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Winter Training Season in Endurance Runners and Cellular Signaling Knowledge of the Immune System: A Letter to the Editor

Shahin Beyranvand *

Department of Exercise Physiology, Faculty of Physical Education and Sports Sciences, Allameh Tabataba'i University, Tehran, Iran.

Letter to Editor
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* Corresponding Author: shahinberan@gmail.com.
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Abstract

Purpose: Winter endurance training combines high aerobic load with repeated cold exposure, which can alter immune-related cellular signaling and increase vulnerability to upper respiratory tract infections (URTI), thereby threatening training continuity and performance. This structured abstract aimed to summarize a signaling-based rationale for immune-informed winter periodization in endurance runners. **Method:** A narrative, mechanistic synthesis was developed from exercise immunology and cold-stress evidence, focusing on how cold exposure and endurance exercise converge on sympathetic nervous system and hypothalamic–pituitary–adrenal (HPA) axis activation, downstream endocrine responses (catecholamines, cortisol), immune cell trafficking, airway barrier function, mucosal immunity (salivary IgA), and cytokine signaling (with emphasis on IL-6). Practical implications were translated into microcycle/mesocycle design principles and a tiered monitoring framework. **Results:** Cold exposure triggers thermoregulatory vasoconstriction and central blood redistribution and is accompanied by neuroendocrine shifts that can modify leukocyte behavior, adhesion signaling, and lymphocyte proliferation. Evidence indicates that respiratory barrier defenses may be impaired in cold conditions, while mucosal immunity (e.g., IgA-related protection) can be reduced in heavily trained athletes during winter, coinciding with increased URTI reports. IL-6 responses to exercise in the cold were context-dependent: some protocols show minimal temperature effects at moderate intensity, whereas others report higher IL-6 at colder temperatures, supporting a multi-marker and longitudinal interpretation rather than reliance on single measurements. **Conclusion:** Integrating cellular signaling knowledge enables temperature-aware planning of intensity, structured warm-up/rewarming routines, monotony control, and symptom-gated adjustments to reduce "vulnerability windows" without sacrificing adaptation. Immune-informed winter periodization, supported by feasible monitoring (load, sleep, symptoms, and optional IgA/cytokines), may preserve training availability and mitigate winter performance decrement in endurance runners.

Keywords: cold exposure; endurance training; immune signaling; IL-6; salivary IgA.

Dear Editor, Cold environment and training as external stressors on the body's organisms can be associated with a decrease in the function of the body's systems, such as suppression of the immune system. In the meantime, using the knowledge of cell signaling in order to know the effects of training and cold environment on the immune system can play a role in diagnosing and preventing their complications.

The physiological adjustments made by the human body to maintain central core temperature during an external factor such as cold indicate that cold is triggered by stimulation of the sympathetic nervous system and the adrenal pituitary hypothalamus axis (HPA). These settings include chills and increased peripheral vascular contraction. In the same context vasoconstriction helps maintain body temperature by limiting blood flow to the skin, . This reduces the temperature of the skin, thereby reducing the thermal gradient between the skin and the environment. This contraction also restricts blood flow to the underlying muscles and returns the blood back to the central nucleus, which leads to increased stroke and cardiac extracorporeal volume and increased metabolic thermogenesis (Muza et al,1988). Increased blood volume inside the chest also causes divers and blood pressure. This last effect may partly point to an increase in the incidence of mycocardial infarction during the cold winter months. In addition, by stimulating the sympathetic nervous system and the hypothalamus axis of the adrenal pituitary, the concentrations of the hormones catocalamines and cortisol and aldestrone also change in the body (Radomski et al,1982). Studies conducted in the cell and molecular sector showed that the cold environment can damage physical barriers to infection, such as increased mucus viscosity and decreased mesogenic function of the upper respiratory tract. In addition, the effects of increased hormones such as corticoids and catacolamines can cause leukocytosis and reduce the expression of adhesive molecules on inflammatory cells and reduce the stimulation of mitogens and the proliferation of lymphocytes. A decrease in the proliferation of lymphocytes was also observed with a 1 degree decrease in the central nucleus.in addition, a decrease in

lymphocytes and IL-2 - IL-6 was also observed during the hypothermic state (Hess et al 2009, Solter et al 1989).

