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Skeletal Muscle as an Endocrine and Paracrine Organ in Breast Cancer Biology: A Narrative Review

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Abstract:
Upon muscle contraction during exercise and resistance training, skeletal muscle releases an array of bioactive molecules called myokines, allowing it to function as a dynamic endocrine organ. Myokines promote systemic and local effects, influencing metabolism and organ function. These myokines may also promote anticancer activity, particularly in breast cancer, and especially in ER/PR-positive, HER2-negative disease. Myokines may influence breast cancer tumor growth, progression, and treatment outcomes. Exercise oncology offers a unique means of optimizing the endocrine and paracrine actions of skeletal muscle-derived myokines within the clinical management of breast cancer. Therefore, the aim of this review is to integrate mechanistic, preclinical, and clinical evidence linking exercise-induced myokines to breast cancer biology, identify breast cancer-specific regulators of myokine signaling, and outline practical implications for exercise oncology prescription and future clinical trial design.

Introduction
Skeletal muscle, in addition to its role in stabilizing and moving the body, functions as the largest dynamic endocrine organ in the human body, capable of secreting a diverse array of bioactive molecules collectively termed myokines **(Table 1)**. These myokines are released in response to myocyte contraction, exerting both endocrine (systemic) and paracrine (local) effects, which influence both local metabolic homeostasis and the biology of distant organs[1]. Acute exercise appears to cause a significant release of myokines, but data remains mixed on the differing impact of type and length of exercise[2].

The importance of exercise both during and after breast cancer treatment to reduce the side effects of treatment like lymphedema and improve strength, muscle mass, mobility and balance has led it to become a standard of care in many survivorship programs[3–5]. The ability of exercise to enhance treatment efficacy or increase survival after treatment for breast cancer remains unknown in human studies, particularly due to issues enrolling the large number of patients that would be required to assess this question. As a result, myokines serve as a potential surrogate, due to a plethora studies showing their anti-cancer effect in both in vivo and in vitro studies.

In the context of breast cancer, and especially in ER/PR-positive, HER2-negative disease, exercise-induced myokines influence tumor growth,

patterns of progression, and patient outcomes after treatment. Exercise oncology and the ability to engage breast cancer patients in intense resistance training and exercise regimens offers a unique means of optimizing the endocrine and paracrine actions of skeletal muscle-derived myokines within the clinical management of breast cancer. Therefore, the aim of this review is to
integrate mechanistic, preclinical, and clinical evidence linking exercise-induced myokines to breast cancer biology, identify breast cancer-specific regulators of myokine signaling, and outline practical implications for exercise oncology prescription and future clinical trial design.

Mechanisms of Endocrine and Paracrine Myokine Action in Breast Cancer

Exercise-induced myokines are released into the circulation, where they can reach distant tissues and exert systemic (endocrine) effects on breast cancer. Myokines with established anti-tumor activity include interleukin-6 (IL-6), IL-7, IL-15, oncostatin M (OSM), secreted protein acidic and rich in cysteine (SPARC), irisin, decorin, and chemokines such as CXCL1, IL-10, and CCL4. Quantitatively, acute bouts of high-intensity resistance training in breast cancer survivors have been shown to increase serum levels of decorin, IL-6, and SPARC by 9% to 47% immediately post-exercise, with IL-6 remaining elevated for at least 30 minutes post-intervention[1]. OSM levels are specifically elevated after resistance training and remain increased at 30 minutes post-exercise in this modality. These acute increases in myokines are associated with a 19% to 29% reduction in the in vitro growth of MDA-MB-231 breast cancer cells when exposed to post-exercise serum. IL-15 has been shown to be released at varying levels postexercise based on the exercise regimen and length, as well as the age and body composition of the individuals exercising[6]. Additionally, human versus animal studies reveal similar discrepancies of IL-15 secretion[7].

Similar findings have been observed in exercise-conditioned serum derived from breast cancer patients after a 12-week structured strength and conditioning program[8]. When exposed to patient derived exercise-conditioned serum, MCF-7 and MDA-MB-231 breast cancer cell lines showed decreased metabolic activity, and increased apoptotic activity as demonstrated by expression of apoptotic proteins caspase-7 and caspase-8. Both breast cancer patient-derived serum and serum from healthy women who underwent resistance training followed by high intensity spinning decreased hormone-sensitive and hormone-insensitive breast cancer cell viability *in vitro*[9].

