Footstrike is the major cause of hemolysis during running

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00631.2001.—There is a wide body of literature reporting red
cell hemolysis as occurring after various forms of exercise.
Whereas the trauma associated with footstrike is thought to
be the major cause of hemolysis after running, its signifi-
cance compared with hemolysis that results from other cir-
culatory stresses on the red blood cell has not been thoroughly
addressed. To investigate the significance of footstrike, we mea-
sured the degree of hemolysis after 1 h of running. To control for
the potential effects of oxidative and circulatory stresses on the
red blood cell, the same subjects cycled for 1 h at equivalent
oxygen uptake. Our subjects were 10 male triathletes, who each
completed two separate 1-h sessions of running and cycling at
75% peak oxygen uptake, which were performed in random
order 1 wk apart. Plasma free hemoglobin and serum haptoglo-
bin concentrations were measured as indicators of hemolysis.
We also measured methemoglobin as a percentage of total
hemoglobin immediately postexercise as an indicator of red cell
oxidative stress. Plasma free hemoglobin increased after both
running ($P < 0.01$) and cycling ($P < 0.01$), but the increase was
fourfold greater after running ($P < 0.01$). This was reflected by
a significant fall in haptoglobin 1 h after the running trials,
whereas no significant changes occurred after cycling at any
sample point. Methemoglobin increased twofold after both run-
ning and cycling ($P < 0.01$), with no significant differences
between modes of exercise. The present data indicate that,
whereas general circulatory trauma to the red blood cells asso-
ciated with 1 h of exercise at 75% maximal oxygen uptake may
result in some exercise-induced hemolysis, footstrike is the
major contributor to hemolysis during running.

erythrocytes; cycling; haptoglobin; methemoglobin; iron status

EXERCISE-INDUCED HEMOLYSIS has been reported for more
than 50 years (11). In particular, distance running has
been associated with significant destruction of red
blood cells (RBC) with RBC turnover being substan-
tially higher in runners compared with untrained con-

trols (29). Several groups have suggested that mechan-
ical damage to RBC occurs as they pass through the
capillaries of the foot during the footstrike phase (5, 8,
10, 20, 29). However, studies on athletes involved in
sports in which foot impact does not occur have also
found evidence of exercise-induced hemolysis. These
activities include swimming (25), weight lifting (2, 24),
and rowing (9). Given that factors other than footstrike
cause hemolysis during exercise, these variables as
causes of hemolysis during running cannot be elimi-
nated. Thus the contribution of footstrike per se to
running-induced hemolysis is unclear.

Apart from footstrike, several other mechanisms
may contribute to hemolysis during exercise. Because
of their continuous exposure to high-oxygen flux, RBC
are extremely vulnerable to oxidative damage (3, 26).
Under normal conditions, the superoxide radical is
generated from the autooxidation of oxyhemoglobin to
methemoglobin in RBC at a rate of 3%/day (17). Be-
cause superoxide generation appears to be propor-
tional to oxygen flux (26), oxidative stress may in turn
increase in proportion to the oxygen uptake ($\dot{V}O_2$) as-
sociated with exercise. Oxidative stress has been im-
plicated in the normal "aging" of RBC (3), and there are
numerous reports of increases in "footprints" of oxida-
tive damage and antioxidant depletion in RBC after
exercise (26). Perturbation of osmotic homeostasis is
another potential contributor to exercise-induced he-
molysis. Because exercise-induced changes in RBC
density (16) and mean cell volume (13) have been
reported, these osmotic changes may render the RBC
more susceptible to membrane damage and hemolysis,
especially during their passage through the microcir-
culation where cells undergo a constant swelling and
shrinking cycle. Finally, it is also possible that com-
pression of large muscle groups on capillaries may
accelerate hemolysis of older RBC (18).

The aim of this study was to test the hypothesis that
footstrike is the major cause of hemolysis during run-

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ning. To measure the effect of footstrike, it was necessary to control other potential hemolytic factors, namely oxidative and other stresses imposed in transit through the circulation. To do this, we have compared the hemolytic responses in athletes as they ran and cycled at equivalent metabolic loads, with the main difference and aspect of interest being in the uniqueness of footstrike to the running medium.

