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# SARS-CoV-2 infection decreases cardiorespiratory fitness and time-trial performance even two months after returning to regular training — Insights from a longitudinal case series of well-trained kayak athletes

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ABSTRACT

*Objective:* The aims of this study were to examine the effect of SARS-CoV-2 infection on cardiorespiratory fitness (CRF) and time-trial performance in vaccinated well-trained young kayak athletes.
*Methods:* This is a longitudinal observational study. Sixteen (7 male, 9 female) vaccinated kayakers underwent body composition assessment, maximal graded exercise test, and 1000-m time-trial tests 21.9 ± 1.7 days before and 66.0 ± 2.2 days after the SARS-CoV-2 infection. The perception of training load was quantified with Borg's CR-10 scale before and after the infection return to sport period.
*Results:* There were significant decreases in peak oxygen uptake (−9.7 %; effect size [ES] = 1.38), peak oxygen pulse (−5.7 %; ES = 0.96), and peak heart rate (−1.9 %; ES = 0.61). Peak minute ventilation, and minute ventilation/carbon dioxide production slope were unchanged after infection compared to the pre-infection values. In the entire 1000-m, the impaired tendencies were found in completion time, mean power, and mean speed (−2.4 to 1.2 %; small ESs = -0.40 to 0.47) as well as significant changes in stroke rate and stroke length (−4.5 to 3.7 %; ESs = -0.60 to 0.73).
*Conclusion:* SARS-CoV-2 infection decreased CRF and time-trial performance even two months after return to regular training in vaccinated athletes.

## 1. Introduction

Acute respiratory infections (ARinf), which represent the prevailing form of acute illness in athletes, account for nearly 50 % of disease instances during major sporting events.[1,2] Athletes afflicted with ARinf, primarily instigated by viruses including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), confront heightened physical burdens, imparting formidable obstacles upon their return to sport and competitive activities.[1–5] Moreover, the term "return to sport" (RTS), conventionally applied in the context of athletes after an injury, has recently been redefined as a continuum that spans the progression starting from return to training to complete recovery of prior athletic performance levels.[2] However, issues including the potential negative effects of ARinf on athletic performance and the feasibility of recovering previous performance levels through RTS after ARinf are a challenge for athletes, sports scientists, and clinical practitioners alike.[1,2,4,5]

Cardiorespiratory fitness/endurance (i.e., CRF, with the primary variable being peak oxygen uptake [$VO_{2peak}$]) was an essential element of the physical performance in the athletic population, especially during the continuous waves of the coronavirus disease 2019 (COVID-19)

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pandemic.[5,6] However, the effects of ARinf on CRF within the athletic population still produce mixed outcomes.[4–6] Current studies presented significant methodological limitations such as high heterogeneity,[3–6] lack of longitudinal data, the long interval between the tests (i.e., different training phases), and the absence of training load data before and after infection.[4,5] Strictly, the available results to support the causality of ARinf's potential effect on CRF in athletes should be interpreted and generalized with considerable caution due to the aforementioned limitations.[3–6] Furthermore, we recently showed that short-term SARS-CoV-2 infection does not decrease neuromuscular and anaerobic performance in well-trained athletes.[7] In fact, the effect of ARinf on "sports performance" among athletes including measures or variables of athletic success such as race completion times, changes in kinematics, and reactions/adaptations during the training period has been consistently overlooked.[1,2,4] In addition, the constant evolution of the SARS-CoV-2 poses ongoing challenges to global public health.[8] The major variants of SARS-CoV-2 include Delta, Gamma, and Omicron, and different variants may cause different symptoms and disease severity.[9,10] Understanding the impact of different variants on athletes is vital for assessing potential performance challenges and health risks.[1,3] However, most of the previous studies were conducted before the emergence of the Omicron variant,[3] and these results may not be applicable to the present world situation (i.e., most of the prevalent Covid strains were variants of Omicron, such as the current JN.1).[8,10] Therefore, there is an imperative need for better quality and high-homogeneity evidence-based longitudinal data to verify the effects of the new variants (i.e., Omicron) on CRF and sports performance in vaccinated athletes,[3–6] which is likely to be prevalent among a substantial proportion of future scenario.[10,11]

On the other hand, the SARS-CoV-2 infection may cause athletes to cessation of training and disruption of the original training schedule, thus reducing the confidence of athletes and coaches in preparing for their training and competitions in different training phases.[12,13] However, previous longitudinal studies were unable to inform about the effects of SARS-CoV-2 infection in athletes at specific training stages (e.g., preparation or competition period) due to the large heterogeneity of data collection (i.e., the pre-and post-tests were not conducted at the same training phases).[11,14] Receiving more information on the consequences of infection at different training phases will assist athletes, sports/exercise practitioners, and clinical teams in developing RTS and competition strategies after infection.[1–5] Therefore, this study aimed to examine the effect of SARS-CoV-2 on CRF and 1000-m time-trial performance in vaccinated well-trained young kayak athletes.

## 2. Methods

### 2.1. Study design and participants

Sixteen (7 male, 9 female) well-trained sprint kayakers (age 17.6 ± 1.6 yrs, height 175.8 ± 7.2 cm, training experience 2.9 ± 1.6 yrs, typical training volume 21.0 ± 2.7 h $wk^{-1}$; mean ± SD) volunteered to participate in the study. All participants hailed from a provincial training center for water sports in XXX and held the status of competitors at the top level of the national competition within their respective age cohorts. These athletes underwent regular monitoring by the training staff, and individuals who had contracted the virus were included as participants in this study. Every participant in the study provided clear written consent after receiving comprehensive information regarding the study's objectives and extent. The research procedures involving human participants underwent thorough review and gained approval from the ethics committee at Shanghai University of Sport (number of approval: 102772022RT102).

