QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

KRUSZYŃSKA, Zuzanna, ALESHCHYK, Anastasiya, ADAMCZAK, Michał, MAJEWSKI, Marcel, LIBURA, Bogumił, LAZAR, Martyna, SZKARADOWSKI, Łukasz, BARANOWSKI, Szymon, LEWAŃSKA, Julia and BOGDAŃSKI, Krzysztof. Aerobic Exercise as a Cardioprotective Strategy in Breast Cancer Patients Undergoing Anthracycline-Based Chemotherapy: A Narrative Review. Quality in Sport. 2026;68:74425. https://doi.org/10.12775/QS.2026.68.74425

ARTICLE TIMELINE
Received: 18.07.2026. Revised: 02.08.2026. Accepted: 02.08.2026. Published: 13.08.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Aerobic Exercise as a Cardioprotective Strategy in Breast Cancer Patients Undergoing Anthracycline-Based Chemotherapy: A Narrative Review

Zuzanna Kruszyńska
ORCID https://orcid.org/0009-0001-2905-9642
e-mail: zuzanna.kruszynska@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Anastasiya Aleshchyk
ORCID https://orcid.org/0009-0006-5368-6343
e-mail: anastasiya.aleshchyk@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Michał Adamczak
ORCID https://orcid.org/0009-0002-3693-1731
e-mail: michal.adamczak@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Marcel Majewski
ORCID https://orcid.org/0009-0002-6658-235X
e-mail: marcel.majewski@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Bogumił Libura
ORCID https://orcid.org/0009-0006-6889-1569
e-mail: bogumil.libura@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Martyna Lazar
ORCID https://orcid.org/0009-0001-5973-846X
e-mail: martyna.lazar@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Łukasz Szkaradowski
ORCID https://orcid.org/0009-0001-3108-3051
e-mail: lukasz.szkaradowski@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Szymon Baranowski
ORCID https://orcid.org/0009-0000-5877-4875
e-mail: szymon.baranowski@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Julia Lewańska
ORCID https://orcid.org/0009-0005-0102-0075
e-mail: julia.lewanska@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Krzysztof Bogdański
ORCID https://orcid.org/0009-0005-1023-6257
e-mail: krzysztof.bogdanski@stud.umed.lodz.pl
Medical University of Lodz, Lodz, Poland

Corresponding Author:

Marcel Majewski
e-mail: marcel.majewski@stud.umed.lodz.pl

Abstract

Background: Advances in breast cancer diagnosis and treatment have significantly extended patient survival, leading to this cancer being increasingly viewed as a chronic disease. However, the use of classic anthracycline-based chemotherapy is associated with a high risk of cardiotoxicity. With extended survival post-therapy, cardiovascular complications emerge as the leading non-oncological cause of mortality among breast cancer survivors

Aim: Summary of the current state of knowledge regarding the role and mechanisms of aerobic training as a cardioprotective strategy in breast cancer patients undergoing anthracycline chemotherapy.

Materials and methods: Analysis of contemporary scientific literature, preclinical studies, and guidelines from international oncology and cardiology societies (e.g., ASCO, ACS, ICOS-CORE).

Results: Anthracyclines damage the myocardium and microcirculation by interacting with topoisomerase IIβ (TOP2B), activating the TGF-β/Smad3 axis, and generating oxidative stress. Aerobic exercise effectively mitigates this cardiotoxicity at the cellular level by strengthening antioxidant defense systems, preserving mitochondrial ultrastructure, promoting biogenesis, and inhibiting apoptotic pathways and myocardial atrophy. Implementing regular exercise during treatment improves therapy tolerance, VO2 peak, and vascular and muscular function.

Conclusions: Regular, structured aerobic training (exercise preconditioning) demonstrates a strong, multifaceted cardioprotective effect. Cardio-oncology rehabilitation programs (CORE) should be an integral component of oncology care and a further research focus.

Key words: breast cancer, anthracyclines, aerobic training, cardiotoxicity, cardioprotection, cardio-oncology.

1. Introduction

Breast cancer presently represents a paramount challenge to contemporary oncology, being the most frequently diagnosed malignancy and the dominant cause of cancer-related mortality among women worldwide [1]. However, over the past few decades, significant progress has been made in early diagnosis, widespread screening, and the development of personalized cancer therapies. Thanks to modern screening methods and advanced combination therapies, the five-year survival rate for patients with early-stage disease exceeds ninety percent [3]. Although the primary goal of anticancer therapy is complete cure or delaying disease recurrence to prolong survival, breast cancer is increasingly treated and viewed as a chronic disease. Therefore, greater attention should be paid to the long-term consequences of systemic treatment [2].