METHODS

Subjects. Ten male triathletes volunteered for the study. The physical, hematological, and training characteristics of the subjects are presented in Table 1. RBC concentration, hematocrit, and hemoglobin concentration were measured before the exercise tests, and all individuals' values fell within the normal reference ranges. The study was approved by the Ethics Committee of the Australian Institute of Sport, and subjects gave written, informed consent.

Exercise trials. Each subject attended the laboratory on three separate occasions. First, a graded running test to exhaustion on the treadmill (AusTredEx, Preston, Victoria, Australia) was used to determine peak $\dot{V}O_2$ ($\dot{V}O_{2\ peak}$). An initial speed of 9 km/h at 0% gradient was increased 2 km/h every minute to 15 km/h followed by a 1% increase in gradient every minute until the individual reached volitional exhaustion. A Hans Rudolph 2700 valve (Hans Rudolph, Kansas City, MO) was used, and volume of inspired air was measured by a Morgan Ventilometer (PK Morgan, Gillingham, Kent, UK). Samples of inspired air were drawn continuously through a dessicant (calcium chloride) and into Applied Electrochemistry oxygen and carbon dioxide analyzers (AEI Technologies, Pittsburgh, PA). Pre- and posttest calibration of the ventilometer and gas analyzers occurred by using a Tissot spirometer (Warren E. Collins, Braintree, MA) and gravimetric standard gases (BOC Gases, Wetherill Park, NSW, Australia). Heart rates were recorded via ECG transmitted to a Quinton Instruments (Bothell, WA) ECG data computer. $\dot{V}O_{2\ peak}$ was designated as the highest rate of oxygen consumption recorded in a 30-s period, and every subject exceeded a respiratory exchange ratio of 1.10. The cycling ergometry was performed by using a Repco airbraked ergometer (Repco Cycle, Mulgrave, Victoria, Australia) modified for racing cycling body positioning and pedals.

The standardized running and cycling trials began ~1 wk later. Each subject completed two 1-h exercise bouts (running

and cycling) at 75% of their $\dot{V}O_{2\ peak}$; one-half of the group was randomly selected to perform the cycle test first, thus preventing any bias due to order. The alternative exercise bout was performed 1 wk later. Whereas individual athletes varied in their weekly training programs, each was careful not to vary their own training in the weeks leading up to their cycling and running trials. A further condition was that no training was carried out 24 h before testing. Athletes were also instructed to consume the same preexercise diet on the day of both of their trials. All subjects wore medium-weight training shoes of a similar type, with all shoes being in good condition. The warm-up was consistent and consisted of 10 min of jogging at ~14 km/h or cycling at ~150 W followed by four efforts of 30 s at the speed or power of their 1-h following effort. The speed of the treadmill required to elicit 75% $\dot{V}O_{2\ peak}$ was estimated from the graded treadmill test, and the cycle ergometer power required was estimated from tables of cycling oxygen cost (1). Our aim was to elicit very similar oxygen consumption in the 1-h period for each mode of exercise on an individual basis to closely approximate potential oxidative and circulatory stress to the RBC. $\dot{V}O_2$ was measured after 5 min of exercise for a duration of 5 min, after which adjustments were made if necessary to the speed or power. The $\dot{V}O_2$ was then checked again in the following 5 min and adjusted again if necessary to produce the designated 75% $\dot{V}O_{2\ peak}$. Measurement of oxygen consumption was repeated at the 20- and 40-min marks of exercise. Trials with several subjects had indicated that this method successfully elicits the required $\dot{V}O_2$ values using each mode of exercise. In addition, heart rates were measured via telemetry (Sports Tester PE 4000, Polarelectro). To verify that the hemolysis found after running and cycling was caused by exercise and not due to temporal variation or damage during blood collection, methemoglobin, free hemoglobin, and haptoglobin were also analyzed in blood samples collected from a nonexercising group over the same time course as the exercising group. As we were interested in controlling any potential temporal effect and the handling of blood samples, we did not need to involve the triathletes in further blood sampling, and we recruited a separate group of five subjects [mean age 29.8 ± 8.2 (SD) yr, height 179.6 ± 5.8 cm, and weight 76.2 ± 8.9 kg], all moderately active and of similar age to the triathletes.

