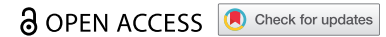







ORIGINAL RESEARCH



# Clinical manifestations of different viral respiratory infections in athletes: implications for risk assessment and return-to-sport - AWARE VII study in 116 cases

Marcel Jooste <sup>a,b</sup>, Nicola Sewry <sup>a,c</sup>, Maarit Valtonen <sup>d</sup>, Marlise Dyer<sup>a</sup>, Esme Jordaan <sup>e,f</sup>  
and Martin Schwellnus <sup>c</sup>

<sup>a</sup>Sport, Exercise Medicine & Lifestyle Institute (SEMLI), University of Pretoria, Pretoria, South Africa; <sup>b</sup>Section Sports Medicine, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa; <sup>c</sup>International Olympic Committee (IOC) Research Centre of South Africa, School of Medicine, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa; <sup>d</sup>Finnish Institute of High Performance Sport KIHU, Jyväskylä, Finland; <sup>e</sup>Biostatistics Unit, South African Medical Research Council, Cape Town, South Africa; <sup>f</sup>Statistics and Population Studies Department, University of the Western Cape, Cape Town, South Africa

## ABSTRACT

**Objectives:** The aims of this study were to describe the etiology of acute respiratory infections (ARinf) in athletic individuals, and to identify differences in the clinical presentation, evidence of possible multi-organ involvement, and illness classification between common pathogen groups.

**Methods:** One-hundred-and-sixteen cases of confirmed ARinf in athletic individuals were evaluated  $\leq 5$  days of the onset of an ARinf. Nasopharyngeal swab multiplex PCR testing was performed to identify a causative pathogen. Symptomatology, clinical examination findings, results of selected blood tests, and the clinical syndrome and illness severity classifications were compared between four common pathogen groups.

**Results:** The etiologies of ARinf in this cohort were: rhinovirus = 34(29%), influenza = 17(15%), SARS-CoV-2 = 15(13%), common coronavirus = 13(11%), 'unidentified' = 16(14%), 'dual pathogen' = 9(8%), and 'other' = 12(10%). Clinical presentation differed among the four common pathogen groups as follows: Influenza had more *total symptoms*, *lower respiratory & regional symptoms*, and *systemic & non-respiratory symptoms* than rhinovirus ( $p \leq 0.002$ ) and common coronavirus ( $p < 0.05$ ). Influenza and SARS-CoV-2 had higher *total symptoms* and *systemic & non-respiratory symptom severity scores* than rhinovirus ( $p \leq 0.0006$  and  $p < 0.03$  respectively) and common coronavirus ( $p \leq 0.03$  and  $p = 0.02$  respectively). Evidence of *other non-respiratory organ involvement* on clinical examination was highest for influenza (53%). Illness classification for pathogen groups differed: common coronavirus had the highest percentage (%) of *rhinitis-like ('common cold')* illnesses (69%), and influenza had the highest % of '*flu-like*' illnesses (82%). Influenza had the highest % of *severe illnesses* (88%) and common coronavirus the lowest (31%). 41% of rhinovirus presented with severe illness.

**Conclusion:** Influenza and SARS-CoV-2 had greater number and severity of symptoms than rhinovirus and common coronavirus. Among the four common pathogen groups, influenza had the highest percentage of abnormal clinical examination and serological findings and severe illnesses. Knowledge of the causative pathogen and the clinical presentation may add value to the risk assessment and guide clinical decision-making in return-to-sport following ARinf in athletic individuals.

## ARTICLE HISTORY

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## KEYWORDS

Respiratory infections; respiratory viruses; return-to-sport; athletes; athletic individuals; clinical assessment; pathogen-specific

## 1. Introduction


Acute respiratory infections (ARinf) are the most common illness among athletes and the leading non-injury-related reason for missing training and competitions [1–3]. Despite this, there are limited data on the etiology and clinical manifestations of pathogen-specific ARinf in athletes.

Of the limited studies, other than those on SARS-CoV-2, most are based on suspected ARinf, often relying on self-reported symptoms, which do not accurately confirm infections [4]. There is also a discrepancy between physician and laboratory-diagnosed infections in athletes [5]. Confirmed ARinf are diagnosed by a physician with laboratory evidence

confirming an infection [6]. To our knowledge, only eight studies have used multiplex polymerase chain reaction (PCR) testing for detecting causative pathogens among 145 athletic individuals with confirmed ARinf [5,7–13], but the clinical presentation and evidence of multi-organ involvement in these studies has not been well documented.

Recognising the significance of ARinf in athletes, the International Olympic Committee (IOC) convened a consensus group to improve our understanding of these illnesses in athletes. One of the recommendations for future research proposed in the consensus statement was to use diagnostic methods to determine the specific pathogens causing ARinf in athletes.

**CONTACT** Martin Schwellnus  [mschwell@iafrica.com](mailto:mschwell@iafrica.com)  International Olympic Committee (IOC) Research Centre of South Africa, School of Medicine, Faculty of Health Sciences, University of Pretoria, Hatfield, Pretoria 0020, South Africa

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Specifically, the IOC consensus recommendation was that studies be conducted to identify whether the clinical presentation, risk of multi-organ involvement, and illness severity classification differ between pathogen-specific ARinf in athletes [6].

Data from the COVID-19 pandemic show that the clinical presentation may guide clinical decision-making in return-to-sport (RTS) following ARinf in athletic individuals with SARS-CoV-2 infection. Specifically, the clinical presentation (types of, and number of symptoms) of ARinf caused by SARS-CoV-2 has been associated with RTS outcomes [14–18] and evidence of multi-organ involvement in athletes [19].

From a clinical viewpoint, a better understanding of the etiology and clinical manifestations of ARinf in athletic individuals can help inform SEM physicians to stratify the severity of ARinf in athletes, as this may form the basis of RTS decision-making [6]. There is, therefore, a clear need for well-controlled prospective studies involving objective laboratory assessment of the etiology and clinical manifestations (symptoms, clinical signs, results of special investigations) of pathogen-specific ARinf in athletes.

