Effect of Hydration Status on High-Intensity Rowing Performance and Immune Function

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Purpose: This study determined the effect of dehydration and rehydration (DR) on performance, immune cell response, and tympanic temperature after high-intensity rowing exercise. Methods: Seven oarswomen completed two simulated 2000-m rowing race trials separated by 72 h in a random, cross-over design. One trial was completed in a euhydrated (E) condition and the other using a DR protocol. Results: The DR condition resulted in a 3.33 ± 0.14% reduction in body mass ($P < .05$) over a 24-h period followed by a 2-h rehydration period immediately before the simulated rowing race. There was a greater change in tympanic temperature observed in the DR trial ($P < .05$). There were increases in the blood concentration of leukocytes, lymphocytes, lymphocyte subsets (CD3+, CD3+/4+, CD3+/8+, CD3+/16+, CD4+/25+; $P < .05$) and decreases in lymphocyte proliferation and neutrophil oxidative burst activity immediately following the simulated race ($P < .05$) in both trials. Blood leukocyte and neutrophil concentrations were greater after exercise in the DR trial ($P < .05$). Whereas most immune measures returned to resting values after 60 min of recovery in both trials, lymphocyte proliferation and the concentrations of CD3+/4+ and CD4+/25+ cells were significantly lower than before exercise. Blood leukocyte and neutrophil concentrations were significantly higher before and after exercise in the E trial. Conclusion: The effects of dehydration/rehydration did not negatively influence simulated 2000-m rowing race performance in lightweight oarswomen but did produce a higher tympanic temperature and had a differential effect on blood leukocyte, neutrophil, and natural killer (CD3−/16+) cell concentrations after exercise compared with the euhydrated state.

Keywords: neutrophil oxidative burst activity, lymphocyte proliferation, dehydration, tympanic temperature.

High-intensity and/or long-duration exercise has been shown to suppress certain aspects of the immune system.\(^1\) It has been proposed that transient alterations in immune function following strenuous exercise can lead to an increased risk of...
contracting an illness, particularly colds or upper respiratory tract infections. Gleeson and Bishop\(^3\) suppose that this increased susceptibility to contract an illness is more likely the result of a combination of factors than due to a single factor. Both hydration status\(^4\) and circulating levels of stress hormones\(^3,5\) have been linked to changes in immune function following exercise. Furthermore, increased heat stress due to exercising in a dehydrated state has been shown to increase stress hormone concentrations and have varying effects on immune function.\(^6–10\)

It has been reported that rowers have higher rates of illness compared with athletes in other endurance events and that this may be partly due to an altered immune function as a result of the intensity of the competition and the physical preparation involved in the sport.\(^11,12\) In addition, lightweight rowers, especially women, have been known to adopt unsafe eating practices and short-term dehydration procedures to achieve transient or temporary body mass reduction before their event.\(^13,14\) The potential health risks of these behaviors have been detailed in the ACSM position stand on “weight cutting”\(^15\) and include impaired thermoregulation, immune function, hormonal status, protein nutritional status, growth and development, psychological state, and others. Indeed, body mass reduction in female athletes has been shown to be immunosuppressive,\(^16–18\) and in rowers the practice of weight cutting may negatively affect simulated rowing race performance time.\(^13,14\) However, the influence of fluid restriction to induce body mass loss, followed by rehydration on immune cell concentration and function and rowing performance in lightweight oarswomen has not been well examined.

Therefore, the purpose of the study was to investigate the effect of a 24-hour period of fluid restriction and subsequent rehydration on 2000-m simulated rowing race performance, tympanic temperature, and immune cell concentration and function in lightweight oarswomen. It was hypothesized that the completion of a simulated 2000-m rowing race following a dehydration/rehydration protocol would impair performance time, elicit a greater elevation of tympanic temperature, and result in greater disturbance of the immune response indicative of postexercise immune suppression compared with a euhydrated condition. There are a wide range of immune parameters that can be assessed, and we chose to include a well-established measure of innate immunity (neutrophil function) and T cell function (proliferation) in addition to more standard clinical immune characterization (phenotype concentrations in blood) as recommended by Albers.\(^19\)

**Methods**

**Participants**

Seven healthy females (age = 22.4 ± 3.6 y, weight = 61.1 ± 3.4 kg, height = 164.4 ± 3.0 cm) volunteered for the study. All participants were competitive rowers (mean experience = 2.6 y) and competed in the lightweight category during the race season. Each participant completed a demographic questionnaire and signed an informed consent form after the purpose and procedures of the study had been
fully explained. A university research ethics board had previously approved all experimental procedures.