Athletes underwent two tests before and after the infection (Table 1). The pre-test was a regular physical assessment performed during the commencement of the general preparation period (Fig. 1). Based on the suggestions of previous studies,[1,2,4,15] the process of complete RTS was defined as a continuum consisting of three stages in this study. (1) The "return to training (RTT)" is defined as the time (days) from the onset of disease, detraining to start training again after the ARinf.[1,16] (2) The RTS is defined as the time (days) from the start of training again to the first exercise/sports performance test after the ARinf.[1,16] (3) The "return to performance (RTP)" is defined as the time (days) from the onset of the disease to return to previous levels of full exercise/sports performance for professional athletes. All athletes were in identical training stages and possessed similar fitness levels, maintaining their regular training program before the infection. Participants detrained in succession due to the symptom onset (December 17 to 23, 2022). The training center was closed, and the regular training programs were suspended from December 23, 2022, to January 1, 2023; during this period, all participants refrained from any physical activity. They were advised by medical staff to rest in their dormitories, and no individuals required hospitalization or medication. The RTS routine commenced following the infection (January 2, 2023). Regarding the maximal intensity testing early in the post-infection period poses potential risks to the health of the participants, this study used an extended RTS strategy to enable the athletes to fully recover to their health status. The post-test took place after infection and an 8-week RTS period. The settings of all pre- and post-tests were the same. All tests were carried out under standardized resting conditions at an identical time of day, with a 24-h interval between the maximal graded exercise test and the 1000-m time-trial test. Daily assessments were conducted to evaluate the perception of training load before and during the RTS period following infection. The athletes' overall training structure remained unaltered before and after infection, as outlined in our previous study.[7]

**Table 1**
Participants characteristics.

| | | N = 16 |
|---|---|---|
| Age (years) | | 17.6 ± 1.6 |
| Female (n,%) | | 9 (56.3 %) |
| Height (cm) | | 175.8 ± 7.2 |
| Vaccination status (dose) | 1 | 2 (12.5 %) |
| | 2 | 11 (68.8 %) |
| | 3 | 3 (18.8 %) |
| SBP/DBP (mmHg) | | 124.7 ± 8.1/70.2 ± 7.5 |
| $SpO_2$ (%) | | 98.4 ± 0.7 |
| Symptoms duration (days) | | 3.0 ± 1.0 |
| RTT (days) | | 10.4 ± 1.9 |
| RTS (days) | | 46.3 ± 5.1 |
| Interval between pre-test and detected infection (days) | | 21.9 ± 1.7 |
| Interval between detected infection and post-test (days) | | 66.0 ± 2.2 |

Abbreviation: RTT, return to training; RTS, return to sport; SBP, systolic blood pressure; DBP, diastolic blood pressure; $SpO_2$, peripheral oxygen saturation.

All participants were first infected with the SARS-CoV-2 of cluster third-wave outbreak. Between December 1, 2022, and March 2, 2023, the XXX Center for Disease Control and Prevention (chinacdc.cn) reported 20,551 valid genome sequences of SARS-CoV-2 cases in XXX, all of which were identified to the Omicron variant. Athletes underwent nasal swab antigen testing conducted by the medical staff at the training center after the emergence of COVID-19-related symptoms to detect the infection. All participants in this case series were vaccinated before SARS-CoV-2 infection with the type of inactivated vaccine provided by the government. The status of vaccination received by the subjects is shown in Table 1. Our cases were categorized as confirmed general (upper/lower) mild to moderate ARinf.[1,4] To assess the illness's severity, a specific questionnaire was employed to collect data on symptom duration, type of symptoms, and vaccination status. All athletes had no persistent previous symptoms at the post-test. Participants with a medical history (e.g., heart disease, blood vessel disease, asthma, etc.), smoking history, significant musculoskeletal injury, resting blood pressure exceeding 150/90 mmHg (YE655A, YuWell, China), or resting peripheral oxygen saturation ($SpO_2$ [%]) below 95 (YX306, Yuwell, China) were excluded. Blood pressure and $SpO_2$ were measured thrice, with 2-min intervals, calculating mean values. All participants underwent a

