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Exosome and miRNAs of skeletal muscle: the role of strength training

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Highlights:

- Extracellular vesicles that carry muscle miRNAs, called myomiRs, are suitable as biomarkers.
- The myomiRs are dynamically influenced during strength training.
- There is a cross-talk among myomiRs and other organs.

Abstract: Extracellular vesicles of which exosome belong are packet of microRNAs, particular interest the muscle-derived, which are critical regulators of intercellular communication in response to exercise. Strength training modifies muscle physiology and influences the expression and release of a class of microRNA called myomiRs that mediate systemic adaptations. Aim: This review aims to explore the relationship between strength training and skeletal muscle-derived extracellular vesicles–miRNAs. Methods: A comprehensive literature review was conducted, focusing on studies examining the expression, packaging, and function of myomiRs in response to strength training in both human and animal models. Key Findings: Strength training influences the expression and extracellular vesicle-mediated release of myomiRs. These myomiRs may regulate muscle hypertrophy, regeneration, and extracellular signaling. However, most available data remain correlational and not causal. Significance: Understanding the regulation and the systemic role of EVs-microRNA in response to strength training uncovers two aspects: first, the identification of novel biomarkers for the validity of the strength training, and second, the identification of a therapeutic strategy for musculoskeletal health.

Keywords: myomiRs; microRNAs; muscle mass; extracellular vesicles; strength training

1. Introduction

Skeletal muscle represents a fundamental part of our organism, as it is the most extensive tissue in terms of percentage and functions, locomotion, stability, and the regulation of energy metabolism,

glycemia, and lipidemia [1,2]. Skeletal muscle exhibits remarkable plasticity, adapting to various stimuli such as endurance, resistance, or combined training through distinct physiological responses [3]. When properly managed, physical exercise induces beneficial systemic adaptations [4], provided there is adequate recovery, nutrient availability, and support for tissue repair and growth. Even the central nervous system is affected by exercise-induced changes, all of which align with the concept of allostatic load [5]. Among various exercise modalities, strength training has distinct effects on muscle structure and function, promoting hypertrophy, fiber-type transformation, and enhanced force production [6–8].

Therefore, it exhibits remarkable plasticity, and among various exercise modalities, strength training has distinct effects on muscle structure and function, promoting hypertrophy, fiber-type transformation, and enhanced force production [7–9]. Consequently, training and nutrition are fundamental binomials for skeletal muscle condition and for an adequate and productive response to it at exercise [10–12]. A muscle in good condition, *i.e.*, well-trained, hydrated, and supplied with the correct nutrients [5], expresses an optimal epigenetic framework, influencing the whole body [12]. In this frame, recent studies have highlighted the role of Extracellular vesicles (EVs), particularly exosomes, as important mediators of such systemic communication [13–17]. EVs carry biologically active molecules, including microRNAs (miRNAs), that can modulate gene expression in target cells. A subset of miRNAs, termed myomiRs, are enriched in skeletal muscle and play essential roles in muscle development, regeneration, and adaptation [18]. Notably also lipoproteins are carriers of miRNAs [19]. Despite growing interest, the mechanistic understanding of how exercise, specifically strength training, alters myomiR profiles—and the physiological consequences of these changes—remains incomplete. Understanding the regulation and the systemic role of EVs-memoir's in response to strength training uncovers two aspects: first, the identification of novel biomarkers for the validity of the strength training, and second, the identification of a therapeutic strategy for musculoskeletal health. Of note is that maintaining active living counteracts cognitive decline behind to physical shape [13,16,20,21].

The objective of this review is to examine the current literature on strength training-induced modulation of skeletal muscle-derived EVs-miRNAs, evaluating their potential functional roles in both local muscle adaptation and systemic communication with other tissues.

