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The relationship between low carbohydrate availability, mucosal immunity, and respiratory illness in endurance athletes: mechanisms and practical implications: A Narrative Review

Jakub Czmok

ORCID: <https://orcid.org/0009-0006-0869-9305>

Kubel2003@gmail.com

Medical University of Lublin

Lublin, Poland

Katarzyna Półtorak

ORCID: <https://orcid.org/0009-0001-1440-6023>

kasiapoltorak013@gmail.com

Wrocław Medical University

Wrocław, Poland

Dominik Tarczyński

ORCID: <https://orcid.org/0009-0004-5307-0130>

dominik.tarczynski02@gmail.com

Medical University of Lublin

Lublin, Poland

Natalia Zięba

ORCID: <https://orcid.org/0009-0005-4554-9536>

natalia.zieba03@gmail.com

Medical University of Lublin

Lublin, Poland

Mateusz Czech

ORCID: <https://orcid.org/0009-0008-6980-6930>

mateuszczech102@gmail.com

Medical University of Lublin

Lublin, Poland

Miłosz Cislak

ORCID: <https://orcid.org/0009-0006-3999-6185>

miłosz.cislak@gmail.com

Medical University of Lublin

Lublin, Poland

Michał Chojeła

ORCID: <https://orcid.org/0009-0007-0911-971X>

michalchojeta9@gmail.com

Medical University of Lublin

Aleje Racławickie 1; 20-059

Lublin, Poland

Dominika Ciesielska

ORCID: <https://orcid.org/0009-0007-8548-1112>

dciesielska600@gmail.com

Medical University of Lublin

Lublin, Poland

Anna Mazur

ORCID: <https://orcid.org/0009-0001-2568-0781>

mazurania2801@gmail.com

Medical University of Lublin

Lublin, Poland

Mikołaj Brzusek

ORCID: <https://orcid.org/0009-0005-6266-6988>

mrib3002@gmail.com

Medical University of Lublin

Lublin, Poland

Katarzyna Kociuba

ORCID: <https://orcid.org/0009-0003-1265-2452>

katarzyna.kociuba1115@gmail.com

Medical University of Lublin

Lublin, Poland

Corresponding Author: Jakub Czmok, Kubel2003@gmail.com

Abstract

Background:

Low carbohydrate availability (LCA) is frequent in endurance sports because of high training load, train-low strategies, and body composition goals. Mucosal immunity, especially salivary secretory IgA, is a key barrier against upper respiratory illness.

Aim:

To summarize links between LCA, mucosal immunity, and respiratory illness in endurance athletes and outline practical implications.

Materials and methods:

Non-systematic search in PubMed and Google Scholar (2015-2026) using terms related to endurance athletes, LCA, LEA, mucosal immunity, sIgA, respiratory illness, cortisol, and glycogen depletion.

Results:

Evidence suggests that reduced carbohydrate availability, particularly when glycogen stores are low, may increase stress-hormone responses, limit fuel for immune cells, and impair salivary defense. Intervention and

observational studies indicate possible carbohydrate-specific effects on immune markers, but direct causal evidence for confirmed infection outcomes remains limited.

Conclusions:

Periodized carbohydrate intake, timely refueling around key sessions, and monitoring of sleep, stress, and symptoms may help preserve mucosal immunity and reduce respiratory illness risk.

Key words:

low carbohydrate availability; mucosal immunity; salivary IgA; endurance athletes; upper respiratory tract infection; exercise immunology; relative energy deficiency in sport; cortisol; glycogen depletion; periodized nutrition; train low

1. Introduction

Endurance athletes, including runners, cyclists, triathletes, rowers, and cross-country skiers, engage in prolonged, high-volume training that places substantial demands on energy systems, particularly carbohydrate stores in the form of muscle and liver glycogen [1, 2]. Low carbohydrate availability (LCA) occurs when dietary carbohydrate intake is insufficient to support training demands and replenish glycogen stores, either chronically or acutely during specific sessions ('train low' strategies). This situation is distinct from, but often overlaps with, low energy availability (LEA), defined as energy intake minus exercise energy expenditure relative to fat-free mass below ~30 kcal/kg/day [3, 4].

Mucosal immunity represents the first line of defense in the respiratory and gastrointestinal tracts. Secretory IgA (sIgA) in saliva and mucosal secretions neutralizes pathogens, prevents adhesion to epithelial cells, and modulates inflammation. Saliva flow rate and sIgA concentration/secretion rate are key markers; reductions are associated with increased risk of upper respiratory tract infections (URTI) or symptoms (URTS) [5, 6, 7, 8]. Endurance athletes are particularly susceptible to URTI/URTS due to high training loads, which can cause transient immunosuppression ('open window' period post-exercise), combined with factors like travel, stress, sleep disruption, and environmental exposures [9, 10].

