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Effect of induced erythrocythemia on aerobic capacity, ventilatory threshold, and run performance

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Human subjects participated in this study after giving their free and informed consent. This research has been conducted in compliance with all applicable Federal Regulations governing the Protection of Human Subjects in Research.

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ABSTRACT

Purpose: To determine the effects of induced erythrocythemia upon aerobic capacity ($\dot{V}O_{2max}$), ventilatory threshold (VT), and aerobic performance (3-mile track run, [3MT]). **Methods:** Six trained male distance runners (age = 34.8 ± 5 yr, hematocrit [Hct] = 39.6 ± 1.8%, $\dot{V}O_{2max}$ = 4.10 ± 0.56 L $O_2 \cdot min^{-1}$), received two infusion treatments in a double-blind, counterbalanced study. Treatments: 760 mL autologous resuspended red blood cells (Hct = 43.5%) (BT) and 250 mL isotonic saline (ST) were administered 7 days apart. **Results:** BT significantly ($P < 0.01$) increased $\dot{V}O_{2max}$ (0.46 ± 0.2 L $O_2 \cdot min^{-1}$), VT (0.36 ± 0.23 L $O_2 \cdot min^{-1}$), and significantly ($P < 0.05$) decreased 3MT time (19.8 ± 16.0 s or 2.0 ± 1.6%). Blood lactate after treadmill tests (8.0 ± 2.0 mmol·L^{-1}) were unchanged by BT but were significantly ($P < 0.01$) lower at rest. Hct was unchanged 24 h following BT, but a wk later had increased significantly ($P < 0.05$) to 44.0%. The delayed increase in Hct suggests an initial increase in blood volume may have contributed to the increase in $\dot{V}O_{2max}$ at 24 h. ST had no significant effects on any measure. Hct and $\dot{V}O_{2max}$ were not different from baseline 4 wk after BT. Blood parameters did not change significantly ($P > 0.05$) after BT; ATP, 2,3-DPG, p-50, MCV, MCH, MCHC, RBCs, and WBCs. Reticulocytes were depressed significantly ($P < 0.05$) (0.28 ± 0.2%) 2 wk after BT, but were normal (0.5 ± 0.2%) all other times. **Conclusion:** Blood loading increases $\dot{V}O_{2max}$ and enhances aerobic performance (3 mile run). Selected hematological parameters remain normal at all times.

Key Words: blood loading, reinfusion, erythrocythemia, oxygen capacity

In the study of physiological limits to physical work capacities and performance, researchers have tested the efficacy of various protocols for induced erythrocythemia, also known as blood doping, blood boosting, and blood loading (Gledhill 1982; Gledhill 1985; Hodgdon and Campbell 1982; Sawka et al. 1987b; Williams 1981). Early studies (Ekblom 1972; Ekblom 1976; Gullbring 1960; Robinson et al. 1966; Williams et al. 1978) produced mixed results, depending on the duration of the postinfusion recovery period, volume of blood infused, fitness level of subjects, and experimental design. Subsequent studies indicated that a successful blood-loading protocol requires the individual to undergo one or more phlebotomies to collect a minimum of 2 units (900 mL) of whole blood. The red cells are then separated and stored using the high glycerol freezing technique (Valeri 1976). Six or more weeks later, when the hematocrit (Hct) has returned to normocythemia, the individual receives an autologous infusion of 2-3 units of resuspended red blood cells (RBC). This procedure increases the oxygen (O_2) carrying capacity of the blood and produces an overnight increase in maximal aerobic power (VO_{2max}) of 4-12% (Buick et al. 1980; Celsing et al. 1987; Ekblom et al. 1976; Muza et al. 1987; Robertson et al. 1984; Sawka et al. 1987b; Spreit et al. 1986). This protocol also has increased performance of short, exhaustive treadmill tests (Buick et al. 1980; Robertson et al. 1982), an intense cycle ergometer test (Robertson et al. 1984), a 5-mile treadmill run (Williams et al. 1981), and a 10-km track race (Brien and Simon 1987).

Possible explanations for the ergogenic effect of blood loading on $\dot{V}O_{2max}$ include greater arteriovenous O_2 difference, increased cardiac output, and increased O_2 delivery to the muscles (Spreit et al. 1986; Thomson et al. 1982). The physical capacities of a given individual will determine the primary limiting factor.

Blood loading has been defined as an ergogenic aid, banned by the International Olympic Committee, and declared unethical for competitors by the American College of Sports Medicine (1987). However, the inability to unequivocally detect athletes who are blood loaded, using routine hematological measures (Berglund et al. 1989; Birkeland and Hemmersbach 1999), makes its use a matter of personal integrity. Few studies have measured both performance and physiological parameters under double-blind, crossover design conditions (Brien and Simon 1987; Buick et al. 1980; Williams et al. 1981). The present double-blind, crossover design study used trained, male runners to test the effect of blood loading on $\dot{V}O_{2max}$, ventilatory threshold (VT), and aerobic performance (a 3-mile track run, 3MT). Our second objective was to determine if blood loading produced hematological values (i.e., Hct, hemoglobin ([Hb]), erythropoietin, reticulocyte index), outside the normal range for this population, that could be used to identify blood-loaded individuals.

METHODS

Subjects.

Six male, trained distance runners, actively engaged in competitive distance running for several years, were recruited for this study (Table 1). Subjects gave

their informed written consent to participate in a study approved by the San Diego State University Committee for Protection of Human Subjects. Subjects' body fat was estimated using the 7-site skinfold thickness method (Jackson and Pollock 1978).

Experimental Design.

The subjects underwent two phlebotomies (450 mL each) spaced 8 wk apart. Erythrocytes were separated from whole blood and stored using the high glycerol freezing technique (Valeri 1976). After the second phlebotomy, subjects continued training 60-100 miles per week for approximately 4 months before receiving the first reinfusion treatment. Blood samples were collected from an antecubital vein weekly and before and after each phlebotomy, reinfusion, and treadmill test. These data were used to establish baseline values for each subject and ensure normocythemia before phlebotomies and reinfusion treatments. Blood samples were analyzed for [Hb], Hct, ATP, 2,3-DPG, lactate ([HLa]), P-50, erythropoietin concentration, and reticulocyte count. Blood samples were taken immediately (30-90 s) after completing each treadmill test, described below, to determine the circulating [HLa] at the point of volitional exhaustion (peak lactate).

