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The role of physical activity in the prevention of colorectal cancer - a review.

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Abstract

Background: Colorectal cancer (CRC) is the second leading cause of cancer mortality. Modifiable factors, primarily physical inactivity, account for 70% of cases. Physical activity (PA) reduces CRC risk by 20–25%, yet precise molecular pathways require synthesis to refine prevention.

Aim: To synthesize biological mechanisms of PA in CRC mitigation and evaluate optimal intensity, dose, and sedentary behavior impact for targeted recommendations.

Materials and Methods: A literature review (PubMed, Scopus; 2008–2024) was conducted, integrating meta-analyses and clinical guidelines on the insulin/IGF-1 axis, myokine signaling, and gut microbiota metagenomics.

Results: Four pathways were identified: 1) Metabolic: modulation of insulin/IGF-1 axis and sensitivity, 2) Immunological: reduced visceral adiposity and inflammation via myokines and NK-cell surveillance, 3) Mechanical: accelerated transit and improved bile acid metabolism, 4) Microbial: increased butyrate-producing bacteria and mucosal integrity; Protection follows a dose-response relationship, peaking at >300 min/week, especially combining aerobic and resistance training. Interrupting sedentary behavior is a critical independent factor for colonic niche health.

Conclusions: PA acts as a systemic biological intervention that inhibits the initiation of neoplastic transformation. Maximum risk reduction requires multimodal training (aerobic and

resistance) and the mitigation of prolonged sedentary behavior. "Precision Exercise Oncology" should prioritize individualized prescriptions based on molecular and genetic profiles.

Keywords: colorectal cancer, physical activity, insulin, IGF-1, gut microbiome, exercise oncology, sedentary behavior.

1. Introduction

Colorectal cancer (CRC) remains a significant global health burden, ranking as the third most commonly diagnosed malignancy and the second leading cause of cancer-related mortality worldwide [1]. Despite the implementation of advanced screening and development of treatment strategies, the incidence of CRC is projected to increase significantly by 2040 [2]. While genetic predisposition plays a role in disease etiology, it is estimated that approximately 50% to 70% of CRC cases are associated with modifiable lifestyle factors, including dietary habits, obesity, and physical inactivity [3].

Physical activity (PA) is defined by the World Health Organization (WHO) as any bodily movement produced by skeletal muscles that requires energy expenditure [4]. PA is recognized not merely as a supportive intervention but as a primary preventive strategy capable of modulating the systemic and local colonic environment. Previous epidemiological meta-analyses have consistently demonstrated a 20% to 25% reduction in CRC risk among the most active individuals compared to those with sedentary lifestyles [5]. Insufficient PA affects 27.5% of the global adult population [6], and in developed countries may be responsible for up to 5% of CRC [7]. However, the shift from observations to an understanding of how exercise influences colonic carcinogenesis is crucial for developing targeted preventive recommendations.

2. Molecular and biological landscape of colorectal cancer prevention

The protective effect of PA against colorectal carcinogenesis is not attributed to a single physiological change but rather to a complex synergy of systemic and localized biological alterations. These mechanisms can be broadly categorized into metabolic regulation, immune system enhancement, mechanical effects on the gut, and the modulation of the intestinal microbiota. By influencing these pathways, regular exercise creates a physiological environment that is inherently hostile to the initiation and progression of neoplastic cells [8].

2.1. Metabolic and endocrine regulation: the insulin and IGF-1 Axis

The most well-established mechanism linking PA to CRC prevention is the modulation of metabolic health. Hyperinsulinemia and elevated levels of insulin-like growth factor 1 (IGF-1) are potent mitogens that promote cellular proliferation and inhibit apoptosis in colonic epithelial cells [9]. Regular PA increases the expression of glucose transporter type 4 (GLUT4) in skeletal muscles, enhancing systemic insulin sensitivity and lowering circulating insulin levels. Furthermore, exercise increases the concentration of IGF-binding protein 3 (IGFBP-3), which sequesters free IGF-1, thereby limiting its bioavailability and binding to the IGF-1 receptor (IGF-1R) on pre-neoplastic cells [10]. PA also reduces the risk of insulin resistance by reducing ectopic fat, which is an independent etiopathological factor. Lower volume of visceral fat also positively impacts the health of the liver and pancreas – crucial elements of glucose metabolism [11,12].