**Experimental Design**

This study was completed in the off-season of the annual training cycle at a stage when body mass is not strictly controlled by the rowers. Because of this, the mean body mass reported in the current study (61.1 kg) was slightly higher than the upper limit of ~59 kg set by the International Rowing Federation for female lightweight rowers. Each participant performed two 2000-m simulated rowing race trials individually under two different experimental conditions. The euhydrated (E) trial included the maintenance of body mass with usual fluid and food intake. The dehydrated/rehydrated (DR) trial involved a body mass reduction of 3.5% by restricting fluid intake but not food consumption over a 24-hour period and a 2-hour rehydration phase immediately before the simulated race trial. A random cross-over design was used to assign the treatment order for each participant, resulting in 3 rowers completing the DR followed by the E, and 4 rowers completing the trials in the reverse order. The two trials were performed at the same time of day and were separated by at least 3 but no more than 5 days to provide adequate recovery between each of the 2000-m trials. Participants were blinded to the finishing time of the simulated 2000-m rowing race trials until after the study was completed.

The DR protocol for the reduction in body mass was similar to that used by Burge et al\textsuperscript{13} with the exception that participants in the current study were asked not to exercise the evening before the experimental trials. Baseline body mass was determined as the mean of 3 measurements on 3 consecutive mornings before the experimental conditions. Each participant was asked to achieve a target body mass reduction of no greater than 3.5% over the 24-hour period immediately preceding the experimental simulated race trials. The body mass loss was accomplished by reducing the volume of any type of fluid intake during the 24-hour period preceding the experimental trials. To do this, each participant was provided with a scale and the body mass goal and asked to monitor the progress of their body mass reduction over the 24-hour period. The rowers reported to the laboratory at 0900 after an overnight fast and were weighed using the same scale. To more accurately mimic weight-cutting procedures practiced by rowers for the DR trial, each rower performed light rowing exercise (a HR of <140 beats·min\textsuperscript{-1}) in a track suit (long sleeve top and pants) for no longer than 30 minutes and were weighed periodically to achieve as close as possible to a body mass reduction of 3.5% in the provided time period (<30 minutes). Subsequently, each participant was given a 2-hour rehydration period at rest during which they were provided with ≤250 mL of cool water every 15 minutes until body mass was reestablished to baseline levels prior to the DR protocol. Following the 2-hour rehydration period, the rowers were reweighed before performing the simulated 2000-m rowing race. Blood samples were taken from each participant at 0900 on the day of the trial, immediately before the simulated 2000-m rowing race, immediately after completion of exercise, and after 60 minutes of recovery. The E condition was identical to the DR
condition except that body mass was maintained through regular hydration in the 24-hour period before and morning of this experimental trial.

**Rowing Performance**

The simulated 2000-m rowing race was completed on a Concept IIC rowing ergometer (Morrisville, VT). This test simulates an on-water 2000-m rowing race under controlled laboratory conditions. Briefly, the trial involved rowing 2000 m as fast as possible following a standardized 10-minute warm up. The time required to complete the 2000-m distance was recorded, as well as heart rate, stroke rate, and average power output every 200 m throughout the test. The ambient conditions in the laboratory were the same for simulated 2000-m races during both experimental trials: temperature = 21 to 22°C and relative humidity = 25 to 30%.

**Blood Collection and Analyses**

Four separate 10-mL blood samples were obtained from a forearm vein using an indwelling 22 gauge Cathelon kept patent with a sterile saline solution (~0.5 mL of 0.9% NaCl). A 1.0-mL blood draw was made and discarded to remove any dilution effects of the saline at the time each sample was taken. Each sample was separated into an EDTA-treated Vacutainer (Becton-Dickinson; Mississauga, ON) for CBC analysis (~3 mL) and a heparin-treated Vacutainer (~7 mL) for detailed immune analysis. Blood hematocrit was measured using the microcentrifuge method in duplicate immediately after each blood sample was taken. Note that there were no significant changes in blood hematocrit observed during exercise in either experimental trial. All samples were kept on ice and analyzed once the final sample was obtained. All immune assays have been previously detailed for our laboratory.

