ARE ATHLETES (REALLY) PRONE TO INFECTIONS?

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INTRODUCTION AND BACKGROUND

It is almost common sense among sport physicians that athletes under considerable training and/or competition load are prone to infections. In the light of plausible underlying pathophysiological mechanisms even a lack of convincing epidemiological evidence for such an effect is quickly attributed to methodological problems: too small samples of available elite athletes, too long observation times necessary for a sufficient number of infections, lack of existing registries etc. Even terms like an "immunodepression of athletes" (likely more appropriate: immune compromisation) can be heard in discussions. However, is this really correct? Shouldn´t we use a more careful explanatory model that distinguishes between sport disciplines, training phases and possibly even properties of the athletes we are dealing with? This article tries to give some answers and pleas for a more differentiated approach.

There are two different models that give rise to the assumption that athletes are prone to infections (for details: see preceding article "The Immune Response to Exercise"):

- Repeated time periods of impaired immunity within hours after (intense) exercise - as symbolized by the "open window for infections"[1].
- A generally increased infection rate associated with repeated (intense) exercise - as symbolized by the "J-shaped curve"[2].

Either numerous phases of compromised immunity after repeated intense training stimuli (open window) or a generally compromised immune response due to repeated training bouts (underlying mechanism of the J-shaped curve) would inadvertently lead to infections. In fact, there are alternative explanations for the changes found in the blood which underly the "open window" theory as well as for an increased reporting of infection symptoms which constitutes the J-shaped curve. This is a result of the type of studies investigating injuries in athletes: They are either observational/descriptive or – when experimental – aiming at surrogate parameters because experiments with human beings who are intentionally exposed to infectious agents are (correctly) considered unethical. Still the absence of conclusive investigations cannot be used to declare them superfluous and carry theories – in this case ones of general infection proneness of athletes – forward without proper evidence.

THE EVIDENCE UNDERPINNING THE ASSUMPTION OF INFECTION PRONENESS IN ATHLETES

Epidemiology

When discussing the epidemiology of infections in sport, a comparison between studies is challenging due to heterogeneity of reporting[3]. Whereas the numerator of the incidence (infections) can be acceptably registered, this is more difficult for the denominator (exposure) because exposure is not limited to the athletic activity alone but likely even higher outside of the sport setting. Among other procedures, reporting per athlete days and per training days can be found. In addition, even the definition of an infection (numerator) can be challenging. Frequently, confirmation by laboratory values (or physician diagnosis) is not available and then replaced by symptom checklists which are subject to individual sensitivity. However, sensitivity is typically assumed to be higher in athletes than in the general population for any physical complaint.

Given the rarity of infections (in Norwegian Olympic athletes: 8.9 days per year of time loss due to illness[4]), small samples - as they are realistic in elite sport - can be considered too limited to generate sufficient numbers of infectious diseases to fulfill criteria of statistical power. This has been criticized within a recent meta-analysis[3] which has stated that the duration of many studies was short and that rarely proper non-athlete control groups were employed. In addition, many infection studies only represent an addendum of injury studies which are typically conducted by orthopedic doctors and physiotherapists with a clear focus on traumatology. Therefore, the overall diligence in diagnosing and reporting infectious episodes can be assumed to be suboptimal (at least below levels of injury reporting).

Illustration

Experiments

A large number of available (quasi-) experimental studies has exposed athletes only to very few bouts of exercise and registered responses of immune parameters that can be measured in the blood or saliva. An open window has typically been observed after either very long, repeated or very intense exercise[5,6,7]. However, what does it mean when salivary IgA or NK cells in the blood fall below baseline levels? Are they downregulated or engaged elsewhere in the body? The conversion into an acute infectious episode at least needs the presence of an infectious agent. Also, this infectious agent has to overcome (remaining) defense mechanisms which do not only consist of salivary IgA and NK cells.

When vaccines were used as "defined immune stimulating stimuli"[8,9,10,11] (in contrast to an infection, even the antigen dose is defined), it was shown for influenza and SARS-CoV-2 that vaccine-specific immune responses were NOT relevantly different between elite athletes under regular training and the general population. In fact, some aspects of the humoral and cellular response were even stronger in athletes for the tetravalent influenza vaccine. This illustrates that available evidence from exercise experiments will remain mechanistic only when there is no proper epidemiological underpinning.

It is important to note that other factors often present in elite athletes also increase the risk of infections: sleep, travel, nutrition, psychological stress, and others[12,13]. Whether the reported alterations in immune cells and biomarkers, in fact, lead to an increased risk of infections is still under debate. It is plausible (but not proven) that the decreases found in the first few hours after exercise indicate that immune function is indeed compromised. The fact remains that numerous study groups have reported an increased incidence of upper respiratory tract infections during or after periods of intense training or after competition in professional athletes. However, it is unlikely that this observation alone is sufficient to construct a general infection proneness of athletes.

ARE LESS INFECTIONS A SUCCESS FACTOR IN ELITE ATHLETES?

There exists an complementary explanation for differing infection rates in athletes: The very best ones (obviously a small group) have less infections than their less successful opponents despite similar training loads[14]. This may come from better resilience to threats from repeated intense bouts of exercise and infectious agents and may even represent a "talent factor". The interpretation aligns very much with one for injury proneness: The highest level of athletes is more resistant to injuries – let it be based on genetic endowment or trained factors. This relationship has been described as "S-shaped curve"[15] (as opposed to the J-shaped curve) indicating a positive turn of the curve for the very elite athletes.