Cardiotoxicity induced by oncological therapies, particularly classic anthracycline chemotherapeutics, represents one of the most critical complications [4]. Myocardial and vascular injury significantly compromises functional capacity, induces chronic fatigue, and elevates the risk of progression to overt, irreversible heart failure [2]. Epidemiological analyses show that in patients who have successfully undergone breast cancer therapy, the risk of cardiovascular death becomes the leading non-oncological cause of cancer-free mortality, exceeding the risk of death from cancer recurrence in the years following treatment completion [5].

Given these considerations, a critical imperative of contemporary medicine is to identify effective and safe cardioprotective strategies that do not compromise the efficacy of oncological therapies. In addition to traditional pharmacological interventions, regular, structured physical activity has proven to be a promising, universal strategy free from negative side effects [6]. The aim of this review is to synthesize the current state of knowledge regarding the role and mechanisms of aerobic training as a cardioprotective strategy in breast cancer patients undergoing anthracycline-based chemotherapy.

2. Anthracyclines and Heart: Mechanisms of Cardiotoxicity.

2a. Anthracyclines - A Standard in Breast Cancer Treatment

Anthracyclines, the most commonly used of which are doxorubicin and its derivative epirubicin, are cytotoxic antibiotics that have been the gold standard and foundation of breast cancer chemotherapy for decades [7]. Despite the rapid advancement of targeted molecular therapies and immunotherapies, conventional anthracycline-based chemotherapy remains an

indispensable component of neoadjuvant and adjuvant regimens due to its established efficacy in eradicating aggressive malignancies [8]. However, their clinical use is limited by cumulative dose-dependent myocardial toxicity. These toxicities may manifest as acute hemodynamic disturbances immediately following infusion or progress asymptomatically over several decades, ultimately culminating in symptomatic heart failure and a decline in left ventricular ejection fraction (LVEF) [9].

2b. Pathophysiology of Anthracyclines

The mechanism of anthracycline-induced cardiotoxicity (AIC) is multifaceted and involves complex interactions at the molecular, cellular, and tissue levels. Although previous theories have focused primarily on the hypothesis of excessive reactive oxygen species (ROS) production, the current literature integrates these assumptions around topoisomerase IIβ (TOP2B) as the primary target for anthracycline molecules in the myocardium [10]. Drug binding to TOP2B triggers a cascade leading to DNA damage, mitochondrial dysfunction, and metabolically induced cardiomyocyte apoptosis [10,11]. Recent scientific reports reveal that doxorubicin directly increases TOP2B protein levels. Consequently, this disrupts the function of the histone methyltransferase SMYD1, which may explain the tissue-specific toxicity observed in the heart. SMYD1 is a muscle-specific histone methyltransferase and serves as a pivotal regulator in normal myocardial growth and differentiation, as well as in human dilated cardiomyopathy [12]. It is also worth noting that the pathogenesis of AIC extends beyond cardiomyocytes and also involves the cardiac microvasculature. Doxorubicin tends to accumulate strongly in cardiac endothelial cells, where it activates the TGF-β/Smad3 signaling pathway [13]. Stimulation of the TGF-β/Smad3 axis also induces a local inflammatory response, which leads to adverse cardiac remodeling and progressive ventricular dysfunction [13]. These processes initiate subclinical, asymptomatic left ventricular dysfunction, which, driven by cumulative myocardial injury over time, can progress to overt, end-stage heart failure [11].

3. Mechanisms of The Cardioprotective Effects of Aerobic Exercise

Aerobic exercise (AE) can be defined as structured, long-term activity that involves a sustained increase in heart rate, resulting in increased oxygen consumption and utilization [4]. Aerobic exercise represents an important non-pharmacological strategy to counteract the cardiotoxicity induced by anthracyclines such as doxorubicin (DOX), protecting the heart structure and preventing systolic and diastolic dysfunction. The cardioprotective benefits of physical activity

are based on stimulating numerous physiological adaptations at the cellular and functional levels.

