國立體育學院九十一學年度研究所博士班入學考試試題

體育運動論文評論（生化營養組）　　（本試題共六頁）

※注意：答案一律寫在答案卷上，否則不予計分

論文評論題目：Effect of caffeine and ephedrine ingestion on anaerobic exercise performance

每題 20 分

1. 受試者在服用受試品或安慰劑前研究人員對他們做了哪些篩選工作及測試前準備？

2. 作者如何定出 125% $\dot{V}O_2$ peak 作爲力的輸出？

3. 作者依據哪些實驗結果 (或/及看法)，認爲 Ephedrine 是經由刺激 CNS 而達到增強無氧性的運動表現。

4. 作者依據哪些實驗結果 (或/及看法)，認爲 Caffeine 是經由增強肌肉代謝而達到增強無氧性的運動表現。

5. 設有營養增補劑 (或中草藥) 名稱爲 A，爲瞭解其與運動相關之功效，試設計一實驗以探討之。敘述內容 (亦可以圖解) 應包括：(1) 受試者 (人或動物) 的數目 (2) 實驗方法與進行步驟 (3) 運動相關之功效可探討如生理方面或荷爾蒙方面或免疫方面或運動相關方面其中之一即可。

Effect of caffeine and ephedrine ingestion on anaerobic exercise performance

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ABSTRACT

BELL, D. G., I. JACOBS, and K. ELLERINGTON. Effect of caffeine and ephedrine ingestion on anaerobic exercise performance. *Med. Sci. Sports Exerc.*, Vol. 33, No. 8, 2001, pp. 1399–1403. **Purpose:** Ingestion of a combination of caffeine (C) and ephedrine (E) prolongs time to exhaustion during high-intensity aerobic exercise. CNS stimulation by C and E was proposed as part of the mechanism for the improvement. It was thought that this arousal might also be of benefit during anaerobic exercise. The purpose of this study was to investigate the effect of C, E, and C+E ingestion on performance of anaerobic exercise. **Methods:** Two groups were used to evaluate the effect of C and E on anaerobic performance. Group 1 (WIN) consisted of 16 healthy untrained male subjects who performed a 30-s Wingate test. Group 2 (MAOD) consisted of 8 healthy untrained male subjects who performed a supramaximal (125%$\dot{V}O_{2peak}$) cycle exercise trial to exhaustion to determine maximum accumulated oxygen deficit. The trials commenced 1.5 h after ingesting either C (5 mg·kg^{-1}), E (1 mg·kg^{-1}), a combination of C+E, or a placebo (P). All trials were randomized and double blind. Blood samples were assayed for lactate and glucose post drug ingestion just before exercise, and again 3, 5, and 10 min post exercise. Catecholamines were measured in the preexercise and 10-min postexercise blood samples. **Results:** Ephedrine increased power output during the early phase of the Wingate test, whereas C increased time to exhaustion and O_2 deficit during the MAOD test. C, E, and C+E increased blood lactate, glucose, and catecholamine levels. **Conclusion:** The improvement in anaerobic exercise performance is likely a result of both stimulation of the CNS by E and skeletal muscle by C. **Key Words:** ERGOGENIC AIDS, DOPAMINE, CATECHOLAMINES, WINGATE TEST, MAOD

Ergogenic aids are usually considered in terms of their effects on competitive sports performance, but they also have potential applications for the enhancement or sustaining of a soldier's performance. Unlike sports, where the use of pharmacological ergogenic aids is unethical, such cheating would not concern the military. It is likely that a safe ergogenic aid would find application in a military setting if the performance benefits increased the chances of mission success.

