The Effects of Caffeine on the Maximal Accumulated Oxygen Deficit and Short-Term Running Performance

Michael Doherty

The purpose of this study was to evaluate the effect of acute caffeine ingestion on the maximal accumulated oxygen deficit (MAOD) and short-term running performance. Nine well-trained males performed a preliminary assessment and, at least 4 days later, a supramaximal run to exhaustion. Their VO\textsubscript{max} values were determined, and the MAOD test at an exercise intensity equivalent to 125% VO\textsubscript{max} was performed. Caffeine (5 mg · kg\textsuperscript{-1}) or placebo was administered 1 hr prior to the MAOD in a double-blind, randomized cross-over study. In comparison to the placebo condition, subjects in the caffeine condition developed a significantly greater MAOD and increased their run time to exhaustion. However, post-MAOD blood lactate concentration ([HL\textsubscript{a}]) was not different between trials for caffeine and placebo. Caffeine ingestion can be an effective ergogenic aid for short-term, supramaximal running performance and can increase MAOD. However, these results do not appear to be related to an increased [HL\textsubscript{a}].

Key Words: short-term high-intensity exercise, anaerobic, supramaximal, ergogenic, blood lactate

Because of athletes’ use of caffeine and reports of its ergogenic properties, a number of governing bodies of sport have included caffeine on their list of banned substances. In recent years, the effect of caffeine on endurance performance has been of particular interest, as witnessed by a number of reviews of its physiological and metabolic effects (6, 7, 11, 22, 27, 32, 36). However, information concerning short-term (2–5 min), high-intensity (>100% VO\textsubscript{max}) exercise (STHIX) has been mostly neglected and in some cases dismissed. For example, Williams (38) concluded that the use of caffeine to improve performance in activities requiring strength and short-term endurance appears unwarranted. Tarnopolsky (32) stated it is unlikely that caffeine is ergogenic when used during exercise so intense that it lasts less than 15 min. Vandenberghhe et al. (34) claimed, “The weight of evidence today indicates that caffeine has no direct effect on high-intensity exercise performance.” However, a number of well-designed studies have indicated that caffeine may indeed aid performance in athletes who participate in STHIX (1, 5, 15, 37). In recent

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reviews of caffeine and performance, Graham et al. (11) and Spriet (27) concluded that caffeine can enhance performance of STHIX lasting ~5 min.

Short-term, high-intensity exercise is synonymous with the measurement of anaerobic capacity, which has been defined as the maximal amount of adenosine triphosphate (ATP) that can be resynthesized via anaerobic metabolism during a specific mode of short-duration maximal exercise (13). This exercise intensity requires maximal provision of energy from both aerobic and anaerobic sources (28). Despite agreement among coaches and sport scientists that the ability to produce energy during STHIX is highly dependent on anaerobic energy production, relatively little information exists on the contribution of anaerobic metabolism to athletic success. This shortfall is mostly due to methodological constraints.

To accurately assess the anaerobic capacity of a muscle group during STHIX, measurements of anaerobic substrates (ATP, creatine phosphate, and carbohydrates), intermediates, and the products of anaerobic pathways are required from muscle biopsies obtained before and after an exhausting work bout (28). The total amount of lactate that has escaped the active muscle must also be estimated using arterial and venous catheters and blood flow measurements (28). Due to the invasiveness of this approach, more practical techniques for indirectly estimating anaerobic capacity during STHIX have been developed. These anaerobic "work" tests are mostly derived from ergometric assessments, for example, the Wingate anaerobic test (12). However, the validity of these tests has been questioned on a number of counts, including, for example, their inability to quantify the contribution of aerobic energy supply to work output (10, 12, 13).