The potential link between LCA and impaired mucosal immunity arises because immune cells (lymphocytes, neutrophils) rely on glucose as a primary fuel, and glycogen depletion can amplify the

hypothalamic-pituitary-adrenal (HPA) axis response, elevating cortisol levels that may suppress sIgA production and immune cell function [8, 11, 12, 13]. Recent studies suggest that some immunological effects attributed to LEA/REDs may be driven more by concurrent low carbohydrate availability than by energy deficit per se [11, 14, 15, 16].

This narrative review synthesizes current evidence on definitions and prevalence of LCA in endurance athletes, the role of mucosal immunity, epidemiology of respiratory illness, biological mechanisms linking LCA to immune suppression, direct and indirect evidence from studies, practical implications for periodized nutrition and monitoring, limitations, and future directions. The goal is to provide evidence-based guidance while highlighting gaps requiring further research.

2. Research materials and methods

This narrative review is based on a non-systematic literature search in PubMed and Google Scholar covering 2015–2026. The search terms included endurance athletes, low carbohydrate availability, low energy availability, mucosal immunity, salivary IgA, respiratory illness, cortisol, and glycogen depletion. Observational, interventional, mechanistic studies, and reviews were considered during manuscript preparation.

2.1 AI

AI was utilized for two specific purposes in this research. Text analysis of the manuscript and source-linked claims was used to identify linguistic patterns associated with overstatement or weakly supported statements. Assistance in refining the academic English language of the manuscript ensured clarity, consistency, and adherence to scientific writing standards. AI was used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of evidence strength, and conclusions were determined by human authors with expertise in sports medicine, nutrition, and scientific writing. The AI tools served primarily to enhance efficiency in text processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

3. Definition and measurement of low carbohydrate availability

Low carbohydrate availability refers to a state in which carbohydrate intake fails to meet the requirements for maintaining glycogen stores and supporting the fuel demands of training sessions. Practical thresholds include daily intakes below 5 g/kg body mass for moderate training or failure to provide 30–90 g/h during prolonged high-intensity exercise (>90 min) [1, 17]. 'Train low' protocols deliberately restrict carbohydrate before or during selected easy-to-moderate sessions to promote metabolic adaptations such as increased fat oxidation and mitochondrial biogenesis, but frequent or poorly managed application can lead to cumulative deficits [12, 15].

LCA differs from LEA in that the former specifically concerns carbohydrate substrate, while the latter encompasses total energy deficit. Athletes can experience LCA even in energy balance if the proportion of carbohydrate is too low, or LEA with adequate carbohydrate percentage but overall insufficient calories. Recent research indicates that immunological and hormonal disturbances observed in LEA contexts may be partly attributable to low carbohydrate availability rather than to energy restriction alone [11, 14, 15, 16].

LCA commonly appears in weight-sensitive endurance sports, during intensified training blocks, in athletes following popular low-carbohydrate or ketogenic diets for perceived performance or body composition benefits, or due to poor nutrition knowledge and under-fueling relative to expenditure. Its significance lies in potential compromises to high-intensity performance, recovery, training quality, hormonal health (particularly in females via REDs spectrum), and possibly immune function and illness susceptibility [3, 4, 18]. Measurement in research often involves dietary logs combined with training/energy expenditure estimates, or controlled interventions manipulating pre-exercise glycogen or daily carbohydrate intake. [1, 2, 12, 17]

4. Mucosal immunity in the context of exercise

Mucosal surfaces of the upper respiratory tract are protected by a multi-layered barrier: physical (epithelium and mucus), chemical (antimicrobial peptides, lactoferrin, lysozyme), and immunological (secretory IgA produced by plasma cells in the lamina propria and transported via the polymeric immunoglobulin receptor). sIgA is the predominant antibody at mucosal sites; it agglutinates pathogens, neutralizes viruses and toxins, and promotes their clearance without triggering strong inflammation [5, 6].

Saliva serves as an accessible window into mucosal immunity. Both sIgA concentration and secretion rate (flow rate $\times$ concentration) are important; low levels or flow are linked to higher URTI incidence in athletes and the general population. Exercise, particularly prolonged intense efforts, can acutely reduce saliva flow (sympathetic activation) and sIgA output or concentration, contributing to the 'open window' of increased infection susceptibility lasting hours to days post-exercise [7, 8, 9, 10]. Chronic heavy training may lead to more sustained suppression if recovery is inadequate. [19]

Carbohydrate availability influences these responses. Adequate carbohydrate intake during or after prolonged exercise can blunt the rise in cortisol and catecholamines, potentially preserving aspects of mucosal immunity. Conversely, training with low carbohydrate availability may exaggerate the stress response and impair immune markers [11, 12, 13].