Subjects received two infusion treatments spaced 7 days apart. One treatment (BT) was an infusion of 760 mL resuspended RBCs at a Hct of 43.5% (i.e., 330 mL RBC). The other treatment (ST) was a sham infusion of 250 mL isotonic saline. To prevent subjects from knowing which treatment was being administered, they were blindfolded and listened to music through headsets during

equivalent duration infusions. To create a double-blind, counterbalanced study, the subjects were divided into two groups of three; Group 1 received the ST prior to the BT and Group 2 received the treatments in the reverse order. Researchers involved with tests and analyses were blinded to the treatment assignments. One of the investigators (NC), assigned treatments, maintained the codes, scheduled subjects for all tests and procedures, but was not involved with tests or analyses.

Metabolic Measures.

Aerobic capacity. Subjects performed a series of four treadmill tests to determine their $\dot{V}O_{2max}$. The first test took place 1 day before the first infusion treatment, the second occurred 24 h after the first infusion, the third 24 h after the second infusion, and the fourth approximately 4 wk after the last infusion. Subjects performed a standard 7-min warm-up followed by a 10-min rest period prior to each treadmill test. Tests began at a velocity of 200 m·min and increased 10 m·min until the subjects reached a velocity of 340 m·min. At that point, the treadmill speed was held constant and the grade increased 1.5% per minute until subjects reached volitional exhaustion. $\dot{V}O_{2max}$ was calculated as the average of the two highest consecutive 30-s measures of $\dot{V}O_2$.

The metabolic measures were made using a system similar to that described by Wilmore and Costill (1974). $\dot{V}O_2$ was measured using open-circuit spirometry employing an Applied Electrochemistry model S-3a oxygen analyzer and a Beckman model LB-2 carbon dioxide analyzer. Expired gas was collected using a Daniels (1971) valve and minute ventilation ($\dot{V}_E$) measured by a

Parkinson-Cowan model CD-4 dry gas meter. Data were collected for fractional expired O_2 (FeO_2), fractional expired carbon dioxide ($FeCO_2$), $\dot{V}_E$, and HR every 30 s. Gas analyzers were calibrated before and after each test using room air and a standard gas of known O_2 and CO_2 concentrations as determined by the Micro-Scholander method.

Ventilatory threshold (VT). VT was used as an indirect indicator of the exercise-related increase in [HLa]. The VTs were determined from the treadmill tests by plotting the gas exchange variables; $\dot{V}_E$, ventilatory equivalent for O_2 ($\dot{V}_E/\dot{V}O_2$), FeO_2, $FeCO_2$, and CO_2 production ($\dot{V}CO_2$) against time. The VT was described as the running speed and $\dot{V}O_2$ at the time of departure from linearity for $\dot{V}_E$, $\dot{V}CO_2$, and a systematic change in $\dot{V}_E/\dot{V}O_2$ and $FeCO_2$ accompanied by a lack of change in $\dot{V}_E/\dot{V}CO_2$ and $FeCO_2$, respectively (Davis et al. 1979). The points of departure for $\dot{V}_E$ and $\dot{V}_E/\dot{V}O_2$ from linearity were used as the primary criteria of VT. However, when the criteria did not agree, the VT was based on the averages for the VT points determined from the plots of gas exchange variables.

Hematological Measures.

The time course of changes in a variety of blood parameters that may be altered in response to increased red cell mass were monitored (Hct, [Hb], ATP, 2,3-DPG, P-50, MCV, MCH, MCHC, RBC count, WBC count, reticulocyte count, [HLa], and erythropoietin). Hct was determined by the microhematocrit centrifuge method (corrected for trapped plasma). Hct and [Hb] values are reported as the mean of duplicate determinations. The [Hb] was determined by the

cyanmethemoglobin technique (Sigma kits No. 525 and No. 525-18, St. Louis, MO). O_2 carrying capacity, was calculated using the formula (O_2 mL = [Hb] x 1.34 mL O_2). Oxygen carrying capacity was compared before and after infusion treatments. The 2,3-DPG levels were determined using the method described by Lowry (Lowry et al. 1964), using Sigma kits No. 35-UV and No. 366-UV. ATP was determined using Bucher's phosphorylation reaction coupled with dephosphorylation/oxidation reaction modified by Adams et al. (1967). [HLa] was measured using Sigma kits No. 826-UV and 826-10, as described by Henry (1968). A Hem-o-scan O_2 dissociation analyzer (American Instrument Co., Silver Spring, MD) was used to determine P-50 values. Reticulocyte counts were determined from blood smears using the methylene blue histological staining method, and a corrected reticulocyte count was calculated according to Rapaport (Rapaport 1971). A subsample of plasma was analyzed for erythropoietin by a local clinical laboratory.

Aerobic Performance Test.

For an aerobic performance measure, subjects ran three solo 3MTs on a standard 440-yd outdoor track. The only performance feedback given was the time to complete the first 440 yd of each 3MT. Subjects were not informed of their performance times for the 3MT until the end of the study. An observer, blinded to the treatments, recorded the time for each of the 12 laps and noted the wind speed, temperature, and cloud cover. The first run took place 3 days prior to the administration of the first infusion to establish a baseline. The second run took

place 3 days after the first infusion, and the third occurred 3 days after the second infusion.

Statistical Analyses.

Standard descriptive statistics (mean ± SD) were performed on metabolic and performance variables. Changes in dependent measures from baseline were assessed by an analysis of variance (ANOVA), using a within-subjects, repeated-measures design. When ANOVA revealed significant F-ratios, paired *t*-tests were used to identify differences. Because of the small sample size, the Wilcoxon Signed Ranks Test was used to test for significance in $\dot{V}O_{2max}$ and performance runs. Statistical significance was set at $P \leq 0.05$.

RESULTS

Metabolic Measures.