2. Overview on microRNAs biogenesis and muscle physiology in exercise adaptation

MiRNAs are non-coding small sequences of RNA, counting for 20–25 nucleotides. Some miRNAs are derived from exonic or intronic regions of non-coding RNAs, while specific genes encode others. They are initially transcribed as ~70–80 nucleotide pri-miRNAs with a hairpin structure and later processed into mature miRNAs in the cytoplasm (Figure 1). They were thought to be by-products without function, but are now known to act as messengers, carried in protective vesicles to target cells where they regulate immunity and protein synthesis [22–24].

The main biochemical mechanism of miRNAs is the downregulation of protein synthesis; more than 30% of messenger-RNAs are expected to be regulated by miRNA, thereby influencing numerous biological pathways [20]. The miRNAs are linked to physiological, and overall health, like antioxidant status [22–26]. They are also dynamically regulated by diet and lifestyle [27–29]. A key feature of miRNAs is that they are found not only in organs and tissues but also in all body fluids [30] giving them the connotation of biomarkers in several pathological conditions, in the case of skeleton muscle, the sarcopenia [31].

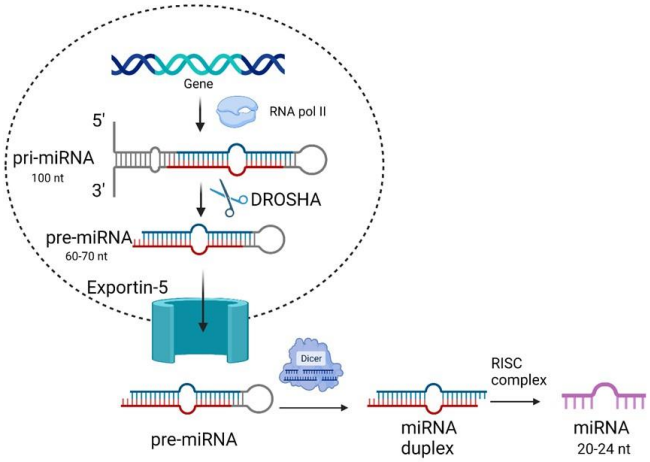

Figure 1. Biogenesis of microRNA. The miRNAs are transcribed from DNA by RNA polymerase II as primary transcripts (pri-miRNA), which are then processed in the nucleus by the enzyme DROSHA into precursor miRNAs (pre-miRNA). These hairpin-shaped structures (~60–70 nucleotides) are exported to the cytoplasm via Exportin-5. In the cytoplasm, Dicer further processes them into ~22-nucleotide miRNA duplexes. One strand is incorporated into the RNA-induced silencing complex (RISC), guiding gene silencing by targeting complementary mRNAs.

Recent evidence suggests that miRNAs involved in muscle adaptation are not only active within muscle cells but can also be secreted into circulation via EVs, both exosomes or microvesicles [32]. These extracellular vesicles protect miRNAs from degradation and enable their transport to distant target tissues [33]. In this context, miRNAs carried by exosomes may act as intercellular messengers, complementing the effects of key stimuli such as insulin-like growth factor 1 (IGF1), insulin, leucine, and physical exercise by influencing pathways like protein kinase B (PKB also known as AKT)–mammalian Target of Rapamycin (mTOR), AMP-activated protein kinase (AMPK)–PPARG coactivator 1 alpha (PGC1α), and transforming growth factor-beta (TGFβ) signaling. From an evolutionary perspective, humans have evolved to be physically active, with motor skills influencing the development of the musculoskeletal system and the brain, facilitating complex coordination [34,35]. Regular physical activity, including both structured exercise and daily movement, should be considered essential for maintaining good health. In sports, understanding how the body adapts to physical activity is crucial. The concept of super-compensation explains how gradually increased training, paired with proper rest and nutrition, improves performance [5,36]. The key mechanism in response to resistance training involves the mTOR pathway and the autocrine isoform of the mechano growth factor (MGF) [37,38]. The pathway also plays a role in endurance training, where PGC1α is more prominent, promoting mitochondrial biogenesis and fusion as it was also evidence by our recent meta-analysis [39]. Myostatin, also known as growth differentiation factor 8 (GDF8) and TGFβ pathways are also essential for muscle homeostasis and adaptation [39–43]. Additionally, exerkins refer to all molecules released by skeletal muscle in response to exercise, including other interleukins, cytokines, lactate, and nitric oxide [44–48]. Among these, also miRNAs, encapsulated in EVs, which act as key mediators of intercellular communication [44,49]; they are called myomiRs. The sorting of miRNAs into exosomes involves RNA-binding proteins (RBPs), which recognizes specific sequence motifs like GGAG in miRNAs [50]. Its function is regulated by post-translational modifications such as sumoylation and O-GlcNAcylation, and it interacts with membrane proteins like caveolin-1 [51,52]. Once released, the miRNA-loaded exosomes are taken