Recent studies demonstrated that a mixture of caffeine (C) and ephedrine (E) ingested approximately 1.5 h before exercise significantly improved exercise performance (5–8). In one study (6), C and E significantly improved cycle ergometer exercise time to exhaustion; furthermore, the benefits of combining C+E were additive (7). In a field study where subjects ran 3.2 km as quickly as possible while wearing military "fighting order" (i.e., webbing, backpack, full canteen, ammunition, and carrying a rifle), run times were also significantly improved by C+E ingestion (5). The percent improvement in the 3.2-km run (5) was reduced compared with the ride time to exhaustion (6), and this was believed to be related to the intensity of effort and the type of test. The estimated intensity of effort during the 3.2-km run was predicted at 90% $\dot{V}O_{2peak}$ from heart rate data (5), whereas the intensity of effort for the bike ride was held constant as 85% $\dot{V}O_{2peak}$. Further, the bike test was a test to exhaustion, whereas the run test was a time to completion. In a follow-up study, the dosage of C+E administered was reduced and a history of caffeine use was also investigated as a possible confounding variable (8). The findings from this study showed that time to exhaustion was significantly prolonged after ingestion of C+E compared with placebo, and the magnitude of the performance improvement was greatest in individuals who ingested caffeine regularly (8). This latter finding was attributed to an "overdosing" of individuals who were not accustomed to caffeine. Taken together, these data indicate that ingestion of both C+E may improve one's ability to sustain high-intensity aerobic work.

It was speculated that the primary mechanism resulting in improved performance after C+E ingestion was increased central nervous system (CNS) stimulation (6). It is tempting to speculate whether such CNS stimulation would also enhance anaerobic exercise performance. However, the data are equivocal in regard to separate administration of C or E on anaerobic performance. For example some studies on C report no improvement (10,11,15), whereas other studies report ergogenic effects (2,9,12). Research on E is sparse and nonsupportive (13,20). Moreover, there are no reported studies investigating the combination of C+E on anaerobic performance. The physical fitness demands of many military tasks involve the performance of supramaximal exercise, i.e., intensities that exceed maximal aerobic power; thus, there could be very broad benefits of an ergogenic aid that was shown to improve both aerobic and anaerobic exercise performance.

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Thus, the purpose of this study was to investigate the effect of ingesting C, E, and their combination on anaerobic exercise performance. It was hypothesized that, compared with a placebo, a combination of C+E would enhance anaerobic performance beyond any possible enhancement seen with separate administration of either drug.

METHODS

General design. To evaluate various aspects of anaerobic performance, two study groups were used in this investigation. The first group (WIN) performed a Wingate test (4), whereas the other group (MAOD) performed a maximal accumulated oxygen deficit test (19). Subjects reported to the laboratory for seven test sessions. During the first session, subjects underwent a medical screening and were given a caffeine intake questionnaire before signing an informed consent document. Once completed, subjects had their $\dot{V}O_{2peak}$ determined on an electronically braked cycle ergometer (Ergomed 920/930, Siemens-Elema, Solna, Sweden) using a ramped cycle protocol (25 $W \cdot min^{-1}$). Visits 2 and 3 served to familiarize subjects to the performance tests they would be performing throughout the study; sessions 4–7 were the treatment trials. All sessions were separated by at least 1 wk.

Treatment trials. These trials were double blind and randomized. Subjects reported to the laboratory after an overnight 12-h fast. They also had refrained from drinking alcohol and caffeine for 24 h before testing. Two hours before commencement of the trials, a 400-mL sport drink (Gatorade®) was consumed. After the 2 h, a venous catheter was inserted in an antecubital vein (Insyte®, Deseret). Subjects were then instructed to rest for 15 min, after which a blood sample was drawn. Ten mL of blood were drawn at each sample time: 5 mL were expelled into a tube treated with EGTA (90 $mg \cdot mL^{-1}$) and glutathione (60 $mg \cdot mL^{-1}$) (Cat-A-Kit, Upjohn, Kalamazoo, MI). This sample was used for catecholamine and drug analyses. The remainder was expelled into an EDTA tube. Between samples, the catheter was kept patent by flushing it with heparinized saline. After the first blood sample, the subject ingested one of the following four treatments: 5 $mg \cdot kg^{-1}$ body weight of C, 1 $mg \cdot kg^{-1}$ body weight E, a combination of these doses C+E, or a placebo (P) that contained a dietary fiber (Metamucil®). All treatments were ingested in opaque gelatin capsules. Ninety minutes after ingestion (pre-exercise) another blood sample was drawn. After this sample, the subjects completed the performance test protocol. Upon completion of the test protocol, the subject moved to a stretcher and lay in a supine position for blood sampling. Here, blood samples were drawn 3, 5, and 10 min after the termination of the test.