The aims of this study were to describe the etiology of ARinf in athletic individuals and to identify differences in the clinical presentation, evidence of possible multi-organ involvement, and illness classification between four common pathogen groups. These data could provide valuable insights into the unique clinical manifestations of each pathogen, which could potentially guide pathogen-specific RTS clinical decision-making.

## 2. Methods

### 2.1. Study design and setting

This cross-sectional descriptive study was conducted at a Sport and Exercise Medicine (SEM) clinical service facility. Data were collected over 18 months from March 2023 - October 2024 to include two Southern Hemisphere winter seasons during which the incidence and pathogenicity of certain respiratory viruses are higher [20]. (See Supplementary Figure S1 for timeline of data collection). Research ethics approval was obtained from the Research Ethics Committee of the Faculty of Health Sciences at the University of Pretoria before data collection (REC 729/2022 and 409/2020). All participants provided written informed consent before participation.

### 2.2. Participants and data collection

Potential participants in this study were athletic individuals who presented to the SEM clinic with symptoms of ARinf. Inclusion criteria for potential participants were: ages 16–60 years, training  $\geq 4$  hours a week, and evaluation by a SEM physician  $\leq 5$  days after onset of symptoms. Participants could be included in the study more than once, provided that the subsequent ARinf episode started  $>3$  weeks after the previous ARinf and  $>1$  week after returning to full performance (i.e. the pre-illness level of performance) following the prior ARinf. Twelve participants were included more

than once. Each ARinf episode will be referred to as a case for the remainder of this manuscript.

#### 2.2.1. SEM physician evaluation

A SEM physician (principal investigator) evaluated all potential participants; this entailed a detailed and systematic history of the current respiratory illness, physical examination, and selected special investigations to verify that the symptoms were due to an ARinf. History included demographics (including age and sex), level of sport participation (professional=elite level/full-time, or amateur=part-time/hobby), years of participation in current sport, type of sport (endurance or power/mixed) and symptoms. A standardized symptom list, based on the IOC consensus [6] was used to record the presence and severity of all the symptoms experienced since the onset of the illness. Thirty-three types of symptoms were documented, with the option of reporting 'other' symptoms that were not on the symptom list (Supplementary Table S1). Symptoms were categorized into three anatomical regions [1,6]: 1) *upper respiratory*, 2) *lower respiratory & regional*, and 3) *systemic & non-respiratory* symptoms. Participants were required to score each symptom as follows: 0=absent, 1=mild, 2=moderate, or 3=severe [9,21,22].

A standardized physical examination was conducted, and clinical signs indicating involvement of specific anatomical regions were documented (Supplementary Table S2). The anatomical regions were 1) *localised* (nose & sinus, oral & throat), 2) *regional* (eye, ear, or lymphadenopathy), 3) *lower respiratory tract (LRT)*, and 4) *other non-respiratory organs* (cardiovascular, abdominal, neurological, dermatological or musculoskeletal). Resting heart rate (RHR) was measured by a 12-lead electrocardiogram (ECG) (custo cardio 110 BT, custo med GmbH) after resting supine for 5 minutes. Tympanic temperature was measured using an ear thermometer (Braun ThermoScan®). A venous blood sample (5–10 ml) was taken from each potential case using standard venepuncture techniques and processed at an accredited pathology laboratory.

The following selected serological tests were performed: white cell count (WCC) with differential (diff), C-reactive protein (CRP) and selected biomarkers of potential non-respiratory organ involvement, including liver [aspartate aminotransferase (AST), alanine transaminase (ALT)], renal [Urea, Creatinine, estimated Glomerular Filtration Rate (eGFR)], cardiac muscle [high-sensitivity cardiac troponin-T], and skeletal muscle [creatinine kinase (CK)].

#### 2.2.2. Pathogen identification

A nasopharyngeal swab, using a flocked swab (FLOQSwabs™, COPAN, Italy), was performed by a SEM physician, to identify a causative pathogen for each potential case. Nasopharyngeal swabs are more sensitive than throat swabs; flocked swabs additionally improve viral detection [23]. The specimen was processed on-site using the BioFire® FilmArray® (BioFire Diagnostics; Salt Lake City, USA) point-of-care device with the Respiratory 2.1 *Plus* panel [24]. This multiplex polymerase chain reaction (PCR) panel detects the following 16 viruses: adenovirus; coronaviruses 229E, OC43, HKU1, and NL63; Middle East respiratory syndrome coronavirus (MERS-CoV); severe acute respiratory

syndrome (SARS-CoV-2); human metapneumovirus; human rhinovirus/enterovirus; influenza A and B; parainfluenza viruses (type 1–4); and respiratory syncytial virus (RSV); and four bacteria: bordetella parapertussis; bordetella pertussis; chlamydia pneumonia; mycoplasma pneumonia. The panel does not differentiate between rhinovirus and enterovirus. Therefore, these rhinovirus/enterovirus specimens were sent to an independent accredited pathology laboratory under recommended conditions for further processing to distinguish between the two viruses.

### 2.2.3. Confirmation of ARinf

The confirmation of cases with an ARinf was based on pathogen testing, abnormalities in selected blood tests and abnormalities in clinical findings (Figure 1). All potential cases with symptoms suggestive of an ARinf underwent testing to detect the pathogen. If no pathogen was identified on multiplex PCR testing, an oropharyngeal swab was taken for bacterial microscopy and culture, which was processed in an accredited pathology laboratory. If no pathogen was detected, a review of the blood test results was done. An infective etiology was confirmed by any of the following: an abnormal WCC, lymphocytes or neutrophils [defined as values outside the established reference ranges for our laboratory (population and sex-specific)] [25]; a raised CRP ( $\geq 5$  mg/l) [26]. If blood tests were normal, cases where systemic symptoms (Supplemental Table S1) or signs of non-respiratory organ involvement (Supplemental Table S2) due to the ARinf were included. The rationale for including these cases was that systemic symptoms have been shown to be the best predictor of infectious vs noninfectious respiratory illnesses among athletes [5].

In total, 122 potential cases were evaluated during the study period. In 100 cases, a specific pathogen was identified. Of the remaining 22, six were excluded for not meeting the criteria for a confirmed ARinf (Figure 1). Therefore, 116 cases

with confirmed ARinf were included in the analysis for this study.

