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PHYSICAL ACTIVITY AND IMMUNE FUNCTION: IMPLICATIONS FOR RESPIRATORY INFECTIONS AND VACCINE IMMUNOGENICITY – A NARRATIVE REVIEW

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ABSTRACT

Background: Physical activity is widely recognized as an important modifier of immune function. Numerous studies have examined exercise-induced changes in cellular and mucosal immunity and their implications for susceptibility to acute respiratory infections and vaccination outcomes. However, the clinical relevance of post-exercise immune alterations remains debated.
Aim: The review aimed to synthesize current evidence regarding the influence of acute and chronic physical activity on immune regulation, respiratory infection risk and vaccine immunogenicity in adults.
Material and Methods: A narrative literature review was conducted using PubMed, PubMed Central and Google Scholar. English-language studies involving adults were included, with priority given to articles published from 2010 onward. Particular attention was paid to relationships between physical activity, immune function, upper respiratory tract infections and vaccine responses.
Results: Regular moderate physical activity is associated with improved immune surveillance and higher resting mucosal immunity markers. Although strenuous exercise induces transient immune alterations, evidence for a clinically meaningful increase in infection risk remains inconsistent. Epidemiological data suggest that physically active individuals generally experience fewer symptomatic days and reduced illness severity. Long-term exercise participation is linked to modestly improved post-vaccination antibody responses, especially in older adults. Single exercise sessions performed around immunization show often minimal effects.
Conclusions: Habitual moderate physical activity supports more efficient immune regulation and may reduce respiratory disease burden. Short-term post-exercise immune fluctuations appear largely adaptive rather than clinically immunosupressive. Physical activity may serve as an accessible adjunct strategy to enhance vaccination outcomes, however, further high-quality trials using objective clinical endpoints are required.

KEYWORDS

Physical Activity, Exercise Immunology, Mucosal Immunity, Vaccine Immunogenicity, Open Window Theory, Upper Respiratory Tract Infection, Immune Cell Trafficking

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1. Introduction:

Seasonal respiratory tract infections represent a substantial and recurring public health challenge, particularly in temperate climates. Adults commonly experience several episodes each year, with repeated illnesses occurring within a single season [1]. Respiratory infections are among the most common causes of medical consultations and sickness-related work absence, generating a considerable cumulative social and economic burden [2].

Given the scale and impact, growing attention has been directed toward identifying accessible, low-cost and scalable strategies capable of strengthening host defense and reducing susceptibility to infection. Among potential non-pharmacological interventions, regular physical activity has emerged as a particularly promising candidate due to its broad systemic influence on immune regulation [3].

The scientific discipline addressing these interactions is exercise immunology. Over the past decades, research has evolved from descriptive reports of leukocyte fluctuations after exertion toward integrated models emphasizing immune cell redistribution, enhanced immunosurveillance and functional consequences for disease risk [3, 4, 5]. A key question remains whether these exercise-induced immunological alterations translate into clinically meaningful outcomes. Contemporary research focuses on endpoints such as incidence and severity of respiratory infections, duration of illness and responsiveness to vaccination.

Despite the rapidly expanding literature, uncertainty persists regarding the magnitude and durability of exercise effects, the influence of intensity and training status, and the interpretation of transient post-exercise immune changes. A synthesis of available evidence spanning cellular, mucosal, epidemiological and vaccination perspectives may help clarify their clinical significance and guide future research.

2. The impact of physical activity on cellular and mucosal immune function

2.1. Mucosal immunity

Salivary and upper airway immune markers have been extensively investigated as non-invasive indicators of host defense against respiratory pathogens. Much of this work has focused on secretory immunoglobulin A (sIgA), the principal antibody at mucosal surfaces [3,6].

Early descriptions of exercise–immune interactions frequently emphasized reductions in mucosal antibodies after strenuous exertion. Summaries of the literature noted that prolonged or intensive activity was commonly accompanied by decreases in salivary and nasal IgA concentrations, forming part of a broader constellation of transient immune perturbations observed in athletes [4]. Reductions on the order of 20–30% following endurance competition or heavy training periods were historically interpreted as evidence of diminished mucosal defense [3].