WIN group. Sixteen healthy male volunteers, aged 32 ± 4 yr (mean ± SD), mass of 80.0 ± 9.2 kg, and $\dot{V}O_{2peak}$ 47.7 ± 6.2 $mL \cdot kg^{-1} \cdot min^{-1}$ performed the Wingate test. Eleven were coffee drinkers, i.e., consuming greater than the equivalent of 1 cup of coffee per day. The procedures for this test follows. A 5-min warm-up ride was performed on a mechanically braked ergometer (Cardionics AB, Stockholm, Sweden). The warm-up resistance was set at 1.5 kg and a pedal frequency of 60 revolutions per minute. Twice during this warm-up, the subjects were instructed to sprint as hard and fast as possible while the resistance was increased to 4–6 kg for about 3–5 s. The warm-up was followed by a 2-min period of stretching. Next, the Wingate test was performed with the resistance adjusted to the subject's body weight (0.08 $g \cdot kg^{-1}$) (4). The subjects were instructed to remain seated while cycling and to cycle as quickly and as forcefully as possible through the entire 30-s duration of exercise. Upon the command to begin exercise, subjects were allowed 2–3 s to overcome the inertia of the flywheel before the full resistance was applied and the 30 s commenced. Once full resistance was applied power output was averaged every 5 s over the 30-s duration of the test via a computer system supplied with the ergometer. Verbal encouragement was provided throughout the ride.

MAOD group. Eight healthy male volunteers, aged 32 ± 5 yr, mass of 86.8 ± 9.1 kg, and $\dot{V}O_{2peak}$ 43.5 ± 3.4 $mL \cdot kg^{-1} \cdot min^{-1}$ performed the MAOD test. All were coffee drinkers, i.e., consuming greater than the equivalent of 1 cup of coffee per day. During the initial visit these subjects also had their oxygen consumption measured on the electronically braked cycle ergometer while performing four consecutive submaximal intensities approximating 50%, 60%, 70%, and 80% $\dot{V}O_{2peak}$. Each submaximal ride was 4 min in duration. A linear regression equation was calculated for each subject from these submaximal oxygen consumption and power output values. The MAOD power output equivalent of 125% $\dot{V}O_{2peak}$ was then calculated from this equation. This power output was then used in the treatment trials. Procedures for the treatment trials were as follows. The electronically braked cycle ergometer was used during the MAOD tests. A warm-up ride at 60 W was performed for 5 min at a cadence selected by the subject (60–80 rpm). At the end of this 5-min warm-up, resistance was immediately increased to the power output calculated to correspond to 125%$\dot{V}O_{2peak}$, and collection of expired gas was initiated. Expired gas was collected in a 300-L wet spirometer (Collins Gasometer, Braintree, MA). After determining the temperature and volume of the expired gas, a sample from the spirometer was directed to the oxygen and carbon dioxide analyzers (Ametek models S3A and CD3A, Pittsburgh, PA) for the determination of these gas fractions. For the MAOD test oxygen deficit was calculated as the difference between the oxygen demand, as determined from the linear regression equation and oxygen uptake during the high-intensity component of the test. Verbal encouragement was provided throughout the test, but elapsed time was not revealed. Cadence was allowed to range between 60 and 100 rpm. The test was terminated by the experimenter when the cadence dropped below 60 revolutions per minute.