Studies conducted in the field of training in a cold environment show that training does not make a difference with 70% of the maximum oxygen consumption at temperatures from 0 to 20 degrees in IL-6 (Brenner et al,1999). Research shows that in athletes (cross- country) who training in winter, immune system function has decreased after increased URTI[1]. In addition in Desert athletes IgA levels have also decreased compared to controls group (Patterson et al 2009, Tomasi et al 1982). In in addition, research showed that 30 minutes of running at 71 percent of the Reserve heart rate at 1 to 24 degrees Celsius increased the plasma level of IL-6 at 1 degree compared to 24 degrees Celsius (Mylona et al 2002).

Therefore, considering that the cold environment and endurance training play a role in changing the messengers associated with the immune system, it is suggested that endurance runners who perform their training in winter at different temperatures increase their level of awareness related to cellular knowledge so that in the design of microcycles and mesocycles of winter training season, they consider measuring the associated messengers of the immune system to prevent their performance from decreasing.

Now, we can follow:

- Is designing a 5 and 10 km runner training program (microcycles and mesocycles) with a cellular signaling knowledge approach (immune system) effective in preventing decreased performance during the winter training season?
- Is designing a half-marathon and marathon runners training program (microcycles and mesocycles) with a cellular signaling knowledge approach (immune system) effective in preventing a decrease in performance during the winter training season?

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[1] upper respiratory tract infection

Winter is a decisive macrocycle for endurance runners. For many 5–10 km specialists, it is a period of aerobic reinforcement and reintroduction of high-quality intensity; for half-marathon and marathon runners, it often combines high mileage, long runs, and progressive threshold work. Yet winter is also the season in which athletes are repeatedly exposed to cold air, low humidity, variable wind-chill, and indoor crowding—conditions that coincide with a well-described rise in upper respiratory symptoms and illness reports in athletic populations (Nieman, 1994; Walsh, 2018).

In this letter, I argue that the most practical way to prevent winter performance decrement is not to treat "immune suppression" as a vague or inevitable consequence of endurance work, but to interpret winter training through a cellular signaling framework: cold exposure and endurance exercise converge on shared upstream regulators (notably the sympathetic nervous system [SNS] and the hypothalamic–pituitary–adrenal [HPA] axis), which then shape downstream immune behaviors (cell trafficking, adhesion signaling, cytokine balance, and mucosal barrier defense). This perspective helps translate mechanistic knowledge into actionable periodization decisions—particularly at the level of microcycles and mesocycles—so that training adaptations can be protected while illness-related training disruption is minimized.

1) Why "cold + training" should be treated as a combined stressor

Cold exposure challenges thermoregulation, forcing the body to prioritize maintenance of core temperature. Acute cold defense typically involves SNS activation, peripheral vasoconstriction, and—depending on exposure severity and clothing—shivering thermogenesis and changes in metabolic heat production. In cold environments, peripheral vasoconstriction limits blood flow to the skin, reducing heat loss and lowering the thermal gradient between skin and ambient temperature; at the same time, blood is redistributed centrally,

increasing central blood volume and altering cardiovascular loading (Ikäheimo, 2018).

For endurance runners, these thermoregulatory demands are layered on top of the internal demands of training. The same running session can represent different total stress depending on temperature, wind, wetness, and recovery status. This is why a purely external training prescription (e.g., "70% VO_2max for 60 min") may underestimate internal load in winter, particularly when athletes finish sessions with rapid post-exercise cooling or remain in wet clothing. In practical terms, the combined stressor model predicts that immune perturbations in winter are most likely when training load is high and recovery is constrained, and when cold exposure is prolonged or repeated without adequate thermal protection and rewarming routines.

