International Journal of General Medicine

Dovepress
Taylor & Francis Group

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REVIEW

Fasting and Fasting-Mimicking Diets as Adjunctive Strategies in Cancer Therapy: Mechanisms, Evidence, and Clinical Implications

Sahibzada Salman Wali

Rehman Medical College, Rehman Medical Institute, Peshawar, Pakistan

Correspondence: Sahibzada Salman Wali, Rehman Medical College, Peshawar, Pakistan, Email salmanatrmc@gmail.com; salmannext1234@gmail.com

Background: Cancer remains a leading cause of global morbidity and mortality. Although conventional therapies such as chemotherapy, radiotherapy, and immunotherapy improve survival, their effectiveness is often limited by resistance and toxicity. Fasting and fasting-mimicking diets (FMDs) have emerged as potential adjunctive interventions due to their effects on cellular metabolism, stress responses, and tumor-host interactions.

Aim: To critically evaluate the biological rationale, molecular mechanisms, and clinical evidence supporting fasting and FMDs as adjuncts in cancer therapy.

Methods: A comprehensive literature review was conducted using PubMed, PubMed Central, and Google Scholar to identify relevant preclinical and clinical studies on fasting, caloric restriction, and FMDs in cancer.

Results: Preclinical studies demonstrate that fasting induces metabolic and oxidative stress, selectively sensitizing cancer cells while protecting normal cells. Early-phase clinical trials suggest that fasting and FMDs are safe, feasible, and capable of influencing metabolism and immune responses, with potential improvements in treatment tolerance and efficacy. However, these studies are limited by small sample sizes and heterogeneous designs.

Conclusion: Fasting and FMDs represent promising adjunctive strategies in cancer therapy. While mechanistic data and preliminary clinical findings are encouraging, larger, well-designed trials are required to confirm their efficacy, safety, and long-term benefits.

Keywords: fasting, fasting-mimicking diet, cancer therapy, metabolic reprogramming, autophagy, chemotherapy

Introduction

Cancer represents one of the most pressing global health challenges, accounting for millions of deaths each year and imposing substantial morbidity worldwide.[1] In 2020 alone, nearly 10 million individuals succumbed to this disease, representing approximately one in every six deaths globally.[2] Although conventional therapies such as chemotherapy, radiotherapy, and immunotherapy have improved survival outcomes, their efficacy often remains limited, and they are frequently associated with significant toxicity to healthy tissues. This highlights an urgent need for novel, complementary strategies that enhance treatment effectiveness.[3]

Dietary and lifestyle factors have emerged as pivotal determinants of cancer risk, with growing evidence linking specific behaviours to disease onset, progression, and therapeutic response.[4] Obesity is associated with an increased risk of multiple cancer types through mechanisms involving dysfunctional adipocytokine signaling.[5] Similarly, high consumption of ultra-processed foods has been consistently linked to an increased risk of several cancers, including colorectal and breast cancer.[6] Physical inactivity and sedentary behaviour independently elevate cancer risk, with underlying mechanisms involving altered sex hormone levels, reduced insulin sensitivity, and chronic inflammation.[7] Tobacco use remains one of the most potent and well-established carcinogenic risk factors, with smoking cessation offering the greatest potential among modifiable behaviours for reducing cancer-related mortality globally.[8]

https://doi.org/10.2147/IJGM.S604182
Received: 19 February 2026
Accepted: 1 May 2026
Published: 7 May 2026