5. Respiratory illnesses in endurance athletes

Upper respiratory tract infections (URTI) or upper respiratory symptoms (URTS) are common in endurance athletes during heavy training periods, and many episodes are viral, although non-infectious URTS related to cold/dry air, pollution, or allergy also occur [5, 7, 8, 10].

Risk factors include high training volume and intensity (especially monotony or spikes in load), intensified training blocks or taper/competition periods, inadequate sleep, psychological stress, travel (jet lag, new pathogens), environmental factors, and nutritional deficits including energy and specific macronutrient shortfalls. Low energy availability and REDs have been associated with increased self-reported illness symptoms and suppressed immune markers in observational studies of athletes [16, 18, 19].

The clinical and performance impact is significant: missed training days, reduced quality of sessions, potential for more severe or prolonged illness if training continues, and psychological burden. Distinguishing infectious from non-infectious URTS is important for management but often challenging in practice [8, 19].

6. Mechanisms linking low carbohydrate availability with weakened immunity

Several interconnected mechanisms explain how LCA may impair mucosal immunity and increase respiratory illness risk:

Low carbohydrate availability reduces glucose availability and may compromise immune-cell energy supply during and after exercise, when demand is elevated. A triathlete study examining sleeping with reduced glycogen stores reported only minimal effects on selected immune markers and sleep, suggesting that recovery-phase glycogen availability remains relevant to mucosal immunity and recovery [11, 13, 18, 20].

Training or exercising with low pre-exercise glycogen stores can exaggerate HPA axis activation, leading to higher cortisol and catecholamine responses to the same workload. Elevated cortisol may suppress sIgA and aspects of immune-cell function; recent controlled studies show that short-term low carbohydrate (but not necessarily low energy) diets alter cortisol responses and immune/iron markers in elite athletes [10, 11, 12, 13].

In contexts overlapping with LEA, LCA may contribute to suppressed reproductive hormones, low triiodothyronine (T3), and altered growth factors, which can indirectly affect immune regulation and mucosal barrier integrity [15, 16]. Chronic elevation of stress hormones may also increase oxidative stress in immune cells [16].

Lower sIgA output or concentration and altered saliva flow have been reported in some carbohydrate-manipulation interventions, although findings are not uniform [8, 12, 13, 19].

The combination of metabolic stress, hormonal suppression, and barrier weakening can facilitate pathogen entry, replication, or symptom manifestation. Observational and intervention data link LEA/REDs, often occurring alongside carbohydrate restriction, with higher rates of self-reported illness and reduced sIgA in endurance athletes [13, 14, 15, 16, 18, 19]. However, causality is not fully established, and individual variability (genetics, microbiome, training status, sex) is high [14, 15, 16, 18, 19].

7. Direct and indirect evidence from studies

Direct evidence specifically examining LCA and mucosal immunity or confirmed URTI in endurance athletes is limited but growing. McKay et al. (2022) demonstrated that six days of low carbohydrate availability (independent of energy availability) in elite athletes altered immune and iron responses to exercise, supporting carbohydrate-specific effects [11]. Terink et al. (2021) in a crossover study found that a low-carbohydrate, high-fat diet attenuated the exercise-induced cortisol response after adaptation and changed post-exercise sIgA and workload responses [12].

Studies assessing mucosal markers in elite endurance athletes show variable sIgA responses, and the effects appear to depend on training load and nutritional context [8, 13, 19]. LEA and REDs literature provides indirect evidence: observational studies report higher illness symptom scores, increased URTI incidence, and suppressed sIgA or other immune parameters in athletes classified as low energy available [14, 15, 16, 18, 19].

Carbohydrate availability around prolonged exercise is often recommended because intervention studies and reviews suggest that adequate intake can blunt stress-hormone responses and support recovery-related immune markers compared with low-carbohydrate conditions [1, 11, 12, 13, 17].

Overall, while large-scale RCTs with hard endpoints (lab-confirmed infections, longitudinal sIgA monitoring) are lacking, the biological plausibility is high, and convergent evidence from controlled interventions and observational athlete cohorts supports caution with chronic or poorly managed LCA [8, 11, 12, 13, 16, 19].

Table 1. Summary of selected studies included in the review.