Additionally, tumorigenesis was reduced by 50% in preincubated MCF-7 breast cancer cells inoculated into NMRI-Foxn1^{nu} mice. This study also revealed the inhibition of breast cancer cell viability from exercise-induced epinephrine and norepinephrine production, which was confirmed with tumor-bearing mice after exercise on a running wheel. It also has been suggested that key exercise-induced factors are specifically secreted from skeletal myotubes that undergo mechanical stress.[2] Despite these known acute effects, the long-term effect of myokine levels with exercise is less studied and represents a need for future research.

The systemic actions of these myokines include direct inhibition of tumor cell proliferation, induction of apoptosis via increased caspase 3/7 activity, and enhancement of anti-tumor immunity through the recruitment and activation of natural killer (NK) cells and cytotoxic T lymphocytes[1,10,11]. IL-6 and IL-15, both upregulated by exercise, support the proliferation and maturation of NK and CD8+ T cells, which are essential for immune surveillance and the elimination of micrometastatic disease[11]. The endocrine effects of myokines are particularly relevant for the suppression of metastatic outgrowth, as they can modulate the systemic immune environment and metabolic state, creating conditions less favorable for tumor progression and dissemination[11–13].

Within the breast tumor microenvironment, exercise-induced myokines may act in a paracrine fashion to directly influence tumor cell behavior and the surrounding stroma (Figure 1). OSM, for example, is robustly upregulated in skeletal muscle and serum following exercise and has been shown to inhibit proliferation and induce apoptosis in ER/PR-positive breast cancer cells via caspase activation[14]. In vivo, blockade of OSM with neutralizing antibodies inhibits the exercise-induced delay in mammary tumor development, suggesting a causal role for OSM in mediating local anti-tumor effects[13–15]. SPARC and decorin, both acutely increased after resistance or high-intensity interval training (HIIT), modulate the extracellular matrix (ECM), inhibit angiogenesis, and reduce the invasive potential of tumor cells[1,16]. Irisin decreases the viability and migration of malignant breast epithelial cells by inducing apoptosis and suppressing NFκB signaling, with these effects being most pronounced in the local tumor milieu[17,18].

Exercise-induced myokines also modulate the immune landscape of the tumor microenvironment. Acute exercise increases the infiltration of cytotoxic immune cells (CD8+ T cells and NK cells) and decreases immunosuppressive myeloid-derived suppressor cells (MDSCs) within the tumor, shifting the immune profile toward a more anti-tumorigenic state[13,19]. These paracrine effects are central to the suppression of primary tumor growth and may also enhance the efficacy of immunotherapies by transforming immunologically "cold" tumors into "hot" tumors with increased immune cell infiltration[19].

Quantitative Clinical Evidence and Exercise Prescription

Post-diagnosis exercise is associated with significant reductions in breast cancer recurrence, breast cancer-specific mortality, and all-cause mortality[20]. Meeting aerobic exercise guidelines (≥2.5 hours per week) after breast cancer diagnosis is associated with approximately a 50% reduction in all-cause mortality, with further incremental benefits up to about 4.5 hours per week. Even lower levels of exercise (around 1 hour per week) confer a 25% reduction in hazard for all-cause mortality. Five-year all-cause mortality rates are 11% for no/minimal exercise, 4% for insufficient exercise, and 3% for those meeting guidelines[20]. These benefits are observed across subtypes but appear most pronounced in those with ER/PR-positive, HER2-negative disease, for whom adjusted hazard ratios for recurrence and breast cancer-related death are 0.63 (95% CI, 0.45–0.88) and 0.57 (95% CI, 0.37–0.86), respectively[21].