3a. Reduction of Oxidative Stress

One of the main mechanisms of doxorubicin (DOX) cardiotoxicity is the increase in oxidative stress through the formation of reactive oxygen species (ROS) [14]. Preclinical studies indicate that aerobic training performed before or during DOX administration reduces oxidative damage, as evidenced by reduced levels of lipid peroxidation, malondialdehyde (MDA) concentrations, and protein carbonylation in cardiac tissue [14]. These changes result from the enhancement of cellular antioxidant systems by physical exercise. Training increases the activity of antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). Exercise also enhances the glutathione system, increasing cellular glutathione (GSH) concentrations and decreasing the concentration of the oxidized form (GSSG). At the transcriptional level, moderate exercise-induced oxidative stress stimulates the activation of the Ref1 pathway and subsequently the Nrf2 factor, which stimulates the transcription of nuclear antioxidant genes and indicates that exercise induces adaptations in cardiac tissues that maintain redox balance in cardiomyocytes [15].

3b. Protection of mitochondrial structure and function

Doxorubicin exhibits a strong affinity for cardiolipin in the inner mitochondrial membrane, leading to its accumulation, impairment of the electron transport chain (ETC), and impaired ATP synthesis [16]. Electron microscopy (TEM) analyses confirm that aerobic training protects cardiac ultrastructure by preventing drug-induced vacuolation, mitochondrial swelling, and loss of mitochondrial cristae [14]. This cardioprotection results, among other things, from the fact that exercise reduces DOX accumulation in the cardiac mitochondria. This mechanism is associated with increased expression of ATP-binding cassette transporters (ABCs), responsible for drug efflux from mitochondria [14]. Concurrently, training enhances mitochondrial biogenesis by increasing the level of the coactivator PGC-1alpha, which, in cooperation with Nrf1/2 factors, stimulates the expression of mitochondrial transcription factor A (TFAM), a protein essential for transcription and replication of mitochondrial DNA (mtDNA) [14].

3.c. Inhibition of Apoptotic and Proteolytic Pathways

Doxorubicin induces programmed cell death (apoptosis) [17]. Aerobic training effectively inhibits these processes, reducing the percentage of apoptotic cells in the heart. Physical

activity reduces the DOX-induced increase in the expression of proapoptotic proteins (Bax, Bak, caspases) while maintaining or increasing the level of the antiapoptotic protein Bcl-2 [19]. Doxorubicin also activates intracellular proteolytic systems that destroy myofibrillar proteins. This excessive activation of protein degradation systems leads to cardiac mass loss and atrophy [18]. Although the precise molecular mechanisms by which physical exercise regulates these processes have not yet been fully elucidated, scientific studies indicate its clear protective effect. Aerobic training counteracts the doxorubicin-induced increase in the expression of autophagy markers (such as Beclin 1, ATG, and LC3) and lysosomal proteases (cathepsins B and L), effectively stabilizing protein turnover in cardiomyocytes [14]. At the proteolytic level, aerobic training also inhibits the FOXO signaling pathway, which reduces nuclear transcription of genes responsible for myocardial atrophy [20].

4. Aerobic Training in Clinical Practice

Implementing aerobic training in clinical practice in breast cancer patients undergoing anthracycline-based chemotherapy is an important, non-pharmacological element of a cardioprotective and supportive strategy [21]. Preclinical studies in animal models suggest that structured exercise training can partially alleviate structural and functional cardiac dysfunction caused by direct doxorubicin toxicity during systemic treatment [22]. The emerging discipline of "exercise cardio-oncology" defines structured exercise as a promising therapeutic method, stimulating numerous biochemical and physiological adaptations in the cardiovascular system and myocardium [23]. Regular activity has been shown to improve cardiorespiratory fitness, physical function, quality of life, and reduce chronic fatigue. There is also emerging evidence of improved treatment tolerance and a reduced risk of long-term cardiac events [23]. A decrease in exercise tolerance and VO2peak is a strong predictor of increased mortality in older patients after breast cancer therapy, particularly with exposure to high cumulative doses of anthracyclines [24]. Modeling analyses show that in high-risk patients, the main determinant of exercise intolerance is impaired peripheral oxygen diffusion in muscle (DMO2), accounting for as much as 81.7% of the variance in reduced V2peak, in addition to impaired convective oxygen delivery (QO2) [24]. This indicates the need to design long-term aerobic interventions that prioritize improving vascular and muscular function [24]. Current guidelines from international scientific societies, including the American Society of Clinical Oncology (ASCO) and the American Cancer Society (ACS), recommend regular physical activity in breast cancer patients [25, 26]. According to these guidelines, patients should aim to achieve at least 150 minutes of moderate aerobic activity or 75 minutes of vigorous aerobic training per week,

supplemented by resistance training twice a week [26]. Although currently less than 15% of patients meet these criteria, initiating training during the active treatment phase yields better clinical results than postponing intervention until after therapy [26]. Due to the documented risk of early and late anthracycline-induced cardiotoxicity, training under the supervision of a qualified specialist is recommended to prevent undesirable stress [25, 26]. A comprehensive approach combining early imaging diagnostics (global longitudinal strain assessment – GLS) with a structured exercise program is the foundation of modern cardio-oncology for the prevention of heart failure [21].

