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Opinion

Sport and exercise during viral acute respiratory illness—Time to revisit

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Athletes are commonly advised not to compete or train during major symptoms of a viral acute respiratory illness (ARI), which most commonly is a common cold. It has been traditionally thought that heavy physical stress could induce the worsening of symptoms of ARI and possibly cause potentially severe complications like myocarditis or sudden cardiac death (SCD). In addition, viral ARI may decrease athlete's performance.[1] These concerns have been recently stressed during the coronavirus disease 2019 (COVID-19) pandemic.[2] However, it is anecdotally well-known that athletes commonly compete while experiencing common colds, most commonly due to picornaviral ARIs. In this perspective, we discuss what is known or not known regarding the risks of exercising during the viral ARI.

1. Common cold: The most common acute illness in athletes

The common cold is typically associated with sore throat, sneezing, a runny nose, congestion, and a cough. Fever is usually absent or mild. Symptoms last 5–7 days.[3–5] In a 1-year prospective upper respiratory tract sampling study there were 6.3 (mean) virus-positive episodes per person per year in 26 participants aged between 18 and 39 years. Seventy three percent (4.6) of these episodes were symptomatic.[6] Recently, the incidence of an ARI in athletes was reported to be 1.7 per year.[7] Up to 18% of elite Olympic athletes have been considered highly susceptible to an ARI suffering ≥4 ARIs (resulting in ≥2 days of restricted training) during 18 months, thus markedly disturbing training.[8] In our study during a 2-week major winter sports competition, the athletes had a 7-fold increase in the risk of a common cold compared to normally exercising control subjects (38% *vs.* 6%).[4] It is well-established that rhinoviruses and seasonal coronaviruses constitute up to 60%–80% of the cases.[5] Rhinoviruses and seasonal coronaviruses are not known to be cardiotropic viruses like enteroviruses and adenoviruses (infect cardiomyocytes and can be cleared from the heart) or cardiotoxic viruses like influenza viruses (indirectly trigger myocarditis by activating the immune system).[9] Importantly, cardiotropic viruses are uncommon causes of the common cold in young adults outside military service and are rarely detected in athletes with acute respiratory symptoms.[1,3–5]

2. Animal studies on exercise and myocarditis

Experimental murine studies conducted over 50 years ago showed that exercise markedly increased the risk of systemic picornavirus disease, specifically coxsackievirus B3 (CVB3)-induced cardiomyopathy. After intraperitoneal inoculation of CVB3, 5.5% of the nonexercised mice died of cardiomyopathy. When infected mice were forced to swim for 30 min in the morning and in the afternoon, 50% of the mice died of myocardiopathy. Concomitantly, the replication of CVB3 in the heart was markedly augmented.[10] Another murine study found that mice running 70 min on a treadmill at 48 h after CVB3 inoculation experienced significantly increased myocardial damage but not lethality.[11] These studies created a dogma that strenuous exercise during a viral infection may induce myocarditis and rest during infections was recommended. A recent systematic review and meta-analysis of animal studies concludes that moderate exercise before or after viral inoculation (herpes simplex virus type 1, H1N1 influenza virus, or BK polyomavirus reduces morbidity with no change in symptom severity or mortality, although exercise until fatigue tended to cause increased symptom severity and mortality.[12] Exercise-induced cardiomyopathy during enterovirus or other respiratory virus infections has not, however, been reported in humans.

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3. Myocarditis in athletes

Myocarditis is the main potentially severe health risk in athletes with a viral ARI. In the general population, myocarditis occurs most commonly in men between 20 and 40 years of age with a rate of 5−10 cases per 100,000 men and is self-limiting. Most cases are diagnosed clinically since endomyocardial biopsy and viral diagnostics are seldom performed. Molecular techniques have found parvovirus B19 and human herpes virus 6 to be the most common viruses detected in the endomyocardial biopsy specimen.[9] Parvovirus B19 and human herpes virus-6 do not cause ARI. One study employing nucleic acid amplification tests nasal swabs collected from 24 children with myocarditis detected viral RNA in 16 (67%) including rhinoviruses, influenza A and B viruses, respiratory syncytial viruses, seasonal coronaviruses, parvovirus 19, adenoviruses, coxsackievirus B5, enterovirus, and parainfluenza type 2 virus.[13] However, such findings provide only indirect evidence of causality. The etiologies of viral myocarditis in athletes are not well established, although multiple studies have reported COVID-19-associated myocarditis in athletes.[14,15] The overall incidence of clinically diagnosed myocarditis in 6138 athletes with COVID-19 in 12 studies was 1.2%, compared to 4.2% in the general population.[16] The clinical manifestations in the great majority of the cases were mild with full recovery after 3−6 months of rest.[2] In a large-scale 1-year prospective follow-up study on 3675 athletes, all 21 (0.6%) athletes with myocardial or pericardial involvement were cleared to return to sport.[17]