**Leukocyte Concentrations.** Differential cell count measurements for blood leukocyte concentrations were completed using a Coulter STKS Hematology Flow Cytometer (Beckman Coulter; Fullerton, CA).

**Lymphocyte Isolation.** Each blood sample was diluted with warm, sterile lysis buffer (phosphate-buffered saline; PBS) and gently inverted for 5 minutes. Peripheral blood mononuclear cells (PBMCs) were isolated and purified using a density gradient of Histopaque 1077 (Sigma Chemical Co., St. Louis, MO) as previously described. The isolated lymphocyte samples were counted and diluted to a concentration of $1.0 \times 10^9/L$ for all immune phenotyping and proliferation assays. Trypan blue exclusion was used to determine cell viability, which was greater than 98% for all samples.

**Lymphocyte Phenotyping.** An immunofluorescence assay was used to identify the cell types in blood. Whole blood was added to a preconditioned microtiter plate, to which warm lysis buffer was added to each sample to lyse red blood cells. This process was repeated twice to ensure removal of all red blood cells. Double color immunofluorescence was performed and the relative proportion of cells determined by flow cytometry as previously described. The monoclonal antibodies (mAb; Sigma, St. Louis, MO) were conjugated to either biotin, phycoerythrin, or fluorescein isothiocyanate and used to identify the following cell
types: CD3+ cells (total T cells), CD3+/4+ cells (helper/inducer T cells), CD3+/8+ cells (cytotoxic/suppressor T cells), CD3−/16+ cells (natural killer cells), and CD4+/25+ cells (IL-2 receptor α subunit; activated helper/inducer T cells). Resulting percentages were corrected for background fluorescence (0% to 2%) determined by incubating the cells with goat antimouse IgG (no MAb) followed by antigoat isothiocyanate, biotin/streptavidin, or phycoerythrin. An estimation of absolute concentration of the different subpopulations of lymphocytes was obtained by multiplying the relative concentrations determined by immunofluorescence by the concentration of lymphocytes determined from differential cell counts measurements.

**Stimulated Lymphocyte Proliferation.** An ex vivo proliferation assay was chosen to estimate the ability of lymphocytes to proliferate in response to mitogens during the different experimental conditions. Each 200-μL sample of isolated lymphocytes (1.0 × 10⁹ cells/L) was cultured for 48 hours in 96-well microtiter plates with either concanavalin A (Con A; 5mg/L; ICN, Montreal, PQ) or phorbol myristate acetate (PMA; 40 μg/L; ICN), or no mitogen as previously described.²¹ Eighteen hours before harvesting the cells, each well was pulsed with [³H]thymidine (18.5 kBq; Amersham, Oakville, ON) and harvested on glass fiber filters using a multiwell harvester (Skatron, Lier, Norway) and counted using Ecolite⁷ in a Beckman betacounter (LS 58017, Beckman Instruments Inc.). All samples were performed in triplicate. A stimulation index (SI) was determined by comparing the response of the stimulated cells to that of the nonstimulated cells through the following formula: SI = [³H thymidine (cpm) incorporated by stimulated cells] − [³H thymidine (cpm) incorporated by unstimulated cells] ÷ [³H thymidine (cpm) incorporated by unstimulated cells].

**Neutrophil Oxidative Burst Activity.** The ability of neutrophils to produce free radicals was assessed by stimulating whole blood with phorbol myristate acetate (3.2 × 10³ nM) and assessing the oxidation of dihydrorhodamine-123 using flow cytometry, as previously described for our laboratory.²³ A stimulation index (SI) was calculated as follows: SI = (Mean channel fluorescence time B) − (Mean channel fluorescence time A) ÷ (Mean channel fluorescence Time A).

**Tympanic Temperature**

Tympanic temperature was recorded as a mean of duplicate measurements using a Thermoscan⁷ infrared thermometer (Braun, USA) placed in the right ear of all participants. The device was checked for accuracy before testing using a thermometer and temperature-controlled heat source over the range of 35 to 45°C. Measurements were made by inserting the head of the thermometer into the ear canal of the participant immediately before each blood sampling time point. Measurement of tympanic temperature has been shown to be positively correlated to rectal temperature and to be reliable for use during exercise.²⁴,²⁵,³²

**Statistical Analysis**

Standard descriptive statistical methods were used to calculate means and standard deviations. Independent t tests were used to determine any significant differences in rowing performance time between the E and DR and the order of these
tests. Two-way ANOVA with repeated measures was used to compare the 2 conditions (ET and RT) by the measurement time points (repeated measures) for blood variables and change in tympanic temperature and body mass. A Newman–Keuls multiple comparison procedure was used to further explore any significant $F$ ratios. The level of significance was set a priori at $P < .05$ for all statistical analyses. All statistical procedures were conducted using a commercially available software package (Statistica Version 6, STATSOFT; Oklahoma City, OK).