Subsequent evaluations have revealed marked variability in these responses. In particular, it has been highlighted that changes in IgA concentration may be influenced by alterations in saliva flow rate, hydration status or sampling methodology, complicating interpretation of functional immune protection [3]. Moreover, several datasets summarized in contemporary reviews did not consistently demonstrate that short-term peri-exercise reductions in sIgA preceded the development of upper respiratory symptoms [3].

Intervention studies provide a somewhat different perspective. A meta-analysis of seven trials involving 435 participants demonstrated that regular moderate-to-vigorous physical activity (typically involving 30 min of exercise performed 3 times per week) was associated with significantly higher resting sIgA concentrations compared with control conditions (standardized mean difference 0.756, 95% CI 0.146–1.365) [7].

Acute responses may further depend on prior training status. When data from trained individuals were pooled, exercise elicited a significant increase in IgA levels (effect size 0.68, 95% CI 0.34–1.02), corresponding to a moderate effect. In contrast, no significant change was detected among untrained participants undergoing comparable exercise bouts (effect size 0.16, 95% CI −0.32 to 0.64) [8].

In comparison to the predominant focus on oral mucosal immunity, an experimental study conducted by Elkhatib et al. examined immune defense directly within the nasal mucosa using nasal lavage fluid (NALF) obtained at rest, after 45 minutes and after 180 minutes of moderate-intensity exercise [9]. Prolonged exercise significantly increased total cell yield recovered from the nasal cavity, with elevations observed in epithelial cells, neutrophils, monocytes and lymphocytes [9]. In contrast, the shorter 45-minute bout did not meaningfully modify cellular composition [9]. Despite these cellular changes, functional analyses demonstrated that the acellular component of NALF possessed marked antiviral activity against influenza A virus at baseline. Importantly, this antiviral capacity remained preserved following both 45 and 180 minutes of exercise [9].

2.2. Cellular immune responses to physical activity

Physical exercise induces rapid and well-documented alterations in circulating immune cell populations. The magnitude of these responses is closely related to exercise intensity and duration [3,5,10].

One of the most characteristic immediate responses to acute exercise is leukocytosis. Increased leukocyte counts are mainly driven by elevations in neutrophils, lymphocytes, and natural killer (NK) cells [3,4]. Importantly, this mobilization is selective. Cells characterized by strong cytotoxic potential are preferentially released into the circulation, particularly NK cells and CD8+ T lymphocytes [5]. Exercise bouts lasting less than one hour consistently produce enhanced recirculation of neutrophils and cytotoxic subsets essential for rapid antimicrobial defense [5].

Current evidence suggests that the post-exercise decline in circulating lymphocytes reflects their migration to peripheral tissues. Cells are believed to relocate to sites with higher likelihood of pathogen exposure, particularly mucosal surfaces and the lungs [3]. This redistribution may transiently strengthen immune surveillance. Neutrophil dynamics follow similar principles. Post-exercise neutrophilia has been attributed to demargination from endothelial surfaces together with recruitment toward tissues exposed to mechanical and inflammatory stimuli [4].

Acute physical activity appears to promote a continuous exchange of leukocytes between compartments [5]. This mobilization is proposed to enhance host defense capability, potentially facilitating more rapid recognition and elimination of infected targets, which contributes to reduced infection risk among regularly active individuals [3,4,5].

Exercise also influences immune regulation through modifications in T helper cell balance. In older adults moderate workloads are commonly associated with patterns that support Th1-mediated mechanisms responsible for intracellular pathogen control [11].

From an epidemiological perspective, these cellular responses correspond with observations that individuals engaging in regular moderate physical activity tend to report fewer upper respiratory tract infections [4]. Furthermore, ultramarathon runners have been shown to experience approximately 1.5–2.8 sick days annually compared with around 4.4 days in the general population [3].