Aerobic capacity. Results of a maximal treadmill test for both treatments are listed in Table 2. Table 3 shows the acute effects of both experimental treatments on each individual. The data indicate a significant increase in $\dot{V}O_{2max}$ ($P < 0.002$) and Hct ($P < 0.001$), after the BT. $\dot{V}O_{2max}$ increased by 0.46 ± 0.20 L · min^{-1} (or 11.9 ± 6.2%) after BT. There were no significant correlations between absolute or percent increase in $\dot{V}O_{2max}$ and the age, initial $\dot{V}O_{2max}$, or 3MT performance of the subjects after BT.

Ventilatory threshold. VT, expressed as $\%\dot{V}O_{2max}$, decreased after BT by 0.7 ± 5.7% and ST by 2.1 ± 3.9%, but neither was significant (Table 3). However,

after BT, the absolute $\dot{V}O_2$ at VT increased significantly by 0.36 ± 0.23 L O_2 · min^{-1}, approximately 80% of the total amount (0.46 L O_2 · min^{-1}) that $\dot{V}O_{2max}$ was increased. Likewise, the percentage increase in $\dot{V}O_2$ at VT (10.9%) approximated the increase in $\dot{V}O_{2max}$ (11.9%). Thus, BT did not affect the $\%\dot{V}O_{2max}$ at the VT but did increase the absolute amount of L O_2 · min^{-1} at VT. As with $\dot{V}O_{2max}$, there was no correlation between the magnitude of increase in VT and the increase in aerobic performance on the 3MT.

Hematological data. Hct and [Hb] values were marginally, but not significantly, elevated ($\bar{x}$ = 0.27 ± 0.28), 24 h after BT. However, 7 days after BT, Hct and [Hb] had increased significantly ($P < 0.001$) by 4.7 ± 1.3% and 5.8 ± 1.7%, respectively. The unexpected small increase in Hct (1.2 ± 1.3) observed 24 h after BT did not allow for the clear separation of the effects on $\dot{V}O_{2max}$ of increased blood volume from increased red cell mass and oxygen carrying capacity (i.e., increased [HB]). Four weeks after BT, the Hct averaged 41.7 ± 1.3%, or 6.9% above baseline (Figure 1). ST produced an immediate decrease in Hct ($\bar{x}$ = 2.4 ± 2.1), but it returned to pre-infusion values 24 h later. Corrected reticulocyte counts were significantly ($P < 0.05$) depressed ($\bar{x}$ = 0.28 ± 0.2%) 1-2 wk after BT, but were within normal limits (0.5 ± 0.2%) at all other times. There were no other significant changes in RBC parameters (MCV, MCH, MCHC, and P-50) or erythropoietin after either treatment.

During the first 24 h postinfusion, the calculated oxygen carrying capacity increased from 18.6 to 19.7 mL O_2·dL^{-1} as a result of the increased [Hb]. Seven

days later, the [Hb], and hence oxygen carrying capacity, had increased by 10% over baseline (20.5 mL $O_2 \cdot dL^{-1}$) and was unchanged 4 wk later (19.9 ± 0.48 mL $O_2 \cdot dL^{-1}$).

Blood lactate. Resting lactate levels were significantly lower than baseline (1.0 vs 0.7 $mmol \cdot L^{-1}$) during the week after BT, suggesting greater tissue perfusion at rest (Figure 2). Peak [HLa] after all maximal treadmill tests averaged 8.0 ± 2.0 $mmol \cdot L^{-1}$ and did not differ significantly between treatments and baseline (Figure 3).

Aerobic Performance.

Time to perform the 3MT decreased significantly ($P < 0.001$) by 19.8 ± 16.0 s after BT but was unchanged after ST (Tables 3 and 4). This represents an average improvement of 2.0 ± 1.6% in running performance. Compared with the baseline run, the time to complete each of the 3 miles after BT was faster by 0.34 ± 3.2% for mile one, 1.6 ± 1.2% for mile two, and 3.6 ± 1.9% for mile three (Figure 4). During treadmill running at 265 m·min, the mean HR decreased nonsignificantly by 7.7 ± 6.9 and 4.2 ± 3.0 bpm, after BT and ST, respectively.

DISCUSSION

To our knowledge, this is the first double-blind, crossover design blood-loading study to document significant improvements in $\dot{V}O_{2max}$ and aerobic performance of trained runners in a non-competitive field setting. Previous studies have relied upon a variety of physiological, hematological, aerobic capacity measures, and laboratory performance tests (i.e., treadmill or cycle ergometer)

while at their VT to determine the efficacy of various reinfusion protocols. Only one study (Brien and Simon 1987), included a track race as a field measure of aerobic performance. However, the results of that study may have been influenced by the fact that the participants competed against each other during the performance tests and were familiar with each other's performance ability. To avoid this potential influence, the subjects in our study ran the performance tests individually.

Even though most blood-loading studies have demonstrated increases in $\dot{V}O_{2max}$ and performance in the laboratory, the transfer of these benefits to field settings has not been well documented. In this study, we demonstrated that infusing 760 mL of autologous, resuspended RBC (Hct = 43.5%) into trained distance runners significantly increased $\dot{V}O_{2max}$ by 11.9 ± 6.3% (0.46 ± 0.20 L O_2 min^{-1}) compared to baseline. This degree of improvement in $\dot{V}O_{2max}$ was at the high end of the range (4-13%) reported by others (American College of Sports Medicine 1987; Gledhill 1985; Sawka et al. 1987b). Additionally, we found that $\dot{V}O_2$ at VT increased by 78% of the increase in $\dot{V}O_{2max}$. However, when VT was expressed as $\%\dot{V}O_{2max}$, it was unchanged. This suggests that blood loading increases the absolute $\dot{V}O_2$ but not the relative $\dot{V}O_2$ (i.e., $\%\dot{V}O_{2max}$) at an individual's VT. To achieve increases in $\%\dot{V}O_{2max}$ at VT requires metabolic adaptations derived from weeks of exercise training near the VT (Keith et al. 1992). We found only one study reporting the effects of blood loading on VT or related measures of [HLa] accumulation. In that study, trained runners were

infused with 3-5 units of blood and exhibited a small but significant increase in running velocity (0.3 km·hr) at an [HLa] of 4 mmol·L^{-1} (Celsing et al. 1987). The VO_2 at this running velocity increased only an average of 100 mL, or 24% of the total increase in VO_{2max}. In contrast, the trained runners in our study running at VT used 360 mL or 78% of their total increase in VO_{2max}. This difference may result from our using VT, an indirect measure of the lactate threshold, instead of [HLa]. Also, it is possible that the percentage of the increase in VO_{2max} resulting from blood loading, which can be used in a performance event, may vary, depending on the individual's training, physiological state, and genetics. More studies are needed to determine the relationship between enhancement of VO_{2max} and its relative effect on performance at the anaerobic threshold (i.e., VT, lactate threshold).