Blood collection and measurements. Blood samples were collected before and immediately after each running and cycling trial and at 1, 6, and 24 h postexercise. At each sampling point, blood was collected via venipuncture of the antecubital vein, with the athlete lying down. To control for plasma volume shifts due to postural change, sampling after the running and the cycling tests was carried out in identical fashion, with the athletes in a supine position. Extreme care was taken during collection of blood to avoid the possibility of hemolysis: a 21-gauge needle was used together with minimal stasis and the gentle movement of the syringe's plunger. Blood was gently expelled down the side of three separate collection tubes, which had the vacuum seals removed. Three milliliters of blood were collected in an EDTA tube for a full blood count. Blood to be used for plasma free hemoglobin determination was collected in a 5-ml heparin tube. Plasma was prepared by gentle centrifugation at 2,000 rpm for 20 min. The plasma was then removed and centrifuged at 11,000 g for 2 min. Three milliliters of blood were transferred into a serum tube and allowed to clot at room temperature. The blood was then centrifuged at 3,000 rpm for 10 min. Serum was removed and frozen at −20°C for haptoglobin determination. One milliliter of blood was collected into a

Table 1. *Characteristics of the 10 male triathletes*

Characteristic	Mean	SD
Age, yr	27.8	6.6
Height, cm	181.5	4.2
Weight, kg	74.7	7.4
$\dot{V}O_{2peak}$, ml·kg^{-1}·min^{-1}	64.5	5.2
Hematological measures		
RBC, $10^6/\mu l$	5.11	0.25
Hematocrit, %	45.3	1.9
Hemoglobin concentration, g/dl	15.3	0.9
Plasma ferritin, μg/l	71.8	13.9
Training volume, km/wk		
Run	64.8	12.6
Cycle	180	106
Swim	7.2	2.9

The blood measurements shown were taken at rest, just before the treadmill test to measure the peak oxygen uptake ($\dot{V}O_{2peak}$) on the treadmill. RBC, red blood cell count.

heparin syringe for methemoglobin determination, an indicator of oxidative stress to the RBC.

Plasma free hemoglobin was assessed spectrophotometrically by the cyanomethemoglobin method (21). Serum haptoglobin concentrations were determined in the clinical pathology laboratory of the Canberra Hospital by using rate nephelometry (Array 360, Beckman Coulter, Miami, FL). RBC count, hematocrit, and hemoglobin concentration were measured by using a semiautomated 5550 Coulter Counter (Beckman Coulter). Blood lactate concentration was determined by using the YSI model L-lactate analyzer (Yellow Springs Instrument). Methemoglobin was measured by using a 270 Corning CO-oximeter (Ciba Corning Diagnostics, Medfield, MA) into which 125 µl of blood from a heparinized syringe were injected within 2 min of sampling. In this machine, dilution, calibration, and calculation of results, using five measurements per sample, are performed automatically under microprocessor control. This instrument detects the optical absorbance of each of the hemoglobin fractions (oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin) from which it calculates the values of interest to our study (total hemoglobin and methemoglobin as a fraction of total hemoglobin). Plasma ferritin was measured as previously described (27).

Statistical analysis. The statistical analysis involves a comparison between profiles of repeated measurements (15) obtained in the running and cycling trials. The overall observed significance level is determined by undertaking an appropriate repeated-measures analysis of variance on the pooled data. Mean profiles are shown graphically, and variability between means is represented by least significant difference (LSD). (LSD is $2 \times$ standard error, and any pair of means differing by more than the LSD are significantly different at $P = 0.05$). The statistical package Genstat (2000; 5th edition; VSN International, Oxford, UK) was employed for statistical computation.