Mechanistic studies suggest that exercise-induced myokines mediate these benefits. Serum collected from breast cancer survivors after a single bout of resistance or HIIT contains elevated levels of decorin, IL-6, SPARC, and OSM, and this serum suppresses the growth of breast cancer cells in vitro by 19% to 29%[1]. A 12-week resistance training intervention increases muscle mass and strength, and post-intervention serum inhibits breast cancer cell growth and promotes apoptosis in vitro; similar effects are observed in the presence of recombinant myokines including CXCL1, IL-10, and CCL4[1,10]. Similar studies have revealed the release of IL-7, IL-15, and other myokines after acute bouts of exercise and decreased breast cancer viability after exposure to post-exercise serum containing these myokines[6]. These data support the role of myokines as mediators of the anti-tumor effects of exercise, although the specific attribution of improved long-term clinical outcomes to myokine-mediated pathways, as opposed to other exercise-induced benefits, remains inferential rather than definitive[1,10,20].

Exercise Regimens for Optimal Myokine Induction

Different exercise modalities, intensity, and length appear to uniquely promote myokine production with regard to type and quantity. For instance, acute bouts of resistance training and HIIT are particularly effective for inducing robust increases in anti-tumor myokines, with some studies revealing that HIIT produced a more pronounced acute increase in IL-6 and resistance training sustaining OSM elevations for a longer period post-exercise[1]. Other data reveal similar changes across exercise modalities, as a comparison of thrice weekly high intensity resistance training for three months versus HIIT revealed significant and similar increases in myokine production, along with similar in vitro breast cancer cell growth reduction[22]. Additional data reveal that chronic exercise interventions, such as 12-week resistance training programs, increase muscle mass and strength and enhance baseline myokine secretion, with both acute and chronic effects contributing to sustained anti-tumor benefits[10]. IL-7, for instance, is secreted after strength training and promotes immune cell regulation[23]. Other myokines, such as IL-15, appear to be secreted after acute exercise sessions, irrespective of exercise type and participants training history. In this large meta-analysis, chronic exercise, defined as 2 weeks or longer, did not result in a baseline secretion of IL-15[24].

The impact of resistance training regimens that incorporate conditioning via manipulation of rest time and time under tension is less studied. However, a large meta-analysis of 62 studies revealed that both aerobic and resistance exercise promoted an increase in myokine levels, however, no significant difference between training modes nor magnitude of change was seen[6]. The considerable heterogeneity within the literature was likely responsible for the lack of significance with some of the findings.

The benefits of myokine production and hormonal changes appear to be from acute exercise production. For instance, serum derived from breast cancer survivors engaging in chronic exercise over six months had no effect on breast cancer cell viability in vitro[25]. However, serum taken from individuals after two hours of acute exercise promoting a significant increase in epinephrine, norepinephrine, serum lactate, and cytokines including IL-6, reduced viability of two breast cancer cell lines versus resting serum. Other data reveal different responses to different exercise modalities based on the cancer subtype, but breast cancer response to exercise appeared greater after a prolonged intense exercise regimen, showing both the acute and chronic impact of intense exercise[26].

While the impact of specific exercise protocols, ranging from purely cardiovascular exercise, to HIIT, to purely resistance training remains unclear at this point, the overarching evidence clearly points to the ability of acute exercise to increase myokine production. However, the release of myokines appears to be enhanced with higher intensity and longer aerobic or higher intensity resistance training. Thus, this further places clinical emphasis on the ability to allow patients to safely but effectively engage in intense exercise.

Data in trained individuals reveals that supervised exercise programs are generally more effective than unsupervised or home-based interventions, particularly for resistance training, as they ensure proper technique, progression, and adherence and lead to greater strength gain, power output, muscle mass gain, and training load increase increased in weight lifted[27–29]. Our group has shown previously that individuals who gained significant muscle mass and lost fat mass loss these benefits when transitioning to an at home program[30], presumably from decreased intensity versus the in person intense regimen. Additionally, a large meta-analysis of breast cancer exercise studies revealed that exercise adherence was strongly associated with contacts with other participants, a supervised element, an exercise prescription, and social support[31].

Exercise prescriptions should be individualized based on patient assessment, comorbidities, and treatment status, with special attention to age, menopausal status, and baseline muscle mass, as these factors influence both the secretion and anti-tumor efficacy of myokines[32]. Movement analyses should ensue to ensure individuals are not at increased risk with resistance training, particularly with intense regimens utilizing compound movements. Our group utilizes the Functional Movement Screen, Y-Balance Test, and Klatt test. Breast surgery often involves resection of axillary lymph nodes, and accordingly lymphedema screening is warranted prior to initiating an exercise program.