Quantitative modelling approaches further support the importance of intensity and duration of exercise. Predictive analyses demonstrate that these two parameters are strong determinants of post-exercise leukocyte behavior [10]. Prolonged or exhaustive physical activity is associated with a reduction in circulating lymphocyte numbers together with a temporary functional alteration [4]. The physiological interpretation and clinical relevance of this phenomenon will be discussed in the following subsection dedicated to the open-window theory.

2.3. The Open window theory

The “open window” theory describes a transient period of altered immune function after prolonged or high-intensity endurance exercise [14] during which susceptibility to opportunistic infections, particularly upper respiratory tract infections (URTI), is considered to be increased [3,12]. Early studies supporting this model reported higher frequencies of post-competition illness symptoms in athletes together with reductions in salivary immunoglobulin A, decreased lymphocyte proliferation, diminished natural killer cell activity, and transient changes in leukocyte distribution [5]. These findings contributed to the J-shaped relationship between exercise load and infection risk, where moderate activity was associated with lower illness incidence than either sedentary behavior or excessive training [4]. Mechanistic explanations have linked these responses to elevations in stress hormones and shifts in regulatory cytokines. In addition natural killer cell (NK) function has been reported to decline following exhaustive exercise [13].

Observational syntheses in athlete populations continue to describe increased respiratory illness during periods of intensified training [12] and pooled marathon data indicate a modest but significant rise in URTI incidence after competition (OR 1.18, 95% CI 1.05–1.33) [14]. However, studies incorporating laboratory diagnostics have shown that many symptom-defined URTI episodes lack microbiological confirmation [3]. In addition, athletes engaged in endurance events are frequently exposed to travel, crowd contact, psychological strain, nutritional challenges, and sleep disruption. All these factors may independently influence immune regulation [3]. Furthermore, post-exercise lymphocytopenia observed 1–2 h after vigorous exercise has been described as transient and interpreted as redistribution of effector cells to peripheral tissues rather than an absolute loss of immune competence, with cell counts returning toward baseline within 24 h [3]. Investigations of respiratory mucosal compartments further demonstrate that antiviral activity may remain detectable despite exercise-induced alterations in cellularity [9].

Across the literature, strenuous exercise is typically followed by short-term perturbations in cellular and mucosal immune markers, whereas evidence linking these changes to clinically verified infection remains inconsistent across study designs [3].

3. The impact of physical activity on the incidence, severity and duration of acute respiratory infections

Across epidemiological studies and randomized trials, regular participation in physical activity is generally associated with a reduced risk of experiencing upper respiratory tract infections. Large population-based datasets and epidemiological investigations consistently indicate that individuals adhering to current moderate-to-vigorous physical activity recommendations tend to report fewer infection episodes than sedentary counterparts [15,16]. However, evidence from meta-analyses of randomized controlled trials remains more conservative. A Cochrane synthesis found little or no reduction in the number of acute respiratory infection episodes per person-year, despite trends favouring exercise groups [17].

Although the impact on incidence is sometimes modest, findings tend to be more consistent for indicators of illness burden. Randomized and observational research indicates that physically active individuals accumulate fewer symptomatic days and may experience milder clinical manifestations [15,16,18]. In older adults reductions in total days of illness (241 vs 453 in control groups) and fewer missed workdays (32 vs 67) have been demonstrated even when differences in episode duration were small [18].

Many studies describe the presence of a dose–response relationship. Greater exercise frequency and longer habitual duration are typically linked to lower infection rates [16,19]. In contrast, exercise intensity often follows a non-linear pattern. Both narrative and population-based analyses describe U- or J-shaped curves, where moderate workloads confer the greatest protection, while very high training loads may attenuate or even

reverse protective benefits of physical activity [19,20]. Importantly, individuals exposed to combinations of high intensity and frequency exercises appear to demonstrate the highest infection probability among active groups [19].