It is generally agreed that the maximal accumulated oxygen deficit (MAOD), while still requiring further examination (2, 10, 13), is a unique and reproducible physiological characteristic (23) that represents the most accurate indirect, noninvasive determination of ATP production from anaerobic metabolism during STHIX (10, 13, 25, 28). MAOD is the difference between the predicted energy (or O$_2$ demand) and O$_2$ uptake that accumulates during STHIX (21). It requires a supramaximal effort at a predetermined exercise intensity of between 110% and 150% VO$_{max}$, and because of this the test duration normally lasts 1 to 5 min. Whether MAOD accurately quantifies anaerobic processes remains open (2), but there is little doubt that it does represent some measure of anaerobic performance. The MAOD is higher in anaerobically trained subjects compared to endurance-trained or sedentary subjects (21), and it is also sensitive to anaerobic training (20, 31). The MAOD remains unchanged during hypoxic exercise (21) and does not change with exercise durations of 2 to 16 min (19). Finally, the MAOD is related to several other measures of anaerobic performance (10, 13). Only one investigation of the effects of caffeine on MAOD has been reported, and this was performed with cycling exercise (16). Thus, the aim of the present study was to assess the acute effects of caffeine ingestion on MAOD and short-term running performance. It was hypothesized that caffeine would increase MAOD and improve run time to exhaustion.

Methods

Subjects

Nine male subjects (24.0 ± 3.2 years old [mean ± SD], 1.79 ± 0.07 m in height, 76.7 ± 6.4 kg in weight) volunteered to participate in this study. All subjects were active athletes (VO$_{max}$ = 60.0 ± 4.5 ml·kg$^{-1}$·min$^{-1}$) participating in a variety of multiple-
sprint sports that required a combination of different training methods. The subjects were recruited on the basis of their fitness and running ability rather than for uniformity in their caffeine habits. As a result, their daily caffeine consumption ranged from 30 to 850 mg. All subjects were sport science students and were familiar with treadmill running and all data collection procedures.

**General Procedures**

The nature, purpose, experimental procedures, and possible risks and benefits were outlined for each subject both verbally and in writing. The procedures were approved by a departmental committee for ethics in research, and all subjects provided written informed consent.

The subjects were tested on three separate occasions, consisting of one preliminary test session and two supramaximal test sessions. The tests were scheduled at the same time of day for each subject with a minimum of 4 days separating test sessions. The subjects were instructed to refrain from physical activity, to avoid caffeine in the 24 hr prior to the tests, and to present themselves at the laboratory in a 4-hr fasted state. One hour prior to the supramaximal test, subjects consumed one of two beverages: (a) 5 mg · kg⁻¹ of caffeine (Roche, Welwyn Garden City, UK) in 200 ml of an artificially sweetened citrus drink (caffeine), or (b) 200 ml of artificially sweetened drink (placebo). Caffeine and placebo beverages were assigned in a random and double-blind manner. Subjects drank the assigned beverage within 2 min and then relaxed for 1 hr prior to the supramaximal warm-up.

**Preliminary Test Session**

Supramaximal energy expenditure was determined using an extrapolation method adapted from Procedure 3 of Medbo et al. (21). In order to relate oxygen uptake ($\dot{V}O_{2}$) to running velocity, each subject performed three 6-min treadmill (PowerJog, Cranlea & Co., UK) runs of increasing exercise intensity ranging between 80% and 90% $\dot{V}O_{2max}$ with 10-min rest periods intervening. All tests were conducted on a 10.5% incline (20). Respiratory gases (Douglas bag, open circuit spirometry), heart rate (beats · min⁻¹; SportsTester, Polar Electro, Finland), and rating of perceived exertion (RPE; 6- to 20-point Borg scale) were recorded during the final minute of each run. Immediately following the third and final run, the treadmill belt was increased by 0.14 m · s⁻¹ and thereafter each minute until volitional exhaustion. This took on average another 3 to 6 min of running. During these final minutes, both respiratory gas (for subsequent determination of $\dot{V}O_{2max}$) and heart rate data were collected continuously. Attainment criteria for $\dot{V}O_{2max}$ included any two of the following (14):

