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The Role of Circulating Therapeutic MicroRNAs in Pulmonary and Muscular Function in Post-COVID-19 Athletes

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ABSTRACT

SARS-CoV-2 infection causes significant acute and long-term morbidity, including persistent pulmonary and muscular dysfunction in athletes. Physical exercise alters circulating microRNA (miR) profiles, and specific microRNAs have documented roles in inflammation, immune regulation, muscle metabolism, and regeneration. This study characterizes circulating microRNAs relevant to COVID-19 pathogenesis and post-viral recovery, including miR-155, miR-146a, the let-7 family, miR-21, miR-424, miR-1, miR-133, miR-499, miR-208, miR-486, and miR-22, and examines how different exercise modalities may modulate these microRNAs to support pulmonary and muscular function in post-COVID-19 athletes. miR-155 and miR-146a are highlighted as modulators of innate and adaptive inflammatory signaling and as mediators of cytokine responses implicated in severe COVID-19. Moreover, several microRNAs, such as miR-21, miR-155, miR-146a, and the let-7 family, converge on NF-κB and related pathways, linking altered miR expression to immune dysregulation and cytokine-driven tissue injury. Additionally, muscle-enriched and metabolism-associated microRNAs regulate myogenesis, mitochondrial biogenesis, and key metabolic pathways (PGC-1α, AMPK, mTOR)-processes essential for muscle repair, endurance recovery, and respiratory muscle support after SARS-CoV-2 infection. Different types of exercise produce distinct miR signatures; notably, moderate-intensity exercise consistently promotes anti-inflammatory and pro-repair miR patterns. We emphasize the therapeutic potential of moderate-intensity exercise as a non-pharmacological strategy to regulate miR expression, reduce cytokine-mediated damage, and support functional recovery in post-COVID-19 athletes. To our knowledge, this is the first study to link exercise-driven miR changes with functional pulmonary and muscular recovery in athletic populations recovering from COVID-19, supporting moderate-intensity exercise as a promising strategy for rehabilitation and performance restoration.

Keywords: COVID-19; Exercise; MicroRNA; Immune system; Inflammation

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INTRODUCTION

The COVID-19 pandemic resulting from the SARS-

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CoV-2 virus led to considerable investigation into the mechanisms of its pathophysiology.[1] Patients experienced acute hyperinflammation leading to acute respiratory distress syndrome (ARDS) and subsequent musculoskeletal problems.[2] Post-acute sequelae of SARS-CoV-2 infection (PASC), also referred to as long COVID, may have various signs and symptoms that include decreased pulmonary function, fatigue, and intolerance to exercise.[3] Several microRNAs (miR) that are thought to modulate physiological responses associated with exercise, recovery, and lung health,[4] have been suggested to be important players in the rehabilitation of post-COVID-19 and therefore hold promise for further investigation. Recent literature has suggested that exercise positively affects the immune system and patterns of inflammation during viral infections.[5] Several microRNAs are now thought to be important regulators of gene expression influenced by physical activity and involved in all biological processes mediated by viral diseases.[6] It is thus important to establish the relationship between microRNAs, exercise capacity, and lung function for the purposes of developing the best possible recovery strategies for athletes suffering the effects of COVID-19. It is indeed possible that novel rehabilitation strategies directed towards selective modulation of specific microRNAs by personalized exercise programs may offer novel avenues for recovery. This present review will discuss the interplay between microRNAs, exercise, and COVID-19 and highlight the possibility for the development of directed therapeutic modalities using exercise to alleviate disease severity and optimize recovery endpoints.

miRNAs: Key Regulators in COVID-19

Circulating miRs are among the smallest non-coding RNA molecules and can be detected in body fluids such as blood. They serve as potential biomarkers for various physiological and pathological conditions.[7] These miRs are released from different tissues and participate in key cellular processes-including inflammation, apoptosis, and tissue repair-all of which are directly linked to the host response in COVID-19.[8] Given the respiratory complications associated with COVID-19, alterations in miRs related to pulmonary health are particularly important. Certain miRs can target genes involved in lung repair and inflammatory regulation.

