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ORIGINAL ARTICLE

Aqua cycling for immunological recovery after intensive, eccentric exercise

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Abstract

Purpose Alterations in immunological homeostasis induced by acute exercise have been frequently reported. In view of the growing amount of repetitive exercise stimuli in competitive sports, quick recovery plays a superior role. Therefore, we examined whether aqua cycling affects cellular immunological recovery.

Methods After performing 300 countermovement jumps with maximal effort male sport students ($n = 20$; 24.4 ± 2.2 years) were randomized into either an aqua cycling (AC) or a passive recovery (P) group. AC pedaled in chest-deep water without resistance, while P lay in a supine position. Each recovery protocols lasted 30 min. Blood samples were taken at *Baseline, Post-exercise, Post-recovery* and *1 h (h), 2 h, 4 h, 24 h, 48 h* and *72 h* after recovery. Outcomes comprised white blood cell (WBC) counts, lymphocyte (LYM) counts and LYM subsets (CD4/CD8 ratio). Additionally, cellular inflammation markers (neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR) and systemic immune-inflammation index (SII)) were calculated.

Results In both groups, WBC, NLR and SII were significantly increased compared to *Baseline* up to and including *4 h* after recovery. Significant interaction effects were found for WBC (*Post-recovery*, *2 h* and *4 h*), NLR (*Post-recovery*), SII (*Post-recovery*) and CD4/CD8 ratio (*2 h*) with values of AC being higher than of P.

Conclusions Interestingly, AC provoked a stronger but not prolonged immunological disturbance than P. NLR and SII may present simple, more integrative markers to screen exercise-induced alterations in immune homeostasis/recovery in athletes and clinical populations. More research is warranted to elucidate the clinical and practical relevance of these findings.

Keywords Exercise · Recovery · Aqua cycling · Inflammation · Immune cells

Abbreviations

AC	Aqua cycling
CD4 cell	T-helper cell
CD8 cell	Cytotoxic T cell
CMJs	Countermovement jumps
IL	Interleukin
LYM	Lymphocyte
NK-cells	Natural killer cells
NLR	Neutrophil/lymphocyte ratio
P	Passive recovery
PLR	Platelet/lymphocyte ratio
SII	Systemic immune-inflammation index
URTI	Upper respiratory tract infection
WBC	White blood cell

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Introduction

In competitive sports, quick recovery after strenuous exercise is of high interest to restore performance capacity as soon as possible. Beside reduction in performance capacity and delayed onset of muscle soreness this recovery period is also characterized by transient alterations in immune

function (Pizza et al. 1995). In this context, the most common approach is the so-called 'open-window' hypothesis, suggesting immune suppression (e.g., decreased lymphocyte (LYM) counts) within 24 h following exercise, thus, possibly resulting in a higher susceptibility to upper respiratory tract infection (URTI) (Kakanis et al. 2010; Pedersen and Toft 2000). However, this theory is discussed controversially; recent evidence suggests that decreased immune cell counts post-exercise might rather represent a redistribution to peripheral target tissues than an actual loss of cells (Edwards et al. 2007; Campbell and Turner 2018).

Early studies on the acute exercise-induced alterations in immune function focused predominantly on cellular responses. The most commonly described cellular response is an increase in WBC counts during exercise lasting for several hours, followed by a return to baseline within *24 h* (Pizza et al. 1995; Shek et al. 1995; Malm et al. 2000). However, when interpreting an increase in total WBC counts, it is important to consider the divers responses of WBC subsets with different functional characteristics (e.g., NK-cells, T-helper cells, etc.). Additionally, cytokine levels, such as interleukin (IL)-6 or IL-10, have also been assessed to investigate an exercise-induced alteration in immune function (Ostrowski et al. 1999). In comparison to changes in immune cell counts, cytokine levels outline a slightly delayed response. Both, alterations in immune cell counts and cytokine levels, can be described as an immunological disturbance, the meaning of 'disturbance' being neutral in this context.

While most of the literature focuses on the impact of aerobic exercise on immunological homeostasis, there is only limited evidence concerning the effect of resistance exercise on the immune system. So far, it was shown that eccentric resistance exercise increases total WBC counts and (anti-) inflammatory cytokine levels (Nieman et al. 1995; Izquierdo et al. 2009), indicating a similar effect as described in response to aerobic exercise. In this context, the high amount of muscle damage is suspected to provoke a strong immunological response. Since eccentric exercise is characteristic for jumping sports, such as volleyball and basketball (Abdelkrim et al. 2007; Turpin et al. 2008), comparable alterations in immune function might occur frequently following training or competition.