2.2. Systemic inflammation and immune surveillance

Chronic low-grade inflammation is a hallmark of colorectal carcinogenesis. Adipose tissue, particularly visceral fat, acts as an endocrine organ secreting pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha [13]. This inflammatory state facilitates the dysplastic transformation of intestinal tissue through several pathways, including the production of reactive oxygen species (ROS) by activated macrophages. These molecules induce oxidative DNA damage and genomic instability, frequently leading to mutations in key tumor suppressor genes such as Tp53 [14,15]. Such mutations are highly prevalent in inflammation-associated colorectal malignancies, occurring in approximately 63% of cases linked to inflammatory bowel disease [16]. PA effectively reduces visceral adiposity and induces an "anti-inflammatory environment" by stimulating the release of myokines (e.g., IL-6 released acutely from muscles acts differently than chronic systemic IL-6) and increasing anti-inflammatory cytokines like IL-10. Notably, exercise has been shown to reduce visceral fat mass in a dose-response manner, demonstrating a superior effect compared to caloric restriction alone [17,18]. Beyond metabolic changes, acute exercise at moderate-to-vigorous intensity inhibits the production of TNF-alpha and promotes the polarization of macrophages toward the anti-inflammatory M2 phenotype [19,20]. Such interventions are crucial to counteract the hyperactivation of the IL-6/JAK/STAT3 signaling pathway, which otherwise supports the survival and progression of premalignant adenoma polyps—the

precursors for over 95% of colorectal cancers [21,22]. Additionally, acute exercise triggers the immediate mobilization of natural killer (NK) cells and cytotoxic T-lymphocytes, which enhances the body's innate ability to identify and eliminate early-stage malignant cells [23]. This is particularly important as chronic inflammation can lead to the persistent activation of STAT3 in regulatory T cells, which facilitates immune evasion by suppressing CD8+ T cell cytotoxicity [24]. Therefore, a high frequency of PA may be essential to consistently induce an anti-inflammatory landscape and disrupt the formation of pro-tumor microenvironment characterized by the accumulation of stromal components and pathological vascularization [14,25,26].

2.3. Gastrointestinal transit time and bile acid metabolism

PA stimulates gut motility through increased vagal tone and the release of gastrointestinal hormones. This reduction in gastrointestinal transit time limits the duration of contact between the colonic mucosa and dietary carcinogens [27]. Recent literature emphasizes that aerobic exercise significantly accelerates colonic transit, particularly in the ascending colon, which reduces the "exposure window" to fecal carcinogens [28]. This accelerated passage is crucial in preventing the accumulation of DNA-damaging metabolites in the colonic landscape.

Beyond transit, PA profoundly influences bile acid metabolism. Studies indicate that regular exercise alters the bile acid pool by decreasing the conversion of primary bile acids into secondary bile acids, such as deoxycholic acid (DCA) [29]. Elevated DCA levels are known to disrupt the intestinal barrier and promote a pro-tumorigenic niche by inducing oxidative stress and DNA damage. By maintaining a lower concentration of these secondary metabolites, PA preserves the mucosal biological framework and minimizes neoplastic risk within the epithelium [30].

2.4. Emerging role of the gut microbiome

Recent advances in metagenomics suggest that exercise independently modifies the composition and diversity of the gut microbiota, promoting a more resilient "anti-inflammatory landscape." PA is associated with an increased amount of butyrate-producing bacteria, such as *Faecalibacterium prausnitzii* and *Roseburia* [31]. Current longitudinal studies indicate that regular exercise increases the concentration of short-chain fatty acids (SCFAs), particularly butyrate, regardless of dietary fiber intake [32]. Butyrate serves as the primary energy source for colonocytes and plays a crucial role in maintaining mucosal

integrity and inducing apoptosis in CRC cells. It acts as a potent histone deacetylase (HDAC) inhibitor, which leads to the epigenetic down regulation of pro-proliferative genes and the activation of tumor suppressor pathways [33]. Furthermore, exercise-induced microbial shifts contribute to a protective biological framework by reducing the prevalence of pro-carcinogenic species (e.g *Fusobacterium nucleatum*), thereby limiting chronic inflammation within the colonic niche [34].