Subtype-Specific and Patient-Specific Considerations

The anti-tumor effects of exercise-induced myokines, while observed across breast cancer histologies, are most consistently observed in ER/PR-positive, HER2-negative cases[21]. Increased post-diagnosis exercise is associated with a significant reduction in breast cancer-related death and recurrence in this subtype, with adjusted hazard ratios for recurrence and death of 0.63 and 0.57, respectively[21]. Mechanistically, myokines such as OSM, SPARC, and irisin inhibit proliferation, induce apoptosis, and modulate the tumor microenvironment in ER/PR-positive, HER2-negative cell lines (e.g., MCF-7), with OSM identified as a key mediator of exercise-induced tumor suppression[14,15,21]. The paracrine and endocrine actions of these myokines are particularly effective in this subtype, likely due to the interplay between hormone receptor signaling and myokine-mediated pathways[10,12,33].

The effects of exercise-induced myokines in HER2-positive and triple-negative breast cancer (TNBC) appear to be more variable. Some studies suggest that exercise and its associated myokines can inhibit tumor growth and promote apoptosis in triple-negative models (e.g., MDA-MB-231), with irisin and SPARC demonstrating anti-proliferative and anti-migratory effects[1,17,34]. However, the magnitude and consistency of these effects are less robust than in ER/PR-positive, HER2-negative disease, while preclinical models have shown heterogeneity in response, with exercise associated with inhibition, no change, or even acceleration of tumor growth in different TNBC models[35]. The clinical benefit of exercise in HER2-positive and TNBC subtypes is less well established, with some studies indicating potential benefit and others showing minimal or variable impact[21,36].

Patient-specific factors such as age, menopausal status, comorbidities, and baseline muscle mass significantly influence both the secretion and anti-tumor efficacy of exercise-induced myokines. Older age and menopause are associated with reduced muscle mass and blunted myokine responses, necessitating tailored exercise regimens to achieve optimal benefits[12,32]. Comorbidities such as obesity, diabetes, and cardiovascular disease can alter myokine profiles and diminish the efficacy of exercise interventions[12,32]. Baseline muscle mass is a key determinant of myokine secretion, with higher muscle mass associated with greater increases in myokine levels during exercise and enhanced anti-tumor effects[12]. Individualized exercise prescriptions that both aim to increase muscle mass and account for these factors are essential to maximize the therapeutic potential of exercise in breast cancer management[12,32].

Interactions with Standard Breast Cancer Therapies and Ongoing Clinical Trials

Preclinical and translational studies indicate that exercise-induced myokines can interact with standard breast cancer therapies to enhance anti-tumor effects, improve treatment efficacy, and mitigate adverse effects. Exercise has been shown to augment the efficacy of chemotherapy (e.g., doxorubicin) and endocrine therapy (e.g., tamoxifen) in rodent models, with myokines such as irisin increasing tumor cell susceptibility to apoptosis and enhancing the cytotoxic effects of chemotherapeutic agents[17,37]. Exercise-induced myokines also modulate the tumor microenvironment, reduce inflammation, and enhance immune cell infiltration, creating conditions more favorable for the efficacy of systemic therapies[1,11,12]. In the clinical setting, exercise interventions during chemotherapy are safe and associated with increased levels of anti-inflammatory and anti-tumor myokines such as IL-6 and adiponectin, with trends toward increased irisin[38]. However, the optimal type, intensity, and timing of exercise to maximize synergistic effects with systemic therapies remain to be defined, and large-scale randomized controlled trials with mechanistic endpoints are needed to establish causality[28,37,39].