Muza et al. (1987) found no increase in VO_{2max} of a 43 year old male after blood loading and speculated age may be a significant factor. Our study found no support for this hypothesis. We found no significant correlations between changes in any hematological parameter, aerobic capacity, or performance and the subjects' age or initial VO_{2max}. In fact, our oldest subject, a 41-year old, demonstrated the second largest increase (16.6%) in VO_{2max} and a 2.0% increase in 3MT performance (group mean = 2.0 ± 1.6%). An analysis of the data from a blood-loading study by Brien and Simon (1987) also indicated no significant correlation between age and improvement in 10-k race performance of six trained runners. The oldest subject in their study, also a 41-year old male, exhibited a 3.8% improvement in $VO_{2\,max}$ compared with the group mean of 3.4 ± 0.8%. Therefore,

we find no evidence nor physiological reason to suggest age ($\leq$ 43 yr) as a factor affecting an individual's response to blood loading.

The 2.0% increase (Hopkins et al. 1999) in performance of our subjects during the solo 3MT (7 $s \cdot mile^{-1}$ or 4.35 $s \cdot km^{-1}$) is sufficient to provide a competitive advantage to an elite athlete. This small but significant improvement in performance is more than enough to affect the placing of top athletes in most competitive aerobic sports. We also found that the subjects ran significantly ($P < 0.01$) faster during their third and final mile of the 3MT than in mile one or two (Figure 4). This strategy does not produce record performances; therefore, we suggest that even pacing would have produced faster times. It may be that the subjects maintained suboptimal performance during the first 2 miles because they were uncertain of the level of effort they could maintain for the 3MT. We speculate that during the final mile, they discovered that increased effort was possible without inducing fatigue. An analysis of the data from two other blood-loading studies that measured running performance revealed the same pattern (Brien and Simon 1987; Williams et al. 1981). Trained runners in both of these studies also ran the last mile significantly faster than the other miles. These observations suggest that training at race pace under blood-loaded conditions may be necessary to achieve maximal performance benefits of this procedure.

Resting Hcts after the blood infusion suggest that a transient increase in total blood volume occurred, and that it persisted at least 24 h. During the first 24-h postinfusion, the oxygen carrying capacity increased 5.8%, but 7 days later it

was 10% above baseline. The increase in $\dot{V}O_{2max}$ after blood infusion occurred prior to reaching the maximum Hct and oxygen carrying capacity. The unexpected small increase in Hct (mean = 1.2 ± 1.3%), observed 24 h after blood infusion, does not allow for the clear separation of the effects of increased blood volume from increased red cell mass and oxygen carrying capacity. The model of O_2 transport proposed by Warren and Cureton (1989) predicts changes in $\dot{V}O_{2max}$ when [Hb] is altered, and it includes a separate contribution of blood volume. Their model assumes $\dot{V}O_{2max}$ is limited by maximal cardiac output and not the pulmonary system as hypothesized for elite athletes (Dempsey 1986). This model predicts the reinfusion of 760 mL of resuspended RBC would increase $\dot{V}O_{2max}$ by 15%, which is in close agreement with the 11.9 ± 6.3% observed in our subjects. The increase in $\dot{V}O_{2max}$ may result from a combination of a short-term (24-48 h) increase in blood volume and a more persistent (4 wk) increase in oxygen carrying capacity. Additionally, we found no change in P-50 or 2,3-DPG of RBCs, indicating no change in the O_2 dissociation curve and [Hb] affinity for O_2, which is in agreement with others (Brien and Simon 1987; Buick et al. 1980; Williams et al. 1978).

It may be significant that the subject (No. 2) with the smallest increase in $\dot{V}O_{2max}$ (0.16 L $O_2 \cdot min^{-1}$ or 3.6%) also had the lowest resting and maximum HR (33 and 165 bpm, respectively). The small increase in $\dot{V}O_{2max}$ may be explained by the findings of Buick et al. (1980), who concluded that increases in $\dot{V}O_{2max}$ from blood loading resulted primarily from increased stroke volume (SV). Our subject

with the smallest response and lowest maximum HR may have had a very large SV initially to achieve the high cardiac output required for his baseline $\dot{V}O_{2max}$ of 68.9 mL $O_2 \cdot kg^{-1} \cdot min^{-1}$. We speculate that since his initial SV was quite large, his heart may have had limited reserve capacity.

The values for selected hematological parameters (Hct, [Hb], [Hla], ATP, 2,3-DPG, p-50, MCV, MCH, MCHC, RBCs, WBCs, erythropoietin, and reticulocyte index) of blood loaded subjects never fell outside of the normal range. Berglund et al. (1989) attempts to detect blood loading measuring serum iron, bilirubin, and ferritin before and after autologous blood reinfusions and detected only 50% of those who received blood. It appears that even with a set of established baseline values of hematological parameters, the detection probabilities are too low to be an effective control for blood loading with autologous reinfusions (Birkeland and Hemmersbach 1999). Given the ethical and legal issues associated with requiring blood sampling of athletes, and the significant number of false positive and false negatives, blood loading may continue until improved methods become available.