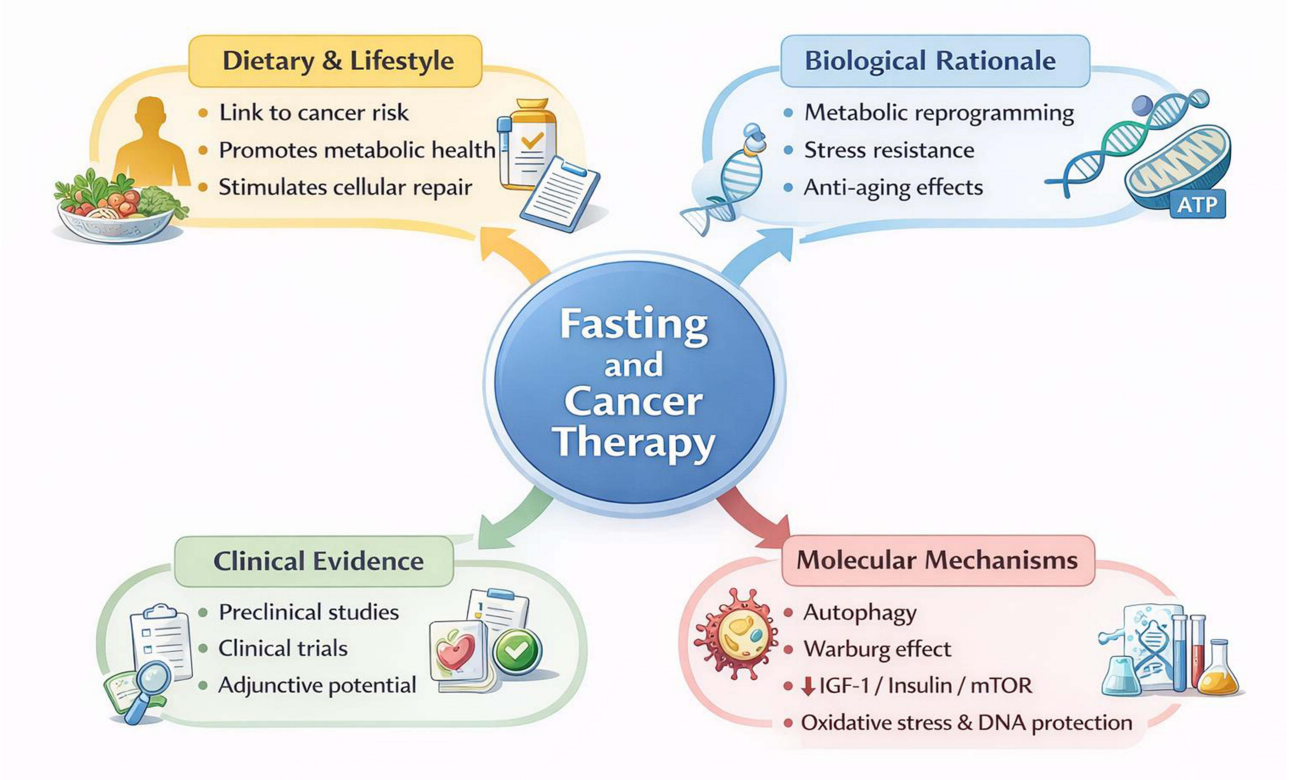

Figure 1 This summarizes how fasting and fasting-mimicking diets support cancer therapy by linking diet and lifestyle to metabolic health, key molecular pathways, and clinical evidence.

Among these, fasting has attracted increasing scientific interest due to its potential to promote overall health and resilience against chronic diseases. Research indicates that fasting can mitigate oxidative stress, suppress inflammation, slow biological aging, and stimulate cellular repair mechanisms, processes that collectively contribute to enhanced longevity and improved metabolic, cardiovascular, and neurological health.[9]

Notably, experimental and clinical findings suggest that fasting may sensitize cancer cells to chemotherapy, enhancing cytotoxicity in malignant tissues while protecting normal cells from treatment induced damage.[10] Consequently, combining fasting or fasting mimicking interventions with conventional chemotherapy may represent a promising adjunctive approach to improve therapeutic efficacy and reduce systemic toxicity (Figure 1).[11]

Methodology

Study Design

This narrative review was conducted to synthesize existing evidence on the biological, clinical, and therapeutic aspects of fasting and fasting-mimicking diets in cancer, with comparative insights from preclinical and clinical studies across different cancer types.

Search Strategy

A comprehensive literature search was performed across major scientific databases, including PubMed, PubMed Central (PMC), and Google Scholar, to identify relevant studies and reports published up to January 2026. The following keywords and Boolean operators were applied: “fasting” OR “intermittent fasting” OR “fasting-mimicking diet” AND “cancer” OR “tumor” AND (“autophagy” OR “metabolic reprogramming” OR “Warburg effect” OR “oxidative stress” OR “DNA protection” OR “clinical trial”).

Study Selection

The initial search yielded a broad pool of articles, which were screened based on title and abstract relevance. Full texts of potentially eligible studies were then assessed for inclusion. A total of 41 articles were ultimately included in the final synthesis based on their relevance to the scope of this review.

Inclusion and Exclusion Criteria

Studies were included if they investigated fasting or fasting-mimicking diets (FMDs) in the context of cancer, including their effects on cancer biology, treatment outcomes, metabolic changes, or therapeutic implications. Only peer-reviewed articles, including narrative or systematic reviews, meta-analyses, clinical trials, case series, and official reports, published in English between 2010 and 2025 were considered. Select landmark studies published prior to 2010 were also included to provide historical context and foundational evidence.

Studies were excluded if they were not peer-reviewed, lacked empirical or analytical content, focused on non-cancer conditions, or were not available in English.

Data Extraction and Synthesis

Data from the selected studies were manually extracted and organized into three main themes: (i) biological rationale of fasting in cancer, (ii) molecular and cellular mechanisms including autophagy, metabolic reprogramming, and oxidative stress/DNA protection, and (iii) clinical evidence and implications. Evidence was narratively synthesized to highlight key patterns, identify research gaps, and inform future research directions. Quantitative meta-analysis was not performed due to heterogeneity in study designs, interventions, and outcome measures.

Ethical Considerations

Since the review relied solely on publicly available literature, no ethical approval was required.