### 2.3. Outcome measures

The following outcome measures are reported for all cases and by pathogen group:

#### 2.3.1. Frequency (%) of causative pathogens and pathogen groups

Pathogen groups were established among cases with an identified pathogen. This was to allow for adequate power for analysis. The four common (seasonal) coronaviruses (OC43, 229E, NL63, HKU1) were grouped as 'common coronavirus.' Influenza A and B viruses were grouped as 'influenza.' Cases with two identified pathogens were grouped as 'dual pathogen.' Participants without an identified pathogen were grouped as 'unidentified.' In our cohort, <10% of the ARinf were caused by pathogens other than the following six pathogen groups: rhinovirus, common coronavirus, influenza, SARS-CoV-2, 'unidentified,' and 'dual pathogen,' therefore the remaining pathogens were grouped as 'other.'

#### 2.3.2. Number of symptoms

The number of symptoms in each anatomical region were counted (maximum of 10 upper respiratory, 6 lower respiratory & regional, and 17 systemic & non-respiratory symptoms) and then added to calculate the total number of symptoms. Only symptoms experienced at rest were included in the analysis to ensure a uniform comparison among cases, as participants who had begun training before the initial consultation might have reported only exercise-related symptoms.

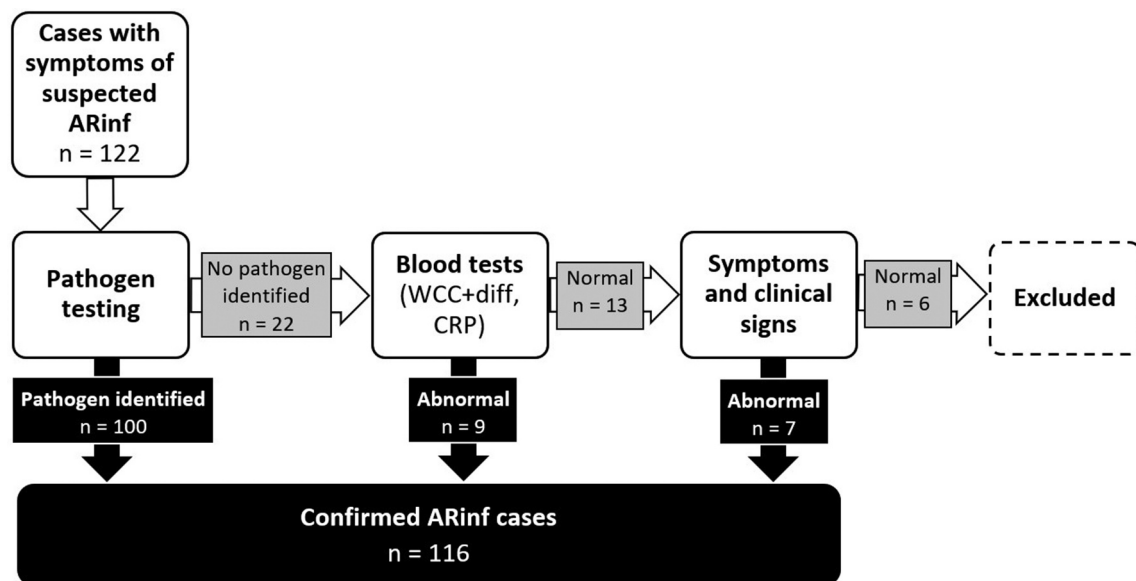


Figure 1. Confirmation of an acute respiratory infection.

ARinf: acute respiratory infection. WCC+diff: white cell count+differential. CRP: C-Reactive protein

### 2.3.3. Symptom severity score

The severity scores for symptoms in each anatomical region were calculated and then summed to create a total symptom severity score [21,22].

### 2.3.4. Frequency (%) of abnormal clinical examination findings

Clinical examination signs were classified as abnormal if resting heart rate (RHR) was  $\geq 70$  bpm or tympanic temperature was  $\geq 37.5^\circ\text{C}$  (abnormal vital signs) and any clinical sign indicating involvement of a specific anatomical region (Supplementary Table S2).

### 2.3.5. Frequency (%) of abnormal blood tests

A blood test value was considered abnormal if the result was outside the established laboratory reference range.

### 2.3.6. Clinical syndrome classification

All cases were classified according to the clinical syndrome classification proposed in the IOC consensus [6]. Presenting symptoms were considered, and cases were classified as follows: i) *predominantly upper respiratory tract: rhinitis-like ('common cold')*, ii) *predominantly upper respiratory tract: flu-like*, iii) *predominantly lower respiratory tract*. See Supplementary Table S3 for the classification used. The classification was done by two investigators (MJ, MS) without the knowledge of the causative pathogen.

### 2.3.7. Illness severity classification

All cases were classified (*mild*, *moderate*, and *severe*) according to the illness severity classification proposed in the IOC consensus [6]. See Supplementary Table S4 for the description of the criteria used, based on i) history, ii) clinical examination findings, and iii) results of selected special investigations. The classification was done by two investigators (MJ, MS) without the knowledge of the causative pathogen.

## 2.4. Statistical analysis

Descriptive data are presented using frequencies [n (%)] or means (SD). Demographics and sport participation variables were compared between the four most common pathogen groups (rhinovirus, common coronavirus, influenza, and SARS-CoV-2). The number of symptoms and the severity scores between the four common pathogen groups were compared using generalized linear modeling (PROC GENMOD). The normal distribution (identity link) was used for total number of symptoms & the upper respiratory severity score, and the negative binomial (log link) distribution for all other outcome variables. Univariate unadjusted means and 95% confidence intervals (95% CIs) were reported, with p-values indicating differences between the groups. Type 3 p-values from the Generalized Estimating Equation analysis assessed overall differences between the four common pathogen groups, accounting for repeated measures due to participants contracting varying pathogens more than once during the study period. The 'dual pathogen' and 'other' groups were excluded in the comparative analysis due to insufficient

sample sizes, and the 'unidentified' pathogen group was excluded as these represent unknown and inhomogeneous pathogens and, therefore, not clinically appropriate as a defined group. Comparative statistics between the pathogen groups were not possible for the remaining outcomes (abnormal clinical examination findings, abnormal blood tests, clinical syndrome and illness severity classification) because the numbers were too few. Data were analyzed using SAS (v 9.4) (SAS Institute Inc., Cary, USA), with a significance threshold of  $p < 0.05$ .