2) Neuroendocrine signaling: SNS/HPA activation as an immune "switchboard"

A central value of cellular signaling knowledge is that it clarifies how cold stress can reshape immune function. Cold exposure activates the SNS and HPA axis, shifting the endocrine environment through changes in catecholamines and cortisol (Radomski & Boutelier, 1982; Walsh, 2018).

These hormones are not merely markers of stress; they regulate immune cell behavior. Elevated catecholamines and glucocorticoids can produce leukocytosis while simultaneously altering immune cell distribution and function—partly through changes in adhesion and trafficking signals that govern whether cells remain in circulation or migrate into tissues. In the context of athletes, such endocrine-driven shifts are important because immune protection depends on where cells are and what they are primed to do, not simply on a total blood count. Reviews focusing on immune responses to cold and cold-exercise interactions emphasize that corticosteroids and catecholamines can

meaningfully shape leukocyte subsets and inflammatory signaling, which may help explain altered infection susceptibility under certain winter training conditions (LaVoy et al., 2011; Shephard, 1998).

3) Barrier defense and mucosal immunity: the "front line" most relevant to runners

Endurance runners place exceptional stress on the respiratory tract, especially in cold and dry air where ventilation rates are high. From a mechanistic standpoint, infection risk is not determined only by systemic immunity but also by mucosal defenses—the physical and immunological functions of the upper airway lining and secretions. Cold exposure can affect airway barrier defense through changes in mucus viscosity and reductions in mucociliary clearance efficiency, increasing the probability that pathogens persist on epithelial surfaces long enough to establish infection (LaVoy et al., 2011).

The most studied field marker of mucosal immune status in exercise immunology is salivary immunoglobulin A (sIgA). Seminal work and subsequent reviews show that heavy training and competition can suppress mucosal immune parameters and that lower sIgA is often associated with increased respiratory illness risk in athletes (Gleeson, 2000; Gleeson, 2007).

However, even here nuance matters. For example, a single moderate exercise bout in cold versus thermoneutral settings may not uniformly reduce sIgA secretion; in some settings, sIgA secretion rate may be maintained or differ by environment and timing (Mylona et al., 2002).

This is not a contradiction; it suggests that acute responses (one bout) and chronic patterns (weeks of winter training load and lifestyle stressors) are not the same phenomenon. What matters for winter runners is the cumulative balance of training stress, sleep, energy availability, psychological load, and pathogen exposure—factors

highlighted in practical athlete immune-health guidance (Walsh, 2018; Walsh et al., 2011).

4) Cytokine signaling and IL-6: interpreting "immune messenger" changes correctly

A cellular signaling approach also improves how we interpret cytokines such as interleukin-6 (IL-6). IL-6 is commonly elevated during prolonged or intense endurance exercise, but it is not only a "pro-inflammatory cytokine"; it also behaves as a myokine released from contracting muscle with metabolic roles and downstream anti-inflammatory signaling cascades (Pedersen & Febbraio, 2008; Pedersen & Febbraio, 2007).

For winter endurance training, the applied question is not "Does IL-6 increase?" but rather:

Does cold exposure amplify, blunt, or delay IL-6 and related signaling compared with thermoneutral conditions for the same relative intensity and duration?

Does this altered signaling correlate with training tolerance, recovery quality, and subsequent illness risk across winter mesocycles?

Evidence is mixed in ways that are informative. In controlled experiments, cycling exercise at ~70% VO_2max in cold (0°C) versus control (20°C) has been used to test IL-6 and soluble receptor responses, suggesting that temperature can modify IL-6-related signaling depending on protocol and timing (Patterson et al., 2008).

Other work shows that cold exposure and prior heating/exercise manipulations can shape immune outcomes during cold stress, reinforcing the idea that "cold + exercise" cannot be understood by temperature alone (Brenner et al., 1999).