Study	Evidence type	Design/Population	Main focus	Main contribution
McKay et al. [11]	Direct LCA/immune	Elite athletes, controlled low CHO vs adequate	Immune and iron response to exercise	Low CHO (not just energy) alters immune/iron markers; supports CHO-specific effects
Terink et al. [12]	Direct diet intervention	Recreational athletes, crossover LCHF vs HC	Cortisol, s-IgA, performance	LCHF attenuated cortisol after adaptation; complex effects on s-IgA
REDs/LEA reviews [14, 15, 16]	Indirect/observational	Elite endurance athletes, LEA classification	Illness symptoms, sIgA	LEA associated with higher illness reports and suppressed mucosal immunity
McKay et al. chronic manipulation [13]	Direct mucosal	Elite endurance athletes, varied CHO availability	sIgA and mucosal markers	Variable impacts; potential impairment under low availability conditions

Source: Own elaboration based on references [7, 8, 11, 12, 13, 14, 16, 18, 19].

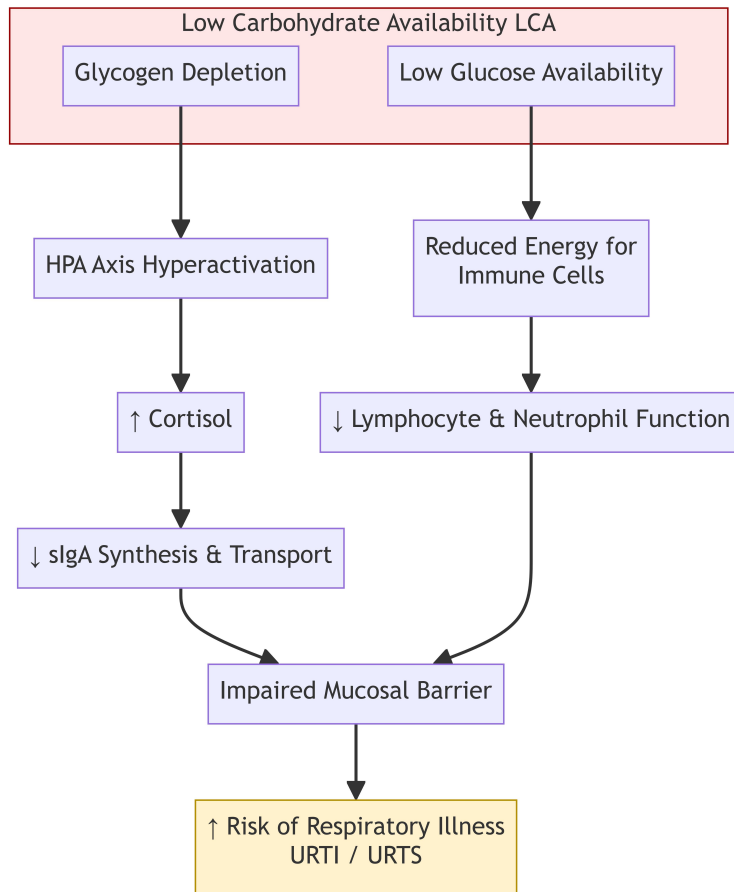


Figure 1. Conceptual model of possible pathways linking low carbohydrate availability with mucosal immune impairment and respiratory illness risk. Own elaboration based on references [1–4, 9, 10, 14–20].

8. Practical implementation and recommendations

Implementation of appropriate carbohydrate strategies requires individualized, periodized approaches rather than blanket high- or low-carbohydrate prescriptions. For high-intensity or key quality sessions (>60–90 min or high glycolytic demand), ensure high carbohydrate availability: pre-load glycogen, provide 30–90 g/h intra-exercise (depending on duration, intensity, and tolerance), and prioritize rapid refueling post-session (1.0–1.2 g/kg/h initially) [1, 12, 17, 21, 22]. For easy aerobic or recovery sessions, selective 'train low' can be used judiciously (e.g., 1–2 sessions per week) to stimulate adaptations without compromising overall recovery or health [12, 21, 22].

To prevent excessive energy or carbohydrate deficit, regularly estimate energy availability (dietary intake minus exercise energy expenditure relative to fat-free mass) using food/training logs or validated tools. Target >40–45 kcal/kg FFM for most endurance athletes during heavy training; monitor body composition trends, resting metabolic rate if available, and hormonal markers if REDs is suspected. Education on 'fueling for the work required' helps athletes avoid under-fueling driven by misconceptions about carbohydrate restriction [3, 4, 14, 15, 16, 18].