## 3. Results

One-hundred-and-sixteen cases with confirmed ARinf were evaluated on average (mean, SD) 2.4 ( $\pm 1.1$ ) (range 1–5) days after the onset of symptoms. A pathogen was identified in 100 (86%) cases. The frequency (%) of causative pathogen groups were as follows: rhinovirus = 34 (29%), influenza = 17 (15%), SARS-CoV-2 = 15 (13%), common coronavirus = 13 (11%), 'unidentified' = 16 (14%), 'dual pathogen' = 9 (8%), and 'other' = 12 (10%). (See footnote of Supplementary Table S5 for a list of pathogens in the 'other' pathogen group).

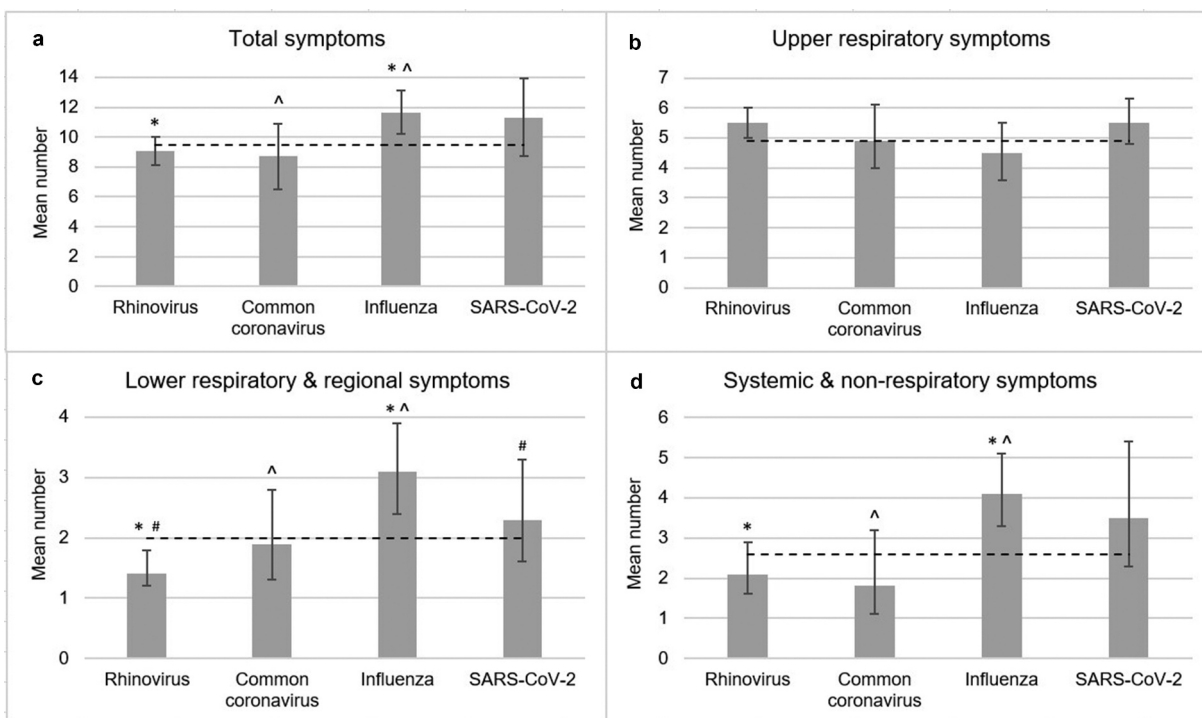
### 3.1. Demographics and sports participation variables

The demographics (age, sex, and BMI) and sports participation variables (level of participation, years of participation, and type of sport) for all cases, and by pathogen groups, are shown in Supplementary Table S5. The mean (SD) age among all cases was 26.1 ( $\pm 8.7$ ) years, the majority (64%) were females, and the mean (SD) BMI was 23.4 kg/m<sup>2</sup> ( $\pm 3.0$ ). 25% of the cases were professional athletes, and the mean (SD) years of participation in sport was 7.2 ( $\pm 3.9$ ). 52% of the cases were classified as endurance athletes, while the other 48% participated in power or mixed-type sports. There were no statistically significant differences in any of the demographic and sport participation variables between the four most common pathogen groups besides BMI ( $p = 0.04$ ). SARS-CoV-2 had a lower BMI compared to the other groups.