Table 1. Summary of biological mechanisms linking physical activity to CRC risk reduction.

Pathway category:	**Biological mechanism:**	**Clinical/oncological impact:**
Metabolic	Insulin/IGF-1 axis modulation	Inhibition of cell proliferation, enhanced apoptosis
Immunological	Myokine release & NK-cell mobilization	Reduced systemic inflammation, enhanced immunosurveillance
Mechanical	Accelerated gastrointestinal transit	Decreased mucosal exposure to fecal carcinogens
Microbial	Increased butyrate production	Improved mucosal integrity, HDAC inhibition
Adiposity	Reduction of visceral fat mass	Mitigation of pro-inflammatory cytokine secretion

3. Characteristics of physical activity: intensity, dose, and sedentary behavior

3.1. The dose-response relationship and global standards

The relationship between PA and CRC risk reduction is characterized by a significant dose-response effect, where higher volumes of activity correlate with stronger protective benefits. According to the WHO, the foundational dose for cancer prevention involves 150-300 minutes of moderate-intensity aerobic PA or 75-150 minutes of vigorous-intensity PA per week [4]. Current evidence highlights that the risk reduction curve is steepest when moving from total inactivity to even modest levels of movement, yet the most profound oncological protection reaching up to 20–24% is observed in cohorts achieving 45-75 metabolic

equivalent task (MET) hours per week [5]. Unlike other health outcomes, CRC prevention appears to exhibit a continuous linear benefit, there is no clearly defined upper plateau where additional activity ceases to offer further protection, emphasizing that for high-risk populations, exceeding the minimum WHO thresholds is clinically advisable [35].

3.2. Impact of exercise intensity: aerobic vs. resistance training

The qualitative aspect of exercise its intensity and type plays a crucial role in activating distinct anti-carcinogenic pathways.

3.2.1. Aerobic exercise

Moderate-to-vigorous PA remains the gold standard in oncological prevention. High-intensity aerobic training (e.g., HIIT) induces rapid systemic physiological shifts, including increased shear stress on vascular walls and the immediate mobilization of cytotoxic NK cells and CD8+ T-lymphocytes. These immune "scouts" are essential for the immunosurveillance of occult malignant cells [36]. Furthermore, vigorous intensity is more effective at reducing systemic levels of bioavailable insulin and C-peptide, thereby neutralizing the pro-proliferative environment of the colonic landscape.

3.2.2. Resistance training

Muscle-strengthening activities provide a complementary biological framework for cancer prevention. Beyond simple calorie burning, resistance training stimulates the release of exercise-induced myokines from skeletal muscle, which have been shown to inhibit colorectal cell proliferation and promote apoptosis. Additionally, by enhancing lean body mass, resistance training improves long-term glucose homeostasis and mitigates the hyperinsulinemia-driven pathways that stimulate adenoma growth [37].

3.3. Prolonged sedentary behavior as an independent risk factor

A critical paradigm shift in exercise oncology is the recognition of sedentary behavior as a distinct risk factor, independent of purposeful exercise. This is often referred to as the "active couch potato" phenomenon, where meeting weekly exercise goals may not fully offset the deleterious effects of 8–10 hours of daily sitting. Prolonged metabolic stasis triggers a pro-inflammatory state characterized by elevated C-reactive protein (CRP) and impaired lipid metabolism [38]. Recent longitudinal studies demonstrate that micro-bouts of light activity (e.g., 2 minutes of walking every 30 minutes) can disrupt this stasis, significantly lowering postprandial glucose levels and restoring a more favorable anti-inflammatory state. Thus, a

dual-action strategy maximizing intentional exercise while minimizing total sitting time is essential for optimizing the colonic niche against neoplastic transformation [39].

4. Discussion and future perspectives

Despite the robust epidemiological evidence supporting the role of PA in colorectal cancer prevention, several challenges remain in clinical translation. A significant area of debate is the "Precision Exercise Oncology" approach. While population-level guidelines are effective, individual responses to exercise vary based on genetic polymorphisms and the molecular subtype of the tumor, such as MSI-high versus MSS status. Recent synthesis of the evidence suggests that the "optimal" prevention strategy requires a shift from generic advice toward individualized "exercise prescriptions" that account for the intensity-dose synergy [40,41].