Blood analysis. Plasma was obtained from aliquots of each blood sample and assayed for glucose (GOD-PAP, Boehringer Mannheim, Germany), norepinephrine, epinephrine, and dopamine (negative ion chemical ionization gas chromatography-mass spectrometry (21). Another aliquot of each blood sample was immediately deproteinized

TABLE . Power output in Watts (mean ± SEM, $N = 16$) during the Wingate test after caffeine (C), ephedrine (E), C + E, or placebo (P) ingestion.

Time (s)	P (W)	C (W)	E (W)	C + E (W)
5	1030 ± 33	1023 ± 31	1036 ± 33°	1047 ± 32°
10	791 ± 21	797 ± 21	803 ± 21°	811 ± 22°
15	683 ± 17	692 ± 19	675 ± 19	682 ± 19
20	584 ± 15	587 ± 15	574 ± 16	582 ± 18
25	501 ± 14	507 ± 16	496 ± 17	497 ± 16
30	444 ± 15	446 ± 17	437 ± 17	440 ± 17

°E trials > non-E trials.

and subsequently assayed for lactate (18). Preexercise plasma samples were also assayed for C and E by mass spectrometry (GC-MS) electron-impact, selective-ion monitoring.

Data analyses. A 2 × 2 factorial repeated measures analysis was used to determine the effects of caffeine (present or absent) and ephedrine (present or absent) on the ride time to exhaustion, oxygen deficit, and accumulated oxygen consumption for the MAOD test. For all other variables a three-factor design was used, i.e., the 2 × 2 factorial repeated measures (caffeine and ephedrine treatments) and a repeated measures over time, to compare the changes in the dependent variables across treatments and time. Commercially available statistical software was used (1). When a *post hoc* comparison was required, a means comparison contrast technique was employed and the Huyn-Feldt-epsilon factors were used to adjust degrees of freedom for multiple comparisons (1). Statistical significance was accepted at the $P < 0.05$ level. Variables measured at exhaustion for the MAOD were used in the analysis; however, those values corresponded to varying times to exhaustion among the subjects.

RESULTS

Caffeine and ephedrine concentrations. Plasma caffeine concentration just before the performance tests after C and C+E ingestion were similar, as were plasma ephedrine concentration after E and C+E ingestion. No caffeine was detectable in the P or E trials, nor was any E detectable in the P or C trials.

WIN group. Table 1 shows that the administration of ephedrine (E and C+E trials) produced a significant increase in power early in the ride compared with the trials when E was not ingested (P and C). After 15 s, this increase in performance no longer existed.

Both C and E treatments caused increased lactate post exercise; C+E lactates were greater than C, E, or P. Also C and E were greater than P post exercise at the 10-min mark (Fig. 1).

Caffeine and ephedrine ingestion significantly increased glucose levels pre- and post-exercise (Fig. 2).

The caffeine treatments (C and C+E) were associated with significant increases in epinephrine levels pre- and post-exercise compared with the noncaffeine trials (E and P) (Table 2). Norepinephrine was increased for the C and E trials post exercise, and the combination was additive. The

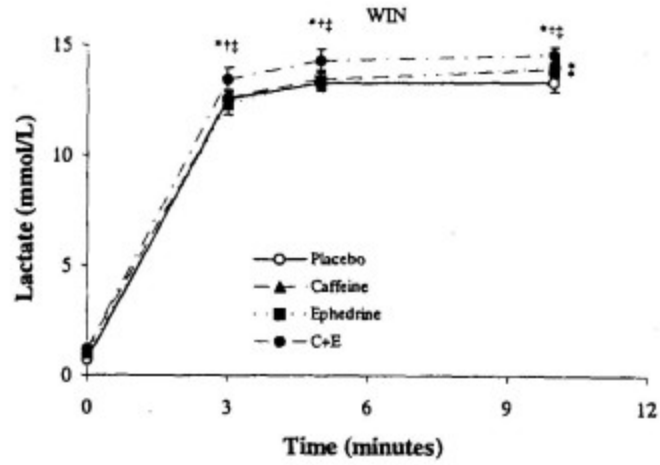

FIGURE 1—Blood lactate (mean ± SEM) pre- and postexercise at 3, 5, and 10 min after performance of the 30-s Wingate test ($N = 16$). * Significantly different from P; † significantly different from E; ‡ significantly different from C.

ephedrine trials showed a significant increase in dopamine post exercise compared with the nonephedrine trials.