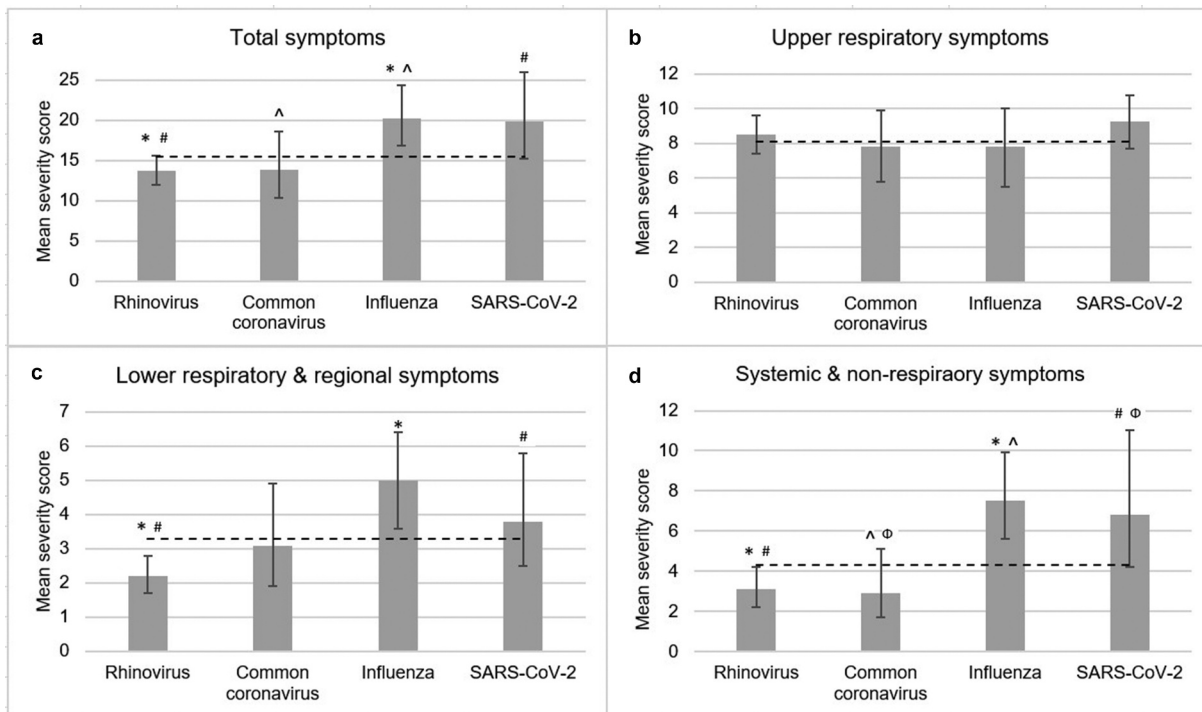
### 3.2. Number of symptoms

Overall, among all 116 cases, the average (mean, SD) number of *total* symptoms experienced per case was 9.5 ( $\pm 3.7$ ; range 1–22), 4.9 ( $\pm 1.7$ ; range 0–8) *upper respiratory* symptoms, 2.0 ( $\pm 1.4$ ; range 0–6) *lower respiratory & regional* symptoms, and 2.6 ( $\pm 2.2$ ; range 0–11) *systemic & non-respiratory* symptoms. The total number of symptoms and per anatomical region for all cases and by pathogen groups is shown in Supplementary Table S6. The comparison between the four common pathogen groups is shown in Figure 2.

Influenza had significantly more *total* symptoms, *lower respiratory & regional* symptoms, and *systemic & non-respiratory* symptoms (11.6; 3.1; 4.1) than rhinovirus (9.1; 1.4; 2.1) ( $p < 0.003$ ) and common coronavirus (8.7; 1.9; 1.8) ( $p < 0.05$ ). SARS-CoV-2 had significantly more *lower respiratory & regional* symptoms (2.3) than rhinovirus (1.4) ( $p < 0.04$ ). There were no significant differences in the number of *upper respiratory* symptoms between groups ( $p = 0.22$ ).



**Figure 2.** The number of total symptoms (a), Upper respiratory symptoms (b), Lower respiratory & regional symptoms (c) and systemic & non-respiratory symptoms (d) in the four common pathogen groups. Shaded bars represent the mean number of symptoms. Error bars represent the 95% confidence intervals. Dashed horizontal lines represent the overall average (mean) among all cases. \*rhinovirus vs influenza  $p \leq 0.002$ . ^common coronavirus vs influenza  $p < 0.05$ . #rhinovirus vs SARS-CoV-2  $p < 0.04$ .



**Figure 3.** The severity score of total symptoms (a), Upper respiratory symptoms, lower respiratory & regional symptoms (c) and systemic & non-respiratory symptoms (d) in the four common pathogen groups. Shaded bars represent the mean severity score. Error bars represent the 95% confidence intervals. Dashed horizontal lines represent the overall average (mean) among all cases. \*rhinovirus vs influenza  $p \leq 0.0006$ . ^common coronavirus vs influenza  $p \leq 0.03$ . #rhinovirus vs SARS-CoV-2  $p < 0.03$ . Φcommon coronavirus vs SARS-CoV-2  $p = 0.02$ .

### 3.3. Symptom severity scores

Overall, the average (mean, SD) *total* symptoms severity score was 15.5 ( $\pm 7.7$ ; range 1–47), the *upper respiratory* symptoms score was 8.1 ( $\pm 3.8$ ; range 0–18), the *lower respiratory & regional* symptoms score was 3.3 ( $\pm 2.7$ ; range 0–12), and the *systemic & non-respiratory* symptoms score was 4.3 ( $\pm 4.3$ ; range 0–26). The total symptom severity score and per anatomical region for all cases and by pathogen groups is shown in Supplementary Table S7. The comparison between the four common pathogen groups is shown in Figure 3.

The *total* symptoms severity score, *lower respiratory & regional* symptoms score and *systemic & non-respiratory* symptoms score were significantly higher in influenza (20.2; 5.0; 7.5) and SARS-CoV-2 (19.9; 3.8; 6.8) vs. rhinovirus (13.7; 2.2; 3.1) ( $p < 0.0006$  and  $p < 0.003$  respectively). Influenza also had a significantly higher *total* symptoms severity score and *systemic & non-respiratory* symptoms score (20.2; 7.5) vs. common coronavirus (13.8; 2.9) ( $p = 0.02$ ). There were no

significant differences in the *upper respiratory* symptoms severity score between the groups ( $p = 0.65$ ).

### 3.4. Abnormal clinical examination findings

Abnormal clinical examination findings (vital signs and clinical signs indicating involvement of specific anatomical regions) for all cases and by pathogen groups is shown in Table 1.

Influenza had the highest percentage (53%) of abnormal vital signs (Table 1). The highest percentage of abnormal *LRT* clinical signs was among 'dual pathogen' (44%) and influenza (24%). No rhinovirus or common coronavirus cases had abnormal *LRT* clinical signs. Influenza had the highest percentage (53%) of clinical signs suggestive of *other non-respiratory organ* involvement, followed by SARS-CoV-2 (33%), whereas rhinovirus (9%) and common coronavirus (15%) had the lowest percentage.

**Table 1.** Abnormal clinical examination findings (vital signs and clinical signs indicating involvement of specific anatomical regions) for all cases and by pathogen groups (values are n (%)).