- An increase in $\dot{V}O_{2}$ with successive exercise intensities of less than 2 ml
- A respiratory exchange ratio (RER) of $\geq 1.10$
- A maximum heart rate of $220 - \text{age} \pm 10$ beats · min⁻¹

Based on the relationship between $\dot{V}O_{2}$ and running velocity, an individual linear regression equation was derived for each subject, which was used to calculate the running velocity at an exercise intensity equivalent to 125% $\dot{V}O_{2max}$. A preexercise oxygen uptake of 5 ml · kg⁻¹ · min⁻¹ (i.e., oxygen uptake at zero treadmill velocity) was assumed to be common for each subject and was used as the intercept in the regression equation (21).
Supramaximal Test

The supramaximal test was preceded by a 6-min warm-up performed at the second treadmill velocity that was used in the preliminary test session (equivalent to ~85% VO$_{2\text{max}}$). The subject then performed a set of stretching exercises, followed by a series of 4- to 8-s bouts of treadmill running at the predetermined supramaximal velocity.

Prior to each supramaximal test, subjects were instructed to continue running until they could no longer maintain the set pace. They were informed that no encouragement or feedback would be given to them during the test. The treadmill belt was then adjusted to the predetermined velocity, and when the subjects were ready, they lowered themselves onto the moving treadmill belt and a digital stop clock was started (start of exercise). Simultaneously, collection of the subjects’ expired air commenced. When subjects could no longer keep pace with the velocity of the treadmill belt, they held onto the handrails, at which point expired air collection was terminated and the treadmill and clock were stopped. The difference between the estimated oxygen demand (determined in the preliminary test session) and the measured oxygen uptake of the supramaximal run (i.e., MAOD) was measured over the whole exercise period. The MAOD in this study was not adjusted for the contribution of the body’s oxygen stores to the energy supply. This contribution has been estimated to be ~10% of MAOD (25).

Five minutes following the end of the test, an arterialized capillary blood sample (50 μl) was taken from an earlobe for subsequent determination of blood lactate concentrations ([HLa]; Analox LM5, Hammersmith, UK).

Respiratory Gas Analysis

In all tests, subjects breathed through a low-resistance nonreturn valve (Hans Rudolph #2700) and were connected to a series of 200-L Douglas bags by means of short, lightweight tubing. One liter of air from each Douglas bag was drawn off to determine fractions of oxygen and carbon dioxide using a paramagnetic oxygen analyzer (Servomex 570A) and an infrared carbon dioxide analyzer (Servomex PA404), both of which had previously been calibrated against gases of known concentration (British Oxygen Co., UK). The remaining volume of each sample was then measured with a Harvard dry-gas meter (Cranlea, Birmingham, UK) that had been calibrated against a Tissot spirometer.

Statistics

Linear regression and Pearson product moment correlation coefficients were used to determine the relationships between measured variables. A series of paired t tests were used to identify differences between caffeine and placebo. An alpha level of .05 was chosen to indicate significant difference. All results are expressed as mean ± SE. All statistical procedures were performed using SPSS-X PC.

Results

Preliminary Test

The procedure used to calculate the supramaximal exercise intensity of 125% VO$_{2\text{max}}$ required a high degree of linearity between VO$_2$ and exercise intensity, and it required
that the submaximal exercise be at least 80% VO$_2$ max (19, 21). In addition, since the VO$_2$ max was determined following three bouts of exercise, it was necessary to verify that maximal values had been achieved. The results showed that all criteria were met. The VO$_2$-exercise intensity relationship determined from the Y-intercept, the three 6-min submaximal runs, and the VO$_2$ max had an $R^2$ of .92 ± .02, and values for the three submaximal runs represented approximately 80%, 86%, and 90% VO$_2$ max (Table 1). The results also showed that all subjects attained at least two of the VO$_2$ max criteria (see the Methods section and Table 1).