## Results

### Body Mass

Table 1 shows that body mass had significantly decreased by $2.0 \pm 0.2$ kg following the dehydration protocol compared with before the dehydration protocol. The 2-hour rehydration period replaced an average of $1.8 \pm 0.4$ kg of the mass lost by the participants, returning their body mass to near their baseline measurement. In the E trial, body mass was not significantly different from baseline or rehydrated body mass.

### Rowing Performance

There was no significant difference in mean time to complete the simulated 2000-m rowing race between the E trial ($487 \pm 19$ s) and the DR trial ($488 \pm 19$ s). As well, there was no significant difference between the first time the simulated 2000-m rowing race was performed ($486 \pm 18$ s) versus the second time ($489 \pm 20$ s).

### Tympanic Temperature

There was no significant change in tympanic temperature between baseline and immediately before the simulated 2000-m rowing race. There was a significant change in tympanic temperature (elevation) in both the E and DR conditions from before to immediately following exercise and after 60 minutes of recovery (Figure 1). The change in tympanic temperature was significantly different between the DR and E trial immediately after and following 60 minutes of recovery.

### Table 1  Body Mass (Means ± SD) of Lightweight Female Rowers at Baseline, After Dehydration, Rehydration, and in the Euhydrated State

<table>
<thead>
<tr>
<th></th>
<th>Baseline (kg)</th>
<th>Dehydrated (kg)</th>
<th>Loss (kg)</th>
<th>Rehydrated (kg)</th>
<th>Gain (kg)</th>
<th>Euhydrated (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>61.1 ± 3.4</td>
<td>59.1 ± 3.3$^a$</td>
<td>2.0 ± 0.2</td>
<td>60.9 ± 3.4</td>
<td>1.8 ± 0.4</td>
<td>61.0 ± 3.5</td>
</tr>
</tbody>
</table>

$^a$Significantly lower than baseline, rehydrated, and euhydrated body mass, $P < .05$. 
Immediately after exercise, the concentrations of total white blood cells (WBC), lymphocytes, and monocytes were significantly higher than before exercise in both groups (Figure 2). However, the mean WBC concentration for the DR trial was higher than that of the E trial after exercise due to a higher concentration of neutrophils ($P < .05$). After 60 minutes of recovery, the WBC count and differential for the DR group had returned to preexercise values. The concentration of total WBC and neutrophils, however, remained significantly higher than before exercise for the E group.

The concentration of CD3⁺, CD3⁺/4⁺, CD3⁺/8⁺, CD3⁺/16⁺, and CD4⁺/25⁺ cells were all significantly elevated in both the E and DR conditions immediately after exercise (Table 2). A significant main effect for trial was observed for

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**Leukocyte Concentrations**

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The concentration of CD3⁺, CD3⁺/4⁺, CD3⁺/8⁺, CD3⁺/16⁺, and CD4⁺/25⁺ cells were all significantly elevated in both the E and DR conditions immediately after exercise (Table 2). A significant main effect for trial was observed for
CD3−/16+ cells between the E and DR, indicating that the concentration of NK cells was higher in the DR condition immediately after exercise. After 60 minutes of recovery, CD3+, CD3+/8+, and CD3−/CD16+ lymphocyte concentrations were not different from before exercise in both trials. However, the concentration of CD3+/4+ and CD4+/25+ lymphocytes were significantly lower than before exercise values in both groups after recovery.

Lymphocyte Proliferation

There were no differences between experimental trials for the lymphocyte proliferation indices. The ability of lymphocytes to respond to both mitogens decreased immediately after exercise in both conditions ($P < .05$, Figure 3). After the 60-minute recovery period, the responses to PMA for both conditions were not different from before exercise; however, the response to ConA remained lower than immediately before exercise ($P < .05$).

