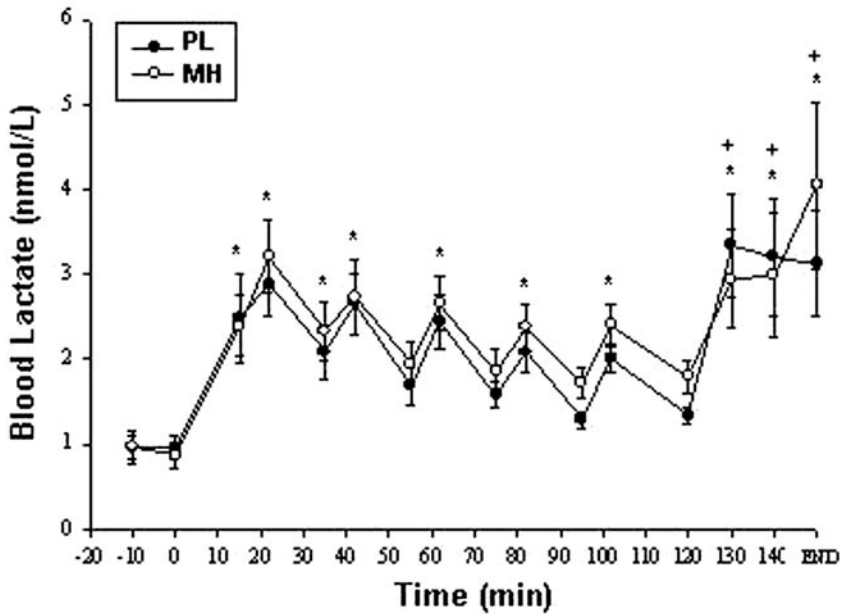


Figure 4 — Whole blood lactate concentrations during rest (-10 to 0 min), pre-time trial (10 to 120 min) and time trial (120 min to END) cycling following the ingestion of Microhydrin (MH) or placebo (PL). Values are means \pm standard error of the mean, $n = 7$. *indicates significantly different from rest (-10 and 0 min) of same trial, in both MH and PL ($P < 0.05$); + indicates significantly different from 120 min of same trial, in both MH and PL ($P < 0.05$).

dant mechanism of MH is the interstitially placed hydride ions via Si-H bonds. In the current study, the internal reflectance infrared (IR) spectroscopy showed a complete absence of Si-H bonds. Also, the established methods used for the hydride derivatization (Si-H formation) of silica present serious limitations for process industrialization and commercialization. Moisture and oxygen, invariably present in the human body, have both been shown to destroy Si-H bonds during some industrial Si-H production methods (14). A recently developed method to synthetically generate Si-H groups on silica also resulted in a very low pure Si-H content yield (5). Therefore, it is apparent that viable silica hydride production on a scale large enough to provide for a commercial nutritional supplement seems most unlikely.

Metabolic Effects: Blood Lactate

Si-H supposedly dissociates in the blood and releases H^- ions into the circulation, which could potentially bind to blood H^+ ions that were produced in conjunction with increased glycolytic activity in the contracting muscle. This, in turn, could maintain blood pH and consequently muscle pH, thus creating a more favorable environment

for muscle function (13). In buffering H^+ produced during exercise, Microhydrin could be used to improve performance during cycling races in which there exist brief periods where anaerobic metabolism is utilized for energy provision.

MH supplementation did not significantly affect blood lactate, glucose, or plasma FFA levels during exercise, suggesting that it had no effect on altering metabolic function and metabolite production. In contrast to previous work, the present study found that MH supplementation did not reduce blood lactate concentration. Purdy Lloyd et al. (13) reported only pre- and post-exercise (5 min post-exercise) measurements, with the MH group having a lower post-exercise blood lactate concentration (2.57 mmol/L with MH vs. 3.37 mmol/L for PL—no standard error values given). It was not clear why blood lactate measurements were not taken throughout exercise. The present study measured blood lactate at rest, and during both the pre-time trial and time trial segments of the exercise test. To simulate “real-life” cycling and to force the muscle to rely more heavily on anaerobic processes with the production of lactate, a series of 2-min simulated “hill climbs” ($\sim 85\% \text{VO}_{2\text{peak}}$) were used (Figure 1). The “hill climbs” transiently increased blood lactate, but without trial differences, clearly demonstrating that Microhydrin had no effect on lowering blood lactate levels.