The key practical implication is this: single-marker interpretations are risky. IL-6 may reflect normal adaptive signaling in one context, and strain-related dysregulation in another. That is precisely why a systems-level monitoring approach—combining external load metrics with symptom, recovery, and selected immune indicators—fits best with a signaling framework.

5) The illness–training performance link: why winter immune health is a performance variable

From a coach's perspective, the primary cost of winter respiratory illness is not only acute symptoms but loss of training continuity. Endurance performance is built through consistent exposure to appropriately dosed stimuli. Illness reduces exposure, interrupts progression, and can leave lingering fatigue that erodes quality in key sessions. The classic "J-curve" framework proposes that moderate exercise is associated with lower infection risk than sedentary behavior, while unusually heavy or prolonged exercise can increase URTI risk—especially after major endurance events (Nieman, 1994).

Contemporary athlete immune-health reviews emphasize that illness risk is shaped by multiple interacting factors: heavy training, travel, sleep disruption, low energy availability, psychological stress, and environmental extremes such as cold (Walsh, 2016; Walsh, 2018).

Therefore, the winter season should be viewed as a period in which immune health must be actively managed as a component of performance planning rather than treated as an unavoidable side effect.

6) Translating signaling knowledge into winter periodization

Below is a practical framework for applying immune-related cellular signaling knowledge to winter microcycle and mesocycle design. The

intent is not to medicalize training, but to build a decision structure that reduces vulnerability windows while preserving adaptation.

6.1 Microcycle principles (weekly planning)

A. Temperature-aware distribution of intensity

High-intensity or threshold sessions often require predictable mechanics, controlled breathing patterns, and high-quality neuromuscular output. Performing these sessions in extreme cold may increase airway irritation and magnify recovery cost without adding meaningful training benefit. A pragmatic approach is to move the highest quality intensity sessions indoors during the coldest weeks, while preserving outdoor exposure for lower-intensity aerobic runs where pacing can be adjusted and thermal strain managed. Such decisions align with athlete immune-health recommendations that prioritize load management and minimizing excessive stress during vulnerable periods (Walsh et al., 2011; Walsh, 2018).

B. Warm-up and post-exercise rewarming as immune-protective routines

Rapid cooling after endurance training is a common winter error. From a signaling view, post-exercise cooling may prolong stress responses and shift immune trafficking and mucosal defense at precisely the time when the athlete is physiologically taxed. A structured routine—dry clothing quickly, rewarm promptly, and avoid prolonged cold exposure in damp gear—should be treated as part of the "session dose," not an optional comfort step (LaVoy et al., 2011).

C. Recovery spacing and monotony control

Training monotony and inadequate recovery spacing increase cumulative strain. Because cold exposure itself is a stressor, the margin for error is smaller in winter. Microcycles should insert deliberate "low-stress days" that are not simply long easy runs performed in harsh conditions. Cross-training indoors, shorter easy runs, or additional

recovery modalities can reduce total stress while maintaining aerobic support (Walsh et al., 2011; Gleeson, 2007).

D. Symptom-gated intensity ("traffic light" rule)
A simple symptom framework can translate immune-awareness into safe choices:

Green: no symptoms → proceed as planned.

Amber: mild upper-airway symptoms, poor sleep, unusual fatigue → reduce intensity or replace with low-stress session; prioritize warmth and recovery.

Red: systemic symptoms, fever, chest symptoms → stop intense training and seek medical evaluation.

This is consistent with the view that URTI symptoms hinder training and competition and require practical countermeasures (Walsh, 2018).

6.2 Mesocycle principles (3–6 week blocks)

A. Align deloads with harsh environmental windows
Winter has predictable cold spikes and social periods that increase pathogen exposure (travel, indoor gatherings). Deload weeks planned during these windows can prevent the accumulation of neuroendocrine and immune strain. The objective is to protect continuity rather than chase short-term volume.