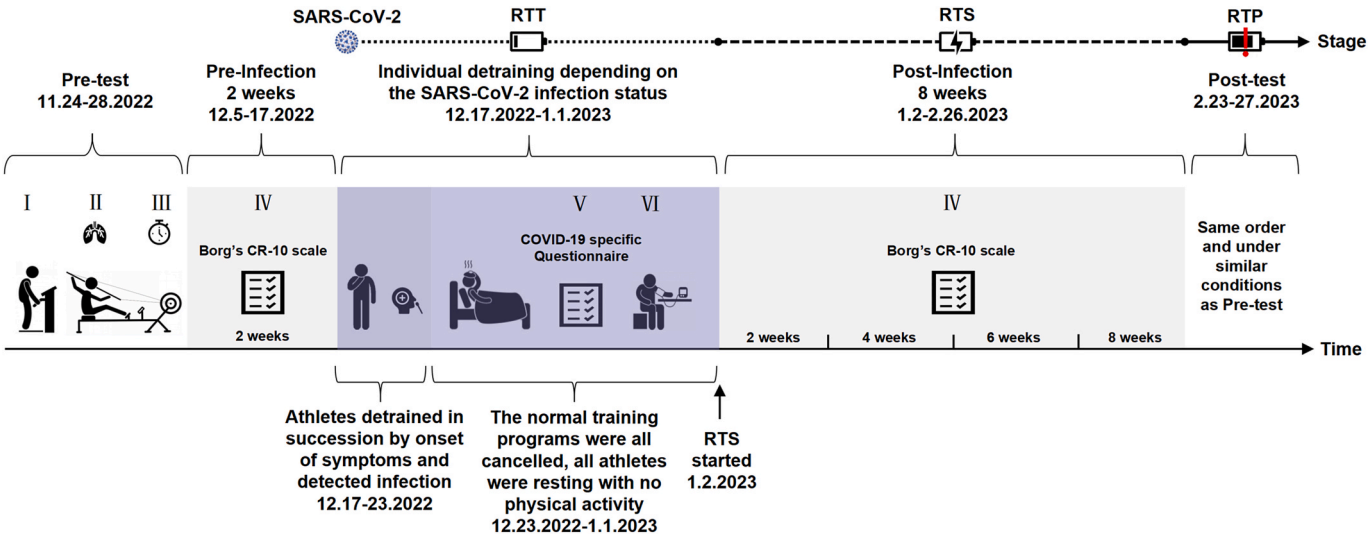


**Fig. 1.** Timeline and data collection. The Roman numerals denote the tests. I, body composition analysis; II, maximal graded exercise test; III, 1000-m time-trial test; IV, daily assessed the perception of training load; V, COVID-19 specific questionnaire; VI, resting blood pressure and peripheral oxygen saturation. Each colored block denotes different periods.
Abbreviation: ARinf, acute respiratory infections; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; COVID-19, coronavirus 2019; RTT, return to training; RTS, return to sport; RTP, return to performance.

Polymerase Chain Reaction test to confirm their negative status (January 11, 2023).

### 2.2. Testing procedures

#### 2.2.1. Body composition analysis

Body composition was measured using a device (IOI353, Jawon Medical, Korea) that utilizes an 8-electrode bioelectrical impedance analysis method, which offers a comparable assessment of body fat mass when compared to Dual Energy X-ray Absorptiometry.[17] Participants were required to fast and abstain from consuming caffeine and alcohol for at least 8-h before the measurement.

#### 2.2.2. Maximal graded exercise test

The athletes performed a 7 × 4-min kayak ergometer maximal graded exercise test (GXT), briefly, which included 6 × 4-min submaximal stages of incremental power output and one final maximal all-out 4-min stage, each stage separated by 1-min of passive recovery.[18] The power output standards (in watts) of each stage and drag-resistance setting (in the arbitrary unit [A.U.]) were determined by the kayakers' sex, age, and performance status, as described before.[18] Before the formal testing, athletes were instructed in separate familiarization tests to guarantee they were familiar with this GXT protocol. All participants were familiar with the kayak ergometer because they frequently used it during the off-season. The GXT proceeded on a kayak ergometer (Dansprint ApS, Hvidovre, Denmark), and the tension in the bungee cords of the ergometer was calibrated using a digital scale (No.6794, XIANGHENG, China) when the drive ropes were extended at 210-cm in length.[18] Before testing, the portable gas analyzer system (MetaMax 3 B, Cortex Biophysik GmbH, Leipzig, Germany) was warmed up 30-min and then calibrated following the manufacturer's instructions with ambient air and a standard gas mixture of known concentration (oxygen [$O_2$]: 15.00 %, carbon dioxide [$CO_2$]: 5.00 %), and the inspiratory flow volume was calibrated with a 3-L syringe. The breath-by-breath ventilatory and gas exchange variables as well as heart rate (Polar H9, Polar Electro, Kempele, Finland) were recorded continuously throughout the test and averaged over 10-s periods. The peak heart rate ($HR_{peak}$) was the peak value obtained during the test. The peak oxygen uptake ($VO_{2peak}$) was defined as the value obtained in the highest continuous 30-s throughout the test (i.e., peak exercise) and calculated using the moving-averages method by the Exercise Threshold App[19] and the time-matched value at peak exercise of variables including $VO_2$/HR, minute ventilation, end-tidal $CO_2$ pressure, and respiratory exchange ratio was defined as peak $O_2$ pulse, $VE_{peak}$, $PETCO_{2peak}$, and $RER_{peak}$, respectively. The minute ventilation/carbon dioxide production slope ($VE/VCO_2$ slope) was determined using least-squares linear regression of data from the initiation of exercise to peak exercise.[20] Each athlete was given powerful and continuous verbal motivation during the GXT by the investigators and coaches.

#### 2.2.3. 1000-m time-trial test

The 1000-m time-trial (TT) test used the same kayak ergometer and the calibration setting with GXT. Before testing, athletes performed a 10-min self-paced pre-race warm-up. After 5-min of passive recovery, the athletes were informed to complete the 1000-m TT in the fastest time possible. The 1000-m TT performance variables such as time, power, distance, and stroke rate were visible on the screen of the ergometer. Each athlete was provided forceful and continuous verbal motivation during the test by the investigators and coaches. The pre- and post-tests for GXT and 1000-m TT used the same ergometer, the same setting of power output standard and drag-resistance for each participant, and the ropes of the ergometer were renewed. The TT performance data was reported as means for each 10-m split value,[21] which comprised completion time (CT), mean power output (MP), mean speed (MS), stroke rate (SR), and stroke length (SL). These variables were analyzed as (1) means over the entire 1000-m, and (2) means in per 250-m splits. The 10-m data for each split were averaged to calculate a mean split value.[21] For instance, the initial twenty-five sets of 10-m data points were averaged, resulting in the mean split value for the first 250-m split.

#### 2.2.4. Training load

Athletes' perception of training load was assessed using the Borg's CR-10 scale. The Borg's CR-10 scale, adapted by Foster et al.,[22] was employed to gauge athletes' session rating of perceived exertion (sRPE) 30 min after each training session. The internal training load (sRPE-TL) was calculated by multiplying an athlete's sRPE value by the session duration in minutes (sRPE-TL = sRPE [A.U.] x session duration [mins]), as previously recommended.[22] Athletes indicated their ratings by touching the appropriate score on a portable computer tablet (iPad©, Apple Inc., California, USA).