Several randomized controlled trials are currently underway to evaluate the effects of exercise interventions during various phases of breast cancer treatment, including neoadjuvant, adjuvant, and metastatic settings. The Neo-ACT trial (NCT05184582, registered 11-9-2022) is a large, prospective, randomized clinical trial enrolling patients with early-stage breast cancer to a home-based program combining HIIT and resistance training, aiming for at least 150 minutes of moderate to vigorous physical activity per week. The primary endpoint is pathological complete response (pCR), with secondary endpoints including quality of life, toxicity, and oncological outcomes[40]. The Neo-Train trial (NCT04623554, registered 6-23-2021) is a two-arm, randomized controlled trial in Denmark, enrolling women with newly diagnosed breast cancer undergoing standard neoadjuvant chemotherapy, with the intervention arm receiving supervised HIIT and progressive resistance training three times per week for 24 weeks[40,41]. In the metastatic setting, the EMBody trial (NCT05468034, registered 4-26-2023) is a phase II, randomized controlled trial enrolling patients with indolent, stable metastatic breast cancer to a virtually delivered, multimodal exercise program three days per week for 16 weeks, with primary and secondary endpoints including cardiorespiratory fitness, physical function, muscle mass, and serologic biomarkers of inflammatory, metabolic, and immune pathways[42]. The NeoChemoPRT study (NCT06858449, registered 1-2-2025) is a randomized study assessing an in-person, progression, and intense resistance training protocol for women with breast cancer undergoing neoadjuvant chemotherapy and randomized to two different post workout protein supplements of 20 versus 60 grams. Body composition, strength, balance, functional mobility, quality of life, anxiety, and depression are being assessed, along with response rates to chemotherapy

Exercise oncology trials are increasingly incorporating mechanistic endpoints, including myokine profiling, to elucidate the pathways by which various exercise regimens may influence tumor biology and clinical outcomes. While direct pharmacologic targeting of myokine pathways in breast cancer remains investigational, the accumulating mechanistic and translational data from ongoing exercise trials are likely to inform the development of future studies that may directly target myokine pathways[43,44].

Limitations and Future Directions

Several significant limitations remain in translating the function of exercise-induced myokines into clinical management of breast cancer. Preclinical models do not fully recapitulate the heterogeneity and complexity of human breast cancer, and the precise mechanisms by which myokines influence tumor biology are incompletely understood[45,46]. There is a lack of standardized protocols for exercise interventions and myokine measurement in both preclinical and clinical studies, with variability in assay sensitivity, specificity, and reproducibility[43]. Large-scale, well-designed randomized controlled trials are required to test the efficacy of structured exercise interventions on breast cancer outcomes, with careful attention to exercise dose, modality, and patient stratification[28,44,47].

The identification and validation of myokines as clinically useful biomarkers for diagnosis, prognosis, or prediction of treatment response is still in its infancy, and the integration of myokine measurements with other omics data may be necessary to fully capture the complexity of muscle-tumor crosstalk[45,48]. Enhanced collaboration across disciplines, improved access to clinical samples, and investment in translational infrastructure are essential to accelerate the translation of preclinical findings into clinical benefit for breast cancer patients[48,49].

Conclusions and Clinical Recommendations

Skeletal muscle serves as an endocrine and paracrine organ in breast cancer biology, with exercise-induced myokines mediating significant anti-tumor effects. Exercise-induced myokines appear particularly beneficial within the context of ER/PR-positive, HER2-negative breast cancer, with more variable effects in HER2-positive and triple-negative subtypes. Quantitatively, moderate to vigorous aerobic and resistance exercise, totaling at least 150 minutes per week, performed 2–3 times per week at intensities of 60–85% HRmax or 60–80% 1RM for a minimum of 12 weeks, is recommended to optimize anti-tumor myokine induction and improve clinical outcomes. The impact of resistance training regimens that incorporate conditioning is less studied. Individualized exercise prescriptions that account for age, menopausal status, comorbidities, and baseline muscle mass are essential to maximize therapeutic benefit.

Ongoing clinical trials are evaluating the impact of exercise interventions on breast cancer outcomes, with increasing emphasis on mechanistic endpoints such as myokine profiling. The integration of structured exercise programs into standard breast cancer care is strongly supported by current evidence.

