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Review

The impact of sport related stressors on immunity and illness risk in team-sport athletes

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ABSTRACT

Objectives: Elite team-sport athletes are frequently exposed to stressors that have the potential to depress immunity and increase infection risk. Therefore, the purpose of this review is to describe how team-sport stressors impact upon immune responses, along with exploring whether alterations in these markers have the potential to predict upper respiratory tract illness symptoms.
Design: Narrative review.
Methods: Salivary secretory immunoglobulin A (SIgA) and T-cell markers have been shown to predict infection risk in individual endurance athletes. Papers discussing the impact of team-sport stressors on SIgA and T-cells were discussed in the review, studies discussing other aspects of immunity were excluded. Journal articles were sourced from PubMed, Web of science and Scopus. Key search terms included team-sport athletes, stressors, immunity, T-cells, cytokines, SIgA and upper respiratory illness.
Results: Most team-sport stressors appear to increase risk for illness. An association between reduced SIgA and increased illness incidence has been demonstrated. Intensive training and competition periods have been shown to reduce SIgA, however, it is less clear how additional stressors including extreme environmental conditions, travel, psychological stress, sleep disturbance and poor nutrition affect immune responses.
Conclusions: Monitoring SIgA may provide an assessment of a team-sport athletes risk status for developing upper respiratory tract symptoms, however there is currently not enough evidence to suggest SIgA alone can predict illness. Team-sport stressors challenge immunity and it is possible that the combination of stressors could have a compounding effect on immunodepression and infection risk. Given that illness can disrupt training and performance, further research is required to better elucidate how stressors individually and collectively influence immunity and illness.

1. Introduction

Athletes are continually exposed to stressors that have the potential to depress immune functions and increase infection risk, particularly upper respiratory tract infections (URTI) or the common cold. Indeed, acute URTI is the most common infectious illness reported in elite athletes.[1] Illness is of great concern for athletes as it can disrupt training and performance.[1] To understand why athletes appear to have a heightened risk for infection, the field of exercise immunology emerged. Studies in this area have been largely concerned with identifying immune markers that can be used to predict athletes' susceptibility to illness, mostly in endurance athletes. Specifically, mucosal immunity and T-cell cytokine responses have been found to be key determinants of URTI risk in athletes participating in individual endurance sports.[2] However, there is comparatively little research in team-sport athletes, therefore whether such immune markers influence a team-sport athletes risk for URTI remains unclear. Team-sport athletes are an important population to investigate as their close proximity to team mates and other athletes (e.g. shaking hands, contact sports, changing rooms) may increase their exposure to infection-causing pathogens. Furthermore, team-sport athletes are continually exposed to a range of stressors which may depress immune responses. Such stressors

Abbreviations: URTI, upper respiratory infection; URTS, upper respiratory tract symptoms; SIgA, salivary secretory immunoglobulin A; T_h, T-helper cell; T_c, T-cytotoxic cell; Treg cells, regulatory T-cells; T1, type 1 T-cell; T2, type 2 T-cell; IFN-γ, interferon gamma; IL, interleukin; HIIA, high intensity intermittent activity; ITP, intensified training period; ICP, intensified competition period; RH, relative humidity.

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include training, competition, travel, environmental extremes, psychological stress, sleep deprivation, poor nutrition and excessive alcohol consumption. It is possible that the combination of these stressors could amplify immunodepression, resulting in greater URTI risk than if each stressor were applied alone. The purpose of this review is to describe how team-sport stressors impact upon salivary secretory immunoglobulin A (SIgA) and T-cell responses, along with exploring whether alterations in these markers have the potential to predict infection risk. Finally, strategies to counteract the potential immunodepression evoked by these stressors will be proposed.

2. Salivary secretory immunoglobulin A (SIgA), T-cells and URTI

The human body is under constant assault by bacteria, fungi, viruses and other infection-causing microorganisms. The immune system provides humans with an array of defence measures to resist such attacks. Humans have three functional divisions that defend the host from infection; the mucosal, innate and acquired immune systems.[1] This review will focus on the markers of SIgA and T-cells which belong to the mucosal and acquired immune systems, respectively. Systemic innate immune responses will not be discussed as in addition to defending the body from infection these cells are involved in tissue damage repair and remodelling.[1] As such, alterations in SIgA and T-cells may be more directly related to the URTI risk.