### 2.3. Statistical analysis

The data were presented as mean ± standard deviation for continuous variables and as numbers (n) with percentages (%) for categorical variables. The normality of the distribution was assessed using the Shapiro-Wilk test. Paired *t*-tests were employed for normally distributed data, while Wilcoxon signed-rank tests were used for non-normally distributed data to evaluate differences between pre- and post-tests. Cohen's d effect size (ES) was calculated to quantify the magnitude of differences between variables, with interpretations categorized as trivial (0–0.19), small (0.20–0.49), medium (0.50–0.79), and large (≥0.80).[23] Linear mixed-effects models with Bonferroni *post hoc* tests were utilized to assess differences in training load variables between the pre- and post-infection periods. Statistical significance was considered at a significance level of $p < 0.05$. The statistical analyses were conducted using IBM SPSS version 27 (IBM, Armonk, New York, USA) and JASP version 0.17.2.1 (JASP, UvA, Amsterdam, Netherlands).

## 3. Results

The prevalence of self-reported acute symptoms was shown in our previous study,[7] and all athletes had no persistent previous symptoms at the post-test. The differences in testing variables pre- and post-infection are shown in Table 2.

Regarding the TT performance variables of the entire 1000-m (Table 2), there were no statistical changes in $CT_{1000m}$, $MP_{1000m}$, and $MS_{1000m}$, as well as a significant decrease in $SR_{1000m}$ and a significant increase in $SL_{1000m}$ pre- and post-infection. For the TT performance variables of splits per 250-m (Fig. 2B, C, D, E, and F), there were significant increases in $CT_{750–1000m}$ (relative changes [Δ] = 1.17 %; $p = 0.034$; ES = −0.584), $SL_{250–500m}$, and $SL_{750–1000m}$ (Δ = 3.88–4.77 %; $p = 0.026$ to 0.028; ES = −0.618 to 0.606) as well as significant decreases in $MS_{750–1000m}$ (Δ = −1.23 %; $p = 0.021$; ES = 0.645), $SR_{250–500m}$, $SR_{500–750m}$, and $SR_{750–1000m}$ (Δ = −5.12 to −4.56 %; $p = 0.004$ to 0.024; ES = 0.629 to 0.847).

Training hours were significantly decreased during the post-infection 4 weeks period compared with the pre-infection 2 weeks period (21.02 ± 2.70 vs 14.66 ± 4.54 [h·wk$^{-1}$]; Δ = −28.66 %; $p < 0.001$), and no significant changes in other period. sRPE was significantly decreased during the post-infection 2 weeks (Δ = −13.14 %; $p = 0.002$) period as well as significantly increased during post-infection 6 weeks (Δ = 14.42 %; $p = 0.016$) period (Fig. 3A). Regarding internal training load (Fig. 3B), there were significant declines in internal training load per session obtained by sRPE ($sRPE\text{-}TL_{session}$) during the post-infection 2 weeks period (Δ = −13.48 %; $p = 0.010$) and internal training load per week obtained by sRPE ($sRPE\text{-}TL_{week}$) during post-infection 2 weeks (Δ = −18.90 %; $p = 0.001$) and 4 weeks (Δ = −31.45 %; $p < 0.001$) period.

## 4. Discussion

To our knowledge, this is the first longitudinal study to definitively demonstrate that mild SARS-CoV-2 infection decreased CRF and TT performance in vaccinated athletes. The key findings were the following: 1) $VO_{2peak}$, peak $O_2$ pulse, and $HR_{peak}$ were significantly decreased but no changes in $VE_{peak}$ and $VE/VCO_2$ slope, the $PETCO_{2peak}$ and $RER_{peak}$ significantly increased after infection; 2) TT performance deteriorated with significant increases in $CT_{750–1000m}$, $SL_{1000m\&250–500m\&750–1000m}$ as well as significant decreases in $MS_{750–1000m}$, $SR_{1000m\&250–500m\&500–750m\&750–1000m}$ after infection.

The athletes in this case series reported shorter symptom durations (~3 days) compared with other acute respiratory illnesses in previous studies (~7 days).[2] A recent systematic review presented that the majority of SARS-CoV-2-infected athletes (~94 %) remained asymptomatic or had mild symptoms.[3] Only 1.2 % of athletes in a large cohort study (n = 3597) had symptoms that persisted >3 weeks.[24] However, we did not continuously monitor symptoms during the RTS period, which might present with new exertional symptoms (e.g., exercise intolerance) in some individuals.[24] Moreover, the Omicron variants usually cause milder symptoms, particularly in vaccinated individuals.[9] Our findings endorsed vaccination's role in preventing ARinf among athletes due to its favorable benefit-to-risk ratio in previous studies.[25] However, different vaccine types (e.g., inactivated virus, adenoviral vector, and mRNA vaccines), doses (e.g., low or high dose), and timing (e.g., before