	All	Pathogen groups						
		Rhino-virus	Common corona-virus	Influenza	SARS-CoV-2	Unident-ified	Dual pathogen	Other
Abnormal vital signs	n = 116	n = 34	n = 13	n = 17	n = 15	n = 16	n = 9	n = 12
(n, %)	(24.1)	(17.7)	(15.4)	(52.9)	(13.3)	(25.0)	(33.3)	(16.7)
<b>Clinical signs indicating involvement of specific anatomical regions</b>								
Localised	105	29	10	16	14	15	9	12
(n, %)	(90.5)	(85.3)	(76.9)	(94.1)	(93.3)	(93.8)	(100)	(100)
Regional	44	13	4	6	7	7	4	3
(n, %)	(37.9)	(38.2)	(30.8)	(35.3)	(46.7)	(46.7)	(44.4)	(25)
Lower respiratory tract	11	0	0	4	0	1	4	2
(n, %)	(9.5)	(0)	(0)	(23.5)	(0)	(6.3)	(44.4)	(16.7)
Other non-respiratory organs	24	3	2	9	5	3	2	0
(n, %)	(20.7)	(8.8)	(15.4)	(52.9)	(33.3)	(18.8)	(22.2)	(0)

Comparative statistics between the pathogen groups were not possible because the numbers were too few.

Abnormal vital signs = heart rate  $\geq 70$  bpm/temperature  $\geq 37.5^\circ\text{C}$ .

Localised only = Nose & sinus/Oral cavity & throat.

Regional involvement = Eye/ear/lymph nodes.

Other non-respiratory organ involvement = Cardiovascular/abdominal/neurological/dermatological/musculoskeletal.

**Table 2.** Abnormal blood tests for all cases and by pathogen groups (values are n (%)).

	All	Pathogen groups						
		Rhino-virus	Comm corona-virus	Influenza	SARS-CoV-2	Unident-ified	Dual pathogen	Other
Abnormal WCC + diff	n = 116	n = 34	n = 13	n = 17	n = 15	n = 16	n = 9	n = 12
(n, %) <sup>a</sup>	(49.1)	(26.5)	(46.2)	(87.5)	(42.9)	(43.8)	(88.9)	(50)
Raised CRP	50	12	4	10	8	5	7	4
(n, %) <sup>a</sup>	(43.9)	(36.4)	(30.8)	(62.5)	(53.3)	(31.3)	(77.8)	(33.3)
<b>Markers of other non-respiratory organ involvement</b>								
Abnormal liver function*	22	7	2	4	2	4	2	2
(n, %) <sup>b</sup>	(19.1)	(20.6)	(15.4)	(25)	(13.3)	(18.8)	(22.2)	(16.7)
Abnormal renal function <sup>^</sup>	33	10	3	7	3	2	4	4
(n, %) <sup>b</sup>	(28.7)	(29.4)	(23.1)	(43.8)	(20.0)	(12.5)	(44.4)	(33.3)
Raised cardiac muscle enzymes (Troponin-T)	1	1	0	0	0	0	0	0
(n, %) <sup>a</sup>	(0.9)	(3.0)	(0)	(0)	(0)	(0)	(0)	(0)
Raised skeletal muscle enzymes (CK)	28	9	3	5	1	4	2	4
(n, %) <sup>b</sup>	(24.4)	(26.5)	(23.1)	(31.3)	(6.7)	(25)	(22.2)	(33.3)

Comparative statistics between the pathogen groups were not possible because the numbers were too few.

Number of cases with missing data: a=2, b=1.

\*Liver function = alanine transaminase (ALT) or aspartate transaminase (AST).

<sup>^</sup>Renal function = urea or creatinine or estimated glomerular filtration rate (eGFR).

Abbreviations: WCC + diff: white cell count and differential. CRP: C-reactive protein. CK: creatine kinase.

### 3.5. Abnormal blood tests

Abnormal blood tests for all cases and by pathogen groups are shown in Table 2.

The highest percentage of abnormal WCC+diff tests was among ‘dual pathogen’ (89%) and influenza (88%), while rhinovirus had the lowest percentage (26%) (Table 2). ‘dual pathogen’ and influenza had the highest percentage of raised CRP tests, abnormal liver function and renal function tests. Only 1 participant had a raised troponin-T. This was a participant with a rhinovirus infection who had mildly elevated troponin-T levels without any other evidence of cardiovascular abnormalities. This was likely due to recent strenuous exercise, as repeat testing revealed normal levels.

### 3.6. Clinical syndrome classification

Among all cases in our cohort, 45% experienced a *rhinitis-like* (“common cold”) illness, 51% had a *flu-like* illness, and only 3% had *predominantly LRT* illness. The percentage of cases in the different clinical syndrome categories by pathogen groups is shown in Figure 4. A detailed description of the number (%) of cases presenting with the different clinical syndromes for all cases and by pathogen groups is available in Supplementary Table S8.

### 3.7. LRT: lower respiratory tract

Influenza presented with the least (%) *rhinitis-like* (“common cold”) illness (6%), and the most *predominantly LRT* illness

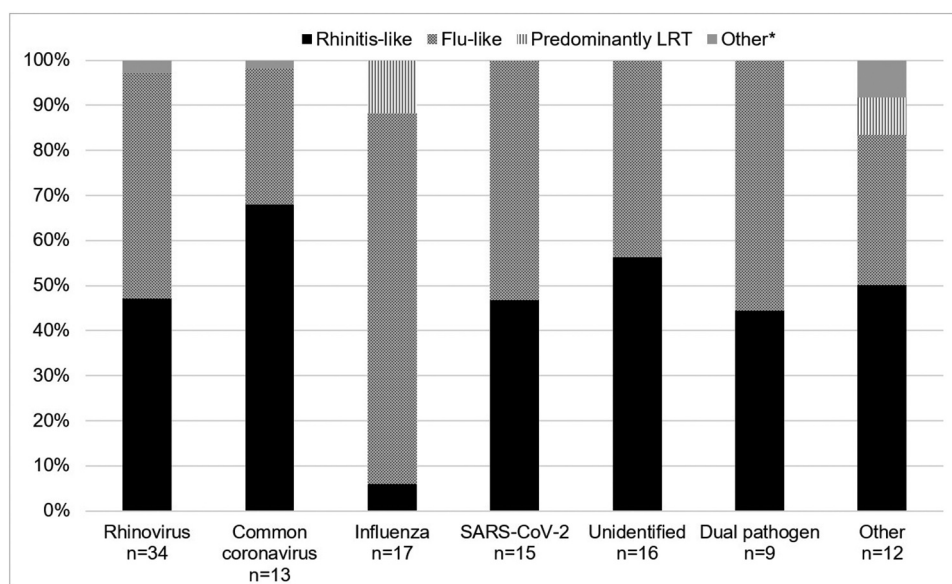


Figure 4. The percentage of cases in the different clinical syndrome categories by pathogen groups. Each 100% stacked column displays the percentage of cases that constitute a clinical syndrome category within a pathogen group. \*other = not classified elsewhere.

