

Effects of Heat Stress on Platelet Function and Coagulatory Potential

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Abstract

The purpose of this study was to investigate changes in platelet function and coagulation following exercise-induced heat stress (PHS) and passive heat stress (PHS) in young active males. Both conditions resulted in significantly elevated Tco (38.4°C and 38.5°C for EIHS and PHS respectively), and was achieved in a similar time period (51.1 and 47.4 min). There were significant increases in platelet number for both conditions (22% and 6% for EIHS and PHS, respectively). In both conditions, ADP-induced time to occlusion and PTT decreased significantly. Additionally, ADP-induced time to occlusion decreased significantly after EIHS. The findings of this study reveal that both EIHS and PHS activate coagulation and platelet function, and suggest that the combination of exercise and heat stress may cause greater activation than heat stress alone.

Introduction

It is well documented that regular physical exercise is associated with a decreased risk of cardiovascular disease (1). Despite all the beneficial effects of regular exercise, an acute bout of strenuous exercise, particularly in persons unaccustomed to physical activity, is associated with an increased risk of sudden cardiac death. Activation of platelets or enhanced coagulatory potential following high-intensity exercise may predispose an individual to the formation of intravascular thrombi. Research suggests that an acute bout of strenuous exercise causes a rise in platelet count, platelet hyperactivity, and hypercoagulability (2, 3, 4). Platelet function and coagulatory changes resulting from exercise-induced heat stress (EIHS) have not been thoroughly investigated. Furthermore, the effect of short term passive heat stress (PHS) on platelet function and coagulation has not been documented.

Purpose

To investigate changes in platelet function and coagulation resulting from EIHS and PHS in apparently healthy active males.

Methods

Study Design

Repeated Measures Design: On separate days subjects completed two trials:

Exercise Induced Heat Stress (EIHS) – cycle in warm-up gear until Tco reached 38.5°C or 60 min. Passive Heat Stress (PHS) – sit in tank of warm water (40°C) until Tco reached 38.5°C or 60 min.

Study Protocol

- Blood samples containing no additives were analyzed for PT and PTT using the Sysmex CA-5000 (Beckman Coulter, Inc; Fullerton, CA, USA).
- Blood anticoagulated with EDTA was analyzed for PT and PTT using the Sysmex CA-5000 (Beckman Coulter, Inc; Fullerton, CA, USA).
- Epinephrine-induced and ADP-induced platelet aggregability was analyzed using a Platelet Function Analyzer (PFA-100, Dade Behring; Deerfield, IL, USA).

Results

Table 1. Effects of EIHS and PHS on core temperature, body weight, thermal sensation, and percent change in plasma volume; n=11

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pre</th>
<th>Post</th>
<th>% Pre-Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature, °C</td>
<td>37.3 ± 0.4</td>
<td>37.3 ± 0.3</td>
<td>1.1 ± 0.3</td>
</tr>
<tr>
<td>Body Weight, kg</td>
<td>80.6 ± 1.5</td>
<td>80.6 ± 1.5</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td>Thermal Sensation</td>
<td>4.2 ± 0.6</td>
<td>4.2 ± 0.6</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td>Time to Achieve min</td>
<td>—</td>
<td>—</td>
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<table>
<thead>
<tr>
<th>Condition</th>
<th>Pre</th>
<th>Post</th>
<th>% Pre-Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet Volume, %</td>
<td>—</td>
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</table>

Values are expressed as the mean ± SD

Discussion/Conclusion

The purpose of this study was to investigate changes in platelet function and coagulation resulting from EIHS and PHS in healthy, active males. Both EIHS and PHS resulted in:

- increased platelet number as evidenced by increased platelet number,
- decreased closure time,
- decreased PTT.

Previous research suggests that moderate exercise suppresses platelet function (platelets take longer to aggregate) while strenuous exercise increases platelet function (platelets take less time to aggregate) (5,6). Sympathetic nervous system activation with exercise, and subsequent catecholamine release is thought to influence platelet aggregation (2).

The present study both EIHS or PHS (moderate activity) caused enhanced platelet function and a procoagulatory state. Thus, it appears likely that heat stress, or exercise in combination with heat stress, exacerbates the risk of an acute thrombotic event. Further research is needed to examine the hemostatic responses to heat stress, particularly EIHS.

Acknowledgements

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References