Salivary SIgA. Saliva collection and analysis is growing in popularity as a non-invasive tool for assessing biomarkers. In the field of exercise immunology SIgA has received considerable attention as it is the main effector of the mucosal immune system.[3] SIgA represents the first line of defence as it prevents colonization and replication of viruses and bacteria on the mucosal surface of the respiratory tract.[3] Although widely examined, methods of collecting, measuring and expressing SIgA vary considerably between studies. The choice of collection method (e.g. cotton swab, passive drool, stimulated, unstimulated) differs between studies as do the quantitative bioassays that are used to measure SIgA (e.g. direct, indirect, functional, ligand-binding assays).[3] SIgA can also be expressed in various ways (e.g. concentration ($mg\,L^{-1}$), secretion rate ($\mu g\,min^{-1}$) and ratio to protein (μg SIgA: mg protein^{-1})). Moreover, it can be expressed as absolute or relative to an athlete's mean healthy SIgA levels. These methodological differences have made it difficult to directly compare studies.

T cells and T1/T2 balance. Emerging evidence suggests T-cell responses may influence illness susceptibility in athletes. T-cell subpopulations of T-helper (T_h), T-cytotoxic (T_c) and Regulatory T-cells (Treg cells) play an important role in immunity as these cells coordinate immune responses, kill infection-causing pathogens and regulate immune responses, respectively.[4] T_h and T_c cells can be further subdivided into type 1 (T1) or type 2 (T2) T-cells based on, in part, the cytokines they produce.[4] Cytokines are small signaling molecules that direct immune responses.[4] T1 cells produce pro-inflammatory cytokines including interferon gamma (IFN-γ), interleukin-12 (IL-12) and IL-2 to promote cell mediated immunity (CMI). CMI is the arm of the immune system responsible for defending the body from intracellular pathogens like viruses.[5] T2 cells produce various anti-inflammatory cytokines (IL-4, IL-5, IL-13) that are essential for the development of humoral immunity.[4] The humoral arm of the immune system primarily targets bacterial and fungal infections.[4] The balance between T1/T2 immunity needs to be maintained for optimal immune defence, when it is tipped to favour T2 immunity athletes may be at an increased risk of viral infection.[5] This is a concern considering most infectious presentations in athletes are of viral origin.[6] Recent studies have also examined Treg cells which are the central anti-inflammatory regulators of the immune response.[4] Treg cells primarily produce IL-10 to suppress the effector functions of immune cells, particularly T1 cells.[4] To assess the functional status of T-cells, researchers tend to use in vitro methods, whereby drawn blood is incubated with a stimulant. Stimulated T-cell cytokine production provides an indication of the in vivo response that would be initiated if an athlete were to come into contact with an infection.[2]

Upper respiratory tractsymptoms. Upper respiratory tract infection is the most common acute illness experienced by athletes.[1] Common URT symptoms (URTS) include sore throat, headache, runny nose and coughing.[6] In the exercise immunology literature, researchers tend to use self-reports or doctors diagnoses to establish URTI. However, such methods are limited as laboratory investigations have shown that a considerable proportion of URTS are not in fact associated with a respiratory pathogen.[7] URTS can result from infectious (viral, bacterial or fungal etiology) or non-infectious and inflammatory (e.g. caused by allergies, asthma and trauma to respiratory epithelial membranes) causes.[6] For further information on the etiology of URTS please refer to a review by Gleeson and Pyne.[6] Irrespective of the underlying cause, URTS have negative implications for athletes as they can impair performance.[6] For the purpose of this review, if illness episodes have not been clinically confirmed as URTI, they will be referred to as URTS.

Immune markers identified as risk factors for URTS. Exercise immunology research has tended to focus on athletes participating in individual endurance sports, in this population SIgA and T-cell cytokine response have been identified as risk factors for URTS. In comparison to healthy endurance based athletes, illness prone athletes have been found to have lower resting SIgA concentrations and unstimulated IFN-γ production, but higher multi-antigen stimulated IL-4 and IL-10 production.[2] Cytokines IL-4 and IL-10 are both antagonistic to IFN-γ and induce immune polarisation towards an anti-inflammatory response. IFN-γ is important for viral defence, therefore athletes may be more predisposed to viral infection when IFN-γ production is reduced.[5] Compared to endurance athletes, relatively fewer studies have investigated these markers in team-sport athletes.[8–10] The next section of this review will explore how stressors associated with team-sport participation influence SIgA, stimulated T-cell cytokine production and infection risk.

3. Stressors that team-sport athletes face on a continual basis

Modern day elite team-sport athletes are being increasingly exposed to a variety of stressors, as summarised in Fig. 1. Given the substantial investment that goes into preparing team-sport athletes for competition, a real demand exists to identify which stressors evoke the greatest immunodepression and subsequent risk for infection. These stressors are not exclusively unique for team-sports, however, this review will adopt a team-sport focused approach as previous research has tended to focus on individual based athletes. Stressors will be separated for clarity on how they impact immunity, although it is appreciated that some stressors are inherently combined.