up by recipient cells through diverse mechanisms. Interactions that mediate this uptake between exosomal surface proteins—including tetraspanins cluster differentiation (CD9, CD63, CD81), integrins, lectins, and heparan sulfate proteoglycans (HSPGs) and their corresponding receptors on target cells [53]. Internalization then proceeds via pathways such as clathrin-mediated endocytosis, caveolin-dependent uptake, macropinocytosis, or phagocytosis [54]. Additionally, lipid interactions, particularly involving phosphatidylserine facilitate this process. The specific route of uptake often depends on the recipient cell type and environmental factors, such as pH and temperature [55]. Skeletal muscle-derived EVs-exosomes play an important role in immune modulation post-exercise. Specifically, the study highlights that exosomes containing miRNAs can influence immune cell signaling pathways, including those related to inflammation, such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), janus kinase/signal transducer and activator of transcription (JAK/STAT), and toll-like receptors (TLRs) pathways. Exercise-induced EVs-exosomal miRNAs have been shown to interact with immune cells such as neutrophils, monocytes, and lymphocytes, altering gene expression and contributing to the anti-inflammatory effects of physical activity [56]. It recently showed that some myomiRs, are encapsulated within muscle-derived exosomes and participate in local skeletal muscle communication [57].

3. MyomiRs and strength training: cross-talk to skeletal-muscle system and other organs

The myomiRs circulate and influence bone, tendons, and ligaments [44] which supports the skeletal-muscle system. Other tissue targets include adipose, as an energy source, heart, which demands oxygen and nutrients during exercise, and the central nervous system, responsible for coordination and control of the movements [45,46]. Together, these interactions suggest that myomiRs serve as systemic mediators of muscle-organ crosstalk, especially in response to physical activity. In strength training, myomiRNAs are important, as modulator of key pathways involved in muscle hypertrophy, protein synthesis, and satellite cell activation [58]. The first miRNA considered specific for skeletal muscle was miR-206 which is the most abundant miRNAs in muscle-derived EVs [59]. It is fundamental for the myogenesis process in the embryonic stage and the regeneration of adult muscle [60]. It has also been observed that circulating miR-206 is linked to osteoporosis, potentially regulating osteoblast proliferation and apoptosis by targeting histone deacetylase 4 (HDAC4) [61]. Additionally, in the context of intervertebral disc degeneration, miR-206 appears to suppress gap junction alpha-1 (GJA1), a gene associated with pro-inflammatory signaling and extracellular matrix degradation [62], suggesting a role in maintaining tissue integrity. Annibalini *et al.*, studied a particular strength training protocol (isoinertial training) and found that miR-206 levels increased within 2 hours post-exercise, while muscle IGF-1Ea propeptide expression decreased. This suggests that miR-206 may initiate early muscle remodeling or inflammation, potentially preceding IGF-1-driven anabolic responses during recovery since plasma IGF-1 levels increased after 24 hours [63]. The miR-206 is expressed in an exclusive manner [64]; it has been seen that it is not essential for myogenesis, however, it is fundamental for the differentiation of satellite cells and for the possible switching of muscle fibers, thus crucial for intense physical exercise and recovery [65].