MAOD group. Table 3 shows that the administration of caffeine (C and C+E trials) significantly improved times to exhaustion by about 8% compared with the non-C trials (P and E). C and C+E significantly increased MAOD by about 7%, as well as significantly increasing postexercise blood lactate (Fig. 3). Caffeine and ephedrine ingestion significantly increased glucose levels. The C+E treatment significantly elevated glucose levels above the C, E, and P levels at all time periods. C and E glucose levels were also significantly elevated above *P*-levels (Fig. 4). The caffeine treatments (C and C+E) were associated with significant increases in epinephrine levels pre- and post-exercise compared with the noncaffeine trials (E and P). Norepinephrine was not changed 10 min post exercise as a result of caffeine and ephedrine ingestion. The ephedrine trials were associated with a significant increase in dopamine levels post exercise compared with the nonephedrine trials (Table 2).

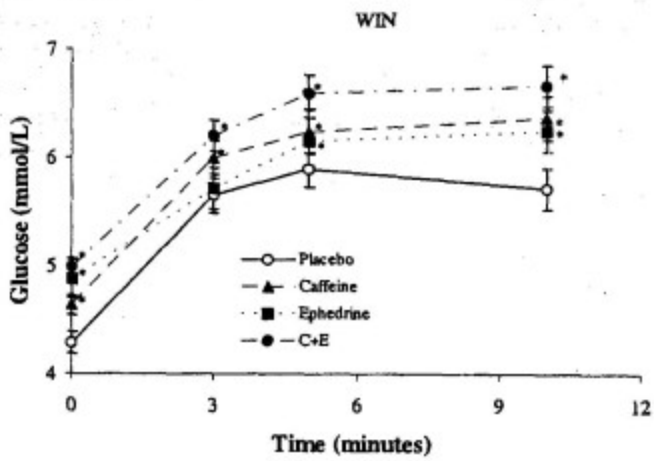

FIGURE 2—Blood glucose (mean ± SEM) pre- and postexercise at 3, 5, and 10 min after performance of the 30-s Wingate test ($N = 16$). * Significantly different from P; † significantly different from E; ‡ significantly different from C.

TABLE 2. Catecholamines (mean ± SEM) pre- and post-exercise at 10 min for the Wingate (N = 16) and MAOD (N = 8) performance tests; caffeine (C), ephedrine (E), C + E, or placebo (P) was ingested 1.5 h before the performance tests.

	Wingate		MAOD	
	Preexercise	Postexercise	Preexercise	Postexercise
Epinephrine (pmol · L^{-1})				
C + E	419.9 ± 19.4[a]	822.8 ± 74.5[a]	376.6 ± 57.6[a]	438.0 ± 61.8[a]
Caffeine	451.6 ± 31.3[a]	895.8 ± 118.4[a]	482.4 ± 56.0[a]	452.3 ± 34.6[a]
Ephedrine	305.0 ± 17.6	596.6 ± 67.6	281.8 ± 56.5	240.8 ± 25.3
Placebo	348.3 ± 24.6	647.8 ± 79.4	267.4 ± 37.8	262.0 ± 20.8
Norepinephrine (nmol · L^{-1})				
C + E	3.19 ± 0.24	10.91 ± 0.95[a,b]	2.58 ± 0.16	4.89 ± 0.60
Caffeine	3.30 ± 0.20	9.52 ± 0.63[a]	2.34 ± 0.36	4.75 ± 0.18
Ephedrine	3.14 ± 0.17	8.73 ± 0.64[b]	2.62 ± 0.25	4.51 ± 0.84
Placebo	2.70 ± 0.21	7.26 ± 0.50	2.01 ± 0.28	3.33 ± 0.36
Dopamine (pmol · L^{-1})				
C + E	571.2 ± 36.8	1490.0 ± 121.1[b]	524.7 ± 54.3	1002.0 ± 178.2[b]
Caffeine	545.9 ± 29.9	769.9 ± 38.6	638.9 ± 85.3	612.8 ± 48.9
Ephedrine	589.2 ± 28.2	1380.7 ± 124.6[b]	659.3 ± 94.0	891.9 ± 103.1[b]
Placebo	498.2 ± 18.5	718.9 ± 32.6	496.1 ± 52.9	729.5 ± 120.1