Acute team-sport exercise. The influence of acute team-sport exercise on SIgA is equivocal. No change in SIgA has been documented following basketball,[11] football[12] and rugby games.[13] Contrastingly, significant reductions in SIgA have been reported following futsal and football matches.[14–17] In fact, previous studies have shown that the decline in SIgA can be substantial (75% decrease)[16] and sustained for several hours post-match (>18 h).[15] In comparison to competitive matches, limited studies have investigated the effects of a single team-sport training session on mucosal immunity. While no alteration in salivary SIgA concen-

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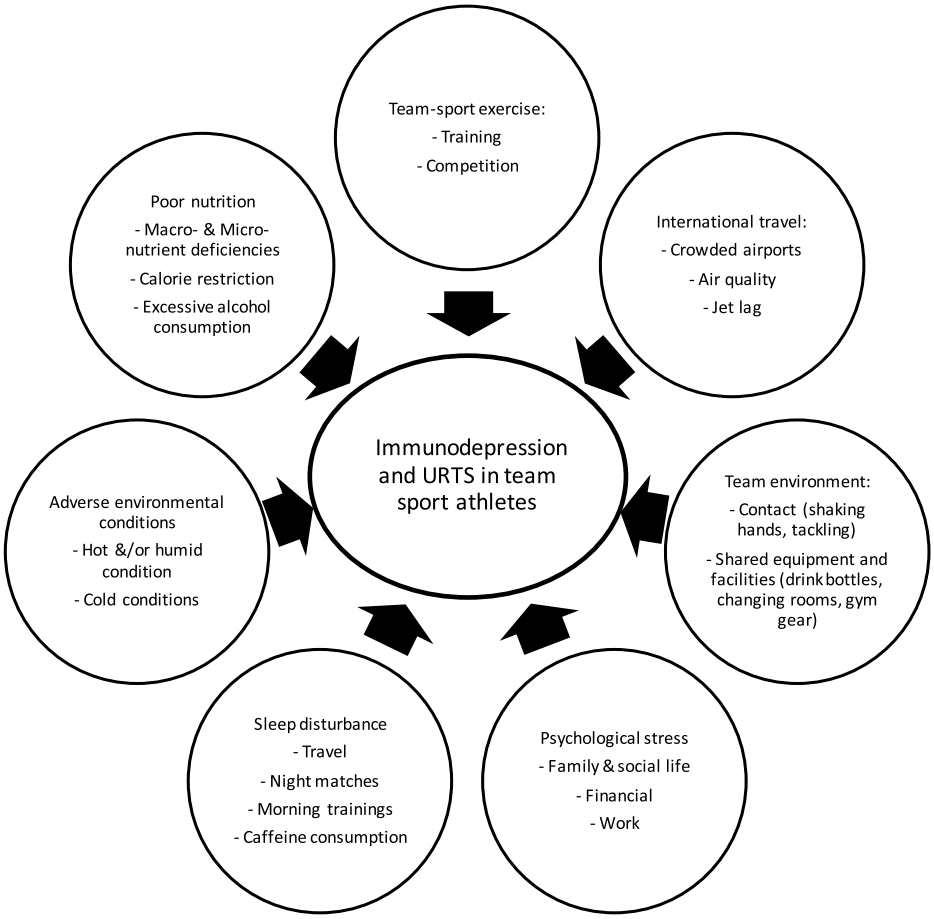

Fig. 1. Stressors that can reduce immunity and increase URTS risk in team-sport athletes (adapted from Walsh [85]).

tration was observed in males following three different basketball training sessions,[18] limited conclusions can be drawn from this study due to the disclosure of what the training sessions consisted of (e.g. intensity, duration). Subsequent research by Owen et al. compared mucosal immune responses to four low intensity (technical, tactical focus) and high intensity (repeated sprint activity, aerobic intervals and small sided games) trainings.[19] Salivary SIgA concentration was consistently lower following all high intensity versus low intensity training sessions. However, this difference only reached significance following the fourth session, suggesting high intensity trainings may have a cumulative suppressant effect on salivary SIgA.[19] The discrepancies in the literature may be explained by methodological differences between studies including the training status of participants, the sport assessed and the method of expressing SIgA.