**Table 2**
Differences of variables pre- and post-infection.

| | Pre-Inf | Post-Inf | MD (95%CI) | Δ% | *p* | *Cohen's d* |
|---|---|---|---|---|---|---|
| **Body composition** | | | | | | |
| BM (kg) | 68.9 ± 10.3 | 70.8 ± 9.1 | −1.9 ± 1.8 (−2.8 to −0.9) | 3.0 ± 2.6 | <0.001[b] | −1.025 |
| BMI (kg·m$^{-2}$) | 22.2 ± 2.1 | 22.8 ± 1.8 | −0.6 ± 0.6 (−0.9 to −0.3) | 2.9 ± 2.6 | <0.001[b] | −1.050 |
| BF (%) | 19.4 ± 4.6 | 20.1 ± 4.1 | −0.7 ± 1.4 (−1.5 to 0.0) | 4.8 ± 7.6 | 0.051 | −0.531 |
| FATM (kg) | 13.3 ± 3.7 | 14.2 ± 3.4 | −0.9 ± 1.1 (−1.5 to −0.3) | 7.9 ± 8.5 | 0.006[b] | −0.796 |
| FFM (kg) | 55.6 ± 9.1 | 56.6 ± 8.0 | −1.0 ± 1.5 (−1.8 to −0.2) | 2.1 ± 2.9 | 0.020[b] | −0.649 |
| SKM (kg) | 31.1 ± 5.2 | 31.6 ± 4.5 | −0.5 ± 1.5 (−1.3 to 0.2) | 2.2 ± 5.1 | 0.160 | −0.370 |
| **GXT** | | | | | | |
| $VO_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) | 49.24 ± 5.27 | 44.32 ± 4.78 | 4.9 ± 3.6 (3.0–6.8) | −9.7 ± 6.9 | <0.001[b] | 1.376 |
| $VO_{2peaka}$ (ml·min$^{-1}$) | 3381.4 ± 556.0 | 3149.1 ± 589.4 | 232.3 ± 193.5 (129.2–335.4) | −7.0 ± 5.7 | <0.001[b] | 1.201 |
| Peak $O_2$ pulse (ml·beat$^{-1}$) | 18.88 ± 3.26 | 17.82 ± 3.36 | 1.1 ± 1.1 (0.5–1.6) | −5.7 ± 6.2 | 0.002[b] | 0.957 |
| $HR_{peak}$ (beat·min$^{-1}$) | 185.4 ± 6.6 | 181.8 ± 7.1 | 3.6 ± 6.0 (0.4–6.8) | −1.9 ± 3.1 | 0.028[b] | 0.608 |
| $VE_{peak}$ (l·min$^{-1}$) | 121.4 ± 23.7 | 120.7 ± 27.7 | 0.7 ± 10.5 (−4.9 to 6.3) | −0.9 ± 9.4 | 0.799 | 0.065 |
| $VE/VCO_2$ slope[a] | 34.4 ± 3.5 | 34.3 ± 3.4 | 0.1 ± 2.5 (−1.3 to 1.4) | 0.1 ± 7.2 | 0.920 | 0.026 |
| $PETCO_{2peak}$ (mmHg) | 34.4 ± 2.5 | 36.1 ± 2.3 | −1.8 ± 1.9 (−2.8 to −0.8) | 5.3 ± 5.7 | 0.002[b] | −0.935 |
| $RER_{peak}$ | 1.04 ± 0.07 | 1.15 ± 0.05 | −0.1 ± 0.1 (−0.1 to −0.1) | 10.4 ± 7.0 | <0.001[b] | −1.562 |
| **1000-m TT test** | | | | | | |
| $CT_{1000m}$ (s) | 290.3 ± 13.9 | 293.5 ± 9.6 | −3.2 ± 8.1 (−7.5 to 1.1) | 1.2 ± 2.8 | 0.133 | −0.397 |
| $MP_{1000m}$ (w) | 141.0 ± 27.2 | 136.1 ± 19.9 | 4.9 ± 13.1 (−2.1 to 11.9) | −2.4 ± 8.9 | 0.158 | 0.372 |
| $MS_{1000m}$ (km·h$^{-1}$) | 12.4 ± 0.6 | 12.3 ± 0.4 | 0.2 ± 0.3 (0.0–0.3) | −1.2 ± 2.8 | 0.083 | 0.465 |
| $SR_{1000m}$ (stroke·min$^{-1}$) | 94.2 ± 11.9 | 89.5 ± 10.2 | 4.6 ± 6.4 (1.2–8.0) | −4.5 ± 6.7 | 0.011[b] | 0.725 |
| $SL_{1000m}$ (m·stroke$^{-1}$) | 2.2 ± 0.2 | 2.3 ± 0.2 | −0.1 ± 0.1 (−0.1 to 0.0) | 3.7 ± 5.8 | 0.031[b] | −0.597 |

Abbreviation: BM, body mass; BMI, body mass index; BF, body fat; FATM, fat mass; FFM, fat-free mass; SKM, skeletal muscle mass; GXT, maximal graded exercise test; $VO_{2peak}$, peak oxygen uptake; $VO_{2peaka}$, absolute peak oxygen uptake; $O_2$ pulse, oxygen pulse; $HR_{peak}$, peak heart rate; $VE_{peak}$, peak ventilation; $VE/VCO_2$ slope, minute ventilation/carbon dioxide production slope; $PETCO_{2peak}$, peak end-tidal $CO_2$ pressure; $RER_{peak}$, peak respiratory exchange ratio; TT, time-trial; CT, completion time; MP, mean power; MS, mean speed; SR, stroke rate; SL, stroke length.

[a] One athlete's (1 female) pre-test data were excluded due to a break in the midsection that did not affect the peak values of other variables.

[b] Indicates significant changes compared with pre-infection ($p < 0.05$).