[a] C trials > non-C trials; [b] E trials > non-E trials.

DISCUSSION

In the present study C and E ingestion was associated with significant improvement in different aspects of anaerobic exercise performance. However, there was no additive effect or synergism between C and E when taken together.

WIN group. Ephedrine improved performance of the Wingate test, which is purported to be a test of anaerobic power. Performance was only improved during the first 10 s of this 30 s test. No improvement in performance was seen with C, although the blood data for norepinephrine, lactate, and glucose were similar for the C and the E trials post exercise. The observation that ephedrine ingestion did not cause changes in the blood chemistry variables, taken together with the sympathomimetic characteristics of ephedrine, suggests that the resulting performance improvement may not originate within the muscle. This would be consistent with earlier speculations (6,7) that E may stimulate the CNS rather than muscle metabolism.

An elevated dopamine level was associated with the E trials, raising the possibility that there may be a nerve factor, as dopamine is a precursor to both epinephrine and norepinephrine within the peripheral nervous system. Further, dopamine is also a important neurotransmitter in the CNS, especially in the area of the hypothalamus, which is known to be important for body arousal. In addition, it is known that ephedrine easily crosses the blood brain barrier and probably stimulates this area. These observations further suggest that ephedrine may be influencing an enhanced CNS drive rather than enhancing muscle metabolism. However, where the increased dopamine is coming from and it's implication for performance enhancement warrants further research.

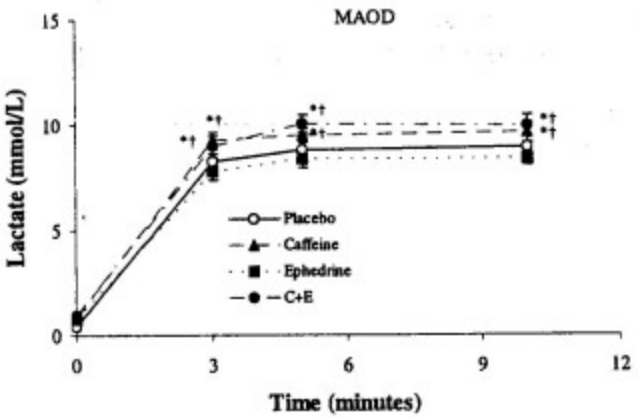

FIGURE 3—Blood lactate (mean ± SEM) pre- and postexercise at 3, 5, and 10 min after the MAOD test (N = 8). * Significantly different from P; † significantly different from E.

The present performance improvement with E conflicts with the works of others (13,20). Much of the discrepancy may be related to the dose of E. The dose of E used in the present study (1.0 mg·kg^{-1}) was 2 to 3 times the level used previously (13,20). Also, Gilles et al. (13) used 120 mg of pseudoephedrine. Pseudoephedrine is only about a third the strength of ephedrine; thus, a 120-mg dose of pseudoephedrine is equivalent to about a 40-mg dose of ephedrine (13), or about a 0.5-mg·kg^{-1} dose in our study.