To restore performance capacity as quickly as possible after training or competition, multiple recovery methods, including active recovery, compression, massage or water immersion, have been investigated (Cheung et al. 2003; Barnett 2006). Aqua cycling (AC) combines several aspects of these methods (Versey et al. 2013; Rewald et al. 2017). Whether AC also affects the restoration of immunological homeostasis has not been examined up to date. While hydrostatic pressure is known to augment during water immersion (Versey et al. 2013), AC links this mechanism to active recovery. In response, cardiac output and blood flow increase and diffusion of metabolic waste products from muscle to blood may be facilitated by improved cellular diffusion gradients (Wilcock et al. 2006). Furthermore, exercise-induced formation of edema may cause capillary constriction, resulting in deteriorated oxygen, nutrient and hormone supply. Due to augmented hydrostatic pressure during AC, development of edema may be reduced or even prevented, thereby possibly abbreviating recovery time (Wilcock et al. 2006). Since AC combines different physiological mechanisms, it may represent a promising recovery strategy that potentially improves restoration of immunological homeostasis.

Against this background, the aim of this study is (i) to investigate the impact of AC as a recovery method on the restoration of cellular immunological homeostasis and (ii) to examine the kinetics of immunological recovery in response to an intensive, eccentric exercise bout. Aside from typically assessed WBC and LYM counts as well as immune cell subsets (CD4/CD8 ratio), cellular inflammation markers (neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR), systemic immune-inflammation index (SII)) are calculated in this study. These inexpensive markers are well known and frequently used in different clinical contexts (Geng et al. 2016; Fest et al. 2018; Yang et al. 2018). Here, we present the first investigation transferring NLR, PLR and SII into the field of exercise immunology. We hypothesize that the intensive, eccentric exercise bout causes an immunological disturbance over time and that AC improves immunological recovery compared to passive recovery (P) indicated by a shorter recovery time.

Methods

This study was approved by the local ethics committee of the German Sport University Cologne. All participants signed written informed consent prior to study participation. A detailed description of the methodological procedures of this study was previously published elsewhere (Wahl et al. 2017).

A randomized controlled study design was applied. In total, 20 healthy male sport students (age 24.4 ± 2.2 years; weight 81.6 ± 7.6 kg; height 184.9 ± 5.9 cm—values as mean ± standard deviation) participated in this study. Participants of other age or sex were not considered for inclusion to obtain a homogenous sample. The testing procedure started between 8:00 and 9:00 a.m. for all participants. First, 300 countermovement jumps (CMJs) without arm motion were performed by all participants to represent an acute bout of intensive physical exercise with a high amount of eccentric load. Participants ought to perform the CMJs with maximal effort every 8 s. Second, participants were randomly allocated into either an AC or P group. Both recovery protocols started 45 min after completion of the 300 CMJs and lasted

30 min. Participants of AC pedaled under thermoneutral conditions (31 °C) in chest-deep water at 65–75 revolutions per minute without additional resistance. Participants of P laid in a supine position for the same period of time (room temperature 21 °C). Blood samples were collected at the following measurement time points: in a resting condition 45 min prior to the 300 CMJs (*Baseline*), immediately after the 300 CMJs (*Post-exercise*), immediately after recovery (*Post-recovery*) as well as *1 h, 2 h, 4 h, 24 h, 48 h* and *72 h* after recovery.

Outcomes and assessments

Blood panel outcomes

The blood samples of all measurement time points were analyzed using an automated hematology analyzer (Sysmex KX-21N Automated, Sysmex Deutschland GmbH, Norderstedt, Germany) to obtain a complete blood panel, providing information on WBC, LYM, neutrophil and platelet counts. Total WBC and LYM counts were used as an outcome. In addition, the cellular inflammation markers NLR, PLR and SII were calculated using total LYM, neutrophil and platelet counts. NLR was calculated as neutrophil counts$_{[10^3/\mu l]}$/lymphocyte counts$_{[10^3/\mu l]}$, PLR was calculated as platelet counts$_{[10^3/\mu l]}$/lymphocyte counts$_{[10^3/\mu l]}$ and SII was calculated as platelet counts$_{[10^3/\mu l]}$ × neutrophil counts$_{[10^3/\mu l]}$/lymphocyte counts$_{[10^3/\mu l]}$.