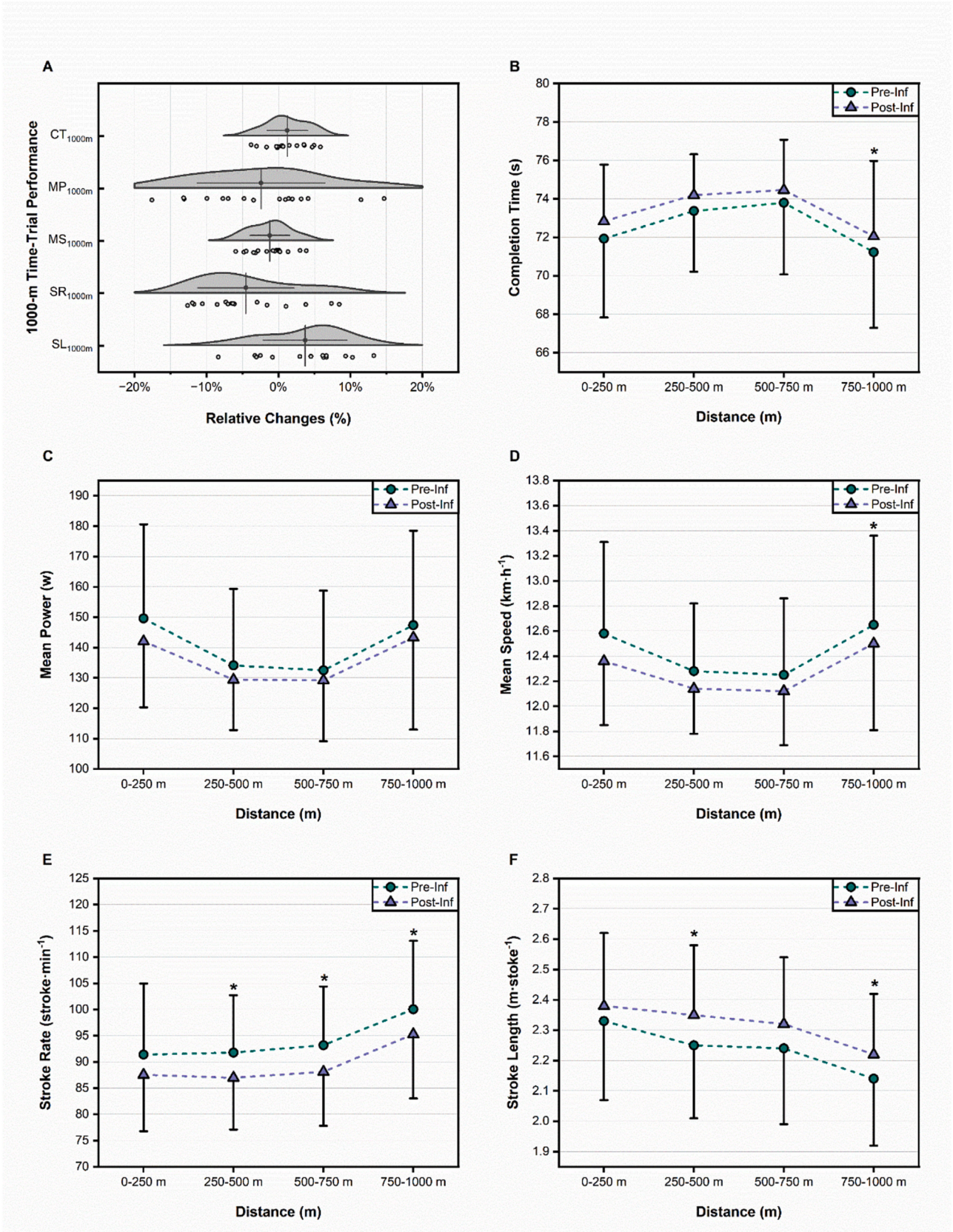


**Fig. 2.** The relative changes of mean values of time-trial performance variables in the entire 1000-m (A) and the differences of performance variables in per 250-m split mean values pre- and post-infection (B, C, D, E, F). B, completion time (CT); C, mean power (MP); D, mean speed (MS); E, stroke rate (SR); F, stroke length (SL). * indicates significant changes compared with pre-infection ($p < 0.05$).