Neutrophil Oxidative Burst Activity

There was no effect of hydration status on the ability of neutrophils in whole blood to produce free radicals after stimulation with PMA (Figure 4). The mean channel fluorescence was significantly lower after exercise at both 10- and 15-min incubation times but was not different from before exercise after 60 minutes of recovery.
### Table 2  Concentration (Means ± SD) of Lymphocyte Subpopulations ($\times 10^7$ Cells/L) Before and After the Performance of the Simulated 2000-m Rowing Race in a Euvhydrated (E) and Dehydrated/Rehydrated (DR) State

<table>
<thead>
<tr>
<th>Subpopulation</th>
<th>DR Before</th>
<th>DR Immediately After</th>
<th>DR After 60 min of Recovery</th>
<th>E Before</th>
<th>E Immediately After</th>
<th>E After 60 min of Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>CD3$^+$</td>
<td>121 ± 42</td>
<td>289 ± 80 $^a$</td>
<td>105 ± 13</td>
<td>132 ± 26</td>
<td>254 ± 51 $^a$</td>
<td>87 ± 21</td>
</tr>
<tr>
<td>CD3$^+$/4$^+$</td>
<td>83 ± 63</td>
<td>134 ± 36 $^a$</td>
<td>57 ± 14 $^b$</td>
<td>79 ± 20</td>
<td>110 ± 21 $^a$</td>
<td>46 ± 13 $^b$</td>
</tr>
<tr>
<td>CD3$^+$/8$^+$</td>
<td>53 ± 36</td>
<td>199 ± 81 $^a$</td>
<td>41 ± 9</td>
<td>48 ± 11</td>
<td>174 ± 51 $^a$</td>
<td>31 ± 8</td>
</tr>
<tr>
<td>CD3$^-$/16$^+$</td>
<td>16 ± 11</td>
<td>180 ± 79 $^a$</td>
<td>22 ± 12</td>
<td>17 ± 4</td>
<td>124 ± 58 $^a$</td>
<td>11 ± 5</td>
</tr>
<tr>
<td>CD4$^+$/25$^+$</td>
<td>6.0 ± 6.7</td>
<td>7.1 ± 4.6</td>
<td>2.9 ± 2.2 $^c$</td>
<td>3.2 ± 2.9</td>
<td>5.7 ± 3.1</td>
<td>2.6 ± 1.5 $^c$</td>
</tr>
</tbody>
</table>

$^a$ = significantly different from before and after 60 min of recovery, $P < .05$.

$^b$ = significantly different from before and immediately after exercise, $P < .05$.

$^c$ = significantly different from immediately after exercise, $P < .05$. 
**Discussion**

It was hypothesized that a reduction in body mass of lightweight oarswomen resulting from a 24-hour period of fluid restriction to produce dehydration, followed by a 2-hour rehydration period, would impair performance of a simulated 2000-m rowing race, induce a greater elevation of tympanic temperature, and initiate a greater suppression in postexercise immune responses when compared with the same exercise bout performed in a euhydrated condition. The experimental protocol used in the current study was sufficient to produce a significant 3.3% reduction in body mass and the 2-hour rehydration period was able to return body mass to baseline before the exercise trial. Contrary to our hypothesis, no significant differences in mean 2000-m rowing race performance times were observed between the dehydration/rehydration and the euhydrated trials. However, tympanic temperature was significantly elevated in the dehydration/rehydration trial compared with the euhydrated trial at all time points, thus supporting this part of our hypothesis. The varying effect of the dehydration/rehydration protocol on some of the immune parameters measured only partially supports our hypothesis that immune cell numbers and function would be affected.

**Figure 3** — Lymphocyte proliferation in response to stimulation with concanavalin A (ConA) or phorbol myristate acetate (PMA), before, immediately after, and after 60 minutes of recovery from a 2000-m simulated rowing race in the euhydrated (E) and dehydrated/rehydrated (DR) trials. Lymphocyte proliferation is presented as a stimulation index (SI). Values are means ± SD. a = significantly different from immediately before exercise for all conditions, $P < .05$. b = significantly different from immediately before exercise for ConA-stimulated response, $P < .05$. 