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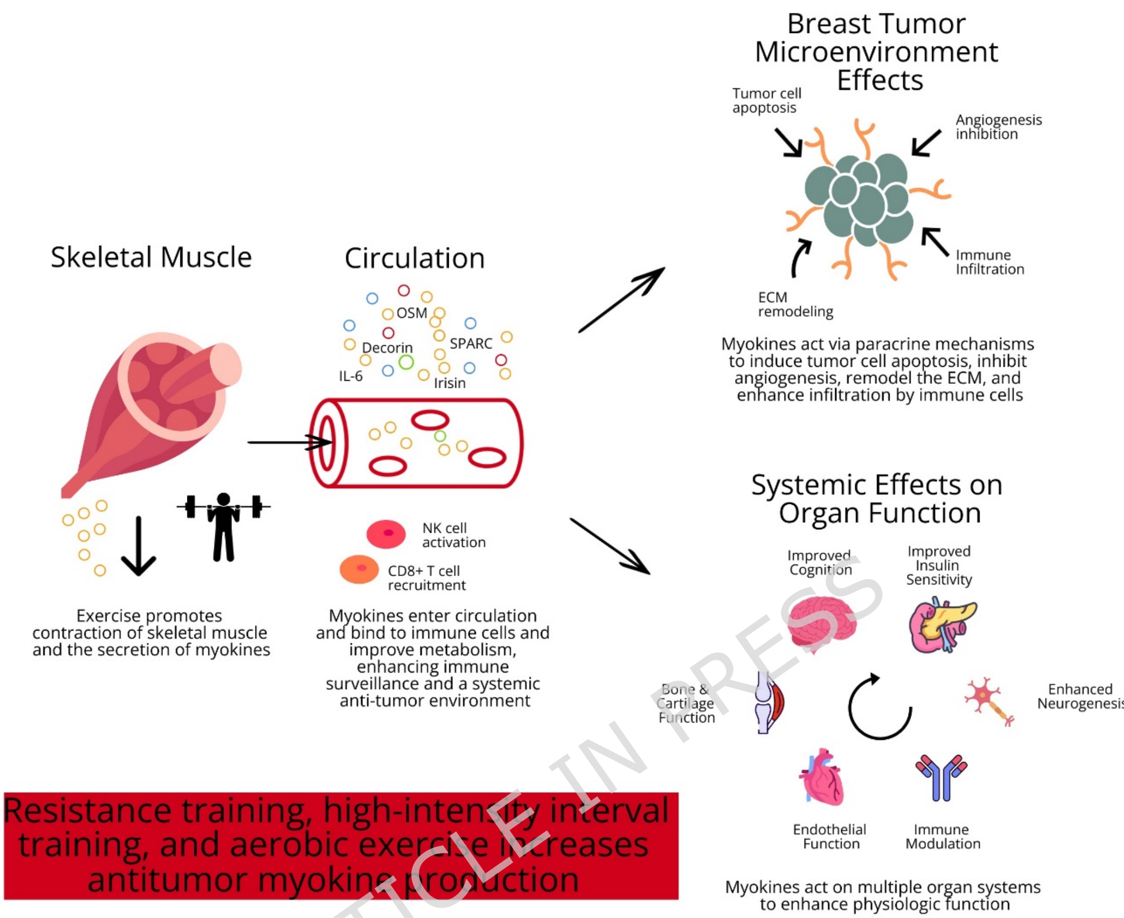
Table 1: Myokine Reference Guide

Myokine	Definition	Main Functions	Potential Benefits
IL-6	Cytokine released from contracting muscle, especially during exercise[50,51].	Regulates metabolism, stimulates glucose uptake, enhances fat oxidation; modulates immune response (pro- and anti-inflammatory depending on context) [50–52].	Improved glucose control, fat metabolism, reduced chronic inflammation risk[50,51].
IL-7	Cytokine released from myotubes during muscle contraction[23].	Immune cell modulation[53].	Improved immune cell function.
IL-15	Myokine produced in skeletal muscles dominated by type II fibers[54].	Enhances skeletal muscle mitochondrial biogenesis, regulates metabolism via PPAR δ and PGC-1α expression. Impacts bone structure via mediation of osteoblasts and osteoclasts. Enhances the innate and adaptive immune system[7].	Improved glucose control, body composition optimization, increased bone density, enhanced immunity[7].
IL-1ra	Anti-inflammatory cytokine released after high intensity exercise that blocks IL-1 signaling[55,56].	Counteracts pro-inflammatory effects of IL-1/IL-1β; helps resolve exercise-induced inflammation[55,56].	Supports tissue recovery, lowers chronic inflammation load[55,56].