MAOD group. Caffeine improved time to exhaustion in the MAOD test, which is considered by some individuals (12,19), but not all (3,14), to be an appropriate test for the indirect noninvasive determination of ATP production from anaerobic metabolism. The enhanced time to exhaustion might suggest that the effect of the caffeine treatments in the current study is related to an enhanced stimulation of skeletal muscle metabolism rather than stimulation of the CNS. This appears to be supported by the fact that the greater O_2 deficit during the C trials was paralleled by a greater increase in blood epinephrine and lactate that was not seen in the E trials. It has been hypothesized previously that skeletal muscle metabolism is enhanced as a result of increased epinephrine levels after caffeine ingestion (10).

The present C result agrees with the works of others that showed caffeine ingestion enhanced anaerobic exercise performance (2,9,12,16,17). However, unlike the present study that found a significant elevation of blood lactate after caffeine ingestion and MAOD performance, Doherty's study (12) showed no significant increase in lactate. Thus, Doherty (12) suggested that an increased rate of anaerobic glycolytic flux, leading to higher production and accumulation of lactate, is not a critical mechanism of action for

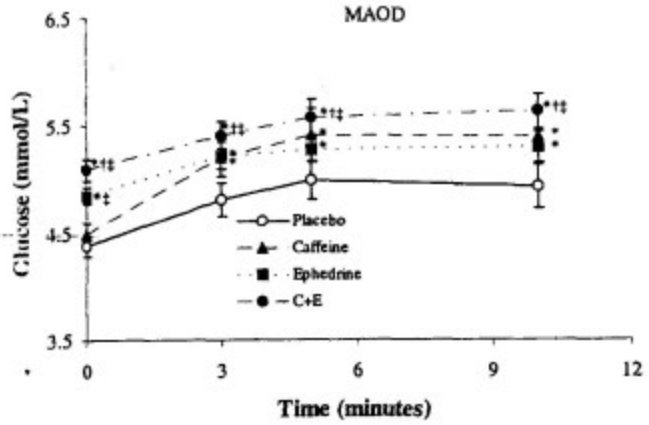

FIGURE 4—Blood glucose (mean ± SEM) pre- and postexercise at 3, 5, and 10 min after the MAOD test (N = 8). * Significantly different from P; † significantly different from E; ‡ significantly different from C.

explaining the ergogenic effects of caffeine. Doherty postulates that there are other central and peripheral elements involved. A probable reason for these ambiguities may be related to the training level of the subjects. In the present study untrained subjects were evaluated, whereas in Doherty's study (12) trained individuals were examined.

In conclusion, taken together with the results of other associated investigations, these data lend strong support to the premise that both aerobic and anaerobic performance is improved after ingestion of C+E. Thus, the combination of C and E could be recommended as an effective ergogenic aid for settings where acute, intense exercise is required, such as occurs in a variety of military operations. Further testing of the C+E is necessary to clarify the effects on cognitive performance and decision making before any actual application should be adapted in such settings.

Finally, in the present study, both caffeine and ephedrine improved different aspects of anaerobic performance. Ephedrine appeared to exert its effect more through an increased arousal, whereas caffeine appeared to enhance muscle metabolism.

TABLE 3. Time to exhaustion, O_2 deficit, and accumulated VO_2 for the MAOD (N = 8) test after caffeine (C), ephedrine (E), C + E, or placebo (P) ingestion; values are mean ± SEM.

	Time (s)	O_2 Deficit (mL)	Accumulated VO_2 (mL)
C + E	113.1 ± 8.3[a]	3301 ± 278[a]	5622 ± 576
Caffeine	117.0 ± 9.3[a]	3757 ± 392[a]	5505 ± 658
Ephedrine	105.3 ± 7.7	3224 ± 258	5073 ± 508
Placebo	108.2 ± 8.9	3214 ± 258	5332 ± 609

[a] C trials > non-C trials.

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