![Figure 3](image-url)
The relative reduction in body mass following the dehydration/rehydration protocol used in the current study exceeded the suggested ≥2% loss of body mass previously reported to be sufficient to produce impairment in exercise performance\textsuperscript{26} as well as other physiological changes, such as a decrease in cardiac output as a result of dehydration\textsuperscript{27}. However, the dehydration/rehydration protocol in the current study did not negatively affect the performance time of the simulated 2000-m rowing race. The simulated 2000-m bout of exercise may not have been long enough to be negatively influenced by the dehydration/rehydration protocol. In addition, the rehydration protocol in the present investigation may have restored a greater relative percentage of the body mass lost due to dehydration and

\textbf{Figure 4} — Neutrophil oxidative burst before, immediately after, and after 60 minutes of recovery from a 2000-m simulated rowing race in euhydrated (E) and dehydrated/rehydrated (DR) trials. Oxidative burst is represented as mean channel fluorescence of neutrophils at 0, 5, 10, and 15 minutes following stimulation with PMA determined by flow cytometric analysis of the oxidation of dihydrorhodamine-123 to rhodamine-123. Values are means ± SD. a = significantly different from all other time points within sample, $P < .05$. b = significantly different from immediately before exercise and after 60 min of recovery at 10-min stimulation point, $P < .05$. c = significantly different from after 60 min of recovery at 15-min stimulation point, $P < .05$. 

The relative reduction in body mass following the dehydration/rehydration protocol used in the current study exceeded the suggested ≥2% loss of body mass previously reported to be sufficient to produce impairment in exercise performance\textsuperscript{26} as well as other physiological changes, such as a decrease in cardiac output as a result of dehydration\textsuperscript{27}. However, the dehydration/rehydration protocol in the current study did not negatively affect the performance time of the simulated 2000-m rowing race. The simulated 2000-m bout of exercise may not have been long enough to be negatively influenced by the dehydration/rehydration protocol. In addition, the rehydration protocol in the present investigation may have restored a greater relative percentage of the body mass lost due to dehydration and
therefore was able to reduce any negative effect on rowing performance. Both Slater et al\textsuperscript{14} and Burge et al\textsuperscript{13} reported significantly reduced rowing performance using dehydration/rehydration protocols, which may have been due to a greater mean body mass reduction of 4.3\textsuperscript{%}\textit{14} and 5.15\textsuperscript{%}\textit{13} in these studies compared with the current study. In addition, Burge et al\textsuperscript{13} and Slater et al\textsuperscript{14} used prior exercise as a means to reduce body mass in their participants to a greater extent than in the current study, and this may have produced a greater amount of residual fatigue, contributing to the impaired exercise performance in their studies.

We were able to show an elevation in tympanic temperature following the simulated rowing race that was significantly higher after the dehydration/rehydration protocol than in the euhydrated trial. It was also noted that tympanic temperature was significantly elevated at all times in the dehydration/rehydration condition, which may have been due to the light exercise performed by several subjects to achieve the required body mass loss. While these observed increases in tympanic temperature did not reach a clinical level known to cause a heat illness, Slater et al\textsuperscript{14} found that rowing performance was decreased in a hot versus neutral environment in their subjects who had followed a body mass restriction protocol. It is important to note that intense exercise outside of a controlled laboratory setting, under different environmental conditions and/or varying levels of body mass reduction using dehydration can amplify physiological changes that can negatively impact performance and intensify postexercise immune suppression.\textsuperscript{9,17,27–29} Further research is certainly required to ensure safe practice in body mass reduction for the variety of both indoor and outdoor sporting events with body mass categories in light of all the potential negative health implications.