In conclusion, it appears that, in well-trained runners, the limiting factor for $\dot{V}O_{2max}$ is the cardiovascular system and its delivery of O_2 to the muscles. However, the limiting factor for any given individual will depend on his level of fitness, physiological, and anatomical capacities. In the elite athlete, $\dot{V}O_{2max}$ may ultimately be limited by the pulmonary system (Dempsey 1986), while in trained endurance athletes, it may be the cardiovascular system (oxygen carrying capacity

and/or cardiac output). In untrained individuals, the limiting factor may be the aerobic capacity of the muscular system (i.e., capillary density, mitochondrial density, and aerobic enzyme concentration). Future research should be designed to characterize the underlying mechanisms, limiting factors, time course of physiological adaptations, and conditions for optimal performance associated with blood loading. Better methods of detecting blood loaded athletes must be developed to deter its potential abuse in sport and destruction of fair competition.

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Table 1. Characteristics of Subjects

	Subject #	Age (years)	Wt. (kg)	Skinfold body fat (%)	VO_{2max} (L•min•kg)	Hct %	Previous Best Marathon
Group 1	1	27	64.8	5.5	68.3 (4.43 L)	39.5	2:26
	2	33	68.9	6.6	64.5 (4.45 L)	39.0	2:30
	3	41	62.5	7.6	52.5 (3.26 L)	37.5	2:44
Group 2	4	36	63.3	8.9	69.1 (4.38 L)	39.0	2:35
	5	33	65.8	7.2	54.0 (3.53 L)	39.5	2:36
	6	39	84.5	10.1	54.0 (4.57 L)	43.0	3:14
	$\bar{x}$	34.8	68.3	7.7	60.4	39.6	2:40
	SD ±	5.0	8.2	1.6	7.7 (4.10 L)	1.8	0:17

Table 2. Results of Infusion Treatments on Aerobic Capacity and Ventilatory Threshold

Aerobic Capacity (VO_{2max})								
Subject	Baseline		24-h post-blood		24-h post-saline		4 wk post-blood	
#	$l\ O_2 \cdot min^{-1}$	ml•kg•min	$l\ O_2 \cdot min^{-1}$	ml•kg•min	$l\ O_2 \cdot min^{-1}$	ml•kg•min	$l\ O_2 \cdot min^{-1}$	ml•kg•min
1	4.43	68.3	4.95	78.7	4.48	70.0	4.11	63.8
2	4.45	64.5	4.61	66.9	4.49	65.6	4.47	63.7
3	3.26	52.5	3.80	59.6	3.33	51.9	3.59	56.8
4	4.38	69.1	4.87	74.8	4.42	70.6	3.93	61.9
5	3.53	54.0	4.27	60.5	4.23	62.8	4.00	59.9
6	4.57	54.0	4.89	57.3	5.01	60.2	4.86	58.3
Mean	4.10	60.4	4.56	66.3	4.33	63.5	4.16	60.7
SD ±	0.56	7.7	0.45	8.8	0.55	7.0	0.44	2.9

Ventilatory Threshold (VT)								
Subject	Baseline		24-h post-blood		24-h post-saline		4 wk post-blood	
#	$l \cdot min^{-1}$	$\%VO_{2max}$	$l \cdot min^{-1}$	$\%VO_{2max}$	$l \cdot min^{-1}$	$\%VO_{2max}$	$l \cdot min^{-1}$	$\%VO_{2max}$
1	3.55	80.1	4.28	82.3	3.73	83.3	3.07	74.7
2	3.42	76.9	3.85	83.6	3.41	76.1	3.72	83.6
3	2.75	84.0	2.88	75.9	2.59	77.8	2.81	78.3
4	3.48	79.4	3.95	81.1	3.20	72.4	2.79	71.0
5	3.02	85.6	3.24	78.7	3.60	85.1	2.89	72.2
6	3.45	75.5	3.60	75.5	3.73	74.4	4.03	82.3
mean	3.28	80.25	3.63	79.5	3.38	78.2	3.22	77.0
SD±	0.32	3.9	0.51	3.4	0.44	5.02	0.53	5.2

Table 3. Acute Effects of Saline and Blood Infusions on Physiology and Performance

A. Saline Infusion

	Ventilatory Threshold (▲ VT)		Aerobic Capacity (▲ VO_{2max})		Hct (▲)	[Hb] (▲)	Aerobic Performance (▲ 3-Mile Run Time)	
Subject #	l O_2•min^{-1}	%VO_{2max}	l O_2•min^{-1}	% ▲	% units	mg/L	Total time (s)	% ▲
1	+0.18	+3.2	+0.05	+1.1	+1.0		+13.0	+1.48
2	-0.01	-0.8	+0.04	+0.9	0.0		+6.0	+0.63
3	-0.16	-6.2	+0.07	+2.1	0.0		+20.2	+2.02
4	-0.75	-7.0	-0.45	-9.2	-0.5		-17.0	-1.75
5	+0.36	-0.5	-0.04	-0.9	+1.8		-17.1	-1.72
6	+0.13	-1.1	+0.12	+2.5	-3.2		-2.1	-0.19
mean	-0.04	-2.1	-0.04	-0.6	-0.15		+0.5	+0.08
SD ±	0.39	3.9	0.21	4.4	1.7		15.5	1.6

B. Blood Infusion

	Ventilatory Threshold (▲ VT)		Aerobic Capacity (▲ VO_{2max})		Hct (▲)	[Hb] (▲)	Aerobic Performance (▲ 3-Mile Run Time)	
Subject #	l O_2•min^{-1}	%VO_{2max}	l O_2•min^{-1}	% ▲	% units	mg/L	Total time (s)	% ▲
1	+0.73	+2.2	+0.52	+11.7	+5.0	+1.3	-21	-2.31
2	+0.43	+6.7	+0.16	+3.6	+5.8	-0.6	-25	-2.63
3	+0.13	-8.1	+0.54	+16.6	+6.3	+0.7	-36	-3.60
4	+0.47	+1.7	+0.49	+11.2	+3.5	+0.4	-26	-2.67
5	+0.22	-6.9	+0.74	+21.0	+2.8	0.0	+11	+1.1
6	+0.15	0.0	+0.32	+7.0	+4.8	-0.2	-22	-1.96
mean	+0.36	-0.7	+0.46	+11.9	+4.7		-20.2	-2.0
SD ±	0.23	5.7	0.2	6.3	1.3		6.2	0.52