Flow cytometry outcome

To further analyze the immunological response to intensive, eccentric exercise, the CD4/CD8 ratio was calculated as a marker for cellular immunological homeostasis. Proportions of CD4 cells and CD8 cells were assessed using flow cytometry (FACS Array, Becton Dickinson, Heidelberg, Germany). Therefore, blood samples were stained with anti-CD3 PE-Cy7, anti-CD4 APC and anti-CD8 PE (Becton Dickinson, Heidelberg, Germany). Gating strategies to identify $CD3^+CD4^+$ T-helper cells and $CD3^+CD8^+$ cytotoxic T cells were applied as presented in Wenning et al. (2013). The CD4/CD8 ratio was assessed at the measurement time points *Baseline*, *2 h* and *72 h*.

Statistical analysis

To detect potential time and/or interaction (time × group) effects separate 2 × 9 mixed covariance analyses (ANCOVA) were performed for the following outcomes: WBC, LYM, NLR, PLR and SII. Concerning the CD4/CD8 ratio, effects were analyzed using a 2 × 3 mixed ANCOVA. Baseline levels of each outcome were used as covariate. All parameters were tested for normality using Shapiro-Wilk test before further statistical analyses were conducted. Sphericity was assessed with Mauchly's test; in case of violation, Greenhouse-Geisser correction was used. Significant ANCOVA results are presented with p and F values, degrees of freedom (dF) and partial eta-squared ($P\eta^2$). In case of significant ANCOVA results, post hoc tests (Bonferroni) were conducted to further investigate within- and between-group differences. Effect sizes (Cohen's d) are presented for post hoc test pairwise comparisons over time. Level of significance is set at equal or less than 0.05. All statistical procedures were conducted using SPSS Version 25 (IBM®, Armonk, NY, USA).

Results

ANCOVA results for WBC revealed a significant time ($p = 0.04$) and interaction effect ($p = 0.003$). Regarding the time effect in AC, values were significantly increased compared to *Baseline* at *Post-exercise* ($p = 0.002$, $d = 3.745$), *Post-recovery* ($p < 0.001$, $d = 5.563$), *1 h* ($p < 0.001$, $d = 6.809$), *2 h* ($p < 0.001$, $d = 10.212$) and *4 h* ($p < 0.001$, $d = 9.496$). Similarly, in P WBC counts were significantly increased compared to *Baseline* at *Post-exercise* ($p < 0.001$, $d = 3.036$), *Post-recovery* ($p = 0.016$, $d = 1.498$), ($p < 0.001$, $d = 3.335$), *2 h* ($p < 0.001$, $d = 3.711$) and *4 h* ($p < 0.001$, $d = 3.926$). Significant interaction effects for WBC were detected at *Post-recovery* ($p = 0.001$), *2 h* ($p = 0.021$) and *4 h* ($p = 0.010$) with values of AC being higher than those of P. A significant time effect ($p = 0.018$) but no interaction effect was revealed examining LYM counts. Post hoc test for AC revealed significant increases compared to *Baseline* at *Post-exercise* ($p = 0.002$, $d = 3.325$), *Post-recovery* ($p = 0.002$, $d = -1.459$) and *1 h* ($p = 0.007$, $d = -1.36$). Regarding P, LYM counts were elevated compared to *Baseline* at *Post-exercise* ($p = 0.017$, $d = 0.961$), *Post-recovery* ($p = 0.001$, $d = -0.958$) and *1 h* ($p = 0.031$, $d = -0.582$). For NLR a significant time ($p = 0.004$) and interaction effect ($p = 0.030$) was found. In AC, values were increased compared to *Baseline* at *Post-recovery* ($p < 0.001$, $d = 7.291$), *1 h* ($p < 0.001$, $d = 7.823$), *2 h* ($p < 0.001$, $d = 6.009$) and *4 h* ($p < 0.001$, $d = 3.795$). In P, values were increased compared to *Baseline* at *1 h* ($p < 0.001$, $d = 8.222$), 2 h ($p < 0.001$, $d = 5.809$) and *4 h* ($p < 0.001$, $d = 6.477$). A significant interaction effect for NLR was only found at *Post-recovery* ($p = 0.031$). For PLR, a significant time effect ($p = 0.039$) but no significant differences compared to *Baseline* were observed. ANCOVA results of SII revealed a significant time ($p = 0.001$) and interaction effect ($p = 0.007$). Concerning the time effect in AC, the values increased significantly compared to Baseline at *Post-recovery* ($p < 0.001$, $d = 6.508$), *1 h* ($p < 0.001$,