In comparison to SIgA, fewer studies have examined T-cell responses to acute team-sport exercise. Nonetheless, a significant reduction in circulating T_c and T_h1 cell counts has been reported after two consecutive futsal games and a soccer match, respectively.[20,21] Reductions in circulating T-cell counts does not necessarily reflect immunodepression, in fact it may be indicative of enhanced immunosurveillance in the organs to which these T-cells traffic to following team-sport exercise.[1] Interestingly, sex differences in T-cell mobilisation following exercise have been reported in amateur soccer players,[22] further research examining the role sex has on T-cell responses is needed. It is apparent that team-sport exercise alters cell redistribution, however, the effect of team-sport matches on T-cell functions remains unknown. Team-sport is characterised by high intensity intermittent activity (HIIA). Only handful of laboratory studies have examined the impact of HIIA on T-cell functions. Bishop et al.[8] found that antigen-stimulated T1 (IFN-γ) and T2 (IL-4) T-cell cytokine transcriptional factors were unaltered by 90 min of HIIA. Similarly a recent study showed that HIIA did not affect super-antigen stimulated IFN-γ production.[9] These findings differ to those reported with acute endurance exercise as stimulated IFN-γ production tends to be decreased post-exercise,[5] thereby suggesting that HIIA may be less disruptive to T1 anti-viral activity than prolonged exercise. However, findings from laboratory based HIIA trials may not necessarily translate to real life competitive matches. Given the limited data, further research is required to explain the impact of acute team-sport exercise on T-cell responses.

Pre-season training and intensified training periods. SIgA resposnes tend to be impaired by strenuous pre-season training and intensified training periods (ITP), which are typically characterised by high training loads over a specified amount of time (weeks-months). Compared to off-season and recovery periods, lower SIgA and a greater number of URTS have been demonstrated in elite rugby and youth soccer players during nine and 12 weeks of pre-season training, respectively.[23,24] In fact, at an amateur level, pre-season football training has been shown to modulate immunity and illness risk, regardless of the time of year. For example, in comparison to the teams average baseline SIgA secretion rate of $54.9 \pm 1.3\ \mu g\ min^{-1}$, SIgA secretion rate was reduced by $14.1\ \mu g\ min^{-1}$ (autumn/fall) and $14.5\ \mu g\ min^{-1}$ (spring) after pre-season training periods.[25] Additionally, during the two preseasons 32–56% of 75 players reported URTS compared to just 5% of players in the offseason.[25] Collectively these studies have demonstrated a link between pre-season training, SIgA levels and URTS, although they failed to examine baseline fitness levels. Previous

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studies have shown that fitness levels influence mucosal immunity and illness rates.[1,26] Further research examining the fitness levels of team-sport athletes returning to pre-season training is needed to understand whether or not it is associated with immunity and risk for illness.

Similar to pre-season training, suppression of SIgA has been documented with ITP. In elite and sub-elite basketball players significant reductions in SIgA concentrations have been reported following 17 day and 4 week ITP, respectively.[27,28] Reduced SIgA has deleterious effects for host protection as pathogens can more easily enter the epithelial surface and establish infection. Furthermore, emerging evidence suggests that the maintenance of SIgA secretion may be important for resistance to viral infection. Yamauchi et al. [29] found that reductions in SIgA secretion rate tended to occur one day before virus-DNA reactivation in collegiate level rugby players during a one month training camp. Moreover, decreased SIgA was associated with increased incidence of URTS.[29] In contrast to previous research, a recent study on male basketball players showed that SIgA concentrations were unaltered by a four week ITP.[30] Interestingly, this study involved youth (15.8 ± 0.8 years) while most previous studies were conducted on adult athletes (>20 years).[27–29] The influence of age or maturity on immune responses to an ITP may require further examination. In addition, future research investigating the impact of pre-season training and ITP on immunity and illness in female team-sport athletes is required as sex differences in SIgA levels and illness incidence have been demonstrated in individual endurance based athletes.[31]

Literature on the impact of pre-season training and ITP on T-cell responses is limited. In university level athletes, no alteration in T_c and T1 cell counts have been demonstrated following a five day intensive training camp,[32] suggesting ITP do not alter anti-viral defence. These findings contradict the significant reductions in T1 cell counts and functions that have been reported following ITP in endurance athletes.[33] In contrast to T1 cells, a 12.9% increase in Treg cells have been reported in elite team-sport athletes following one week of intensive training.[34] Although this increase was not significant it demonstrates a potential link between team-sport training and Treg cells. Treg cells are essential for immune homeostasis as they suppress excessive immune responses to prevent autoimmunity, however, increased frequencies of Treg cells can result in a state of immunosuppression.[34] As such, ITP could potentially increase a team-sport athletes risk for infection via augmenting circulating Treg cells. However, previous team-sport studies have not reported URTS, as such the clinical implications of altered T-cell counts is unknown.