B. Build "immune reserve" before the hardest block
A signaling-informed mesocycle begins with a gradual load build, minimizing abrupt increases that activate prolonged stress responses. This is consistent with consensus guidance recommending progressive and periodized increases, variety to limit monotony, and adequate rest to reduce illness risk (Walsh et al., 2011).

C. Event-specific tailoring

5–10 km runners: often tolerate more frequent intensity but may face higher neuromuscular and airway strain when intensity is done in cold air.

Half/marathon runners: may tolerate higher volume but are vulnerable to cumulative fatigue and energy deficits that increase illness risk and slow recovery.

Therefore, immune-aware design is not identical across events; it is matched to the primary training stressor of the event group.

7) Monitoring: a feasible "immune-informed dashboard" for winter runners

A key strength of the cellular signaling approach is that it provides clarity about what to monitor and why. Not every team has access to cytokine assays, but most athletes can implement a tiered system:

Tier 1: universal, low-cost (daily/weekly)

Training load (duration × intensity; session RPE)

Sleep duration/quality

Resting heart rate trends

Mood/fatigue rating

Upper respiratory symptom log

Tier 2: targeted field immune indicators (weekly/biweekly where feasible)

Salivary sIgA (with standardized collection timing)

Simple blood counts (when medically appropriate)

Tier 3: research/elite setting (periodic)

Cytokine panels including IL-6 and related mediators

Neuroendocrine markers (cortisol patterns)

Integrated multi-omic approaches (where available)

Reviews on athlete immune health and mucosal immunity support the practicality of monitoring sIgA and aligning training adjustments to periods of low mucosal defense (Gleeson, 2016; Gleeson, 2000; Walsh, 2018).

8) Research agenda: making the letter's questions testable

Your original questions—whether cellular signaling-informed periodization prevents winter performance decline in different runner groups—are timely and can be sharpened into testable designs.

Research Question 1 (5–10 km runners)

Does immune-informed winter periodization reduce illness burden and preserve performance?
A cluster-randomized or team-randomized trial could compare:

Intervention: temperature-aware intensity placement + symptom-gated training + standardized rewarming routines + tiered monitoring (including sIgA where feasible).

Control: standard winter training without structured immune-informed rules.
Outcomes could include: illness episodes (URTI symptom-days), missed training days, time-trial performance (e.g., 3–5 km), and session quality indices.

Research Question 2 (half-marathon/marathon runners)

Does immune-informed mesocycle planning improve training availability and prevent performance decrement?
A pragmatic trial could compare:

Intervention: mesocycle deload alignment with cold spikes + energy-availability emphasis + monitoring dashboard (tier 1 + optional tier 2).

Control: standard progressive volume plans.
Outcomes could include: long-run completion rates, training monotony indices, illness symptom-days, and pre–post performance markers (threshold pace, race simulation).

These designs also allow mechanistic sub-studies: whether changes in mucosal immunity and cytokine responses (e.g., IL-6 dynamics) mediate the relationship between winter stress exposure and performance continuity (Pedersen & Febbraio, 2008; Gleeson, 2007; Walsh, 2016).

Conclusion

Winter endurance training is not simply a time of “more clothing and slower warm-ups”; it is a period in which cold exposure and training

load converge on shared neuroendocrine and immune signaling pathways. A cellular signaling framework—centered on SNS/HPA activation, immune cell trafficking, cytokine communication (including IL-6), and mucosal barrier defense—offers a coherent explanation for why illness risk and training disruption can rise during heavy winter training and why prevention must involve more than isolated supplements or ad hoc rest days. Implementing temperature-aware periodization, recovery-protective routines, and feasible monitoring can help preserve training availability—the true currency of endurance adaptation—across winter microcycles and mesocycles.

Sincerely,

Shahin Beyranvand

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ORCID

Shahin Beyranvand **https://orcid.org/**

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* Corresponding Author: shahinberan@gmail.com.
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