$d = 6.698$), *2 h* ($p < 0.001$, $d = 5.205$) and *4 h* ($p = 0.001$, $d = 3.761$). In P, values increased significantly compared to *Baseline* at *Post-exercise* ($p = 0.045$, $d = 2.919$), *1 h* ($p = 0.001$, $d = 5.116$), *2 h* ($p < 0.001$, $d = 5.224$) and *4 h* ($p = 0.001$, $d = 4.620$). The significant interaction effect indicated higher values in AC compared to P at *Post-recovery* ($p = 0.015$). In view of CD4/CD8 ratio a significant interaction effect ($p = 0.044$) was observed at *2 h* ($p = 0.014$), with values of AC being higher than those of P. All results and significant differences are displayed as raw data (mean ± standard deviation) in Fig. 1. Detailed ANCOVA results are shown in Table 1.

Discussion

In accordance with our hypothesis, the applied exercise modality of 300 CMJs resulted in a cellular immune disturbance lasting up to and including *4 h* post-recovery. In AC, cellular immune disturbance was significantly greater than in P and in both groups WBCs, LYMs, NLR, PLR and SII returned to baseline levels within *24 h* after recovery. Therefore, the results of this study suggest a stronger but not shortened immune response in AC contradicting the second part of our hypothesis.

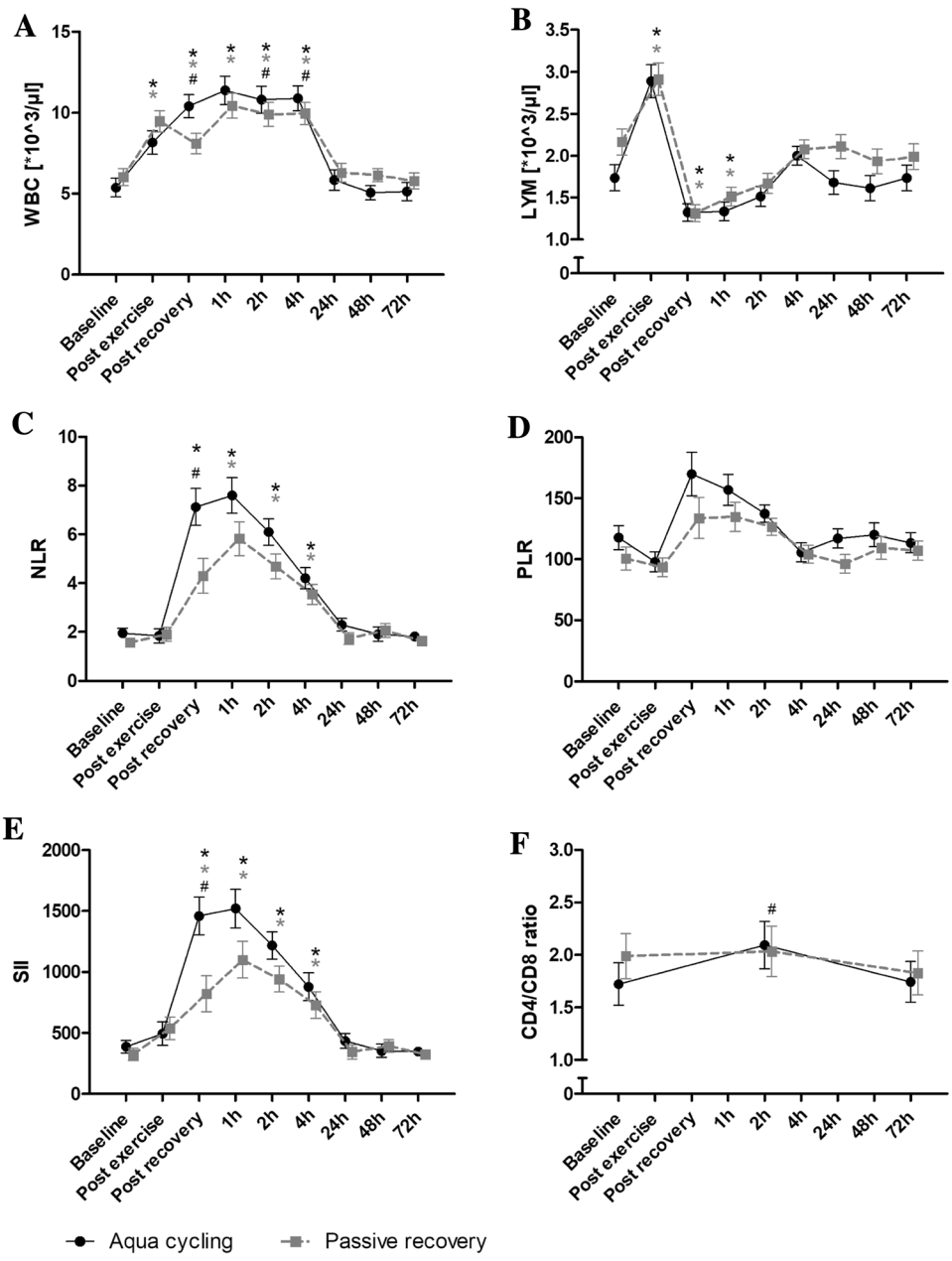

Fig. 1 Kinetics of **a** white blood cell (WBC) counts, **b** lymphocyte (LYM) counts, **c** neutrophil/lymphocyte ratio (NLR), **d** platelet/lymphocyte ratio (PLR), **e** systemic immune-inflammation index (SII) and **f** CD4/CD8 ratio. Values are presented as means ± SE of raw data. Indicated significant differences refer to ANCOVA results. Asterisk denotes significant difference compared to *Baseline* within AC. Grey asterisk denotes significant difference compared to *Baseline* within P. #Significant interaction effect

Table 1 Baseline-adjusted ANCOVA results for all outcome measures

Outcome	Group size	ANCOVA time	ANCOVA time × group