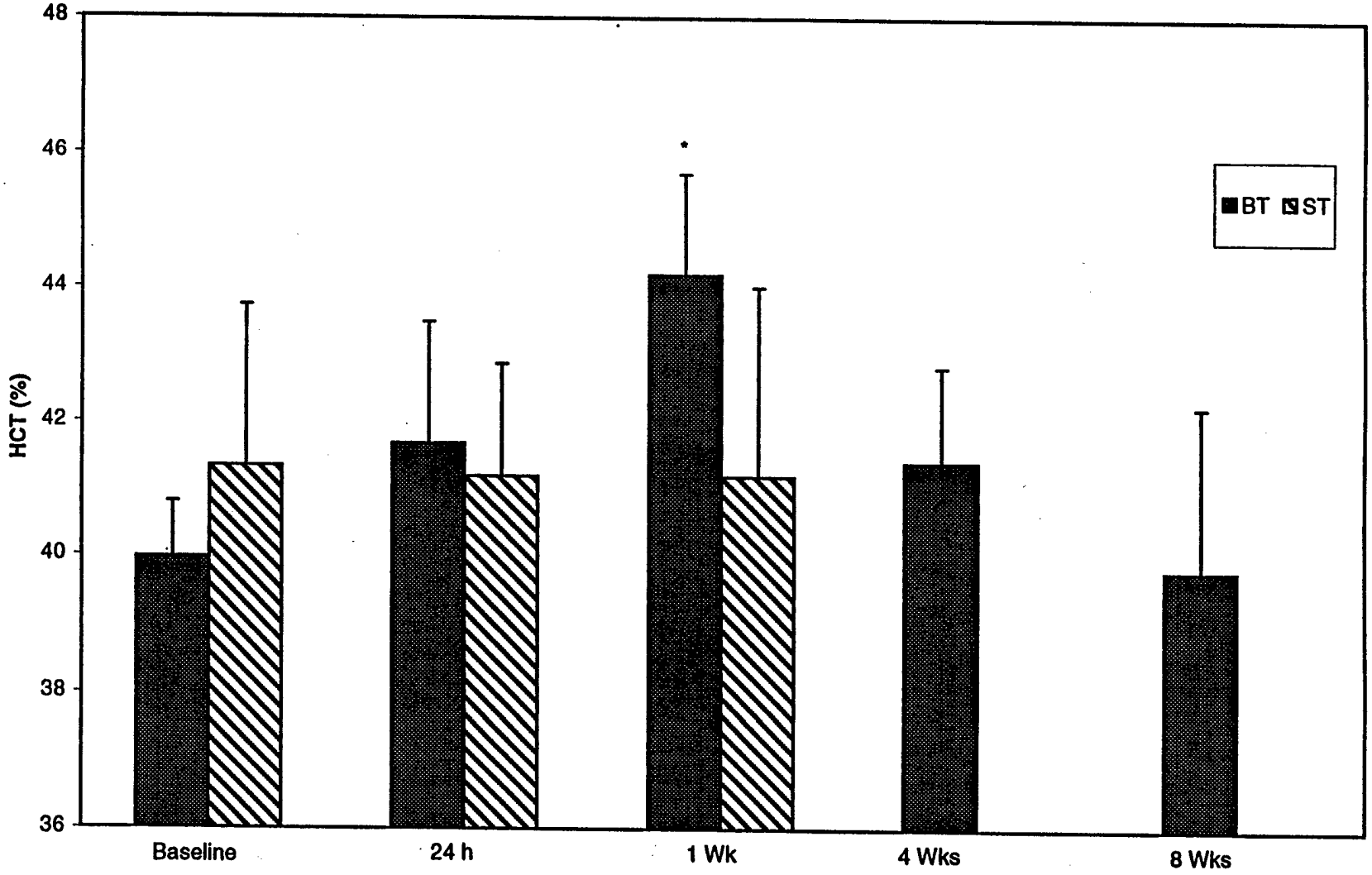

Figure 1. Effects of Infusion Treatments Upon Hematocrit. Values are mean ± SE; BT=6, ST=6; * significant difference from baseline at $p < 0.05$.