**Supramaximal Test**

The ingestion of 5 mg of caffeine per kilogram of body mass 1 hr prior to the supramaximal run resulted in significantly greater MAOD (5.89 ± 0.4 vs. 5.30 ± 0.4 L O$_2$ equivalents for caffeine and placebo respectively; Figure 1) and run time to exhaustion (208.2 ± 13.6 vs. 181.0 ± 9.4 s for caffeine and placebo, respectively; Table 2). Values of MAOD and duration of run times for both placebo and caffeine trials fell within the range reported in previous studies that used a similar exercise intensity (13, 21, 23). In comparison to the placebo condition, all but 1 of the 9 subjects improved their MAOD and run time to exhaustion when consuming caffeine. The range of differences in the run time to exhaustion (i.e., caffeine minus placebo trial) was −3 s to 56 s, with an average improvement of 14% (i.e., 29 ± 6 s; Figure 2). Although MAOD and run time to exhaustion improved with caffeine, [HLa] was not different between trials (13.0 ± 0.7 and 11.9 ± 0.9 mMol for caffeine and placebo, respectively; Table 2). One significant correlation coefficient showed that MAOD was related to run time to exhaustion ($r = .72$), an association that has recently been employed as the basis for a MAOD field test (24).

**Discussion**

The main findings from this study were that both MAOD and time to exhaustion during short-term (i.e., ~3–4 min), supramaximal running performance were increased following caffeine ingestion (5 mg · kg$^{-1}$). The improvements in MAOD were of a similar magnitude to those reported following 6 weeks of aerobic training in recreationally active subjects (21). However, the improvements are lower

Table 1 Preliminary Test Results ($n = 9$)

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<td>0.6</td>
<td>15.1</td>
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than those observed in a well-trained group following anaerobic training (31). The findings are also in accord with the most recent investigations of the effects of caffeine on MAOD and STHIX. For example, in the only other reported study of the effects of caffeine on MAOD, Jacobs et al. (16) showed that caffeine (5 mg·kg⁻¹), in comparison to a combination of caffeine and ephedrine and a placebo trial, improved MAOD during cycle exercise. However, unlike the present study, the increase in MAOD was not accompanied by an increased time to exhaustion. Wiles et al. (37) found that 3 g of caffei-
Figure 2 — Differences in time to exhaustion between placebo and caffeine trials for each subject. Positive number indicates that time was greater in caffeine test. *Mean data for caffeine trial significantly greater (p < .05) than placebo data.

1,500 m in comparison to placebo. In the same study, a separate 1,500-m trial was performed at a predetermined speed and the run was completed with a final 1-min finishing burst at a speed determined by the athlete. Again, caffeine was shown to be ergogenic, resulting in a significantly faster finishing speed (i.e., 23.5 km/hr for caffeine compared with 22.9 km/hr for placebo). In addition, Jackman et al. (15) reported that caffeine ingestion (6 mg/kg) improved cycle time to exhaustion by 16% during STHIX exercise that lasted 4 to 6 min.

The energy required to cover the oxygen deficit during high-intensity exercise that lasts 1 to 5 min is derived mostly from anaerobic glycolysis (~80%) and the ATP and creatine phosphate stores (~16%; 25). Some investigations on the effects of caffeine ingestion (1, 4, 5), but not all (15, 37), have shown an associated elevation in [HLa] following STHIX. In addition, it is known that the release of epinephrine (Epi) from the adrenal medulla following caffeine ingestion augments the rise seen with exercise alone by a factor of two to three (11). Although the metabolic consequences of additional Epi caused by caffeine ingestion remain unclear (35), and the role of Epi in the activation of glycogenolysis during STHIX has not been studied systematically (3), it has been suggested that the elevation in [HLa] following caffeine ingestion is a result of an epinephrine-mediated increase in muscle glycogenolysis (1, 4, 5). Thus, if the MAOD is increased following caffeine ingestion as a direct result of an increase in anaerobic glycolysis, one might also expect to find an elevation in [HLa]. However, although MAOD and short-term running performance were improved in the present study by 10% and 14%, respectively, there was no accompanying rise in [HLa] following caffeine ingestion compared to placebo.