Competition. Substantial reductions in SIgA with intensive competition periods (ICP) have been consistently reported in the team-sport literature.[24,25,28] During competition, the decline in SIgA appears to be more severe when fixtures are congested and recovery time is limited. Indeed, in elite rugby union players Cunniffe et al.[23] observed a greater reduction in mean relative SIgA concentration following an ICP (nine games in eight weeks) compared with a less competitive period (two games in four weeks) (29% vs 9% decrease in SIgA). Furthermore, reductions in SIgA seemed to predict illness risk as the highest incidence of URTS occurred during the 8-week intensive competition period.[23] Similar data have been presented in elite soccer players. Significant reductions in SIgA have been demonstrated during ICP (five matches over 15 days),[35] while an association between decreased resting SIgA concentration and increased illness incidence was demonstrated when players participated in seven soccer matches over 20 days.[36] When competition periods are less intensive, SIgA levels may be maintained. However, regardless of the recovery window between matches, competing on a regular basis appears to have a cumulative suppressant effect on salivary SIgA. This was demonstrated in elite Australian Rules footballers, from which SIgA samples were taken at baseline (pre-competition) and 36 h after 16 matches (6–8 days recovery between matches).[37] From match one to seven SIgA concentration was not significantly altered. However, from match 7 onward resting salivary SIgA concentration was significantly lower than those obtained at baseline.[37] Therefore, team-sport practitioners should be aware that athletes may be more susceptible to illness in the latter stages of the competition period where mucosal immune defences are lowered. Taken together, previous research suggests that competition periods can impair SIgA secretion and subsequently increase a team-sport athletes risk for infection, although, there appears to be a paucity of data on female team-sport athletes. Inconsistent findings have been presented on female collegiate soccer players. In agreement with male studies, an association between reduced SIgA and increased illness incidence has been demonstrated.[38] Conversely, SIgA has been shown not to predict URTS.[39] Further research on females, specifically at an elite level is warranted as previous studies have demonstrated that competition level may influence immune markers and illness incidence.[40,41]

A handful of longitudinal studies have assessed T-cell responses to training and competition periods in team-sport athletes. A reduction in T-cell counts has been demonstrated following 6–11 months of soccer training and competition.[42,43] However, research describing the T1/T2 T-cell response to competition is scarce. Del Giacco et al.[10] examined T1 and T2 cell subsets in Italian premier league football players over an 11-month season. In comparison to cell counts measured in the offseason, equivocal findings were reported at the end of the season as the number of IL-2 and IL-4 producing T-cells were significantly reduced while IFN-γ producing T-cells did not change.[10] Unfortunately, T1 and T2 cell functions (stimulated cytokine production) were not assessed in this study. Alterations in T1 and T2 T-cell counts and functions do not always occur in parallel.[5] Therefore, studies examining the impact of team-sport training and/or competition periods on T1 and T2 cell functions should be conducted.

The feasibility of using salivary SIgA and T-cell responses to predict infection risk in team-sport athletes. To prevent illness, researchers have attempted to identify the timeframe between lowered SIgA and the actual onset of illness. A recent review by Jones et al.[44] suggested that a latency period exists between reduced SIgA and URTI development. The authors proposed that athletes who exhibit sustained suppression of SIgA (7–21 days) following a rapid increase in training load will have a 50% greater risk for contracting URTI.[44] However, shorter time frames have been reported in soccer players with 82% of illness explained by a preceding decrease (within seven days) in SIgA in female players[38] and just days before the appearance of URTS in male players,[45] suggesting that sex differences in alterations in SIgA and illness onset may exist.

Along with establishing a time interval, researchers have sought to identify which SIgA concentrations may predispose an athlete to illness. In endurance athletes pre-season SIgA concentrations of $<40\ mg\ L^{-1}$, between $40–60\ mg\ L^{-1}$ and $>60\ mg\ L^{-1}$ have been associated with high, moderate and low risk for illness over a season, respectively.[46] Data are limited in team-sport athletes, although Fahlman et al.[25] found that team-sport athletes were at an increased risk for developing URTS when SIgA secretion rate dropped below $40\ \mu g\ min^{-1}$. Thus, SIgA appears to be a useful clinical biomarker for practitioners to examine when trying to predict infection risk in team-sport athletes. Practitioners can measure SIgA on a regular basis using point-of-care SIgA tests, which provide results within minutes. However, the usefulness of measuring SIgA may be somewhat limited as high inter- and intra-individual variability has been demonstrated.[3] Therefore, it may be more appropriate to express SIgA in relative terms (i.e. SIgA levels relative to an athlete's mean healthy SIgA) when trying to predict illness risk. Indeed, in elite sailors a SIgA concentration less than 40% rel-