5. Conclusions

The literature review indicates that aerobic training plays an important, multifaceted role in cardioprotection in breast cancer patients undergoing anthracycline-based chemotherapy. Evidence from preclinical studies confirms that aerobic training (so-called exercise preconditioning) effectively counteracts the direct cardiotoxicity of doxorubicin [27]. Through cellular and systemic mechanisms – including reducing oxidative stress, stabilizing mitochondrial structure and function, inhibiting apoptosis, suppressing inflammation, and improving microcirculatory efficiency – physical areobic exercise effectively prevents the decline in cardiac reserve and protects patients from functional impairment [14].

The clinical dimension of these reports is gaining importance in light of the latest international standards developed by expert groups. They emphasize that cardio-oncological rehabilitation, a logical approach based on structured exercise programs (CORE – Cardio-Oncology Rehabilitation and Exercise), should be an integral, not merely optional, element of care for cancer patients. A multidisciplinary approach integrating oncology, cardiology, and exercise rehabilitation is essential to enhance patients' functional capacity, optimize quality of life, and mitigate the risk of adverse cardiovascular complications [28]. Consequently, an optimal cardioprotective strategy in the era of modern medicine cannot be limited to isolated pharmacological measures but must be integrated. Personalized aerobic training should be complementarily combined with advanced pharmacological methods (such as the use of dexrazoxane, beta-blockers, ACE inhibitors, or liposomal anthracyclines) and rigorous imaging surveillance based on advanced echocardiography and global longitudinal strain assessment (GLS) [21]. Future developments in this field should focus on further randomized controlled trials that will allow for precise optimization of the timing, volume, and intensity of exercise interventions in relation to the patient's individual risk profile. The synergy of pharmacotherapy, modern diagnostics, and lifestyle medicine currently represents the most

promising approach to long-term cardiac function preservation in women treated for breast cancer.

Disclosure: Authors do not report any disclosures

Authors' contribution:

Conceptualization: Zuzanna Kruszyńska

Methodology: Krzysztof Bogdański, Martyna Lazar

Investigation: Bogumił Libura, Anastasiya Aleshchyk

Sources: Martyna Lazar, Michał Adamczak

Formal Analysis: Łukasz Szkaradowski, Julia Lewańska

Visualization: Szymon Baranowski

Supervision: Marcel Majewski, Anastasiya Aleshchyk

Validation: Julia Lewańska, Szymon Baranowski

Writing- Original Draft: Zuzanna Kruszyńska

Writing- Review & Editing: Krzysztof Bogdański, Michał Adamczak

Project administration: Marcel Majewski, Bogumił Libura

All authors have read and agreed with published version of the manuscript.

Funding statement: No external funding was received to perform this review.

Conflict of interest: The authors declare no conflict of interest.

Statement of institutional review board: Not applicable.

Statement of informed consent: Not applicable.

Data availability: Not applicable.

Acknowledgments: Not applicable

References:

1. Sung, H., Ferlay, J., Siegel, R. L., Laversanne, M., Soerjomataram, I., Jemal, A., & Bray, F. (2021). Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA: A Cancer Journal for Clinicians, 71(3):209–249. https://doi.org/10.3322/caac.21660

2. Yang, M., Yang, Q., Tse, G., Ma, Y., Lyu, Q. Y., Wang, W., Lai, A. Y., Zhang, Y., Wong, C. W. M., & Liu, X. (2026). Effectiveness of self-management interventions in reducing cancer treatment-related cardiotoxicity in breast cancer survivors: A systematic review. Asia-Pacific Journal of Oncology Nursing, 13(1),100931. https://doi.org/10.1016/j.apjon.2026.100931

3. Guan, T., Zhang, H., Yang, J., Lin, W., Wang, K., & Su, M. (2021). Increased Risk of Cardiovascular Death in Breast Cancer Patients Without Chemotherapy or (and) Radiotherapy: A Large Population-Based Study. Frontiers in Oncology, 10:619622. https://doi.org/10.3389/fonc.2020.619622