RESULTS

No significant differences were found in $\dot{V}O_2$ at any of the equivalent sampling points, which, over all of the measurements, averaged 48.5 ± 3.1 (SE) ml·kg^{-1}·min^{-1} for running and 47.6 ± 2.3 ml·kg^{-1}·min^{-1} for cycling. Similarly, no significant differences were found in postexercise blood lactate concentration, which averaged 3.4 ± 0.3 mM for the running and 3.4 ± 0.2 mM for cycling exercise trials, indicating that potential oxidative and circulatory stress on the RBC was similar for each mode of exercise. In other words, these potential hemolytic effects on the RBC were well controlled. Heart rates averaged 164 ± 3.2 and 161 ± 2.6 beats/min for the running and cycling trials, respectively, with negligible drift and no significant difference between the profiles ($P = 0.81$). There was no significant difference between the running and cycling tests in methemoglobin formation, although there was a twofold increase ($P < 0.05$) in methemoglobin accumulation in both modes of exercise, which was maximal immediately after the 1-h effort (Fig. 1). These values fell progressively back to preexercise levels 24 h after both exercise tests. No significant differences ($P > 0.05$) occurred in any of the hematological variables (hematocrit, RBC concentration, and hemoglobin concentration) before and after each of the exercise trials. However, we did note small, but nonsignificant, in-
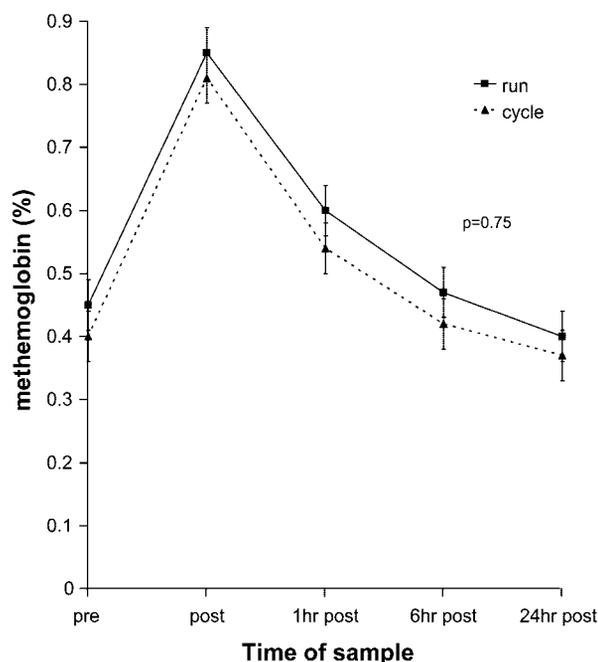

Fig. 1. Comparison of methemoglobin concentration, expressed as a percentage of the hemoglobin concentration, at various stages before (pre) and after (post) running and cycling for 1 h at 75% peak oxygen uptake ($\dot{V}O_{2\,peak}$). Values are means ± SE; $n = 10$ subjects. Statistical comparison of the profiles of the running and cycling data indicated no significant differences existed ($P = 0.75$).

creases in the means of hematocrit, RBC concentration, and hemoglobin concentration immediately after both forms of exercise followed by small, nonsignificant reductions in these variables at the 1-h sampling point (data not shown). Most relevant to this investigation was that the statistical comparison of profiles of the means of each hematological variable for cycling and running was not significantly different (P values of 0.29, 0.48, and 0.37, respectively). Consequently, we were able to assume that, even if a small change in plasma volume had occurred, the similarity of such occurrence for each mode of exercise would indicate that any such change would have no impact on the interpretation of our comparative results.

Although plasma free hemoglobin concentration (an indication of hemolysis) increased significantly ($P < 0.05$) after both running and cycling, the mean increase was fourfold greater ($P < 0.05$) after running (Fig. 2). At 1 h postexercise, mean free hemoglobin was not significantly different from the preexercise mean for the cycling and running trials.