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ative to mean healthy SIgA indicated a 48% chance of developing URTS within three weeks.[47]

Previous studies examining T-cell subpopulations (T1, T2 and Treg cells) in team-sport athletes have failed to make parallel assessments of URTS. Therefore, there is currently no evidence to suggest that alterations in T-cell cytokine production predict infection risk. However, the functional status of T-cells appears to be a promising marker in endurance athletes, as reduced unstimulated IFN-γ production and greater antigen-stimulated anti-inflammatory cytokine (IL-4 & IL-10) production have been associated with URTS.[2] Therefore, further studies on team-sport athletes are required to determine if stimulated T-cell cytokine production influences illness risk.

Travel. Along with exercise stress, elite team-sport athletes are often required to travel across multiple time zones. Travel is associated with a range of challenges that can threaten immunity. The ventilated enclosed environment of an aircraft cabin exposes athletes to hypobaric hypoxia, dry humidity and close proximity to others, thereby increasing risk for infection transmission.[48,49] Furthermore, following long haul travel athletes can experience jet lag which is a physiological condition caused by disturbance of the body's circadian rhythm.[48,49] It seems plausible that desynchronization of rhythmic immune parameters would result following long haul travel as body temperature, hormonal circadian rhythms and sleep wake cycles are all disrupted by international air travel.[49] In athletic populations, it has been demonstrated that travel can increase an athlete's risk for illness. For example, in elite rugby players, changing time zones was associated with increased illness incidence, not the direction of travel or flying per se. Furthermore, travelling to international destinations that were >5 h time zone difference away from an athlete's home country was associated with a 2–3 fold increased risk of illness.[50] Similarly, long haul travel across 11 times zones was found to exacerbate URTS in elite rugby league players.[51] The effect of travel on illness incidence in female team-sport athletes is yet to be elucidated, however in 39 elite cross country skiers, 17 of whom were female, air travel was shown to significantly increase risk for URTS.[52] The current limitation to the studies discussed is that immune markers were not measured alongside the illness reports. This limits our understanding of how travel may impact upon the immune system to subsequently increase risk of URTS.

Environmental Extremes. Elite team-sport athletes are often required perform in challenging environmental conditions including heat/humidity and cold. Exercising in such conditions imposes significant physiological strain. For example, exercise in hot or cold environments elicits a more substantial increase in circulating stress hormones than exercise in thermoneutral conditions.[53] Knowing that immune suppression has been associated with elevations in cortisol and catecholamines,[1] it seems plausible to hypothesise that exercise in hot or cold conditions would evoke greater immune suppression and subsequently higher incidence of URTS. However, exercise in hot (28–38.7 °C, 45–76% relative humidity (RH)) or cold (water: 18–23 °C or air: −6.4–5 °C) environments does not appear to cause further immunodepression than exercise in a thermoneutral environment (air: 18–22 °C, 30–60%RH or water: 35 °C).[53] Specifically, similar SIgA responses to exercise have been demonstrated when performed in hot[54] or cold[55,56] conditions compared to thermoneutral conditions. Although, T1 and T2 T-cell responses to exercise in environmental extremes tend to be more varied. Compared to thermoneutral conditions, T1 and T2 cytokine production has been shown to be attenuated[57,58] or augmented[59] with exercise in the cold. However, no alteration in T1 and T2 cytokine production has been demonstrated following exercise in the heat (35 °C & 60% RH).[60] Given the important role that T-cells play in coordinating immune responses and eliminating infection-causing pathogens further studies are required.

Overall, exercising in adverse environmental conditions appears to have modest effects on immunity. However, previous studies have mostly used steady state exercise protocol. Therefore, findings are not necessarily applicable to team-sport athletes who engage in HIIA. Sari-Sarraf et al.[61] examined the mucosal immune response to 90 min of soccer specific intermittent exercise in a hot environment (30 °C & 40%RH). Equivocal findings were reported as SIgA concentration was unaltered, SIgA secretion rate increased and the SIgA to protein ratio decreased.[61] Unfortunately, no control temperate condition was included in this study. Thus, it remains unclear how thermal stress alters immune responses to team-sport specific exercise. Moreover, this study was conducted on recreationally active males, not team-sport athletes. As many major team-sport events are scheduled in potentially adverse environments future research is warranted to elucidate how performing team-sport exercise in hot and cold conditions influences immunity and risk for infection.