4. Varghese, S. S., Johnston, W. J., Eekhoudt, C. R., Keats, M. R., Jassal, D. S., & Grandy, S. A. (2021). Exercise to Reduce Anthracycline-Mediated Cardiovascular Complications in Breast Cancer Survivors. Current Oncology, 28(5):4139–4156. https://doi.org/10.3390/curroncol28050351

5. He, J., Wang, S., Liu, H., Duan, C., Zhang, H., Wen, F., & Zhang, C. (2023). Competing risk analysis of cardiovascular death in breast cancer: evidence from the SEER database. Translational Cancer Research, 12(12):3591–3603. https://doi.org/10.21037/tcr-23-1163

6. Migliari, M., Fazzini, L., Campana, N., Deidda, M., Dessì, M., & Cadeddu Dessalvi, C. (2025). Current strategies for prevention of cancer therapy-related cardiotoxicity: pharmacological, non-pharmacological and emerging approaches. Frontiers in Cardiovascular Medicine, 12:1668308. https://doi.org/10.3389/fcvm.2025.1668308

7. Cai, S., Yu, S., Lin, X., Zhou, Q., Zheng, X., Chen, H., Ma, T., Chen, X., Sun, H., & Shi, Y. (2026). Adjuvant pegylated liposomal doxorubicin versus epirubicin sequential paclitaxel in triple-negative breast cancer: comparable efficacy with a distinct safety profile. Frontiers in Immunology, 17:1690888. https://doi.org/10.3389/fimmu.2026.1690888

8. Jensen, B. V. (2006). Cardiotoxic consequences of anthracycline-containing therapy in patients with breast cancer. Seminars in Oncology, 33(3 Suppl 8):S15–S21. https://doi.org/10.1053/j.seminoncol.2006.04.022

9. Cardinale, D., Iacopo, F., & Cipolla, C. M. (2020). Cardiotoxicity of Anthracyclines. Frontiers in Cardiovascular Medicine, 7:26. https://doi.org/10.3389/fcvm.2020.00026
10. Vejpongsa, P., & Yeh, E. T. H. (2014). Prevention of Anthracycline-Induced Cardiotoxicity. Journal of the American College of Cardiology, 64(9):938–945. https://doi.org/10.1016/j.jacc.2014.06.1167
11. Vuong, J. T., Stein-Merlob, A. F., Cheng, R. K., & Yang, E. H. (2022). Novel Therapeutics for Anthracycline Induced Cardiotoxicity. Frontiers in Cardiovascular Medicine, 9:872740. https://doi.org/10.3389/fcvm.2022.872740
12. Wang, Q., Zhang, W., Chen, J., Stenson, K. T., Parsai, S., Parthasarathy, P. B., Wang, L., & Zheng, Z. (2026). Novel Mechanism of and Therapeutic Approach for Anthracycline-Induced Cardiotoxicity. Cancer Research Communications, 6(6):1261–1277. https://doi.org/10.1158/2767-9764.CRC-25-0511
13. Cobb, M. S., Tao, S., Shortt, K., Girgis, M., Hauptman, J., Schriewer, J., Chin, Z., Dorfman, E., Campbell, K., Heruth, D. P., Shohet, R. V., & Dawn, B. (2022). Smad3 promotes adverse cardiovascular remodeling and dysfunction in doxorubicin-treated hearts. American Journal of Physiology-Heart and Circulatory Physiology, 323(6):H1091-H1107. https://doi.org/10.1152/ajpheart.00312.2022
14. Dozic, S., Howden, E. J., Bell, J. R., Mellor, K. M., Delbridge, L. M. D., & Weeks, K. L. (2023). Cellular Mechanisms Mediating Exercise-Induced Protection against Cardiotoxic Anthracycline Cancer Therapy. Cells, 12(9):1312. https://doi.org/10.3390/cells12091312
15. Gaytan, S. L., Lawan, A., Chang, J., Nurunnabi, M., Bajpeyi, S., Boyle, J. B., & Sanchez, C. A. (2023). The beneficial role of exercise in preventing doxorubicin-induced cardiotoxicity. Frontiers in Cardiovascular Medicine,14:1133423 . https://doi.org/10.3389/fphys.2023.1133423
16. Montalvo, R. N., Boeno, F. P., Dowllah, I. M., Moritz, C. E. J., Nguyen, B. L., Doerr, V., & Powers, S. K. (2023). Exercise and Doxorubicin Modify Markers of Iron Overload and Cardiolipin Deficiency in Cardiac Mitochondria. International Journal of Molecular Sciences, 24(9):7689. https://doi.org/10.3390/ijms24097689
17. Christidi, E., & Brunham, L. R. (2021). Regulated cell death pathways in doxorubicin-induced cardiotoxicity. Cell Death & Disease, 12(4):339. https://doi.org/10.1038/s41419-021-03614-x