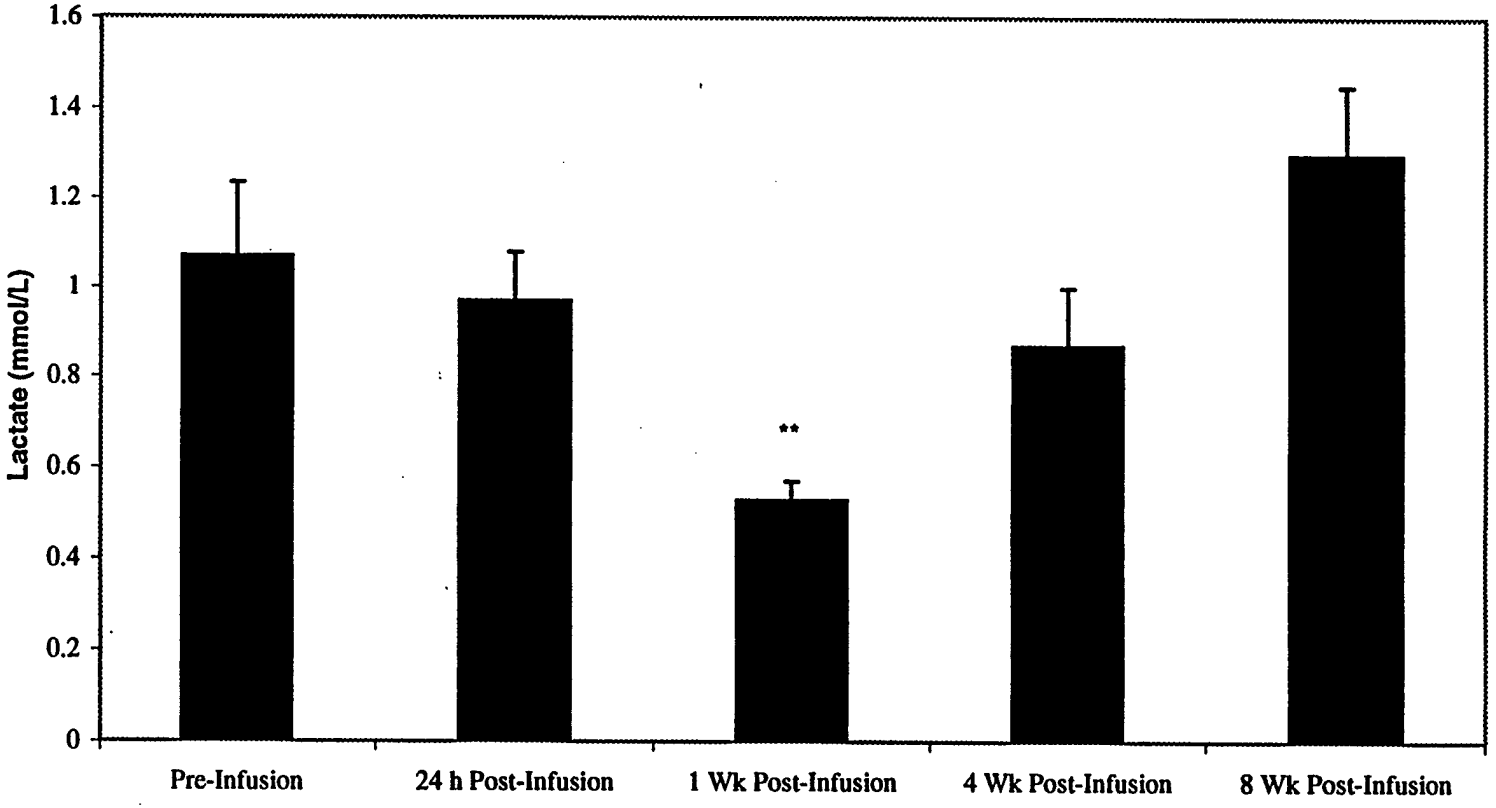

Figure 2. Effect of Blood Infusion Upon Resting Plasma Lactate Concentrations. Values are mean ± SE; BT=6; ** significant difference from baseline at $p < 0.01$.

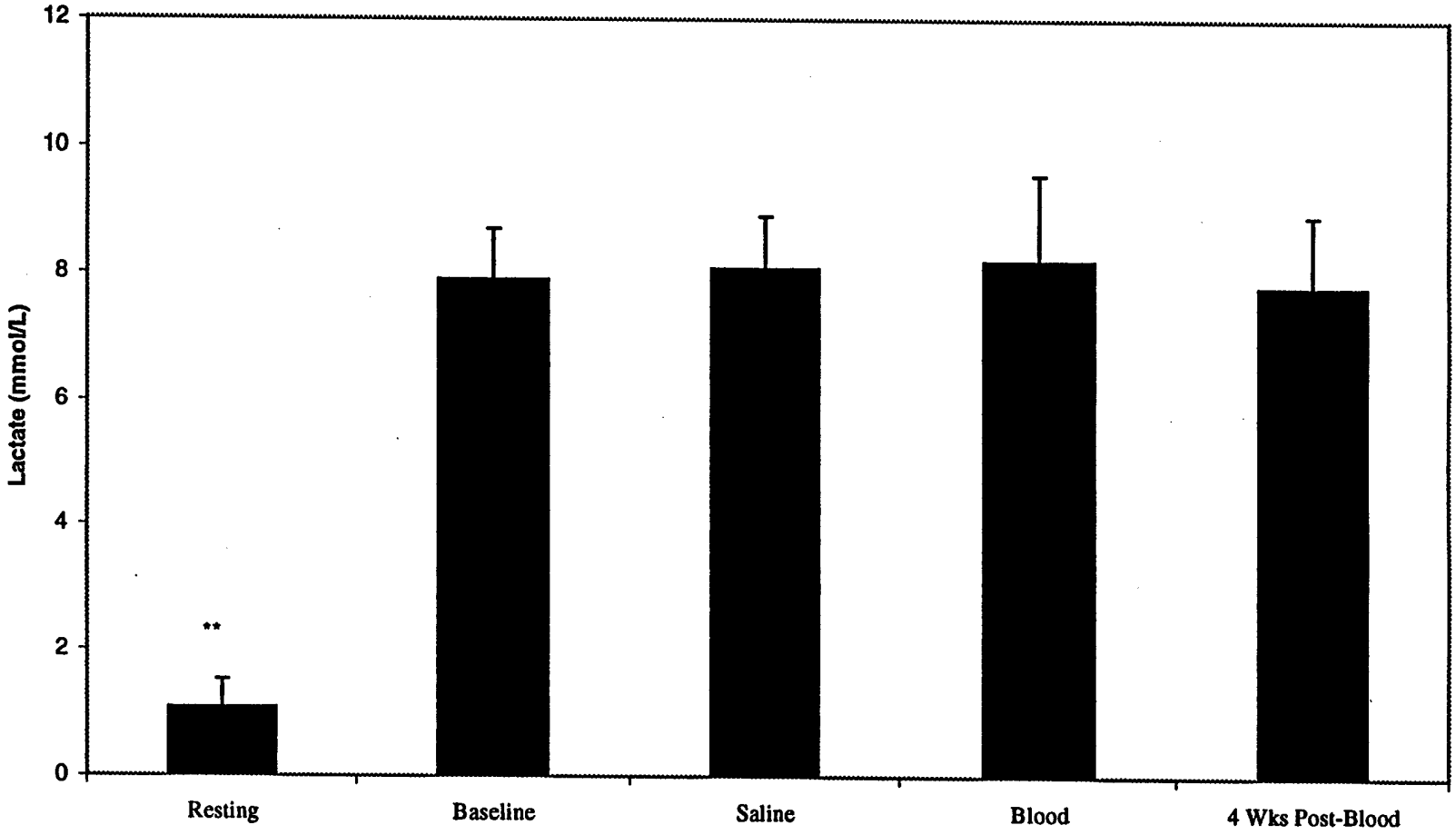

Figure 3. Effect of Infusions on Peak Plasma Lactate Concentrations After Maximal Treadmil Exercise. Values are mean $\pm$ SE; BT=6, ST=6; ** significant difference from baseline at $p < 0.01$.