4.1. Barriers to implementation and the optimal dose requirements

A major limitation identified in recent literature is the low adherence to the recommended PA volumes. Socioeconomic factors, urban infrastructure, and the lack of standardized protocols in primary care settings contribute to persistent sedentary lifestyles. According to the latest reports, achieving the maximal protective effect often requires volumes at the upper limit of global standards (e.g., 300 minutes of moderate-intensity PA per week). However, as highlighted by Orange et al., higher-intensity exercise may offer a more time-efficient "shortcut" to activating systemic anti-carcinogenic pathways, potentially improving adherence among populations with time constraints [41]. Future public health strategies must focus on behavioral interventions that facilitate long-term maintenance of these optimized doses.

4.2. Future research directions: multimodal and molecular insights

The interaction between exercise and the gut-immune axis may be one of the priorities. Studies should focus on identifying the metabolomic signature of exercise - specifically the balance of short-chain fatty acids (SCFAs) and secondary bile acids to develop novel biomarkers for CRC risk assessment [42]. Combining PA with specific dietary patterns, such as the Mediterranean diet, could further enhance the stability of a protective colonic niche [28]. Prospective research should prioritize randomized controlled trials using objective measures of PA, such as accelerometry, to eliminate recall bias. Furthermore, investigating the synergy between aerobic and resistance training modalities emerges as a crucial direction for future investigation [41].

5. Conclusions

Current research confirms that PA is a vital toolaspe in preventing CRC, working through various biological pathways that go beyond simple weight management. Although higher levels of activity provide the strongest protection, combining different types of exercise such as aerobic and strength training may appear a key to improving immune function and reducing systemic inflammation. Future strategies should focus on personalized exercise plans and reducing daily sitting time to better protect the colonic environment. Ultimately, framing PA not only as a lifestyle choice but as a targeted biological intervention may be imperative to mitigate the rising global burden of CRC.

Disclosure

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References:

1. Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin. 2021;71(3):209-249. doi:10.3322/caac.21660

2. Xi Y, Xu P. Global colorectal cancer burden in 2020 and projections to 2040. Transl Oncol. 2021;14(10):101174. doi:10.1016/j.tranon.2021.101174

3. Huxley RR, Ansary-Moghaddam A, Clifton P, Czernichow S, Parr CL, Woodward M. The impact of dietary and lifestyle risk factors on risk of colorectal cancer: a quantitative overview of the epidemiological evidence. Int J Cancer. 2009;125(1):171-180. doi:10.1002/ijc.24343

4. Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behavior. Br J Sports Med. 2020;54(24):1451-1462. doi:10.1136/bjsports-2020-102955

5. Kyu HH, Bachman VF, Alexander LT, et al. Physical activity and risk of breast cancer, colon cancer, diabetes, ischemic heart disease, and ischemic stroke events: systematic review

and dose-response meta-analysis for the Global Burden of Disease Study 2013. BMJ. 2016;354:i3857. Published 2016 Aug 9. doi:10.1136/bmj.i3857

6. Brown KF, Rumgay H, Dunlop C, et al. The fraction of cancer attributable to modifiable risk factors in England, Wales, Scotland, Northern Ireland, and the United Kingdom in 2015. Br J Cancer. 2018;118(8):1130-1141. doi:10.1038/s41416-018-0029-6

7. Guthold R, Stevens GA, Riley LM, Bull FC. Worldwide trends in insufficient physical activity from 2001 to 2016: a pooled analysis of 358 population-based surveys with 1·9 million participants. Lancet Glob Health. 2018;6(10):e1077-e1086. doi:10.1016/S2214-109X(18)30357-7

8. McTiernan A. Mechanisms linking physical activity with cancer. Nat Rev Cancer. 2008;8(3):205-211. doi:10.1038/nrc2325

9. Pollak M. Insulin and insulin-like growth factor signalling in neoplasia. Nat Rev Cancer. 2008;8(12):915-928. doi:10.1038/nrc2536

10. Friedenreich CM, Neilson HK, Farris MS, Courneya KS. Physical Activity and Cancer Outcomes: A Precision Medicine Approach. Clin Cancer Res. 2016;22(19):4766-4775. doi:10.1158/1078-0432.CCR-16-0067

