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Ambient temperature affects thrombotic potential at rest and following exercise ☆

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ABSTRACT

Introduction: During exercise, ischemic risk increases, possibly due to changes in coagulation and fibrinolytic activity. Previous research suggests ambient temperature affects resting thrombotic potential, but the effect of heat and cold on hemostasis during exercise is unknown. The purpose of this study was to assess changes in coagulation and fibrinolysis during maximal exercise in hot and cold temperatures, and to compare those responses to exercise under temperate conditions.
Materials & Methods: Fifteen healthy men completed maximal exercise tests in hot (30 °C), temperate (20 °C) and cold (5° - 8 °C) temperatures. Blood samples were obtained before and immediately after exercise and analyzed for concentrations of thrombin-antithrombin III (TAT), active tissue plasminogen activator (tPA) and plasminogen activator inhibitor-1 (PAI-1). Results were analyzed by ANOVA.
Results: A main effect of time was observed for TAT (temperate = 1.71 ± 0.82 – 2.61 ± 0.43 ng/ml, hot = 1.81 ± 0.73 – 2.62 ± 0.67 ng/ml, cold = 2.33 ± 0.65 – 2.89 ± 0.81 ng/ml, PRE to POST, respectively) and tPA activity (temperate = 0.72 ± 0.44 – 2.71 ± 0.55 IU/ml, hot = 0.72 ± 0.38 – 2.64 ± 0.61 IU/ml, cold = 0.86 ± 0.45 – 2.65 ± 0.77 IU/ml, PRE to POST, respectively). A trend was observed for the PAI-1 response to exercise (temperate = 14.5 ± 23.7 – 12.3 ± 20.2 IU/ml, hot = 15.1 ± 26.5 – 10.0 ± 15.1 IU/ml, cold = 10.5 ± 10.4 – 7.9 ± 9.7 IU/ml, PRE to POST, respectively, $p = 0.08$). TAT concentrations were significantly higher in cold compared to temperate and hot conditions.
Conclusion: Coagulation potential is elevated during exposure to cold temperatures. These data suggest that risk of an ischemic event may be elevated in the cold.

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Introduction

The majority of cardiovascular events, including approximately 80% of acute myocardial infarctions, are precipitated by occlusive thrombi [1,2]. Elevated coagulation activity and reduced fibrinolysis, the process of fibrin clot dissolution, are associated with risk of cardiovascular disease and ischemic events [3,4]. Risk of an adverse cardiovascular event increases during exercise [5], possibly due to the effect of exercise on blood coaguability. Acute exercise stimulates fibrin clot formation [6], and indicators of elevated coagulation activity such as increased plasma concentration of coagulation factor VIII [7] and thrombin:antithrombin III complex (TAT) [8] have been observed following acute bouts of exercise using various modalities and intensities. The fibrinolytic response to exercise may prevent occlusive clot formation. Exertion stimulates an increase in plasma concentration of tissue plasminogen activator (tPA), a stimulator of fibrinolysis, and a decrease in concentration of the primary inhibitor of fibrinolytic activity, plasminogen activator inhibitor-1 (PAI-1) [9,10]. An excessive coagulation response or inadequate fibrinolytic response to exertion may lead to clot formation and a possible subsequent ischemic event.

Rates of cardiovascular events, morbidity and mortality are increased during seasons in which extreme temperatures are common [11-17], leading to the hypothesis that exposure to hot and cold ambient temperatures may enhance thrombotic potential. Previous findings indicate that cold exposure stimulates platelet activation and increased plasma levels of various markers of coagulation [18-20]. Heat stress can also lead to blood composition changes that favor arterial thrombosis. Investigations of heat exposure have shown increases in coagulation markers [21-23], potentially due to increases in blood lactate and plasma cholesterol [22,24] which may enhance coagulation potential. Exercise stress combined with exposure to hot or cold temperatures may result in greater thrombotic risk than exercise under temperate conditions, but the influence of ambient temperature on the coagulation and fibrinolytic responses to exercise are currently unknown.

The purpose of this study was to assess coagulation and fibrinolytic responses to exercise in both cold and hot environments, and to compare those responses to exercise under temperate conditions. It was hypothesized that coagulation potential, as depicted by plasma concentrations of TAT, and fibrinolysis, assessed by plasma levels of

Abbreviations: TAT, thrombin-antithrombin III complex; tPA, tissue plasminogen activator; PAI-1, plasminogen activator inhibitor-1; RPE, rating of perceived exertion.

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active tPA and PAI-1, would be higher during exposure to hot and cold compared to normal, temperate conditions.

Methods

All procedures of this study were approved by the Institutional Review Board at Ball State University. Signed informed consent was obtained from all subjects prior to participation in the study.

Subjects

Fifteen healthy men between the ages of 19–35 years (mean age 25.3 ± 4.3 years) participated in this research study. Subjects were non-smokers, free from any known cardiovascular or metabolic disease and used no medications. Participants had no physical limitations that prevented them from doing high-intensity exercise on a cycle ergometer. Exercise testing was conducted only on subjects who were considered "low risk" according to American College of Sports Medicine guidelines [25].