The greater increase in plasma free hemoglobin concentration immediately after running was subsequently reflected by a greater decrease in haptoglobin concentration. Haptoglobin concentration reached its lowest point 1 h after running (Fig. 3), and analysis of the integrated profiles indicated a significant difference between running and cycling ($P = 0.014$). The average change in haptoglobin concentration between preexercise and 1-h postexercise samples was 0.016 g/l for cycling and 0.085 g/l for running.
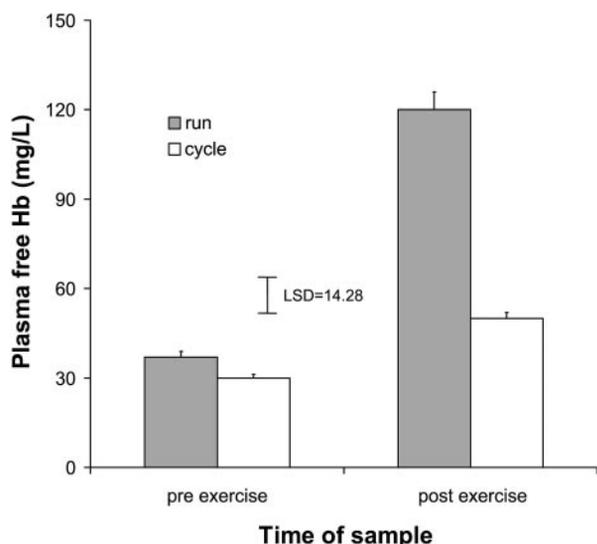
Fig. 2. Comparison of plasma free hemoglobin (in mg/l) before and after running and cycling for 1 h at 75% V̇O$_{2\,peak}$. Values are means ± SE; n = 10 subjects. Least significant difference (LSD) is equivalent to twice the SE of the differences. Any pair of means differing by more than the LSD is significantly different at P = 0.05.

The investigation of temporal variation in the blood samples taken from the nonexercising group (n = 5) revealed no significant differences ($P > 0.05$) in any hemolytic variable in the 2-h measurement period (Table 2), indicating that the previously demonstrated significant changes in this time period were exercise related.

DISCUSSION

Compared with cycling, greater increases in plasma free hemoglobin concentrations and decreases in serum haptoglobin concentrations were found after the
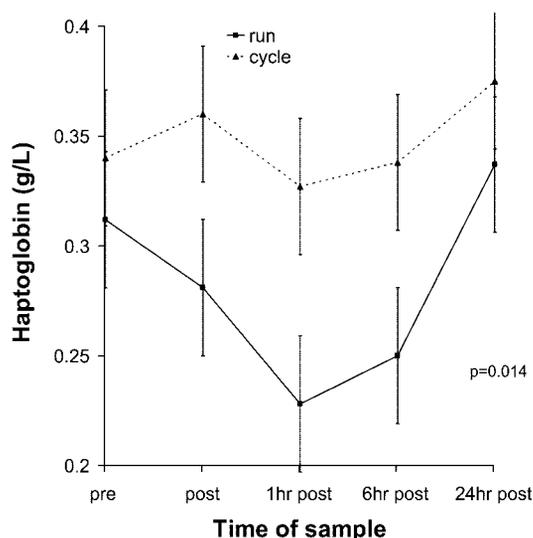

Fig. 3. Comparison of plasma haptoglobin concentration (in g/l) before and after running and cycling for 1 h at 75% V̇O$_{2\,peak}$. Values are means ± SE; n = 10 subjects. A statistical comparison of the profiles of the running and cycling data revealed a significant difference at P = 0.014.