A limitation to the interpretation of the [HLa] found in vivo studies is that elevated lactate can reflect a change in clearance and/or production and release from
muscle (25). While studying muscle metabolite data, Jackman et al. (15) fixed the exercise intensity at 100% VO_{2}max during two 2-min cycling bouts interspersed with 6 min of rest following either caffeine or placebo trials. Muscle biopsies were taken and it was shown that muscle lactate concentrations, but not blood lactate concentrations, were elevated following the caffeine trial \( p < .05 \). It is possible that this also occurred in the present study. Finally, a recent report detailing the acute effects of caffeine ingestion in humans with impaired Epi responses (33) showed that caffeine directly stimulates specific tissues, that is, adipose and peripheral vascular tissue. These effects appear to be independent of Epi secretion.

In attempting to explain the improvements in STHIX following acute caffeine ingestion, most investigators point to the findings of in vitro and in situ studies which have clearly shown that caffeine potentiates muscle force production (7, 9, 11, 27, 38). Evidence for caffeine’s effect on central nervous system stimulation, enhanced neuromuscular transmission, and enhanced muscle fiber contractility is well documented (7, 38). The cellular mechanisms hypothesized to account for these changes include the direct effect of caffeine on calcium exchange by the sarcoplasmic reticulum (29, 30), inhibition of phosphodiesterase (8), and adenosine receptor antagonism (8, 39).

An experiment that could be explored in future in vivo studies to help explain caffeine’s effect on STHIX involves changes in electrolyte handling (in particular, potassium, \([K^+]\)) and glucose metabolism. For example, MacIntosh and Wright (18) observed higher postexercise blood glucose values in a group of swimmers whose 1,500-m swim times improved \( p < .05 \) following caffeine ingestion. The authors suggested that either the rate of glycogenolysis/gluconeogenesis was greater with caffeine present or a higher steady level blood glucose was maintained in the caffeine condition, or both. Loss of \( K^+ \) from skeletal muscle and its accumulation in extracellular spaces have been identified as possible mechanisms of fatigue (26). The increase in \([K^+]\) that is associated with exercise and fatigue may be attenuated by caffeine, possibly as a result of accelerated Na^+/K^-ATPase activity (17). MacIntosh and Wright (18) found lower preexercise plasma \([K^+]\) following caffeine ingestion in their swimming study. Although caffeine ingestion improved swim time over 1,500 m \( p < .05 \), there were no differences in \([K^+]\) at the end of the exercise bout. The authors suggested that the lower preexercise \([K^+]\) concentration permitted a higher intensity of exercise for a given duration before a critical level of extracellular \([K^+]\) was reached.

Graham et al. (11) analyzed urinary caffeine levels and found that the concentrations were below the International Olympic Committee’s acceptable limit (i.e., 12 µg caffeine/ml urine) even after subjects ingested 9 mg/kg of caffeine. Since most studies investigating STHIX have involved lower doses than this (i.e., ~5 mg/kg), it appears that short-term performance can also be improved with caffeine concentrations that are lower than the acceptable limit, as demonstrated in this and other studies (1, 4, 5, 15, 16, 37). This is an ethical issue that has led some authors to call for a total ban on caffeine use by high-performance competitors (11, 27).

In conclusion, caffeine ingestion can be an effective ergogenic aid for short-term, supramaximal running performance and can increase the anaerobic component of exercise. However, the mechanisms to explain these results do not appear to be related to an increased [HLA]. Instead, the evidence suggests that caffeine exerts a direct influence on the central nervous system and/or upon active muscle during short-term, high-intensity exercise.
References


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