WBC ($\times 10^3$/µl)	AC ($n=8$) P ($n=10$)	$F=3.694$ $dF=3.159$ $p=0.017$* $P\eta^2=0.198$	$F=6.635$ $dF=3.159$ $p=0.001^{\#}$ $P\eta^2=0.307$
LYM ($\times 10^3$/µl)	AC ($n=9$) P ($n=10$)	$F=3.388$ $dF=1.502$ $p=0.063$* $P\eta^2=0.175$	$F=1.387$ $dF=1.502$ $p=0.264$ $P\eta^2=0.080$
NLR	AC ($n=9$) P ($n=10$)	$F=5.599$ $dF=2.465$ $p=0.004$* $P\eta^2=0.259$	$F=3.531$ $dF=2.465$ $p=0.030^{\#}$ $P\eta^2=0.181$
PLR	AC ($n=9$) P ($n=10$)	$F=3.115$ $dF=2.786$ $p=0.039$* $P\eta^2=0.163$	$F=1.164$ $dF=2.786$ $p=0.332$ $P\eta^2=0.068$
SII	AC ($n=9$) P ($n=10$)	$F=6.329$ $dF=2.833$ $p=0.001$* $P\eta^2=0.283$	$F=4.681$ $dF=2.833$ $p=0.007^{\#}$ $P\eta^2=0.226$
CD4/CD8 ratio	AC ($n=8$) P ($n=7$)	$F=0.179$ $dF=2$ $p=0.837$ $P\eta^2=0.015$	$F=3.559$ $dF=2$ $p=0.044^{\#}$ $P\eta^2=0.229$

AC aqua cycling, *P* passive recovery, *WBC* white blood cell counts, *LYM* lymphocyte counts, *NLR* neutrophil/lymphocyte ratio *PLR* platelet/lymphocyte ratio, *SII* systemic immune-inflammation index, *F F* value, *dF* degrees of freedom, *p* probability value, $P\eta^2$ partial eta-squared

*Significant time effects

#Significant interaction effects

The significant increase in WBC counts following the 300 CMJs indicates a similar effect as previously reported in studies examining aerobic or resistance exercise (Nieman et al. 1995; Shek et al. 1995). The elevated WBC counts within the 4 h following recovery emphasize that an acute bout of eccentric exercise unbalances the cellular immunological homeostasis. Similar kinetics were found for the inflammation markers NLR and SII. In contrast, LYM counts were significantly increased within both groups at *Post-exercise*, but significantly decreased up to and including *1 h*. This response of peripheral LYM counts is in accordance with previous findings (Pedersen et al. 1998; Campbell and Turner 2018). Since WBC counts and inflammation markers approach baseline levels *24 h* after recovery, restoration of cellular immunological homeostasis within this period can be assumed.