Lifestyle factors. Lifestyle factors associated with team-sport participation including psychological stress, sleep deprivation, poor nutrition and excessive alcohol consumption have the potential to disrupt immunity and increase infection risk. It is well established that psychological stress influences immunity and illness susceptibility.[62] Psychological stress is accompanied by elevations in hormones, cortisol and adrenaline which suppress immune functions and in turn may increase illness susceptibility.[63] Indeed, early work in this area demonstrated that URTI and clinical colds increase in a dose response manner with increased degree of psychological stress.[64] Similarly, an association between psychological stress and illness incidence has been demonstrated in overreached elite youth soccer players.[65] Unfortunately, limited team-sport studies have examined the impact of psychological stress on immune responses and therefore further investigation is required to better understand the mechanisms underpinning increased illness susceptibility.

Along with psychological stress, elite team-sport athletes are frequently exposed to situations and conditions that can disturb sleep quantity and quality (e.g. international travel, caffeine consumption, night matches and morning trainings). Sleep disturbance appears to influence illness susceptibility. Cohen et al.[66] demonstrated that individuals sleeping less than 7 h per night prior to inoculation with an active cold virus were three times more likely to develop a cold than those sleeping 8 h or more. Similarly, an association between lack of sleep/poor quality sleep and increased risk for developing URTS has been demonstrated in endurance athletes.[67] There is evidence to suggest sleep deprivation may cause immunodepression,[68] although, conversely short term changes in sleep disturbance have been shown to prime immunity and enhance the redistribution of T_c cells in response to endurance exercise, which may be indicative of enhanced immunosurveillance.[69] Findings from the general population and endurance athletes are not necessarily applicable to team-sport athletes, therefore research examining the impact of sleep on immunity and illness in team-sport athletes is required.

Similarly, poor nutrition can increase infection risk as deficiencies of protein, energy and particular micronutrients impair immunity.[70] Team-sport athletes may be at an increased risk for nutritional deficiencies when purposefully calorie restricting. Such dietary behaviours typically occur during pre-season periods as athletes seek to improve body composition. In combat-sport athletes, immunodepression has been reported with dieting and rapid weight loss.[71] At present there are limited data on team-sport athletes, although a recent study involving 81 elite athletes, 34 of whom were team-sport, demonstrated a significant association between low energy availability and illness.[72] Thus, calorie restriction whether it be inadvertent or purposeful will likely increase a team-sport athletes risk for illness.

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Table 1
Strategies to reduce illness risk in team-sport athletes.

Stressor	Strategy
Transmission of contagious infection throughout the team	- Promote good hygiene practices (e.g. wash hands, carry anti-bacterial hand gel, shower, do laundry regularly) - Advise athletes to avoid ill people, crowded areas, animals, children and contaminated objects - Discourage athletes from sharing equipment (drink bottles, towels, sporting gear etc.) - Routinely clean shared equipment (e.g. weights) - Advise athletes to have recommended immunisation vaccines - Monitor athletes' illness symptoms and their severity (Jackson common cold scale)[79] - Monitor sickness in the athletes household
Training	- Periodise training: ■ Gradually increase training load & volume (increases of 5–10% per week) ■ Limit monotony & staleness by including cross training ■ Provide sufficient rest & recovery time - Monitor athletes' internal training load, monotony, strain & wellbeing ratings to better predict infection risk - Athletes experiencing URTS should avoid high intensity exercise as it can depress immune functions
Competition	- Ensure prompt recognition and management of athletes experiencing symptoms of illness (e.g. isolate sick athlete, move out roommates) - Following a match ensure athletes have a recovery period of at least 24 h to allow immune variables to return to baseline
Travel	- Advise athletes to have recommended travel vaccines - Provide athletes with disposable face masks - If possible seat athletes away from ill passengers - Encourage athletes to drink plenty of water to keep well hydrated to ensure mucosal secretions are stable and do not dry out - After travel schedule adequate recovery time (≥24) before athletes engage in strenuous training sessions or competition - Avoid flying on the same day as competition, delay homeward travel until the subsequent day - Consider circadian variation and use strategies to shift circadian phase into new time zone[80]
Hot conditions	- Implement strategies to reduce physiological strain: ■ Heat acclimation — see recent review for guidelines[81] ■ Cooling (ice slurries, cooling garments etc.) ■ Ensure athletes are well hydrated ■ Increase carbohydrate intake[81]
Cold conditions	- Implement strategies to reduce physiological strain: ■ Ensure athletes have appropriate cold weather clothing ■ Increase carbohydrate intake - Provide athletes with facial masks to protect airways being exposed to cold air during exercise
Stress	- Monitor athletes stress levels (daily wellness questionnaire) - Provide education around stress management techniques and coping strategies[82]
Sleep	- Monitor sleep quality and quantity - Advise athletes to get a minimum of 8 h of sleep per night - Apply strategies to optimize sleep quantity and quality[83]
Nutrition	*Generally:* - Encourage athletes to eat a well-balanced diet without excessive or inadequate intake of macro- and micro-nutrients. - Discourage crash dieting. - Identify any nutritional deficiencies and target nutritional strategies. *During periods of increased risk (e.g. intensified training periods, competition & travel):* - Consider supplementation (e.g. Probiotics, carbohydrates, flavonoids) (See recent Consensus Statement [70]) *When athletes first start developing URTS:* - Consider Zinc acetate lozenges (80 mg/day) as they have been found to reduce the duration of the common cold [84] *Alcohol:* - Discourage excessive alcohol consumption particularly following exercise - Low alcohol consumption (one or two drinks/day for female or male athletes) does not appear to impair immunity