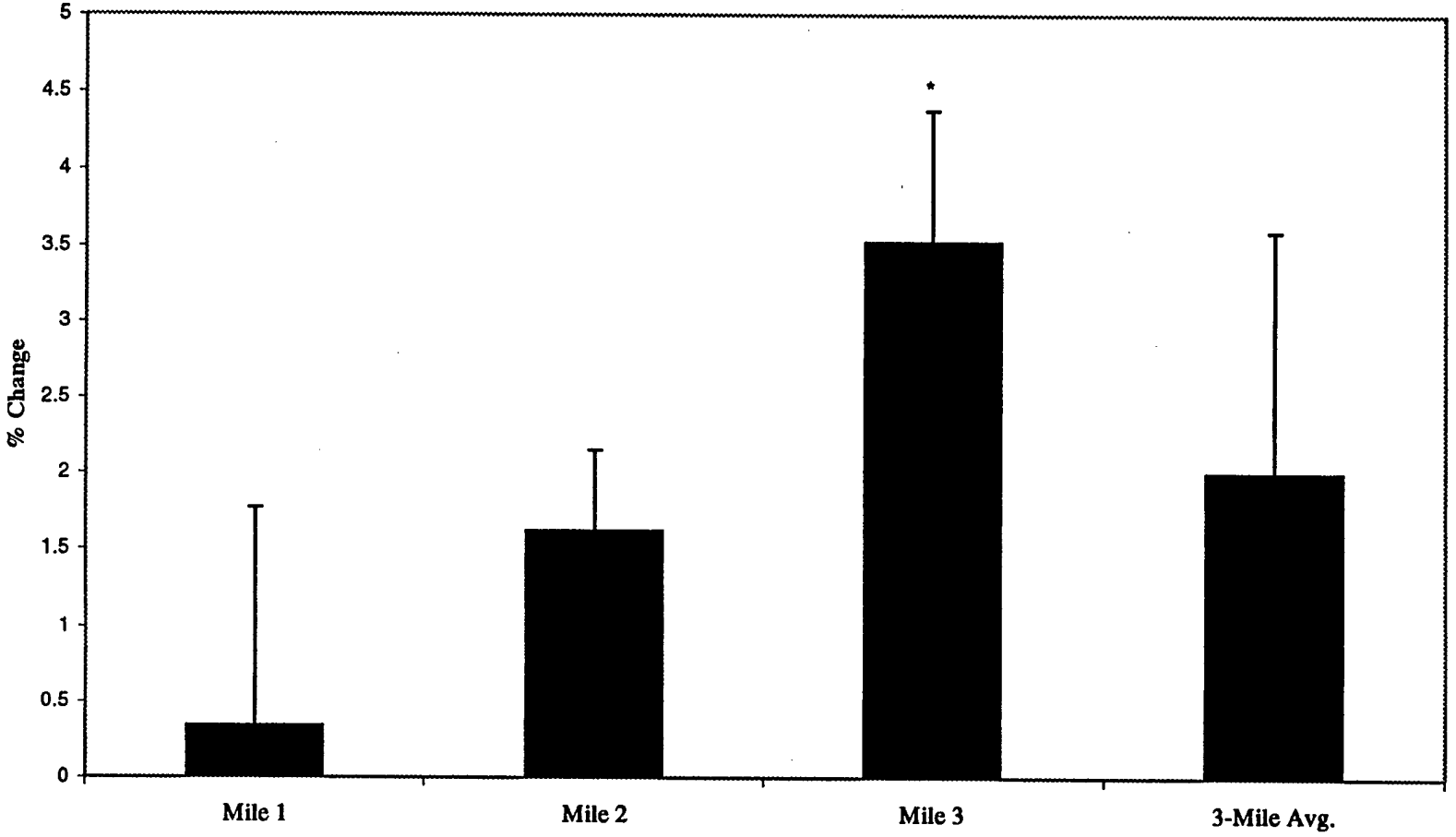

Figure 4. Percent Change in Velocity During 3-Mile Track Run After Blood Infusion. Values are mean ± SE; n = 5; * significant difference from other miles at $p < 0.05$.

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13. ABSTRACT *(Maximum 200 words)* **Purpose:** Document the effects of induced erythrocythemia upon aerobic capacity (VO_{2max}), ventilatory threshold (VT), and aerobic performance (3-mile track run, 3MT). **Methods:** Six trained male distance runners (age = 34.8 ± 5 yr, hematocrit [Hct] = 39.6 ± 1.8%, VO_{2max} = 4.10 ± 0.56 L $O_2 \cdot min^{-1}$), received two infusion treatments in a double-blind, counterbalanced study. Treatments: (BT) 760 mL autologous resuspended red blood cells (Hct = 43.5%) and (ST) 250 mL isotonic saline were administered 7 days apart. **Results:** BT significantly ($P < 0.01$) increased VO_{2max} (0.46 ± 0.2 L $O_2 \cdot min^{-1}$), VT (0.36 ± 0.23 L $O_2 \cdot min^{-1}$), and significantly ($P < 0.05$) decreased 3MT time (19.8 ± 16.0 s or 2.0 ± 1.6%). Blood lactate after treadmill tests (8.0 ± 2.0 mmol·L^{-1}) were unchanged by BT but were significantly ($P < 0.01$) lower at rest. Hct was unchanged 24 h following BT, but a wk later had increased significantly ($P < 0.05$) to 44.0%. The delayed increase in Hct suggests an initial increase in blood volume may have contributed to the increase in VO_{2max} at 24 h. ST had no significant effects on any measure. Hct and VO_{2max} were not different from baseline ± wk after BT. Blood parameters did not change significantly ($P > 0.05$) after BT; ATP, 2,3-DPG, p-50, MCV, MCH, MCHC, RBCs, and WBCs. Reticulocytes were depressed significantly ($P < 0.05$) (0.28 ± 0.2%) 2 wk after BT, but were normal (0.5 ± 0.2%) all other times. **Conclusion:** Blood loading increases VO_{2max} and enhances aerobic performance (3 mile run). Selected hematological parameters remain normal at all times.

14. SUBJECT TERMS
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