11. Schenk S, Saberi M, Olefsky JM. Insulin sensitivity: modulation by nutrients and inflammation. J Clin Invest. 2008;118(9):2992-3002. doi:10.1172/JCI34260

12. Hens W, Taeyman J, Cornelis J, Gielen J, Van Gaal L, Vissers D. The Effect of Lifestyle Interventions on Excess Ectopic Fat Deposition Measured by Noninvasive Techniques in Overweight and Obese Adults: A Systematic Review and Meta-Analysis. J Phys Act Health. 2016;13(6):671-694. doi:10.1123/jpah.2015-0560

13. Nimmo MA, Leggate M, Viana JL, King JA. The effect of physical activity on mediators of inflammation. Diabetes Obes Metab. 2013;15 Suppl 3:51-60. doi:10.1111/dom.12156

14. Grivennikov SI, Greten FR, Karin M. Immunity, inflammation, and cancer. Cell. 2010;140(6):883-899. doi:10.1016/j.cell.2010.01.025

15. Canli Ö, Nicolas AM, Gupta J, et al. Myeloid Cell-Derived Reactive Oxygen Species Induce Epithelial Mutagenesis. Cancer Cell. 2017;32(6):869-883.e5. doi:10.1016/j.ccell.2017.11.004

16. Robles AI, Traverso G, Zhang M, et al. Whole-Exome Sequencing Analyses of Inflammatory Bowel Disease-Associated Colorectal Cancers. Gastroenterology. 2016;150(4):931-943. doi:10.1053/j.gastro.2015.12.036

17. Coker RH, Williams RH, Kortebein PM, Sullivan DH, Evans WJ. Influence of exercise intensity on abdominal fat and adiponectin in elderly adults. Metab Syndr Relat Disord. 2009;7(4):363-368. doi:10.1089/met.2008.0060

18. Recchia F, Leung CK, Yu AP, et al. Dose-response effects of exercise and caloric restriction on visceral adiposity in overweight and obese adults: a systematic review and meta-analysis of randomised controlled trials. Br J Sports Med. 2023;57(16):1035-1041. doi:10.1136/bjsports-2022-106304

19. Gleeson M, Bishop NC, Stensel DJ, Lindley MR, Mastana SS, Nimmo MA. The anti-inflammatory effects of exercise: mechanisms and implications for the prevention and treatment of disease. Nat Rev Immunol. 2011;11(9):607-615. Published 2011 Aug 5. doi:10.1038/nri3041

20. Cullen T, Thomas AW, Webb R, Hughes MG. Interleukin-6 and associated cytokine responses to an acute bout of high-intensity interval exercise: the effect of exercise intensity and volume. Appl Physiol Nutr Metab. 2016;41(8):803-808. doi:10.1139/apnm-2015-0640

21. Grivennikov S, Karin E, Terzic J, et al. IL-6 and Stat3 are required for survival of intestinal epithelial cells and development of colitis-associated cancer. Cancer Cell. 2009;15(2):103-113. doi:10.1016/j.ccr.2009.01.001

22. Aarons CB, Shanmugan S, Bleier JI. Management of malignant colon polyps: current status and controversies. World J Gastroenterol. 2014;20(43):16178-16183. doi:10.3748/wjg.v20.i43.16178

23. Idorn M, Hojman P. Exercise-Dependent Regulation of NK Cells in Cancer Protection. Trends Mol Med. 2016;22(7):565-577. doi:10.1016/j.molmed.2016.05.007

24. Yu H, Pardoll D, Jove R. STATs in cancer inflammation and immunity: a leading role for STAT3. Nat Rev Cancer. 2009;9(11):798-809. doi:10.1038/nrc2734

25. Erez N, Truitt M, Olson P, Arron ST, Hanahan D. Cancer-Associated Fibroblasts Are Activated in Incipient Neoplasia to Orchestrate Tumor-Promoting Inflammation in an NF-kappaB-Dependent Manner. Cancer Cell. 2010;17(2):135-147. doi:10.1016/j.ccr.2009.12.041

26. Petersen AM, Pedersen BK. The anti-inflammatory effect of exercise. J Appl Physiol (1985). 2005;98(4):1154-1162. doi:10.1152/japplphysiol.00164.2004