URT, upper respiratory tract; IFN-γ, interferon gamma.

Excessive alcohol consumption can also directly impair immunity and may subsequently increase susceptibility.[73] High alcohol consumption has been reported in team-sport athletes, particularly males, in the hours following a sporting match as part of the post-game celebration. While low to moderate consumption (one drink (10–12 g) or two drinks (20–24 g) of alcohol for women and men per day) is ok and in some instances, can even have beneficial effects on immunity, high doses of alcohol consumption (>two drinks/day) can be detrimental.[73,74]

Interestingly these additional lifestyle factors all share a common feature in that they appear to modulate T-cell responses. A reduction in CMI and a shift in the T1/T2 balance to dampen T1 immunity and favour T2 immunity has been reported with psychological stress,[75] sleep deprivation,[68] calorie restriction[71] and excessive alcohol consumption.[73] Therefore, a team-sport athlete's risk for viral infection may be increased when exposed to such lifestyle factors. However, previous studies are limited in that they have just examined lifestyle factors in isolation when in reality team-sport athletes may be exposed to several at one time.

4. Interactive effect of stressors on immunity and infection risk

Various stressors that team-sport athletes can be exposed to have been discussed in this review. However, it is important to distinguish the difference between acute and chronic stress; acute stress lasts for minutes to hours, while chronic stress persists for several hours per day for weeks to months.[76] While acute sport

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related stressors may enhance or 'prime' immunity,[69,77,78] evidence suggests that the duration of stress is a determining factor in its influence on immunity and health, with chronic stress having detrimental effects on immunity. Further research examining the impact of both acute and chronic exposure to team-sport stressors is needed to understand the impact and potentially compounding effects on immunity and illness risk.

5. Strategies to reduce immunodepression and avoid illness in team-sport athletes

It is apparent that immune function is influenced by a number of stressors that team-sport players are exposed to on a regular basis. To help maintain player availability and minimise the potential immunodepression evoked by such stressors team-sport practitioners could employ the strategies listed in Table 1.

6. Conclusions

At present, our understanding on how stressors individually and collectively influence a team-sport athlete's immune system is limited as much of the research has been conducted on endurance athletes. Immunodeprresion and increased illness risk have been shown with intensive training and competition periods. Monitoring SIgA may provide an assessment of a team-sport athletes risk status for developing URTS, however there is currently not enough evidence to suggest SIgA alone can predict URTS. Team-sport studies have predominately examined exercise stress, as such the influence of contributing stressors (travel, environmental conditions, psychological stress, sleep deprivation, poor nutrition and alcohol consumption) on immunity and illness risk is less clear. Furthermore, previous team-sport studies are somewhat limited in that they have tended to only examine SIgA or T-cell responses, whereas interactions between these immune markers normally occur. Future studies simultaneously evaluating the effect of team-sport stressors SIgA and T-cell responses are required to gain a more comprehensive insight. In addition, rather than investigating each stressor and immune marker in isolation, future longitudinal studies should be conducted to better elucidate how multiple stressors (training, competition, travel, environmental extremes and additional lifestyle factors) affect various immune functions and illness. If periods of increased risk are evident, strategies can be targeted to reduce a team-sport athletes risk for developing URTS.

Practical implications

- Modern day elite team-sport athletes are being increasingly exposed to a variety of stressors (high training loads, congested match schedules, international travel, environmental extremes and additional lifestyle factors) that have the potential to depress immune functions and increase risk for upper respiratory tract symptoms.
- Salivary SIgA appears to be a useful clinical biomarker for practitioners to monitor team-sport athletes risk status for developing upper respiratory tract infection, however there is not enough evidence to suggest that SIgA alone can predict illness risk.
- There are several strategies practitioners could employ to help minimise immunodepression and keep team-sport athletes healthy.

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