Beyond self-limited illness, physical activity has also been associated with reduced risk of severe respiratory outcomes. In a cohort of over half a million adults, meeting aerobic and muscle-strengthening activity guidelines was linked to substantially lower influenza and pneumonia mortality. The greatest benefit was seen at 301–600 minutes physical activity/week, achieving ≈50% lower mortality risk [21]. Even volumes below current recommendations were associated with a 21% reduction in mortality risk [21].

However, the majority of available literature remains observational and inconsistency in case definitions, exposure measurement, and control of confounding factors continues to limit causal inference [20].

4. The impact of physical activity on the efficacy of influenza and COVID-19 vaccinations:

4.1. The effect of regular physical activity on vaccination efficacy

In a large meta-analysis conducted by Sebastien F. M. Chastin et al., participation in regular physical activity was associated with significantly higher post-vaccination antibody titers (SMD = 0.142; 95% CI 0.021–0.262) including among older adults. The data suggest that engaging in at least 150 minutes per week of moderate to vigorous physical activity, incorporating both aerobic and strengthening exercises, 20 weeks prior to vaccination could have a positive impact on antibody levels [7].

Evidence summarized in a review focused specifically on COVID-19 vaccination in adults and older individuals indicates that structured physical activity is associated with enhanced humoral responses, greater T-cell activation and improved immune cell mobilization. In addition, a reduction in key inflammatory markers, including IL-6 and CRP, was reported. The analyzed exercise protocols typically include aerobic or resistance training performed several times per week for months before or during the vaccination period [22].

Longer participation in exercise programs appeared particularly relevant for older adults. Engagement in regular structured physical activity was linked to both attenuation of chronic low-grade inflammation and partial counteraction of age-related immune decline [22].

Complementary findings were presented in the review by Luca Barni et al., who reported that individuals engaging in structured or habitual exercise tended to demonstrate higher post-vaccination antibody titers compared with inactive controls. The authors highlighted that this effect was not uniform across populations. Greater responses were typically observed in younger participants relative to older adults and were often more pronounced in women than in men [23].

In addition to humoral responses, several reports describe more favorable cellular and inflammatory profiles among physically active individuals. Higher counts of CD4+ lymphocytes, elevated leukocyte responses and increased interleukin-6 concentrations were noted among exercise groups, suggesting a broader stimulation of immune mechanisms [23].

4.2. The effect of a single session of physical exercise performed before or after vaccination

Available evidence assessing exercise performed in close temporal proximity to immunization demonstrates heterogeneous effects on vaccine-induced immunogenicity.

A Cochrane review by Antonio Jose Grande et al., found no consistent differences between individuals performing pre-vaccination exercise and resting controls in antibody titers, influenza incidence, or adverse events. Most protocols involved a single session of aerobic or resistance exercise lasting approximately 25–50 minutes on the day of immunization. The certainty of evidence was judged to be low to very low due to small sample sizes and methodological limitations [24].

However, a 45-minute session of moderate-intensity resistance exercise performed immediately before influenza vaccination reduced local and systemic vaccine reactions among older adult participants but did not influence antibody titers, seroprotection rates, or the occurrence of influenza-like symptoms during six months of follow-up [25].

In an individual participant data meta-analysis, acute exercise did not significantly enhance antibody responses in the overall sample. However, benefits were observed for selected strains, particularly H1, and were influenced by baseline physical activity status, age, and whether the exercised limb corresponded to the vaccinated arm [26].

In contrast, certain studies reported that prolonged aerobic activity undertaken shortly after vaccine administration enhanced antibody responses without increasing reactogenicity [27]. Participants who completed approximately 90 minutes of light- to moderate-intensity exercise exhibited higher follow-up antibody concentrations following both influenza and COVID-19 vaccination [27].

5. Discussion

The present review integrates evidence linking physical activity with immune regulation, susceptibility to respiratory infections and vaccine responsiveness. Across mechanistic and clinical perspectives, the most consistent message is that habitual moderate exercise supports effective immune surveillance, whereas the clinical importance of transient post-exercise immune perturbations appears smaller than traditionally proposed.