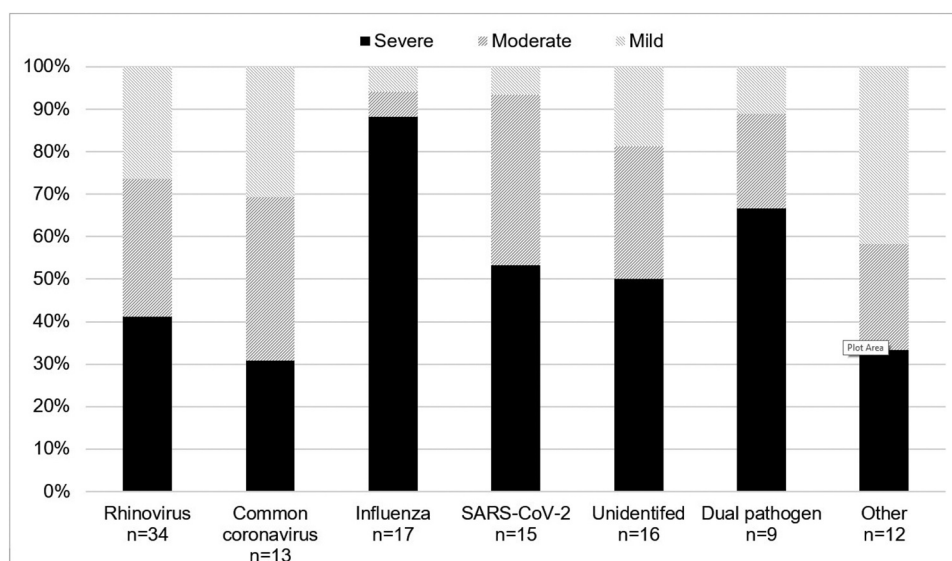


Figure 5. The percentage of cases in different illness severity categories by pathogen groups. Each 100% stacked column displays the percentage of cases that constitute an illness severity category within a pathogen group.

(12%) and *flu-like* illness (82%). Common coronavirus presented with the most (%) *rhinitis-like* ('common cold') illness (69%) and the least *flu-like* illness (31%). Notably, in our cohort, rhinovirus more frequently presented with *flu-like* illness (50% of cases) than with *rhinitis-like* ('common cold') illness (47% of cases).

### 3.8. Illness severity classification

Among all cases, 21% experienced *mild* illnesses, 29% *moderate*, and 51% *severe* illnesses. The percentage of cases in different illness severity categories by pathogen groups is depicted in Figure 5. A detailed description of the number (%) of cases in the different illness severity categories for all cases and by pathogen groups is shown in Supplementary Table S9.

Influenza had the most (%) *severe* illnesses (88%), while common coronavirus had the least *severe* illnesses (31%). Notably, rhinovirus presented with *severe* illness in 41% of cases.

## 4. Discussion

In this study, we describe the etiology of ARinf in athletic individuals and identify differences in the clinical presentation, evidence of possible multi-organ involvement, and illness classification between four common pathogen groups. To our knowledge, this is the largest study in a cohort of athletic individuals comparing symptomatology between the four most common pathogen groups: rhinovirus, common coronavirus, influenza viruses, and SARS-CoV-2. In our cohort, influenza and SARS-CoV-2 were associated with a greater number of and more severe symptoms than rhinovirus and common coronavirus.

### 4.1. Aetiology of ARinf in athletic individuals

There are very few studies identifying different pathogens causing ARinf in athletic individuals. In the previous studies, rhinovirus was the most commonly detected pathogen, followed by influenza (A+B) and common coronaviruses [5,7–13]. All but one of these studies [13] were conducted before the COVID-19 pandemic. In our cohort, the most common pathogens causing ARinf were rhinovirus (29%), influenza (A+B) (15%), SARS-CoV-2 (13%), and common coronavirus (11%). The etiology of ARinf in our cohort is comparable to that among the general public [27,28] and in keeping with the studies in athletic individuals [5,7–13]. About 9% of the ARinf in previous studies were caused by dual pathogens, similar to our findings (8%). We note that the pathogen detection rate in our study (86%) is higher than the average reported among the previous studies in athletes ( $\pm 59\%$ ) [5,7–13]. This may be partly because the two earliest studies, with detection rates of 29% [7] and 53% [5], used cotton swabs, which are inferior to newer flocked swabs [23]. The more recent studies, using similar sampling techniques to ours, report detection rates of 75–82% [9,11,13], similar to our detection rate (86%).

### 4.2. Symptom number and severity

The symptomatology of different pathogen-specific ARinf among athletes have only been described in two studies, both with small sample sizes [9,13]. GrÖnroos et al. [13] described the presenting symptoms of 11 athletes but did not provide details on methods used to report these symptoms. Valtonen et al. [9] presented the median total illness severity score among 15 athletes. While the number of symptoms was not reported, their findings indicated that athletes with influenza had the highest severity scores, while those with rhinovirus and common coronaviruses had the lowest scores. In our study, there were significant differences in the symptomatology between the four common pathogen groups. Rhinovirus and common coronavirus had fewer total number of symptoms, fewer *lower respiratory & regional* symptoms, and fewer *systemic & non-respiratory* compared to influenza and SARS-CoV-2. The differences were even more evident when comparing the severity scores between the pathogens. There are data to show that knowledge of the symptomatology is useful for clinicians, as specific symptom clusters and the number of acute symptoms during SARS-CoV-2 infection have been associated with prolonged RTS [14–18] and evidence of multi-organ involvement in athletes. [19] Whether this association holds for other pathogens causing ARinf, such as those reported in this study, will be explored in future studies.