4. Myocarditis and SCD

Sports activity increases the risk of SCD occurring during or within an hour of exercise. The incidence of SCD or aborted SCD in athletes varies from 0.24 to 2.28 per 100,000 person-years in athletes <35 years of age.[18] A structurally normal heart is often found at autopsy. Sudden arrhythmic death syndrome and myocardial diseases are the most common causes of SCD.[19] The possible role of viral ARI-associated myocarditis as a cause of SCD has been controversial. The occurrence of myocarditis as a cause of SCD has varied from 6% to 10%.[18] A recent systematic review on sports-related SCD attributable to myocarditis included 15 studies comprising 347,092,437 person-years. The incidence was 0.047 per 100,000 person-years or 1 death of myocarditis in 2.3 million person-years.[20] A UK study between 1994 and 2022 identified 756 consecutive adolescent cases of SCD including 128 competitive athletes.[19] Myocarditis was determined as the cause of death in 30 cases (4%) but none among athletes. Out of the 30 cases, 29 died at rest and 1 during exercise.[19] In another UK study on 7702 consecutive cases of SCD between 1994 and 2022, only 82 (1.1 %) were attributed to myocarditis.[21] Overall, 11% died during exertion, including 2 of 5 athletes.[21] The available evidence suggests that SCD-related myocarditis is exceedingly rare.[20] The small risk of SCD is highest in male athletes, black athletes, men's basketball, men's soccer, and American football.[22]

5. Effects of ARI on performance

There is no evidence-based data on the effects of different respiratory virus infections on athletic sports performance. No studies have been carried out during the acute symptomatic phase of naturally occurring viral ARIs. In 1 study experimental rhinovirus type 16 infection induced only minimal changes in maximal exercise capacity.[23] Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a different respiratory virus inducing multiorgan abnormalities, may reduce an athlete's aerobic capacity and cardiorespiratory function affecting endurance performance and cognitive capacities.[24] There is anecdotal evidence that athletes often compete during common colds. To our knowledge, most players in the National Hockey League play despite having a common cold. Last spring, the Norwegian skiers Johannes and Tarjei Bö got first and second positions in the World Cup biathlon competition while being acutely SARS-CoV-2 positive. Recently, Jacob Ingebritsen won the world championship in 5000-m running while suffering from a common cold. In our study retrospective analysis revealed an outbreak of enterovirus D68 in a professional ice hockey team and 4 symptomatic players with the virus had played without any adverse events (W. Grönroos et al, to be published).

6. Return to play after ARI

Guidelines to return to sport after a viral ARI are based on expert opinions. "A neck-check rule" has been commonly used by sports physicians, i.e., exercise is allowed if symptoms are limited to the upper respiratory tract. This rule is nonscientific but may be partly useful. Return to play should be based on etiologic diagnostics. For example, adenoviral tonsillitis, enteroviral pharyngitis, and streptococcal tonsillopharyngitis are upper respiratory tract infections but potentially dangerous in athletes. Adenoviruses and enteroviruses are hematogenic viruses. The etiology, clinical manifestations, viral kinetics, and bacterial complications of ARI in athletes are poorly studied. During the COVID-19 pandemic, an avalanche of expert opinion guidelines for return to sport were published focusing on the risk of myocarditis. The American College of Cardiology recommends that athletes with mild non-cardiopulmonary symptoms withdraw from exercise training until symptom resolution.[2] It is worth stressing that even a short bed rest may rapidly decrease exercise performance which may require weeks to rebuild.[25]

7. Conclusion

There is a lack of evidence that sport and exercise during common colds could cause significant deleterious effects on an athlete's health. However, symptoms of cardiac involvement i.e., chest pain, syncope, shortness of breath, and palpitations are absolute contraindications for exercise and indications for cardiologic investigations. Point-of-care respiratory viral diagnostics should be used more commonly.[3,4] Potentially cardiogenic SARS-CoV-2, adenovirus, enterovirus, or influenza virus infections are reasons for a break in training and restriction of competitions. In other instances, an athlete

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with a common cold could train and compete but the decision is based on a personalized process between the athlete and the medical team which should also contain expertise in infectious diseases. There is clearly a need for clinical and virological studies of the effects of a viral ARI on the health and performance of an athlete.

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Authors' contributions

OR conceived the idea for the paper and wrote an initial draft; MV, MW, RL, and OJH drafted and edited the manuscript. All authors have read and approved the final version of the manuscript, and agreed with the order of presentation of the authors.

Competing interests

The authors declare that they have no competing interests.