Acute bouts of exercise reliably induce leukocyte mobilization, particularly of cytotoxic populations such as natural killer cells and CD8+ T lymphocytes. Although these elevations are followed by temporary declines in circulating counts, the phenomenon is increasingly interpreted as redistribution toward peripheral tissues rather than systemic immunosuppression. Such trafficking may in fact enhance the probability of early pathogen recognition at mucosal entry sites.

A comparable evolution in interpretation concerns secretory immunoglobulin A. While earlier frameworks treated peri-exercise reductions as evidence of impaired protection, contemporary analyses emphasize methodological variability and the weak association between short-term IgA changes and subsequent illness. More relevant clinically may be the observation that individuals engaging in regular training often demonstrate higher resting mucosal antibody levels. Experimental data from nasal compartments further indicate that antiviral activity can remain preserved despite shifts in cellular composition.

Within this context, the traditional open-window hypothesis warrants cautious reinterpretation. Increased reporting of symptoms after endurance competitions has been described, yet many episodes are not laboratory confirmed. Athletes are simultaneously exposed to numerous non-exercise stressors, including travel, crowds, sleep restriction and nutritional challenges, all of which may contribute to infection risk. Moreover, immune cell numbers generally normalize within hours, making prolonged impairment biologically uncertain. Consequently, accumulating literature favours an adaptive model in which exercise promotes dynamic immune surveillance rather than a period of dangerous vulnerability.

Epidemiological observations reinforce this interpretation. Even when reductions in infection incidence are modest, physically active individuals tend to experience fewer symptomatic days and milder disease courses. From a population perspective, decreases in severity and functional limitation may translate into meaningful public health benefits.

At the same time, several analyses describe non-linear dose–response patterns, suggesting that extremely high training volumes could attenuate protective effects. However, inconsistency in exposure measurement and reliance on self-reported outcomes continue to restrict causal inference.

The role of physical activity in shaping vaccine responses appears more coherent when exercise is maintained chronically. Regular participation in moderate-to-vigorous programs is associated with slightly higher antibody titers and more favorable inflammatory environments, particularly among older adults. In contrast, interventions based on a single session performed immediately before or after immunization produce heterogeneous and often minimal effects. These findings support the notion that long-term behavioral adaptation, rather than an acute stimulus, is required to meaningfully influence immunogenicity.

Several methodological constraints should be acknowledged. Many studies depend on symptom definitions without pathogen verification, training exposure is frequently self-reported and vaccination trials often include small samples with diverse protocols. Standardization of clinical endpoints and incorporation of objective diagnostics would substantially strengthen future research.

Overall, current knowledge supports promotion of habitual moderate physical activity as a pragmatic strategy to enhance immune regulation and reduce respiratory disease burden. Short-lived fluctuations observed after intense exercise are better understood as components of physiological adaptation than as evidence of clinically significant immunodeficiency. Further well-designed trials are needed to determine the magnitude of benefit in specific populations and to refine exercise prescriptions that maximize immune resilience.

6. Conclusions

Regular moderate physical activity is associated with favorable immune regulation, including enhanced mucosal protection, efficient leukocyte redistribution and a reduced overall burden of respiratory illness. Acute post-exercise fluctuations in immune markers are typically transient and are increasingly interpreted as adaptive responses rather than manifestations of clinically meaningful immunosuppression. Although periods of intensified or prolonged exertion may coincide with short-term immune perturbations, consistent evidence linking these changes with laboratory-confirmed infection remains limited. Consequently, the traditional

concept of a post-exercise “open window” is being progressively reconsidered. Long-term engagement in physical activity appears to modestly enhance vaccine-induced immune responses, particularly in older adults, whereas single exercise sessions performed around vaccination demonstrate variable effects.

Promotion of habitual physical activity should be regarded as a supportive strategy for strengthening population resilience against acute respiratory infections and for optimizing vaccination outcomes. Further high-quality research incorporating rigorous clinical endpoints is required to refine exercise prescriptions and to identify individuals most likely to benefit.

Disclosure

Supplementary materials

Not applicable

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