Experimental Design

Subjects reported to the laboratory for testing on three separate occasions, separated by a minimum of seven days. All tests were conducted between 6 am and 10 am to control for diurnal variations of the variables under examination. Subjects were asked to abstain from exercise, caffeine, and any food or drink other than water for 12 hours and alcohol for 24 hours prior to testing. Subjects were also free from any fever or illness during the 7 days prior to testing. All testing procedures were conducted in an environmental chamber (Tenny Engineering; Union, NJ) under the following conditions: normal (20 °C), cold (5° or 8 °C) and warm temperature (30 °C), and in random order. Humidity was not controlled during testing. Due to a technical complication with the environmental chamber, six subjects completed the "cold" condition at 5 °C and nine completed the test at 8 °C. A repeated measures ANOVA revealed no differences in any measured variable between these two "cold" temperatures, so results from all of these tests were included in the final analyses. Testing began with the subject sitting in the experimental temperature for a 15-minute period of seated rest followed by acquisition of a baseline blood sample, resting heart rate (HR) and blood pressure (BP). A scripted explanation of the Borg Ratings of Perceived Exertion (RPE) scale was read to each subject. Subjects were then seated on a cycle ergometer and allowed a few minutes of familiarization with the equipment, after which a maximal exercise test on a cycle ergometer was completed. A post-exercise blood sample was then collected.

Exercise testing procedures

Exercise tests were conducted on an electronically-braked cycle ergometer (SensorMedics Ergometrics 800, SensorMedics Corp; Yorba Linda, CA). Subjects were instructed to maintain a pedal rate between 60–80 revolutions per minute (rpm). The intensity was set at zero watts for the first minute, and increased 25 watts per minute until the subject reached volitional exhaustion. During exercise, expired gases were collected for the determination of peak oxygen consumption (peak VO_2) using a metabolic measurement system (Parvo Medics TrueOne® 2400; Sandy, UT) which was calibrated prior to each test. RPE were recorded at the end of every third minute of exercise, and HR was assessed by telemetry (Polar Electro, Kempele, Finland). Criteria for exerting maximal effort included achieving at least two of the following: a respiratory exchange ratio (RER) value of > 1.10, a plateau in VO_2 with an increased work load, a lactate concentration > 8.0 mmol/L, and achieving at least 85% of age predicted maximal HR.

Blood Sampling/Assays

Ten ml of blood were drawn from an antecubital vein using clean venipuncture with minimal stasis prior to exercise and within two minutes after the completion of the exercise test. The blood samples were collected in an acidified citrate buffer (Biopool Stabilyte, Biopool International; Ventura, CA), and centrifuged for 20 min at 1500 ×*g* and 4 °C to obtain platelet-poor plasma. Plasma aliquots were stored at −80 °C until assayed. Enzyme-linked immunosorbancy assays were used to assess plasma concentrations of TAT (Enzygnost TAT Micro, Behringwerke AG, Marburg, Germany), tPA activity (Chromolyze tPA, Biopool International; Ventura, California and PAI-1 activity (Chromolyze PAI-1, Biopool International; Ventura, California). All samples were assayed in duplicate and the average of the two values was used in statistical analyses. Post-exercise values were corrected for changes in plasma volume, determined using pre- and post-exercise hematocrit values [26]. Lactate concentration was measured from whole blood using dry chemistry (Accutrend ®, Roche Diagnostics; Indianapolis, IN) according to manufacturer recommendations.

Statistical Analysis

Statistical analyses were completed using SPSS 10.05 software (SPSS Inc., Chicago, IL). Subject characteristics were depicted using descriptive statistics. Peak exercise HR, VO_2, RPE, lactate, and RER in each of the three temperatures were assessed using a one-way ANOVA. A one-way ANOVA was also used to assess potential differences in baseline HR and blood pressure in the three temperatures. Differences in plasma concentrations of TAT, tPA and PAI-1 were assessed with a two-factor ANOVA, using temperature (normal, hot, cold) and time (pre-, post-exercise) as within-subjects factors. Post-hoc pairwise comparisons were done using Fisher's LSD method, and statistical significance for all analyses was set at alpha = 0.05. Unless otherwise indicated, data are presented as means ± S.D.

Results

Subject characteristics and peak exercise variables are displayed in Tables 1 and 2 respectively. Peak HR was statistically higher in the hot versus cold temperature. There was no significant difference in peak VO_2 or RPE among the three temperatures. Peak lactate concentration was significantly higher in the normal temperature compared to the cold. Respiratory exchange ratio (RER) was higher in the cold (1.23 ± 0.06) when compared with the heat (1.20 ± 0.06). Mean exercise time did not differ among trials, and ranged from 10.0 - 15.7 minutes in the normal temperature, 10.2 – 15.5 min in the hot condition and 10.2 – 15.6 min in the cold.

Ambient temperature did not affect baseline HR (normal = 64.9 ± 11.6 bpm, hot = 67.1 ± 15.9 bpm, cold = 64.7 ± 13.8 bpm) or systolic blood pressure (normal = 115.7 ± 7.6 mmHg, hot = 114.9 ± 9.9 mmHg, cold = 118.8 ± 11.2 mmHg). Resting diastolic blood pressure was significantly lower in the heat (65.9 ± 6.5 mmHg) than the cold (72.4 ± 10.0 mmHg) and normal (70.3 ± 6.3 mmHg) temperatures.

Average intra- and inter-assay coefficients of variation for TAT were 5% and 7%, respectively. Plasma concentration of TAT significantly increased during exercise (see Fig. 1). A main effect of temperature was also observed, as TAT concentrations were found to be

Table 1
Subject Characteristics.

Age (years)	25.3 ± 4.3
Height (cm)	179.4 ± 8.4
Weight (kg)	79.9 ± 11.9
BMI (kg/m^2)	24.8 ± 2.9

Values are displayed as means ± SD. BMI = Body Mass Index.

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