Although results of investigations on exercise-induced alterations in immune function are consistent, their interpretation ranges from immune suppression to immune activation. As mentioned above, recent evidence suggests an enhanced immune activation following acute exercise (Campbell and Turner 2018). In this context, decreased LYM counts post-exercise are rather interpreted as a redeployment of immune cells to target tissues than an actual loss. However, we propose that changes in unspecific immune cell populations, such as WBC or LYM counts, do not provide information on whether the immune function is up- or downregulated. In fact, more specific immune cell subsets and its compositions should be considered to evaluate the impact on immune function. The theory of an exercise-induced immune activation is further supported by the transient inflammatory state indicated by the presented inflammation markers.

To explain the demonstrated immunological disturbance following exercise several underlying mechanisms are discussed. First, the high amount of eccentric contractions leads to extensive muscle damage, provoking a corresponding immunological response. Second, increased circulation and blood pressure induce higher shear forces that may promote a passive mobilization of immune cells from the lymphatic system into peripheral blood flow. Although this flushing of lymphatic organs represents a popular explanation of exercise-induced leucocytosis (Peake et al. 2017), it should be scrutinized in regard of the persistent elevation of WBC counts after exercise (here up to 4 h post-recovery). Third, neuroendocrine–immune interactions may contribute to a mobilization of immune cells. During exercise, levels of catecholamines, especially epinephrine, increase and induce mobilizing effects on leukocyte subsets by β-adrenergic signaling (Walsh et al. 2011).

Against the background of an exercise-induced cellular immune disturbance, the aim of this study was to investigate AC for immunological recovery following an acute bout of intensive, eccentric exercise. Here, we detected significant interaction effects between the two groups for WBC, NLR, SII and the CD4/CD8 ratio within 4 h after recovery. Opposing the assumption that AC could improve immunological recovery, it revealed a stronger cellular immune disturbance than P. In detail, the higher WBC counts, NLR, SII and CD4/CD8 ratios might be caused by the additional exercise performed. Whether this stronger but not prolonged cellular immune response has any beneficial effect regarding immune function remains unclear. However, potential effects of AC on alleviation of muscle damage or delayed onset of muscle soreness could be used without impairing restoration of immunological homeostasis. Since no overland cycling group was implemented, the applied study design does not provide information on the isolated effect of AC. Future studies should apply an active overland cycling group adapting equal exercise modalities. Furthermore, the practical application of AC in training or competition scenarios seems to be questionable, especially regarding costs and effort in

team sports, although it combines different physiological mechanisms that may improve immunological recovery.

In view of the CD4/CD8 ratio, the kinetic seems to be similar compared to those of the cellular inflammation markers, although the smaller number in measurement time points must be mentioned as a methodological limitation. To capture exercise-induced effects on immunological homeostasis, the calculated inflammation markers may represent a suitable option, not only because of its established use in clinical context but also considering different immune cell subsets. In contrast to commonly used methodological procedures in this field (e.g., flow cytometry, enzyme-linked immunosorbent assays), the required financial and temporal resources are comparatively low. Following studies may directly compare the response of the inflammation markers presented in this study with humoral inflammatory mediators and immune cell subsets identified using flow cytometry. Further research with larger and more heterogeneous sample sizes in terms of age, sex and training status is needed to investigate the transferability of our results.

Conclusion

In conclusion, the intensive, eccentric exercise bout, which is related to competitive jumping sports, resulted in a significant cellular immune disturbance up to and including 4-h post-recovery. The investigated recovery method AC provoked a stronger but not prolonged immunological disturbance than P. Further investigation is needed to compare AC with overland cycling and passive recovery. Moreover, this study is the first to transfer cellular immune markers that are mainly used in a clinical context into the field of exercise immunology. These markers may represent a valuable option to routinely examine (anti-)inflammatory responses and conditions in the field of exercise immunology.

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Author contributions PW, PZ and WB developed the study design. PW conducted the interventions. AS, PW, PZ, NJ and DW conducted statistical analyses. AS designed the figures. NJ, DW, PZ and PW drafted the manuscript. All authors read and approved the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare no conflicts of interest.

Statement of human rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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