MicroRNAs Involved in Inflammatory Responses and Pulmonary Function

miR-155

miR-155 is typically known as a pro-inflammatory miR. Increased expression occurs upon stimulation with various inflammatory factors, including those of a microbial origin.[9] miR-155 works on the NF-κB signaling pathway by inhibiting various negative controls of this pathway and thus enhances the expression of pro-inflammatory cytokines.[10] It works by inhibiting *SOCS1* (suppressor of cytokine signaling 1), and enhancing signaling through cytokines such as IL-6 and TNF-α.[11] It exerts its action also on the target gene *PTEN* (phosphatase and tensin homolog), as inhibitory control of *PTEN* exerts pro-survival and proliferative actions in inflammatory scenarios.[12] Furthermore, the expression levels of miR-155 may be influenced by exercise, which may ultimately play a role in influencing respiratory function.[13] In post-COVID-19 athletes, an assessment of the expression levels of miR-155 may be used to assess pulmonary recovery.

miR-21

miR-21 also plays the role of an oncomiR in some cancers and modulates the immune response.[14] There are also cardioprotective properties associated with the expression of miR-21, and it also regulates pulmonary function through inflammation-mediated pathways.[15] Higher levels of miR-21 have been associated with improved pulmonary function and decreased fibrosis during various pulmonary disease states via modulation of the *TGF-β* (transforming growth factor β) signaling pathway.[16] Because of this, a review study reported that miR-21 may prove to be beneficial in helping to decrease the inflammatory signaling in the post-COVID-19 recovery phase while also helping the damaged lung tissue heal, which may help improve exercise tolerance.[17] The down-regulation of *PDCD4* (programmed cell death 4) by miR-21 promotes tumorigenesis and inflammation in an animal study.[18] Also, mirroring miR-155, miR-21 down-regulates *PTEN,* which promotes the proliferative signaling of the cells associated with the immune and inflammation responses.[19]

miR-146a

Moreover, miR-146a is a negative regulator of inflammatory responses and is commonly upregulated in cytokine and microbe-stimulation, diminishing

excessive inflammation.[20] In athletes recovering from COVID-19, miR-146a may assist in restoring pulmonary homeostasis by attenuating hyperinflammatory responses, thus supporting improved lung function and exercise capacity.[21] It primarily modulates the TLR (toll-like receptor) and NF-κB pathways, responsible for providing the feedback in order to diminish the inflammatory response.[22] The suppression of *TRAF6* (tumor necrosis factor receptor-associated factor 6) by miR-146a would thereby reduce the activation of NF-κB and consequently the production of pro-inflammatory cytokines in human umbilical vein endothelial cells.[23] The second common target is *IRAK1*; its inhibition by miR-146a also would attenuate proinflammatory signaling.[24]

miR-let-7

miR-let-7 is associated with various cellular processes, including the regulation of epithelial integrity and inflammatory responses.[25] A systematic review has indicated that miR-let-7 can modulate the expression of cytokines and enzymes that contribute to lung inflammation and injury.[26] Enhanced levels of miR-let-7 during recovery can positively influence exercise tolerance by promoting optimal pulmonary function.[27]

miR-424

Conversely, miR-424 has also been implicated in the regulation of immune responses and inflammation.[28] This miR has been shown to have effects in TNF-α signaling and to affect downstream inflammatory responses. Through targeting cyclin D1, miR-424 is capable of modulating immune cell proliferation.[29]

MicroRNAs and Muscular Function

Recent research determined that microRNAs, such as miR-1, miR-133,[30] and miR-499[31] are engaged in the processes of muscle hypertrophy and regeneration. Their expression appears to differ in infection states, possibly leading to impaired recovery and physical deconditioning in COVID-19 patients.[32] These microRNAs are primarily produced in muscle tissue, and miR-1, for example, promotes muscle differentiation and hypertrophy via downregulation of *HDAC4*. The other target of miR-1 is *MEF2C*, an important factor in muscle cell developmental fate.[33] Also, at this phase of the insulin signaling pathway, the mTOR signaling activities may come into play and possibly modulate the mTOR signaling activities, which are critical to muscle hypertrophy.[34]

Additionally, miR-133 interacts with the mTOR signaling pathway, stimulating muscle cell proliferation and protein synthesis.[35] The inhibition of *HDAC5* by miR-133 contributes to muscle hypertrophy and inhibits muscle cell apoptosis.[36] miR-499 inhibits *atrogin-1*-the major gene contributing to muscle atrophy-inhibiting loss.[37]

The results of cellular and animal study miR-208 is involved in the downstream signaling of skeletal muscles, regulating muscle fiber type determination and causing hypertrophy.[38] It regulates signaling pathways in which cardiac and skeletal muscles grow through adaptation to the stress of exertion.[39] A review study showed that the expression of miR-208 protects the muscles by the action of a signaling cascade involving the downregulation of *MuRF1* (muscle RING finger 1).[40] *GATA4*, which affects cardiac muscle hypertrophy, is another target of miR-208.[39] Also, miR-486 regulates glucose metabolism and facilitates the sensitivity to insulin in muscle cells, an important regulation in the growth and maintenance of muscle. Because it prevents *PTEN* expression, miR-486 promotes insulin signaling, facilitating the transport of glucose into the muscles.[41] Also, recent animal research showed that miR-486 facilitates the growth and hypertrophy effects of muscle cells via the regulation of *Akt* (protein kinase B) signaling.[42]