The miR-1 has similar biochemical action of miR-206, and is most likely circulating in exosomes and with possible action on muscle tissue [66]. Recently, Burke *et al.,* reports the potential EVs-mediated mechanism by which skeletal muscle communicates with adipose tissue and modulates lipolysis via miR-1 following resistance training [67]. During mechanical overload (mimic strength training), in mouse models muscle releases miR-1-enriched EVs that are preferentially taken up by *epididymal white*

adipose tissue (eWAT). The muscle EVs boost eWAT miR-1, revealing a novel mechanism of muscle-driven adrenergic amplification. A similar miR-1 EVs release was observed in humans, indicating a conserved muscle-adipose signaling axis following resistance exercise [68].

The miR-16 in response to strength training acts on the mechanisms that regulate protein synthesis, in particular mTOR and AKT [69], but also on the biofactors that regulate myogenic differentiation [70]; it also has an anti-inflammatory action, which is carried out directly on the inflammasome complex and on the NF-κB [71], very useful in recovery from injuries and in general from the inflammatory state induced by intense training [72].

The miR-21 is probably one of the most studied miRNA, as it has important actions on the regulation of autophagy [73]; it is also expressed by skeletal muscle and is circulating in EVs from plasma [70,74]; it has been reported that in the -5p form in response to physical exercise downregulates the phosphatase ten-sin homolog deleted on chromosome 10 (PTEN) and the mothers against decapentaplegic homolog 7 (SMAD7) pathway with a positive action on exogenesis. Additionally, the reduction in PTEN levels contributes to the indirect regulation of the phosphatidylinositol 3-kinases (PI3K)/AKT/mTOR signaling pathway [75] and the promotion of muscle hypertrophy.

The miR-29, particularly miR-29c promotes muscle growth and protein synthesis during resistance training, enhancing muscle size and strength despite AKT/mTOR inhibition [76]. Wang *et al.*, reports that miR-29 was transported from muscle to kidney by exosomes in murine model reducing muscle atrophy and kidney fibrosis [77]. In addition, skeletal muscle can release miR-29a-3p via EVs in response to resistance training, suggesting a role for the miR-29 family in systemic metabolic communication between muscle and other organs including liver [78]. However, the miR-29 family exhibits isoform-specific functions, highlighting the need to distinguish among family members when evaluating their biological roles [79].

The miR-93 has an action on TGFβ and on the regulation of inflammation, in particular by acting on TLR4 and autophagy related 7 (Atg7) [80]; therefore, the response to physical exercise could translate into a modulation of post-workout inflammation with consequent ease in recovery and super-compensation; it is interesting to note that in the work of Agostini *et al.*, miR-93-5p is downregulated, as a consequence of strength training, performed on sarcopenic elderly [81–83].

The miR-133a/b have regulatory action on various metabolic pathways, in particular regarding lipids [84]; they circulate in exosomes and are characteristic of skeletal muscle; they are secreted in response to both endurance physical exercise promoting cardiovascular function [85,86], and in response to strength exercise, even in post-pathological conditions [87]; they act in particular on PGC1α, promoting the shift of adipose cells towards brown fat [88–90].

The miR-208 a/b have an action in particular on cardiac muscle; the action at the molecular level is mainly on HDAC, on heavy myosin chains and on peroxisome proliferator-activated receptor gamma (PPARγ) [91,92]; it is interesting to note that cardiac miR-208 following an infarction, on the bone marrow, in order to try to cope with the traumatic event [93]; it is regulated by exercise resulting in benign hypertrophy of left ventricle [94].

The miR-222, circulating in exosomes, acts mainly on the PTEN/mTOR pathway [95] but also has an anti-inflammatory action by acting on NF-kB and on the synthesis of Tumor Necrosis Factor alpha (TNF-α) [96]; the combined action with miR-21 in response to physical exercise on elderly

subjects after 8 weeks of moderate training is interesting, and is maintained even after 16 weeks of follow-up [97].

The miR-378 has a limiting action on the differentiation of muscle satellite cells, therefore it is inversely related to muscle well-being ; there is a direct correlation between this miR and myoblast determination protein (MyoD) [98]; it seems to have a trophic effect on vascularization, favoring the response to physical exercise; it has also been shown to have an action on neuropathic pain from inflammation of the sciatic nerve, through the regulation of the enhancer of zeste homolog 2 (EZH2), it also has an action on PGC1α [99,100].