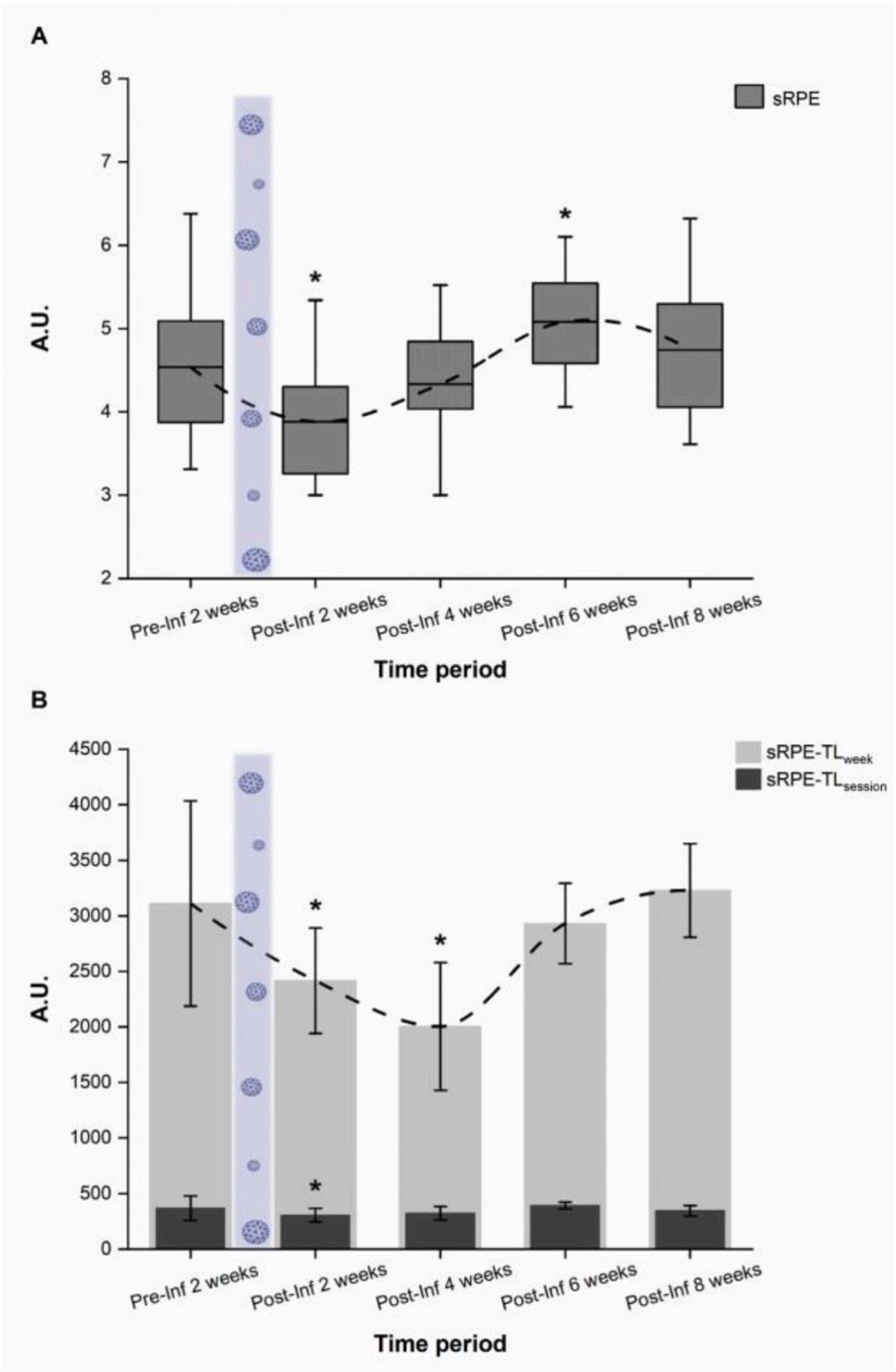


**Fig. 3.** Session rating of perceived exertion (sRPE) (A) and the internal training load obtained by sRPE (B) in different periods of well-trained young kayakers pre- and post-infection. Abbreviation: $sRPE\text{-}TL_{session}$, internal training load per session obtained by sRPE; $sRPE\text{-}TL_{week}$, internal training load per week obtained by sRPE. * indicates significant changes compared with Pre-Infection 2 weeks ($p < 0.05$).

or after infection) of vaccination may affect the effects of prevention and reduction of ARinf symptoms/severity in athletes.[26,27] More evidence is needed to clarify the role of longer-term influences of vaccination (type, dose, and timing) on training in athletes across age and sports disciplines,[25] and new challenges seem to be looming.[8,10] Additionally, fat mass significantly increased after 8 weeks of RTS, which had no significant change during the early-stage RTS period (i.e., after 2 weeks of RTS) in our previous study.[7] This could be attributed to the limitation in appetite caused by symptoms of anosmia/dysgeusia (~47–63 %) in the athletic population after SARS-CoV-2 infection reported by previous studies.[3,24] The abrupt eating habit changes (e.g., overeating) after symptoms may be resolved to result in fat gain.[28] This short-term "feast diet" recently has been demonstrated to cause temporary immunity decline and potential infection exacerbation.[29]

Currently, the available evidence shows mixed results and low confidence regarding the effect of SARS-CoV-2 on CRF in the athletic population.[4–6,30] Our data demonstrated that SARS-CoV-2 infection decreased $VO_{2peak}$ by ~10 % (Table 2). The $VO_{2peak}$ of the infected athletes could be influenced by main three factors including ventilatory/pulmonary, central, and peripheral factors.[6,31] Firstly, the present longitudinal studies indicated that athletes' pulmonary function by spirometry (e.g., forced vital capacity [FVC] and forced expiratory volume in 1 s [$FEV_1$]) did not decline compared to pre-infection data.[32,33] There is also no current evidence that lung diffusing capacity is negatively affected in athletes after SARS-CoV-2 infection.[32,34] Moreover, symptoms of lower respiratory restriction (shortness of breath and chest pain) were minimal in our previous study.[7] The $VE_{peak}$ and $VE/VCO_2$ slope did not significantly change after infection in our case series (Table 2), which is in accordance with previous studies.[32,35] Therefore, in the athletic population, pulmonary/ventilation function might not be the primary limitation of low $VO_{2peak}$ and exercise intolerance in the long term,[5,6] or these factors could be gradually recovered over time.[32,36]

Regarding the central factors, our results showed that Peak $O_2$ pulse and $HR_{peak}$ significantly decreased as well as increases in $PETCO_{2peak}$ after infection (Table 2). The Peak $O_2$ pulse was calculated at this stage of $VO_2$/HR and used to estimate stroke volume (SV).[6,31] According to Fick's equation, the cardiac output (CO) was equal to SV × HR.[31] Due to the previous studies that have examined the positive correlation between $PETCO_{2peak}$ and CO at peak exercise,[37] we hypothesized that in our case series, CO and SV did not decline and may have even been super-normal.[38] Furthermore, deconditioning (usually accelerated HR response) cannot serve as the simple explanation for the low CRF.[6,30,39] There was some evidence that demonstrated the significant decrease in $HR_{peak}$ even after 7 months to >1 year post-infection in the athletic and general population.[35,40,41] We speculated that this might be due to chronic autonomic dysfunction blunting the HR response to exercise,[41,42] which leads to a sub-optimal distribution of supernormal CO in the exercising musculature.[38] Reassuringly, most studies have shown reversibility of CO, SV, and $O_2$ pulse after 3–6 months post-infection.[6,36,40]

Muscle $O_2$ extraction was demonstrated to be more important than central factors ($VO_{2peak}$, CO, and SV) in kayaking performance (i.e., upper-body exercise mode).[43] Previous studies suggest that SARS-CoV-2 might lead to systemic microvascular dysfunction[39,41] and small-fiber neuropathy that induces peripheral shunting.[44] Additionally, recent studies have shown that SARS-CoV-2 disrupts mitochondria as the primary $O_2$ organelles leading to impaired mitochondrial function.[45] Reduced mitochondrial $O_2$ sensing, amount of mitochondrial in muscle cells, and impaired oxidative stress-related thrombocyte function may contribute to oxidative phosphorylation compromise,[45] which might be related to the prolonged anaerobic fatigue (i.e., greater glycolytic metabolism) presented by the larger $RER_{peak}$ in our results (Table 2).[46] We speculated that since no sustained cardiac or pulmonary impairment was observed (which might have been recovered), the $VO_{2peak}$ tested by the upper-body GXT significantly decreased (~10 %) in our well-trained endurance-type athlete's case series might be related to the impaired ability of $O_2$ delivery/extraction[6,30,38,39] However, more studies (e.g., invasive cardiopulmonary exercise testing) are needed to demonstrate the above-postulated mechanisms causing low CRF in athletes after infection.