The significant differences in neutrophil, white blood cell, and CD3\textsuperscript{−}/16\textsuperscript{+} (NK) cell concentrations between the two experimental conditions partially support our hypothesis that the immune system would be differentially altered by a dehydration/rehydration protocol. However, there were no significant differences between the dehydration/rehydration and euhydration conditions for other immune cell concentrations or for the two functional assessments of the immune system (lymphocyte proliferation and neutrophil oxidative burst activity). Our data suggest that the innate and acquired immune function in the immediate postexercise period was suppressed and may also have influenced T cell response during recovery. This was evidenced by the combination of a reduction in lymphocyte proliferation and neutrophil oxidative burst activity despite increases in the blood concentration of these cells, the suppressed response to the T cell mitogen ConA, and the lower concentration of CD3\textsuperscript{+/4\textsuperscript{+}} (helper/inducer T cells) and CD4\textsuperscript{+/25\textsuperscript{+}} (activated CD4\textsuperscript{+} cells expressing the IL-2 receptor) lymphocytes after exercise in both experimental conditions. The concentrations of blood leukocytes and neutrophils were highest immediately following exercise in the dehydration/rehydration trial, but this response was delayed until after 60 minutes of recovery in the euhydrated condition. This is supported by Nielsen et al,\textsuperscript{30} who found that the neutrophil concentration peaked in recovery following an initial bout of short-duration, high-intensity rowing exercise in a euhydrated state. However, it is possible that the significant elevation of CD3\textsuperscript{−}/16\textsuperscript{+} cells during the dehydration/rehydration trial in
the current study was influenced by the elevated body temperature. Other research has shown that exercise in the heat can exacerbate postexercise immune suppression. Furthermore, Kappel et al. showed that elevated body temperature increased the number of circulating natural killer cells but other research showed that 60 minutes of endurance exercise in a hot or cold environment did not differ in natural kill cell activity. Thus, further research is recommended to determine the relative influence of these various stimuli on immune responses and their clinical significance.

Nielsen et al. also reported increases in leukocyte concentrations and increased immune cell activity (both natural killer and lymphokine-activated killer cell activities) following a 6-minute “all-out” rowing exercise bout followed by a return to resting values in most of these variables after 2 hours of recovery. These results along with our data suggest that the postexercise alteration in immune response induced by a single bout of high-intensity, short-duration exercise may persist only for a short period of time. In a different study, Nielsen et al. have also shown that rowing exercise induces an alteration in certain immune responses and suggested that this could potentially put an athlete at greater risk of contracting an illness with consecutive exercise bouts. Furthermore, Shade et al. suggest that frequent body mass cycling may also have negative long-term effects on the immune system, as supported by reduced natural killer cell activity in women who frequently and intentionally lost body mass. Thus, the long-term effects of repeated weight-cutting practices over time (eg, competition or training periods) on the immune system have not been adequately investigated.

There are several limitations of the current study. Due to the restricted availability of competitive lightweight oarswomen eligible to participate in our study, the sample size was small and this reduced the statistical power of our experimental design. Despite the small sample size, each subject completed both experimental procedures to improve our statistical power. Secondly, the use of tympanic temperature has been criticized as it tends to overestimate true core temperature, producing higher body temperature readings than other methods such as rectal measurements. However, the tympanic thermometer was checked for accuracy, duplicate measurements were taken, and the same device was used for all measurements during the randomly assigned experimental protocols. Tympanic temperature is practical and is sensitive to changes in core temperature, and these changes occur in parallel with other core temperature measurements albeit consistently higher in response. As well, some of the observed immune responses may have been partially influenced by performing light exercise before the dehydration/rehydration trial. This was done to achieve the final body mass reduction goal and to simulate current practice of dehydration-induced body mass reduction and has been used in previous research designs. As a result, this may have initiated some changes in the immune system before the experimental trial itself, resulting in the differential timing of peak blood concentrations of immune cells following exercise. Lastly, no direct measurement of body dehydration status was made but by restricting only fluid intake over the previous 24-hour period, and it was assumed that the majority of this body mass loss was due to body fluid loss.
Practical Applications and Conclusions

Castell et al\textsuperscript{11} has found that rowers report a high incidence of illness such as upper respiratory track infections in comparison with several other athlete groups. Since lightweight rowers practice weight cutting before racing, the combination of this and high-intensity exercise may increase the susceptibility of these athletes to contracting these types of illness. The results of this investigation demonstrated that a 24-hour fluid restriction period to produce a significant reduction in body mass followed by a 2-hour rehydration period did not significantly impair simulated 2000-m rowing race performance in female lightweight rowers. However, there was a significantly greater change in tympanic temperature during and after the dehydration/rehydration protocol, suggesting a higher level of heat stress with this protocol. The performance of the high-intensity, short-duration rowing race did elicit significant alterations in some immune cell responses immediately following exercise in both experimental trials. As well, blood leukocyte and neutrophil concentrations were significantly greater in the dehydration/rehydration trial. It was concluded that a single session of body mass reduction using fluid restriction practices did not negatively impact performance but can differentially influence tympanic temperature and some aspects of the immune response following high-intensity rowing. Thus, despite a similar performance outcome, caution is advised to coaches and athletes who practice fluid restriction for body mass reduction before exercise and competition.

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References