The miR-486 following resistance exercise was found in circulating exosomes [101], confirming the regulatory action of exercise on circulating miR; like miR-21 it acts on the PTEN pathway but also by regulating forkhead box O1 (FOXO1) important for insulin regulation, which then translates into an indirect anabolic signal [101,102]. Of note, the myomiR-486 was propose as potential biomarkers of sarcopenia in the older adults [103].

The miR-499 a/b are expressed by skeletal muscle and circulating in exosomes [104]; the predominantly detected action is on TGFβ receptor 1, therefore they share the pathway of other myomiR; the result is generally in myogenesis, satellite cell differentiation, muscle fiber shift and hypertrophy: it is interesting to note that from the work of Zhang *et al.*, the response to strength training by miR-499 is maintained even in elderly subjects [105].

Table 1 summarizes the miRs analyzed, linked to the most involved pathways and to the possible expected effect.

Table 1. MyomiR, pathway and outcomes.

miRNA	Major pathway involved	Expected outcome	Model	Reference
-1	IGF1, MyoD, Myostatin	Muscle mass, muscle regeneration, improvement of bone/adipose tissue/heart	Human	[66–68]
-16	AKT3, mTOR, IGF1 receptor, NF-kB	Tissue remodeling, cell cycle regulation, inflammation modulation	Mice/*in vitro**	[69–72]
-21	SMAD7, PTEN, IL1β, HIF1α	Tissue remodeling, cell cycle regulation, antioxidation status	Mice	[72–75]
-29	AKT/mTOR inhibition	Tissue remodeling, muscle size	Mice	[76–79]
-93	TLR4, Atg7, TGFβ	Cell cycle regulation, glucose transport, inflammation	*In vitro**	[80–90]
-133a/b	IGFR, PGC1α, SRF	Myoblast differentiation, fiber shift, protein synthesis	Human/Mice	[84–90]
-206	HDAC4, MyoD	Muscle regeneration, oxidative stress managing	Human/*in vitro**	[60–65]
-208a/b	HDAC, PPARγ	Fiber shift, muscle hypertrophy, lipid metabolism	Human	[91–94]
-222	PTEN/mTOR, NF-kB, TNF-α	Tissue remodeling, cell cycle regulation, inflammation	Mice/*in vitro**	[95–97]
-378	MyoD, PGC1α	Muscle quality, myogenic differentiation	*in vitro**	[98–100]
-486	FOXO1, PTEN	Myoblast differentiation and fusion	Human	[101–103]
-499a/b	TGFβ receptor 1	Fiber shift, muscle hypertrophy	Human	[104–105]

* cell lines

4. Synergistic roles of miR-1 and miR-133a/b in muscle regulation

The miR-1 and miR-133a are often described as a coordinated pair due to their co-expression from the same bicistronic miR-1/133a cluster gene, which exhibits muscle-specific expression patterns [106,107]. Despite being transcribed together; they play distinct and complementary roles in muscle physiology. The miR-1 primarily promotes myoblast differentiation, targeting genes such as HDAC4, heart- and neural crest derivatives-expressed protein 2 (HAND2), and IGF-1 receptor, which are involved in muscle development and hypertrophy [108]. In contrast, miR-133a enhances myoblast proliferation and regeneration by repressing serum response factor (SRF), a key transcription factor in muscle growth [109]. This functional duality ensures proper progression of myogenesis, where miR-1 encourages differentiation the miR-133a supports proliferation, maintaining a balance crucial for muscle adaptation to exercise [106,109]. Their expression is regulated by myogenic transcription factors such as MyoD, myogenin, and myocyte enhancer factor 2 (MEF2), which bind to promoter regions and activate transcription [110]. Interestingly, HDAC4, a target of miR-1, can repress its expression through chromatin condensation, establishing a negative feedback loop [110,111]. Extracellular signals also modulate miR-1/miR-133 levels. IGF-1 activates the PI3K/Akt pathway, upregulating miR-1 and miR-133a via enhanced activity of myogenic transcription factors [112]. Additionally, non-coding RNAs influence miR-1/miR-133 levels activity. Other non-coding-RNAs can sequester miR-1 and miR-133a [113,114] to have a fine-tune regulatory effects on muscle growth and repair [115].

