Extracellular Matrix Remodeling in Response to Resistance and Endurance Training: Implications for Athletic Performance

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ABSTRACT

Skeletal muscle adaptation to exercise extends beyond contractile proteins to include extensive remodeling of the extracellular matrix (ECM). This review synthesizes findings from studies published between 2015 and 2025 that investigated how resistance and endurance training influence ECM composition and how these changes relate to performance outcomes. Evidence indicates that acute exercise bouts transiently increase matrix metalloproteinases such as MMP-9, proteoglycans including serglycin, and glycoproteins such as tenascin-C, while suppressing tissue inhibitors like TIMP-1 [1,2,7]. Chronic training interventions of 10–14 weeks consistently report upregulation of collagens (types I, III, IV), proteoglycans, and glycoproteins, alongside reductions in MMP-14 [2,3,7]. These structural adaptations are associated with improvements in muscle cross-sectional area, strength, tendon stiffness, and aerobic capacity [3–6]. Importantly, individual variability in baseline MMP-14 levels appears to predict hypertrophic responsiveness [3]. Collectively, the evidence supports a mechanistic link between ECM remodeling and enhanced functional performance in athletes, underscoring ECM as a critical mediator of exercise adaptation.

Keywords: **extracellular matrix, resistance training, muscle adaptation, athletic performance**

1. INTRODUCTION

The extracellular matrix (ECM) of skeletal muscle is a highly dynamic network of collagens, glycoproteins, and proteoglycans that provides not only structural support but also regulates force transmission, cell signaling, and repair processes. In recent years, the role of ECM remodeling in mediating adaptations to physical training has received growing attention. Resistance and endurance exercise both impose mechanical and metabolic stresses on muscle fibers, and the ECM responds through remodeling processes involving matrix metalloproteinases (MMPs), tissue inhibitors of metalloproteinases (TIMPs), and cross-linking enzymes such as lysyl oxidase-like 1 (LOXL1) [1–7].

Why does this matter for athletic performance? Traditional perspectives on training adaptations have emphasized hypertrophy of muscle fibers and neural factors. However, improvements in muscle strength, power, and endurance also depend on the quality and functionality of the surrounding ECM. For example, increased tendon stiffness contributes to more efficient stretch–shortening cycle performance [5], while enhanced collagen turnover supports greater hypertrophic potential [4].

Despite these insights, the literature remains fragmented, with variability across study designs, training protocols, and methods of ECM assessment. To address this gap, the present review synthesizes evidence from interventional studies and systematic reviews conducted between 2015 and 2025. We focus on (1) acute and chronic ECM responses to resistance and endurance training, (2) inter-individual variability in ECM remodeling, and (3) the functional significance of ECM adaptations for athletic performance.

2.Characteristics of Included Studies

Table 1 summarizes the key features of the studies included in this review, highlighting study design, training protocols, ECM measures, and performance outcomes.

Table 1. *Characteristics of included studies (2015–2025)*

Study	Design	Training Intervention	ECM Measures	Performance Outcomes
Hjorth et al., 2015 [1]	Longitudinal intervention	12 wks, 2×/wk, strength + endurance	Biopsy, mRNA seq; serglycin, collagens, elastin, laminins	CSA, strength
Scarpelli et al., 2024 [2]	Longitudinal intervention	10 wks resistance training	Biopsy; MMP-9, MMP-14, TIMP-1, TIMP-2	CSA, type II fiber CSA
Godwin et al., 2023 [3]	Longitudinal intervention	10 wks, whole-body RT, 2×/wk	Biopsy; Collagen I, IV, MMP-14, MMP-2, MMP-9, TIMP-1, TIMP-2	CSA (pQCT, US), fiber CSA
Kritikaki et al., 2021 [4]	Systematic review	RT, endurance, combined, HIIT, 5–13 wks	Biopsy, protein/mRNA; Collagen I/IV, laminin, proteoglycans	CSA, strength, lean mass
Douglas et al., 2017 [5]	Systematic review	Eccentric RT, 5–20 wks	Tendon structure/function	CSA, tendon stiffness, power
Pattamaprapanont et al., 2024 [6]	Controlled experimental (mice + human data)	Aerobic training	Proteomics; LOXL1, ECM proteins	Capillary density (mice), aerobic capacity (humans)
Hyldahl et al., 2015 [7]	Longitudinal intervention	Repeated-bout eccentric	Biopsy; Collagen I/III/IV, tenascin-C, TGF-β	Strength loss

3.Acute Responses of ECM to Exercise

Acute exercise triggers transient but reproducible changes in ECM composition (Table 2). Hjorth et al. [1] showed serglycin upregulation, Scarpelli et al. [2] demonstrated elevated MMP-9 and reduced TIMP-1, while Hyldahl et al. [7] observed dramatic increases in tenascin-C following eccentric bouts.