MyomicroRNAs and Exercise Response

Exercise is known to induce the release of muscle-specific microRNAs, often referred to as myomiRs.[43] After exercise, circulating levels of these myomiRs can change significantly. In athletes, post-COVID-19, increased levels of myomiRs may indicate muscle adaptation, repair, and the restoration of physical function.[44] Improve circulation and oxygenation, enhancing the delivery of signaling molecules that regulate miR expression and contribute to tissue recovery.[45]

Aerobic Exercise

Aerobic exercise reduced oxidative stress and enhanced metabolic demands on muscle tissues, providing a stimulus for adaptive response.[46] The PGC-1α pathway is of importance and is known to regulate mitochondrial biogenesis and energy metabolism.[47]

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miR-1 is activated during aerobic exercise and promotes muscle adaptation by down-regulating *HDAC4* and promoting muscle differentiation in athletes.[48] Exercise-induced miR-133 upregulation assists in repression of *HDAC5* as a key molecule in hypertrophy of muscle cells.[49] Furthermore, PGC-1α is a positive influence on mitochondrial functionality and energy metabolism affected by miR-133.[50]

Resistance Training

On the other hand, based on the results of the review study, resistance training (RT) induces muscle tension and mechanical overload, which gives rise to an adaptive hypertrophic response.[51] mTOR is the regulatory signaling pathway that is activated during RT and is involved in protein synthesis and muscle growth.[34] RT can increase the levels of miR-486 in healthy male[52] and enhance the process of insulin sensitivity-mediated glucose uptake and muscle metabolism in older hypertensive participants.[53] This is thought to promote hypertrophy and cell survival.[54] Promotion of increased *Akt* signaling in miR-486-inhibited *PTEN* is likely to facilitate muscle growth and repair functions.[42]

High-intensity Interval Training (HIIT)

HIIT integrates both aerobic and anaerobic forms of exercise, resulting in immediate metabolic and physiological responses. This involves the activation of various pathways, including those regulated by AMPK (AMP-activated protein kinase) and PGC-1α, which are associated with increased oxidative function.[55] HIIT may elicit a regulation of miR-155 expression, favoring metabolic adaptation in overweight or obese middle-aged women, although inflammation is most closely associated.[56] In addition, *Cox-2* is repressed when targeted by miR-155, which is associated with the regulation of inflammation in connection with HIIT. The biological effect of miR-155 is thought to be associated with increased mitochondrial biogenesis and conditioning adaptations.

Exercise and Immune Function

Review and systematic review studies showing differences in the levels of various microRNAs resulting from moderate-intensity exercise, which provided beneficial changes in the expression of microRNAs and suppression of inflammatory cytokines and stimulation of anti-inflammatory mediators.[57,58] Because COVID-19 patients are often hyper-inflammatory,[59] this is relevant. miR-146a and miR-21 are always upregulated after moderate-intensity exercise and may lessen inflammation by targeting certain pro-inflammatory signaling pathways,[57] while the microRNAs upregulate the anti-inflammatory mediators, adjusting the immune response.[60] Patients with COVID-19, more than most others, have an elevated concentration of inflammatory cytokines, which include IL-6, IL-1β, or TNF-α. The configuration of hyperinflammation subsequently leads to increased lethality, causing damage to tissues and imminent dangers.[61] Probably to obtain recovery or rehabilitation benefits, moderate-intensity exercise might modulate microRNA expression profiles, downregulating inflammatory cytokines and upregulating anti-inflammatory mediators while averting the hyperinflammatory responses pronounced among COVID-19 patients.[62] Increased control of immune processes by exercise may bolster overall recuperation from COVID-19 and mitigate the long-term complications associated with post-viral syndrome.[63]

Exercise Prescription and Rehabilitation Strategies

Incorporating exercise into the pandemic rehabilitation strategy can have a favorable effect on miR expression in patients recovering from COVID-19.[64] Regularly implemented exercise protocols not only improve physical performance but also improve the miR environment necessary for recovery and immune function.[65]

CONCLUSION

Regular aerobic exercise has evolved into a promising adjuvant strategy for the prevention of thrombotic and inflammatory complications in patients with COVID-19, partly by induction of the release of beneficial microRNAs. The association between exercise capacity, microRNAs, and pulmonary function in post-COVID-19 athletes represents an exciting area of inquiry for future investigations. MicroRNAs such as miR-21, miR-146a, and miR-let-7 play key roles in the regulation of inflammatory processes and lung health, which are critical for optimal recovery and performance. Future studies should be aimed at longitudinal assessments to better elucidate the dynamics of miR expression and their effects on exercise and pulmonary health in this unique patient.

STATEMENT OF ETHICS

Not applicable.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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DATA AVAILABILITY

Data are available from the corresponding author upon reasonable request.

AI ASSISTANCE DISCLOSURE

No AI tools were used in this study

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