Table 2. *The variation of blood measurements in samples taken 1 h apart in nonexercised subjects*

Variable	Sample 1	Sample 2	Sample 3	Change
Lactate, mM	0.88 ± 0.03	0.90 ± 0.02	0.86 ± 0.02	0.02 ± 0.01
Methemoglobin, %	0.34 ± 0.01	0.35 ± 0.05	0.37 ± 0.03	0.038 ± 0.02
Plasma hemoglobin, mg/l	13.6 ± 3.7	14.4 ± 2.4	15.9 ± 4.5	3.2 ± 1.9
Haptoglobin, g/l	0.68 ± 0.2	0.70 ± 0.2	0.70 ± 0.2	0.1 ± 0.01

Values are means ± SD. There were no significant differences ($P > 0.05$) in the means of any one variable at any of the sample points.

running trial. Because foot impact is the major difference of relevance between running and cycling, these results provide solid evidence that mechanical trauma to RBC during footstrike is the factor responsible for the greater hemolysis found after running. Because there were no significant differences between the running and cycling trials in the means of measures of V̇O$_2$, heart rate, and lactate accumulation, it is highly unlikely that variance in general circulatory stresses to the RBC can explain the differences. Of interest is a previous finding that shoe type may be an influencing factor in RBC damage because reticulocyte levels were 29% higher in runners training with hard-soled shoes after 18 days of running that covered 429 km, compared with a matched control group that trained with soft-soled shoes (7). The present study, in confirming that footstrike is in fact the major cause of the hemolysis, is consistent with finding of increased erythropoiesis after the endurance running with less cushioning in the shoes.

Many reports have appeared describing sports anemia in athletes (13, 19, 28), with most cases apparently due to an expanded plasma volume and hemodilution (pseudoanemia) rather than true RBC deficiency. However, it has been reported that reinfused autologous ^{51}Cr-labeled RBC survive for an average of only 74 days in runners compared with 114 days in sedentary controls (29), indicating that running reduces RBC life span, which is consistent with a higher rate of RBC destruction and increased reticulocytosis (23). This study confirms that the stress of footstrike has a significant influence on the life span of runners' RBC and might be suspected to increase the susceptibility of those involved in running sports to anemia should responsive erythropoeitic mechanisms be inadequate. However, Green et al. (12) have reported that total body RBC volumes do not differ between trained runners and cyclists. This suggests, in general, that, despite a greater rate of RBC destruction in runners, replacement keeps pace and thus prevents anemia.

Although most iron is salvaged from senescent or damaged RBC and reutilized under normal conditions (6), these conservation mechanisms may fail when subjected to the stress of intensive training. For example, should blood haptoglobin stores be overwhelmed or incompletely replenished during the recovery period, excess hemoglobin will be excreted or broken down. Although a single hemolytic episode is unlikely to cause iron loss of clinical significance (20), daily or

twice-daily hemolytic episodes during hard training may have a cumulative effect that might be significant; athletes such as marathoners with high training volumes would be particularly at high risk. Several studies have indicated that distance runners have compromised iron stores (4, 8, 14, 22). In some cases, iron supplementation is required to prevent or correct anemia. The suggestion that the mechanical trauma of footstrike may be associated with iron deficiency in runners is consistent with evidence that athletes who participate in other foot-impact sports such as basketball and tennis have lower iron stores than cyclists and rowers (27), even after correction for body size and gender.

This present study indicates that hemolysis occurred for reasons other than footstrike, with a small but significant increase in plasma free hemoglobin concentration evident after the cycling trial. This increase was considerably less than that related to running. In fact, there was not a sufficient release of hemoglobin during the cycling to significantly reduce serum haptoglobin concentration at the time points measured. Another possible cause of hemolysis that we have suggested previously to be common to running and cycling is oxidative stress. The small increase in methemoglobin that we observed after running was also found to an equivalent degree after cycling, indicating that the hour of exercise did to some extent increase oxidation of hemoglobin. Taking this into account, together with the finding of only a minor amount of hemolysis resulting from cycling compared with running, we can deduce that oxidative influences alone have little effect, if any, on the hemolysis associated with running.

In conclusion, in controlling for the potential influences of oxidative and circulatory challenges to the RBC, we have demonstrated that the mechanical trauma associated with footstrike is the major cause of hemolysis during running.

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