27. Oettlé GJ. Effect of moderate exercise on bowel habit. Gut. 1991;32(8):941-944. doi:10.1136/gut.32.8.941

28. Song M, Chan AT. Environmental Factors, Gut Microbiota, and Colorectal Cancer Prevention. Clin Gastroenterol Hepatol. 2019;17(2):275-289. doi:10.1016/j.cgh.2018.07.012

29. Mailing LJ, Allen JM, Buford TW, Fields CJ, Woods JA. Exercise and the Gut Microbiome: A Review of the Evidence, Potential Mechanisms, and Implications for Human Health. Exerc Sport Sci Rev. 2019;47(2):75-85. doi:10.1249/JES.0000000000000183

30. O'Keefe SJ. Diet, microorganisms and their metabolites, and colon cancer. Nat Rev Gastroenterol Hepatol. 2016;13(12):691-706. doi:10.1038/nrgastro.2016.165

31. Ortiz-Alvarez L, Xu H, Martinez-Tellez B. Influence of Exercise on the Human Gut Microbiota of Healthy Adults: A Systematic Review. Clin Transl Gastroenterol. 2020;11(2):e00126. doi:10.14309/ctg.0000000000000126

32. Motiani KK, Collado MC, Eskelinen JJ, et al. Exercise Training Modulates Gut Microbiota Profile and Improves Endotoxemia. Med Sci Sports Exerc. 2020;52(1):94-104. doi:10.1249/MSS.0000000000002112

33. Liu G, Tang J, Zhou J, Dong M. Short-chain fatty acids play a positive role in colorectal cancer. Discov Oncol. 2024;15(1):425. Published 2024 Sep 10. doi:10.1007/s12672-024-01313-5

34. Wegierska AE, Charitos IA, Topi S, Potenza MA, Montagnani M, Santacroce L. The Connection Between Physical Exercise and Gut Microbiota: Implications for Competitive Sports Athletes. Sports Med. 2022;52(10):2355-2369. doi:10.1007/s40279-022-01696-x

35. McTiernan A, Friedenreich CM, Katzmarzyk PT, et al. Physical Activity in Cancer Prevention and Survival: A Systematic Review. Med Sci Sports Exerc. 2019;51(6):1252-1261. doi:10.1249/MSS.0000000000001937

36. Patel AV, Friedenreich CM, Moore SC, et al. American College of Sports Medicine Roundtable Report on Physical Activity, Sedentary Behavior, and Cancer Prevention and Control. Med Sci Sports Exerc. 2019;51(11):2391-2402. doi:10.1249/MSS.0000000000002117

37. Momma H, Kawakami R, Honda T, Sawada SS. Muscle-strengthening activities are associated with lower risk and mortality in major non-communicable diseases: a systematic review and meta-analysis of cohort studies. Br J Sports Med. 2022;56(13):755-763. doi:10.1136/bjsports-2021-105061

38. Biswas A, Oh PI, Faulkner GE, et al. Sedentary time and its association with risk for disease incidence, mortality, and hospitalization in adults: a systematic review and meta-analysis. Ann Intern Med. 2015;162(2):123-132. doi:10.7326/M14-1651

39. Dempsey PC, Blankenship JM, Larsen RN, et al. Interrupting prolonged sitting in type 2 diabetes: nocturnal persistence of improved glycaemic control. Diabetologia. 2017;60(3):499-507. doi:10.1007/s00125-016-4169-z

40. Papadimitriou N, Dimou N, Tsilidis KK, et al. Physical activity and risks of breast and colorectal cancer: a Mendelian randomisation analysis. Nat Commun. 2020;11(1):597. Published 2020 Jan 30. doi:10.1038/s41467-020-14389-8

41. Orange ST. What is the optimal type and dose of physical activity for colorectal cancer prevention?. Best Pract Res Clin Gastroenterol. 2023;66:101841. doi:10.1016/j.bpg.2023.101841

42. Fiuza-Luces C, Valenzuela PL, Castillo-García A, Lucia A. Exercise Benefits Meet Cancer Immunosurveillance: Implications for Immunotherapy. Trends Cancer. 2021;7(2):91-93. doi:10.1016/j.trecan.2020.12.003