18. Liu, J., Zheng, H., Tang, M., Ryu, Y. C., & Wang, X. (2008). A therapeutic dose of doxorubicin activates ubiquitin-proteasome system-mediated proteolysis by acting on both the ubiquitination apparatus and proteasome. American Journal of Physiology-Heart and Circulatory Physiology, 295(6):H2541–H2550. https://doi.org/10.1152/ajpheart.01052.2008
19. Ascensão, A., Magalhães, J., Soares, J. M. C., Ferreira, R., Neuparth, M. J., Marques, F., Oliveira, P. J., & Duarte, J. A. (2005). Moderate endurance training prevents doxorubicin-induced in vivo mitochondriopathy and reduces the development of cardiac apoptosis. American Journal of Physiology-Heart and Circulatory Physiology, 289(2):H722–H731. https://doi.org/10.1152/ajpheart.01249.2004
20. Kavazis, A. N., Smuder, A. J., & Powers, S. K. (2014). Effects of short-term endurance exercise training on acute doxorubicin-induced FoxO transcription in cardiac and skeletal muscle. Journal of Applied Physiology, 117(3):223–230. https://doi.org/10.1152/japplphysiol.00210.2014
21. Dao, V., Amin, S., Kammaripalle, T., & Manning, K. (2026). Review on cardioprotective strategies in the setting of chemotherapy-induced cardiotoxicity. Cardio-Oncology, 12(1):63. https://doi.org/10.1186/s40959-026-00480-4
22. Uurasmaa, T. M., Bourdin, P., Nammas, W., Latifi, S., Liljenbäck, H., Saraste, A., & Knuuti, J. (2025). Exercise training partly ameliorates cardiac dysfunction in mice during doxorubicin treatment of breast cancer. Journal of Translational Medicine, 23(1):89. https://doi.org/10.1186/s12967-025-06108-y
23. Kang, D.-W., Wilson, R. L., Christopher, C. N., Normann, A. J., Barnes, O., Lesansee, J. D., & Jones, L. W. (2022). Exercise Cardio-Oncology: Exercise as a Potential Therapeutic Modality in the Management of Anthracycline-Induced Cardiotoxicity. Frontiers in Cardiovascular Medicine, 8:805735. https://doi.org/10.3389/fcvm.2021.805735
24. Haykowsky, M. J., Weeldreyer, N. R., La Gerche, A., Sarma, S., & Foulkes, S. J. (2026). Impaired peripheral oxygen diffusion is the primary determinant of exercise intolerance in high-risk older breast cancer survivors. American Journal of Physiology-Heart and Circulatory Physiology, 331(1):H318–H326. https://doi.org/10.1152/ajpheart.00324.2026
25. Ligibel, J. A., Bohlke, K., May, A. M., Clinton, S. K., Demark-Wahnefried, W., Gilchrist, S. C., & Wollins, D. S. (2022). Exercise, Diet, and Weight Management During Cancer Treatment: ASCO Guideline. Journal of Clinical Oncology, 40(22):2491–2507. https://doi.org/10.1200/JCO.22.00687

26. Rock, C. L., Thomson, C., Gansler, T., Gapstur, S. M., McCullough, M. L., Patel, A. V., & Bandera, E. V. (2020). American Cancer Society guideline for diet and physical activity for cancer prevention. CA: A Cancer Journal for Clinicians, 70(4):245–271. https://doi.org/10.3322/caac.21591

27. Ghignatti, P. V. C., Marschner, R. A., Ribeiro, R. T., Gayger-Dias, V., Da Silva, V. F., Teixeira, L. V., & Schaan, B. D. (2026). Preventive Aerobic Training Protects Against Doxorubicin-Induced Cardiotoxicity by Preserving Redox Status and Attenuating Cardiac Stress-Related Signaling. Cells, 15(5):408. https://doi.org/10.3390/cells15050408

28. Adams, S. C., Rivera-Theurel, F., Scott, J. M., Nadler, M. B., Foulkes, S., Leong, D., & ICOS-CORE Working Group. (2025). Cardio-oncology rehabilitation and exercise: evidence, priorities, and research standards from the ICOS-CORE working group. European Heart Journal, 46(29):2847–2865. https://doi.org/10.1093/eurheartj/ehaf100