The tendency for impairment of TT performance was observed in $CT_{1000m}$, $MP_{1000m}$, and $MS_{1000m}$ (small ESs = −0.397 to 0.465), as shown in Table 2. However, small changes could cause significant alterations in the sports performance of professional athletes.[4] The aerobic contribution has been estimated to be ~75 % in 1000-m kayaking events.[47] Additionally, arm muscles exhibit lower oxidative capacity, impaired oxygen extraction, and capillary muscle delivery, increased blood flow variability, and a higher proportion of type 2 muscle fibers compared to leg muscles.[48] These factors contribute to heightened fatigue.[48] These could explain the significant decrease in performance during the latter part ($CT_{750–1000m}$ and $MS_{750–1000m}$) of the TT (Fig. 2B–D). The impaired ability of $O_2$ delivery/extraction and reduction in $VO_{2peak}$ caused by SARS-CoV-2 infection might be more detrimental to endurance performance in this upper-body exercise pattern (e.g., rowing, swimming, cross-country skiing, etc.).[4,7] Furthermore, $SR_{1000m\&250–500m\&500–750m\&750–1000m}$ and $SL_{1000m\&250–500m\&750–1000m}$ significantly changed (Fig. 2E and F). We cannot determine whether these changes were caused by fatigue and/or kinematic changes due to SARS-CoV-2 infection.[49] However, there were few studies in this area.[4] Furthermore, the impairment of SARS-CoV-2 infection and other ARinf on kinematics and dynamic balance and its relationship to injury/performance might be seriously underestimated in athletes in current studies.[4,49]

The internal training load during the later-stage (post-infection 6–8 weeks) of the RTS period has restored to the pre-infection status (Fig. 3B) but the level of perception significantly increased (Fig. 3A), which indicated that potential exercise intolerance may exist and the perceived recovery might be impaired.[15] Underlying mechanisms for this combined response may encompass multiple systems and symptoms (e.g., endothelial dysfunction, chronic fatigue syndrome and inflammation, etc.).[15,41] Finally, the RTS after ARinf might be the same challenging as after injury due to the complex pathological mechanisms of ever-changing viruses (Fig. 4).[1,2] Therefore, the structure, "pacing/-ramp", and dose of the RTS should be rigorously formulated, continuously supervised, and appropriate for each individual.[50] For professional athletes, subsequent research and reports on RTS could be subdivided into three phases, including "return to training", "return to sport" and ultimately "return to performance" (Figs. 1 and 4), which would facilitate the development of effective RTS strategies and the construction of prediction models of return to performance after ARinf.[1,2,4,15]

Some limitations of this study are required to be identified. These limitations mainly related to collecting samples from athletes during peak contraction periods of COVID-19 have feasibility concerns and limitations (i.e. cannot dictate/predict when the infection will occur, and the severity of symptoms may vary). This study had a small sample size and lacked a control group. The interval of pre- and post-test was approximately 3 months. However, our study group was highly homogeneous in terms of methodology including pre-infection data, timing of infection, and athlete characteristics, and all participants were in the same training phase (i.e., general preparation period). The tests of GXT and TT were performed 24 h apart and this could have influenced TT performance. However, this time interval between the two tests was similar when tested before and after infection.

## 5. Conclusions

This is the first longitudinal study to definitively demonstrate that SARS-CoV-2 infection decreased CRF and TT performance even two months after returning to regular training in vaccinated athletes. These results would improve the understanding of the underlying mechanisms of low CRF after ARinf in athletes and assist in the development of more

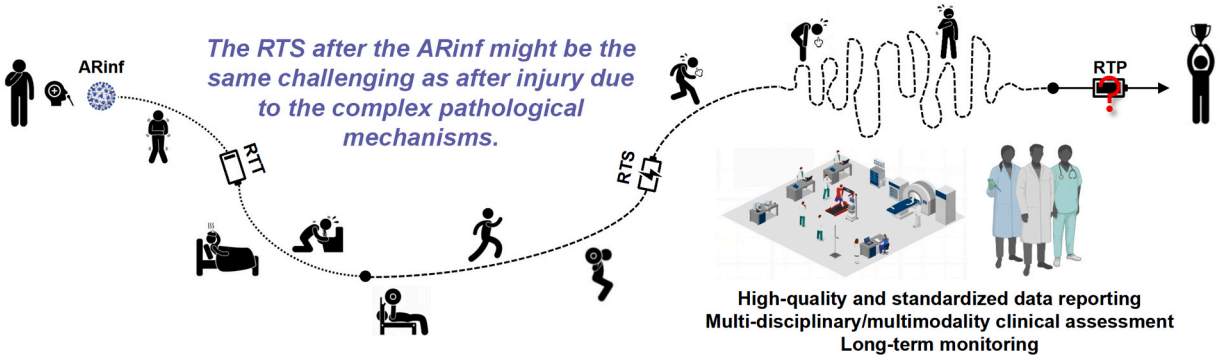


**Fig. 4.** The complete "return to sport" procedure in the athletic population after acute respiratory infections. Abbreviation: ARinf, acute respiratory infections; RTT, return to training; RTS, return to sport; RTP, return to performance.

targeted treatment prescriptions and RTS protocols (e.g., repeated sprint/interval training or altitude/hypoxic training)[51] in athletes. Finally, high-quality and standardized data reporting in response to the International Olympic Committee consensus,[1,2] multidisciplinary and multimodality clinical assessment, and long-term monitoring will support athletes in confronting unpredictable future crises (e.g., new variants such as JN.1, etc.).[8,10]

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## 6. Data availability statement

If reasonably requested the authors of this study will consider providing the data to support their conclusions.

## CRediT authors statement

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## Declaration of competing interest

The authors declare that they have no competing interests.

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