References

1. Ruuskanen O, Luoto R, Valtonen M, Heinonen OJ, Waris M. Respiratory viral infections in athletes: Many unanswered questions. *Sports Med* 2022;**52**:2013–21.
2. Writing Committee; Gluckman TJ, Bhave NM, Allen LA, et al. 2022 ACC expert consensus decision pathway on cardiovascular sequelae of COVID-19 in adults: Myocarditis and other myocardial involvement, post-acute sequelae of SARS-CoV-2 infection, and return to play: A report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol* 2022;**79**:1717–56.
3. Valtonen M, Waris M, Vuorinen T, et al. Common cold in team Finland during 2018 Winter Olympic Games (PyeongChang): Epidemiology, diagnosis including molecular point-of-care testing (POCT) and treatment. *Br J Sports Med* 2019;**53**:1093–8.
4. Valtonen M, Grönroos W, Luoto R, et al. Increased risk of respiratory viral infections in elite athletes: A controlled study. *PLoS One* 2021;**16**: e0250907. doi:10.1371/journal.pone.0250907.
5. Luoto R, Waris M, Valtonen M, Ruuskanen O. Respiratory viral infections – Impact on sport and exercise medicine. *Exerc Immunol Rev* 2023;**29**:7–21.
6. Byington CL, Ampofo K, Stockmann C, et al. Community surveillance of respiratory viruses among families in the Utah better identification of germs – Longitudinal viral epidemiology (BIG-LoVE) study. *Clin Infect Dis* 2015;**61**:1217–24.
7. Derman W, Badenhorst M, Eken MM, et al. Incidence of acute respiratory illness in athletes: A systematic review and meta-analysis by a subgroup of the IOC consensus on "acute respiratory illness in the athlete". *Br J Sports Med* 2022;**56**:630–8.
8. Cuthbertson L, Turner SEG, Jackson A, et al. Evidence of immunometabolic dysregulation and airway dysbiosis in athletes susceptible to respiratory illness. *EBioMedicine* 2022;**79**:104024. doi:10.1016/j.ebiom.2022.104024.
9. Basso C. Myocarditis. *N Engl J Med* 2022;**387**:1488–500.
10. Gatmaitan BG, Chason JL, Lerner AM. Augmentation of the virulence of murine coxsackievirus B-3 myocardiopathy by exercise. *J Exp Med* 1970;**131**:1121–36.
11. Ilbäck NG, Fohlman J, Friman G. Exercise in coxsackie B3 myocarditis: Effects on heart lymphocyte subpopulation and the inflammatory reaction. *Am Heart J* 1989;**117**:1298–302.
12. de Araujo RB, Chacon-Mikhael MPT, Lord JM, Sardeli AV. Exercise effect on symptom severity, morbidity and mortality in viral infections: A systematic review and meta-analysis. *Immunol Exerc Rev* 2022; **28**:133–40.
13. Aljohani OA, Mackie D, Bratincsak A, Bradley JS, Perry JC. Spectrum of viral pathogens identified in children with clinical myocarditis (pre-coronavirus disease-2019, 2000–2018): Etiologic agent *versus* innocent bystander. *J Pediatr* 2022;**242**:18–24.
14. Moulson N, Petek BJ, Baggish AL, et al. The cardiac effects of COVID-19 on young competitive athletes: Results from the outcomes registry for cardiac condition in athletes (ORCCA). *J Cardiovasc Dev Dis* 2023;**10**:72. doi:10.3390/jcdd10020072.
15. Bavishi A, Kliethermes SA, Petek B, et al. Clinical spectrum of COVID-19 complications in young adults: Combined analysis of the American Heart Association COVID-19 cardiovascular disease registry and the outcomes registry for cardiac conditions in athletes. *BMJ Open* 2023;**13**: e069943. doi:10.1136/bmjopen-2022-069943.
16. Hofbauer T, Humann K, Neidenbach RC, Scharhag J. Myocarditis screening methods in athletes after SARS-CoV-2 infection–A systematic review. *Int J Sports Med* 2023;**44**:929–40.
17. Petek BJ, Moulson N, Drezner JA, et al. Cardiovascular outcomes in collegiate athletes after SARS-CoV-2 infections: One-year follow-up from the outcomes registry for cardiac conditions in athletes. *Circulation* 2022;**145**:1690–2.
18. Han J, Lalario A, Merro E, et al. Sudden cardiac death in athletes: Facts and fallacies. *J Cardiovasc Dev Dis* 2023;**10**:68. doi:10.3390/jcdd10020068.
19. Finocchiaro G, Radaelli D, D'Errico S, et al. Sudden cardiac deaths among adolescents in the United Kingdom. *J Am Coll Cardiol* 2023; **81**:1007–17.
20. Quinn R, Moulson N, Wang J, Isserow S, McKinney J. Sports related sudden cardiac death attributable to myocarditis: A systematic review and meta-analysis. *Can J Cardiol* 2022;**38**:1684–92.
21. Bhatia RT, Finocchiaro G, Westaby J, et al. Myocarditis and sudden cardiac death in the community: clinical and pathological insights from a national registry in the United Kingdom. *Circ Arrhythm Electrophysiol* 2023;**16**: e012129. doi:10.1161/CIRCEP.123.012129.
22. Harmon KG. Incidence and causes of sudden cardiac death in athletes. *Clin Sports Med* 2022;**41**:369–88.
23. Weidner TG, Anderson BN, Kaminsky LA, Dick EC, Schurr T. Effect of a rhinovirus-caused upper respiratory illness on pulmonary function tests and exercise responses. *Med Sci Sports Exerc* 1997;**29**:604–9.
24. William Z, Hull JH. Respiratory complications following COVID-19 in athletic populations: A narrative review. *Scand J Med Sci Sports*. 2022. doi:10.1111/sms.14275. [Epub ahead of print].
25. Spiering BA, Weakley J, Mujika I. Effects of bed rest on physical performance in athletes: A systematic and narrative review. *Sports Med* 2023;**53**:2135–46.

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