DOES IT MAKE SENSE TO AVOID INFECTIONS AT ANY RATE IN ATHLETES?

An observation has been made in the seasons after the SARS-CoV-2 pandemic: The courses of infectious diseases had become more severe after all distancing and other hygiene measures that have been applied to avoid Covid transmission[16]. This may illustrate that any immune system needs training. Therefore, even for elite athletes it is likely a trade-off between

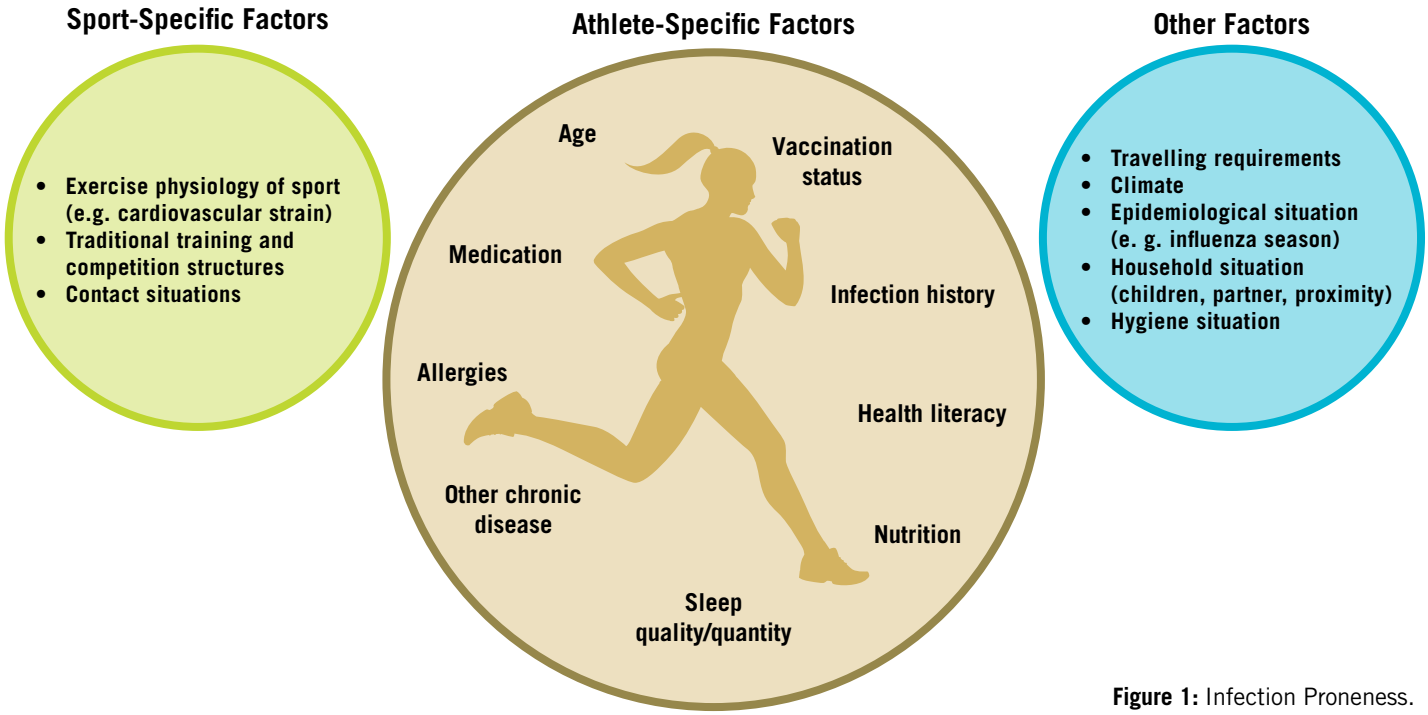

Figure 1: Infection Proneness.

avoiding infections and risking more severe infections later. However, no athlete-specific data confirm this until today. It is more of a clinical observation that has been made in other settings; still transferability to athletes seems likely. In practice, it may mean that athletes should be allowed to live a normal life – at least outside of periods just before and during very important competitions.

A MODEL FOR INFECTION PRONENESS IN ATHLETES AND HOW TO MANAGE IT

Instead of expecting an increased likelihood of infectious diseases in athletes based on generalized training- and competition-induced proneness, one should rather try to be more specific/individualized and analyze sport (typical training and competition schedules, cardiocirculatory strain, time of the season, content of training sessions) and athlete (sex, age, predisposing factors like allergies, known infection proneness etc.) to determine necessary preventative measures. Besides saving resources such a behaviour avoids excessively hiding athletes away from infectious agents and, thereby, in the long term contributing to more severe courses of infections (see preceding paragraph). This has been illustrated in Figure 1.

CONCLUSIONS

It may be time to abstain from simplifying and undifferentiated statements about athletes' proneness to infections. There are certainly disciplines with typical training volumes and patterns being permissive to infections. Also, more challenging training phases (physically and mentally) can be named which may compromise the immunity of athletes temporarily. However, a general susceptibility to infectious diseases has never been convincingly documented and stands in contrast to clinical experience (and would even be hindering a successful career). It follows that there is no need to generally hide away athletes from public contacts. Instead targeted (temporary) measures appear more appropriate for the long-term preventative management for infections, e.g. prior to very important competitions.

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