To support immunity holistically: prioritize consistent high-quality sleep (7–9 h/night), stress management, and environmental hygiene. Consider vitamin D status (common deficiency linked to immune function) and correct if low. During periods of high illness risk, maintain adequate overall energy and carbohydrate around training. Probiotics (specific strains) have some evidence for reducing URTI duration/severity in athletes but are not a substitute for foundational nutrition and recovery [10].

Monitoring should include daily wellness questionnaires (sleep quality, fatigue, stress, motivation, presence/severity of URTS), training metrics (session RPE, performance consistency), and periodic medical checks. Salivary sIgA can be useful in research or high-performance settings but has high day-to-day variability and is not routine for most athletes. If URTS increase or wellness declines coinciding with low-carbohydrate periods, temporarily increase carbohydrate availability, reduce load, and investigate other contributors (sleep, stress, allergens) [9, 10]. Collaboration among coach, nutritionist, and sports medicine physician is essential. [9, 10]

9. Limitations and future directions

This is a narrative review, not systematic; selection bias is possible, and no formal risk-of-bias or meta-analysis was performed. The literature search prioritized 2021–2026 publications, but direct RCTs on chronic LCA with hard clinical endpoints (confirmed infections, longitudinal mucosal markers) remain scarce. Many studies are acute or observational, with confounding by training load, sleep, stress, and overlapping LEA difficult to isolate. Sex differences (females more studied in REDs/LEA contexts) and individual variability require more attention. Dose-response relationships for LCA (how low, how frequent, which session types) on immunity outcomes are not well defined.

Future research should include well-controlled interventions comparing isocaloric high- vs. low-carbohydrate availability over training blocks with primary outcomes of lab-confirmed URTI incidence and sIgA kinetics. Integration of multi-omics, microbiome analysis, and wearable biomarkers could elucidate mechanisms. Personalized approaches accounting for genetics, sex, training phase, and prior illness history are needed. Development of practical digital tools for real-time carbohydrate guidance and symptom tracking would enhance translation. Safety of 'train low' protocols in junior, masters, and clinical populations deserves specific study.

10. Discussion

The accumulated evidence supports a plausible and biologically coherent link between low carbohydrate availability and impaired mucosal immunity in endurance athletes, mediated primarily by amplified stress hormonal responses (cortisol) and reduced glucose substrate for immune cells and glycoprotein synthesis (sIgA). While direct causal proof from large RCTs is still emerging, convergent data from controlled dietary interventions and LEA/REDs observational studies provide a strong foundation for practical recommendations [11, 12, 13, 14, 15, 16, 18, 19].

Importantly, not all low-carbohydrate strategies are detrimental. Strategic, periodized use ('train low' for selected sessions) can promote beneficial metabolic adaptations when overall energy and recovery nutrition are adequate and high-carbohydrate availability is prioritized for key workouts. The key is avoiding chronic or

excessive restriction that leads to cumulative deficits, hormonal disruption, and immune compromise [1, 2, 12, 17].

From a public health and athlete welfare perspective, emphasizing 'fuel for the work required' and holistic monitoring (nutrition, sleep, stress, symptoms) offers a low-risk, high-reward approach that supports both performance and health [1, 2, 10, 12, 17].

11. Conclusions

Low carbohydrate availability in endurance athletes, especially when chronic or mismanaged during heavy training, is associated with mechanisms that can weaken mucosal immunity—primarily through heightened cortisol-mediated suppression of sIgA and metabolic constraints on immune cell function—thereby potentially elevating risk of respiratory illnesses. Key mechanisms center on glycogen depletion amplifying stress responses and limiting substrate availability for optimal immune and barrier function.

In practice, athletes and support teams should adopt periodized carbohydrate strategies that ensure high availability around demanding sessions while permitting judicious low availability for adaptations, alongside vigilant monitoring of wellness, symptoms, sleep, and recovery indicators. This balanced approach supports sustainable training, performance goals, and athlete health while minimizing illness-related disruptions. More robust causal evidence from targeted longitudinal studies is required to refine guidelines and optimize implementation across diverse athlete populations.

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Author's Contribution Statement:

Conceptualization: Jakub Czmok, Mateusz Czech

Methodology: Katarzyna Kociuba, Dominik Tarczyński

Investigation: Anna Mazur, Dominika Ciesielska

Sources: Jakub Czmok

Formal analysis: Mikołaj Brzusek, Michał Chojęta, Miłosz Ciślak

Supervision: Natalia Zięba

Writing—rough preparation: Jakub Czmok, Katarzyna Półtorak

Writing – review and editing: Michał Chojęta, Katarzyna Póltorak

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