5. Temporal dynamics of miRNAs during strength training, disease and supplementation

Cui *et al.*, report that miR-133a levels significantly decreased immediately after exercise, indicating an acute response to muscular stress, returning to baseline within one hour. It suggests a rapid recovery phase. In contrast, miR-133b levels remained stable post-exercise but increased significantly after one hour, indicating its involvement in early muscle remodelling or repair [116]. No significant time-dependent changes were observed in miR-1, miR-206, miR-208b, among others. Additionally, Horak's study examined two distinct strength training protocols. In the Explosive Strength Training (EXPL) group, circulating miR-222 decreased by week 5 and remained low, while miR-16 showed a steady decline throughout the intervention, indicating sustained downregulation. In contrast, the Hypertrophic Strength Training (HYP) group exhibited early increases in miR-93, miR-16, and miR-222 at week 5, followed by a decline below baseline by week 8 suggesting a transient upregulation followed by later suppression as training adaptations progressed [117]. By the way, circulating EVs-miRNAs associated with skeletal muscle are lower in older individuals and increase following resistance exercise training. In this context, several promising research areas are emerging, though many remain in their early stages showing considerable potential as biomarkers for the early detection of muscle-related pathologies, such as sarcopenia and lipedema. MiR-486 stands out as especially promising for therapeutic targeting in sarcopenia and neuromuscular disorders due to their central role in muscle maintenance and regeneration. Also, the integration of nutritional strategies and dietary supplementation tailored to miR profiles may further enhance athletic performance and aid in the prevention of musculoskeletal injuries; for example, omega-3 fatty acids could be very useful to modulate inflammation triggered by training [117], monitoring myomiRs related to inflammation could assess their efficacy. In this context, innovative strategies

such as exosome-based delivery systems, miRNA mimics, and antagomiRs merit [118] exploration in preclinical models of metabolic syndrome and muscle wasting.

6. Strengths and limitations

This review highlights the regulatory roles of myomiRs in strength training, supported by mice model and human studies. A key strength is the comprehensive integration of molecular pathways with physiological adaptations, emphasizing the role of EVs-associated miRNAs released by muscle. However, several limitations must be acknowledged, the limited number of human studies in which confounding factors such as age, sex, training status, and genetic background are rarely controlled. These variables can significantly influence miRNA expression patterns. For instance, miRNA regulation may differ between younger and older individuals or between males and females due to hormonal and metabolic differences. Genetic variation in miRNA binding sites or biogenesis machinery may also modulate individual responses.

7. Conclusion

The myomiRs signature here reported (miR-1, -16, -21, -23a/b, -93, -133a/b, -206, -208a/b, -222, -378, -486, and -499a/b) was found circulating and packed in EVs, having an impact on other tissues. The action on adipose tissue, could be explained for the energy supply for physical activity; on the bone because a stronger skeleton can better support muscle function. On the cardiac muscle as in both endurance and resistance training a greater cardiac output is needed; lastly the influence on central nervous system, since strength training needs a strong neuro-muscular coordination. The signature of myomiRs impact also on biochemical mechanisms linked to pathways such as IGF1 and its receptor, mTOR, MyoD, PGC1α and TGFβ as well as on proteins deputy to the regulation of inflammation and management of free radicals such as NF-kB, FOXO1 and PTEN.

Authors' contribution

Conceptualization, RC and EC; methodology, DMAG; data curation, DMAG and FL; writing—original draft preparation, RC; writing—review and editing, RC, DMAG, FL and EC. All authors have read and agreed to the published version of the manuscript.

Conflicts of interests

The authors declare no conflict of interest.

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