LIF	Member of IL-6 family of cytokines produced after repeated exercise stimuli[57,58].	Stimulates muscle repair, involved in muscle regeneration and satellite cell activation, influences neural survival; supports immune modulation[57,58].	Enhanced muscle regeneration after injury or training[57,58].
Irisin	Peptide cleaved from FNDC5 protein during exercise[59].	Promotes 'browning' of white adipose tissue; increases thermogenesis; may improve brain function[59].	Supports fat loss, metabolic health, neuroprotection[59].
SPARC	Exercise-induced glycoprotein produced by muscle and bone[60–62].	Supports muscle integrity through ECM remodeling and mitochondrial biogenesis, inhibits tumor growth (anti-oncogenic); involved in ECM remodeling; supports bone and muscle health[60–62].	Cancer protection, musculoskeletal integrity[60–62].
Decorin	Small leucine-rich proteoglycan secreted by muscle during exercise[63,64].	Binds and neutralizes myostatin (muscle growth inhibitor); influences collagen organization[63,64].	Promotes muscle hypertrophy, tendon and connective tissue strength[63,64].
OSM (Oncostatin M)	Cytokine in the IL-6 family secreted during exercise[65].	Anti-tumor properties; regulates inflammation and tissue remodeling[65,66].	Potential cancer suppression, improved tissue repair[65,66].
Myostatin	Myokine whose production is deceased with exercise[67].	Limits muscle fiber size; maintains muscle mass homeostasis[67].	Lower levels after training = greater potential for hypertrophy[67].
Follistatin	Glycoprotein released during exercise that binds and inhibits myostatin[68].	Promotes muscle growth; supports tissue regeneration[68].	Hypertrophy, recovery from injury[68].

Apelin	Peptide secreted by muscle during high intensity exercise[69].	Enhances cardiovascular function, muscle perfusion, glucose uptake; supports mitochondrial health, offsets oxidation[70].	Improved heart health, insulin sensitivity, endurance[70].
FGF21 (Fibroblast growth factor 21)	Hormone expressed in muscle, liver, pancreas, brain and adipose tissue with mixed response to exercise[71].	Regulates glucose and lipid metabolism; promotes fat oxidation; supports mitochondrial function[71].	Metabolic health, anti-obesity effects, potential longevity benefits[71].
Cathepsin-B	Lysosomal protease released during exercise[72].	Crosses blood–brain barrier; stimulates brain-derived neurotrophic factor production; supports neurogenesis[72].	Cognitive benefits, brain health, neuroprotection[72].
IL-15	Myokine expressed in skeletal muscle with higher levels in type II fibers[73].	Promotes muscle anabolism, fat oxidation; enhances NK cell activity in immune system[74,75].	Lean mass preservation, fat loss, immune defense[74,75].
METRNL (Meteorin-like)	Exercise-induced protein from muscle and adipose tissue[76].	Promotes angiogenesis, glucose/lipid metabolism, modulates immune cell anti-inflammatory activity[76].	Improved metabolic control, reduced inflammation, immune modulation[76].
Myonectin (CTRP15)	Myokine from skeletal and cardiac muscle[77].	Enhances fatty acid uptake into tissues;regulates systemic lipid metabolism[77].	Improved lipid profile, metabolic health[77].

Figure 1:


Breast Tumor Microenvironment Effects
Tumor cell apoptosis
Angiogenesis inhibition
Immune Infiltration
ECM remodeling
Myokines act via paracrine mechanisms to induce tumor cell apoptosis, inhibit angiogenesis, remodel the ECM, and enhance infiltration by immune cells
Skeletal Muscle
Circulation
OSM
Decorin
SPARC
IL-6
Irisin
NK cell activation
CD8+ T cell recruitment
Exercise promotes contraction of skeletal muscle and the secretion of myokines
Myokines enter circulation and bind to immune cells and improve metabolism, enhancing immune surveillance and a systemic anti-tumor environment
Systemic Effects on Organ Function
Improved Cognition
Improved Insulin Sensitivity
Bone & Cartilage Function
Enhanced Neurogenesis
Endothelial Function
Immune Modulation
Myokines act on multiple organ systems to enhance physiologic function
Resistance training, high-intensity interval training, and aerobic exercise increases antitumor myokine production