Table 2. *Acute and chronic ECM responses to training*

ECM Marker	Acute Response	Chronic Response	References
MMP-9	↑ 24h post-exercise	Elevated in trained & untrained	[2]
TIMP-1	↓ 24h post-exercise	Reduced in trained/untrained	[2]
MMP-14	Unchanged acutely	↓ after 10 wks	[2,3]
Collagen I/IV	No change acutely	↑ after 10–27 days	[3,7]
Collagen III	–	↑ 3.2-fold (27 days)	[7]
Serglycin	↑ 2.2-fold	↑ 1.4-fold (12 wks)	[1]
Tenascin-C	↑ 10.8-fold	Attenuated with repeated bouts	[7]
LOXL1	–	Exercise-regulated target	[6]

4.Chronic Adaptations of ECM

Chronic training (≥10 weeks) promotes structural reinforcement of the ECM. Studies consistently demonstrate increased expression of collagens, proteoglycans, and glycoproteins [1,3,4,7], alongside reductions in MMP-14 and TIMP-1 [2,3]. LOXL1 emerges as a novel regulator during aerobic training [6]. These adaptations collectively enhance tissue integrity and resilience.

5.Individual Variability in ECM Responses

Godwin et al. [3] reported that baseline MMP-14 levels predicted hypertrophic responsiveness. Participants with higher pre-training MMP-14 exhibited greater reductions in this marker and larger increases in muscle CSA after training (Table 3).

Table 3. *Individual variability in ECM markers*

ECM Marker	Finding	Performance Association	Reference
MMP-14	Higher baseline in high responders; greater reduction post-training	Greater CSA hypertrophy	[3]

6.ECM–Performance Relationships

ECM adaptations are closely linked to functional outcomes (Table 4). Collagen upregulation supports muscle hypertrophy [4], tendon ECM remodeling improves power [5], and LOXL1 is associated with aerobic capacity [6]. Conversely, excessive tenascin-C correlates with transient strength loss after eccentric exercise [7].

Table 4. *ECM markers and their performance associations*

ECM Marker	Performance Outcome	Association	Reference
Collagen transcripts	CSA, strength	Positive	[4]
Serglycin	CSA, strength	Positive	[1]
MMP-14	CSA hypertrophy	Positive (predictive)	[3]
MMP-9 & TIMP-1	CSA, fiber CSA	Positive	[2]
Tendon ECM	Power, SSC efficiency	Positive	[5]
LOXL1	Aerobic capacity	Positive	[6]
Tenascin-C	Strength (eccentric loss)	Negative	[7]

7.Discussion

This review emphasizes that ECM remodeling is central to skeletal muscle adaptation. Acute exercise promotes transient proteolysis (↑MMP-9, ↑tenascin-C, ↓TIMP-1), creating a permissive environment for remodeling [1,2,7]. Chronic training reinforces ECM through collagen and proteoglycan upregulation while downregulating MMP-14 [2,3,4,7].

Importantly, individual variability—particularly MMP-14 as a biomarker of hypertrophic potential [3]—suggests opportunities for personalized training prescriptions. Functionally, ECM adaptations underpin not only hypertrophy but also tendon stiffness, power, and aerobic performance [4–6].

Limitations include heterogeneity across study designs and reliance on small sample sizes or animal data. More standardized methodologies are needed to strengthen conclusions.

8.Conclusion

Exercise-induced ECM remodeling is a critical mediator of performance. Acute proteolytic activity transitions into chronic reinforcement of ECM structure, correlating with gains in strength, size, tendon function, and endurance. Individual markers such as MMP-14 may predict responsiveness, offering potential for precision exercise interventions. Recognizing ECM as an active determinant of adaptation enriches our understanding of how training drives both structural and functional improvements

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