### 4.3. Abnormal clinical examination and serology findings

We are unaware of any studies, besides those on SARS-CoV-2, that have reported on pathogen-specific clinical and serological findings in athletic individuals. In our study, 23% of all cases had an abnormal vital sign (resting heart rate or raised temperature), 10% had clinical signs indicating *LRT* involvement, 21% indicating *other non-respiratory organ* involvement, and 44% had a raised CRP. Influenza had the highest percentage of abnormal vital signs (53%) and clinical signs indicating *other non-respiratory organ* involvement (53%). Influenza is known to cause fever more often than other respiratory viruses among the general population [21]. 'Dual pathogen' and influenza had the highest percentage of raised CRP tests, abnormal liver function and renal function tests. The impact of 'Dual pathogen' ARinf on clinical outcomes among the general population remains unclear [29]; further study among athletic individuals is also necessary. Specific clinical and serology findings may indicate more severe illness, and IOC consensus guidelines recommend using these parameters to stratify the severity of ARinf in athletes [6].

### 4.4. Clinical syndromes and severity classification

A novel aspect of this study was to describe ARinf according to the clinical syndrome and illness severity classifications proposed in the most recent IOC consensus [6]. To our knowledge, no studies have described pathogen-specific ARinf according to these classifications among athletic individuals. Regarding the clinical syndrome classification,

common coronavirus resulted in the highest percentage of *rhinitis-like* ('common cold') illnesses, while influenza resulted in the highest percentage of *flu-like* illnesses. Regarding the severity classification, common coronavirus resulted in the highest percentage of *mild* and *moderate* illnesses, while influenza resulted in the highest percentage of *severe* illnesses. A noteworthy finding was that 41% of rhinovirus infections in this cohort presented with *severe* illnesses. Therefore, for the SEM physician, it is important to note that rhinovirus infection can be associated with a broad clinical spectrum and does not only cause seemingly insignificant localized illness. The varying clinical manifestations following rhinovirus infections may be due to the multiple serotypes [30]. Assessing the severity of ARinf in athletic individuals is essential as it influences the risk of medical complications during exercise after ARinf and should guide clinical decision-making in RTS following these infections [6]. The relationship between clinical syndromes or illness severity classifications of pathogen-specific ARinf in athletes and RTS needs exploration in future studies.

#### 4.5. Strengths and limitations

To our knowledge, this study is the largest and most extensive report on pathogen-specific ARinf in athletic individuals. In keeping with the recommendations of the IOC consensus [6] these findings address knowledge gaps in the current literature on the etiology of pathogen-specific ARinf and highlight differences in the clinical presentation, evidence of possible multi-organ involvement, and illness classification in ARinf in athletic individuals. The study does have several limitations. One limitation is our grouping of the viral genera. While this was necessary to allow for adequate power to analyze pathogen groups, we recognize that different viral species within a genus (e.g. rhinovirus A, B, C) may cause varying clinical manifestations [31]. While the clinical manifestations of ARinf are mainly due to the immune response to the infection, these may also be affected by other host factors and the infecting pathogen [30]. Also, while we found differences in the clinical manifestations between the pathogen groups, no individual symptoms or clinical findings allow any particular pathogen to be ruled in or ruled out. Therefore, we do not propose that the etiology of the ARinf can be predicted by symptoms or clinical findings. Another limitation is that we do not have baseline data on participant blood tests. As a result, we cannot confirm that the raised values are due to the ARinf. We also acknowledge that our study was conducted on a self-selected convenience sample of athletic individuals presenting with ARinf to a SEM clinic. Therefore, our data need to be interpreted bearing in mind that the sample is not necessarily representative of a general population of athletes with ARinf. Specifically, our cohort may overrepresent a population of more 'severe' cases that had access to physician care. Furthermore, 75% of our sample were amateur athletes, therefore, the findings may not be generalizable to elite athletes.

#### 4.6. Clinical implications

The clinical assessment described in this study, and possibly pathogen identification, can be used in the initial assessment of athletic individuals with ARinf to risk stratify and may form the basis of RTS decision-making. Understanding the differences in symptomatology, clinical examination and serological findings between the common pathogens causing these infections is valuable, as the severity classification is based on these factors. Future studies should explore the relationship between RTS outcomes after ARinf in athletes and specific pathogens, symptom number and severity, clinical syndromes and illness severity classifications.

#### 5. Conclusion

In this cohort of athletic individuals with confirmed ARinf, the most common pathogen was rhinovirus. Influenza and SARS-CoV-2 infections caused a greater number and severity of symptoms than rhinovirus and common coronavirus. Among the four common pathogen groups, influenza had the highest percentage of abnormal clinical examination and serological findings, while rhinovirus and common coronavirus had the least. Knowledge of the specific pathogen causing ARinf in athletes may add value to the risk assessment and clinical RTS decision-making in athletic individuals with ARinf.

#### Author contributions

M.J. was involved in study planning, data collection, data cleaning, data management, data interpretation, manuscript writing (first draft), and manuscript editing. N.S. and M.V. contributed to study planning, data interpretation, and manuscript editing. E.J. and M.D. were involved in data cleaning, data management, data analysis (including statistical analysis), data interpretation, and manuscript editing. M.S. was responsible for the overall content (as guarantor), study concept, study planning, data interpretation, manuscript editing, and facilitating funding. All authors have read and approved the final version of the manuscript and agree with the authors' presentation order.

#### Disclosure statement

The authors report there are no competing interests to declare.

#### Data availability statement

No data are available. Due to ethical considerations, datasets from this project are not publicly available.

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## ORCID

Marcel Jooste  <http://orcid.org/0000-0002-6914-514X>  
 Nicola Sewry  <http://orcid.org/0000-0003-1022-4780>  
 Maarit Valtonen  <http://orcid.org/0000-0001-8883-2255>  
 Esme Jordaan  <http://orcid.org/0000-0002-0361-3473>  
 Martin Schwellnus  <http://orcid.org/0000-0003-3647-0429>

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