Microhydrin and Cycle Performance

In the present study, each laboratory ride ended with a time trial designed to mimic a “final lap” situation where power output would naturally increase with subjects being asked to complete a given amount of work as quickly as possible. The use of a work-based time trial (7 kJ/kg bm) has been shown to be a reliable means of objectively detecting changes in performance between different subjects and trials (4). The average power output during the time trial phase was greater ($\sim 225 \text{ W}$) compared to the pre-time trial phase ($\sim 206 \text{ W}$) with no differences between trials. This final time trial phase of the trial simulates what would occur during the “final lap” of a race.

Given the absence of Si-H bonds, and no effect of Microhydrin on blood lactate levels, it was not surprising that MH had no effect on time trial performance compared to placebo (Figure 3). In agreement with Purdy Lloyd et al. (13), the present study found no differences in average time trial power output between trials. During a 40 km time trial, with no pre-time trial ride or increases in power output, Purdy Lloyd et al. (13) failed to report performance time but did observe no difference in power output between trials.

It should be mentioned that only well-trained ($\text{VO}_{2\text{peak}}, 65.3 \pm 1.2 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) male cyclists were recruited for this study. It has been well-documented that the reproducibility and reliability of time-to-exhaustion measurements is greater among trained subjects (6, 10). Well-trained cyclists can perform to exhaustion during a time trial with less concern over motivation limitations or whether they indeed performed to their maximum.

Cardiorespiratory Effects

During the pre-time trial ride there were no significant differences in VO_2 , RER, or HR between trials, supporting previous findings (13). Ventilation and VCO_2 , however, were significantly increased during the Microhydrin trial compared to

placebo. The increased $\dot{V}CO_2$ could have been caused by increased MH ventilation and might have resulted from increased ventilatory drive because of increased PCO_2 or H^+ in the blood. Interestingly, the opposite would have been hypothesized for MH given that its proposed mechanism is to decrease blood H^+ .

Dosing Considerations

It is possible that a performance-enhancing effect was not seen in the current study because the dosing protocol was not optimized. Although using a different dosing protocol than Purdy Lloyd et al. (13), the current dosing regime was developed under the guidance of professional cyclists to mimic the supplementation protocol currently used in professional cycling. Subjects did consume many times the manufacturer's recommended dosage, therefore decreasing the possibility that an ergogenically ineffective dose was used. ■

Microhydrin also contains small amounts of potassium carbonate ($KHCO_3$), which could act as a blood buffer and induce a slight metabolic alkalosis if ingested in high doses (~ 300 mg/kg body mass) during prolonged submaximal exercise (11). In the present study, potassium carbonate was ingested pre-exercise at a much lower dosage (10 capsules at < 300mg/capsule and 70 kg body mass = ~ 40 mg/kg body mass), decreasing the likelihood of an ergogenic effect. Dosing above the levels employed in the current study is not recommended as high $KHCO_3$ ingestion can interfere with cellular excitability thus leading to cardiac fibrillation or arrhythmia (9). The Canadian Centre for Ethics in Sport was contacted regarding Microhydrin, and although the product was not tested specifically, none of the ingredients on the label are on the International Olympic Committee or the World Anti-Doping Agency banned substance list. Although Microhydrin was not ergogenic with the current studies exercise protocol, it was also not ergolytic.

Conclusion

This study demonstrated that Microhydrin supplementation was not ergogenic. It did not affect performance during prolonged submaximal cycling in well-trained subjects, nor did it alter the normal physiological and metabolic responses to exercise compared to placebo treatment. It is also important to note that Microhydrin did not contain any of the "ergogenically active" Si-H bonds it was purported to contain.

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