Biological Rationale of Fasting in Cancer

Diet and metabolism play crucial roles in cancer development, with obesity significantly increasing cancer risk. Because cancer cells cannot adapt to nutrient deprivation like normal cells, fasting imposes metabolic and oxidative stress that selectively weakens tumor cells. Thus, fasting may alter the tumor environment and enhance the effectiveness of anticancer therapies (Figure 2).[12] Fasting and fasting-mimicking diets (FMDs) exert their anticancer effects primarily through two interrelated mechanisms: differential stress resistance (DSR) and differential stress sensitization (DSS). DSR refers to the ability of normal cells to enter a protective, low-proliferative state during nutrient scarcity, characterized by reduced growth signaling and enhanced stress resistance pathways. In contrast, cancer cells, due to oncogenic signaling and constitutive proliferative drive, fail to fully activate these protective mechanisms and therefore remain vulnerable under fasting conditions. DSS further complements this effect by increasing the susceptibility of cancer cells to chemotherapy-induced damage, as fasting exacerbates cellular stress in tumor cells while simultaneously reducing damage in normal tissues.[13]

Preclinical and early clinical studies suggest that fasting and FMDs can reduce growth-promoting factors such as IGF-1, activate cellular stress-response pathways, and selectively sensitize cancer cells to chemotherapy. These effects collectively may improve therapeutic outcomes while protecting normal tissues from treatment-induced toxicity.[14]

Molecular and Cellular Mechanisms of Fasting in Cancer

Caloric restriction and intermittent fasting promote health and longevity by enhancing autophagy,[15] counteracting the Warburg effect in cancer cells,[16] and selectively impairing tumor cell survival through metabolic reprogramming and increased oxidative stress.[17]

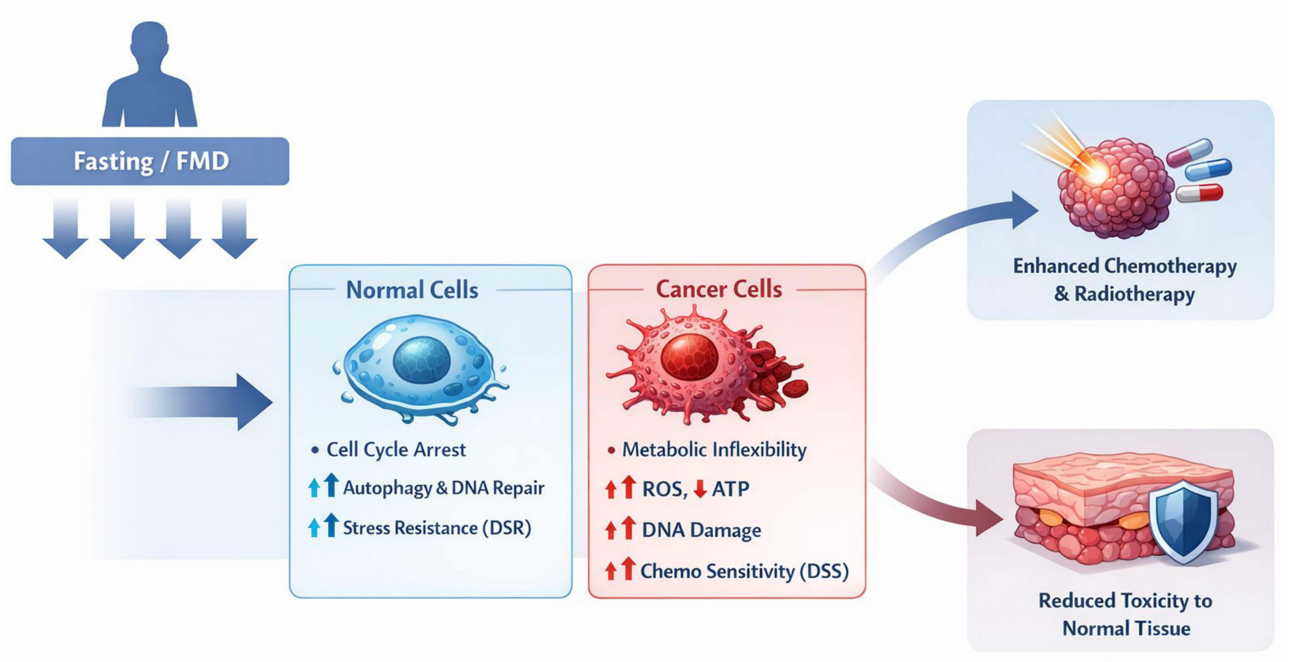

Figure 2 This figure illustrates the contrasting responses of normal and cancer cells to fasting or fasting-mimicking diets in the context of cancer treatment.

Autophagy

Autophagy is a conserved cellular process that degrades and recycles damaged proteins and organelles to maintain cellular homeostasis. While generally protective, excessive or dysregulated autophagy can trigger cell death.[18]

Fasting and fasting-mimicking diets (FMDs) stimulate autophagy, creating metabolic stress that affects normal and cancer cells differently. Normal cells respond by slowing growth and activating adaptive repair pathways, whereas cancer cells, dependent on continuous nutrient supply and proliferative signalling, are less able to cope. This makes tumor cells more susceptible to cell death and enhances their sensitivity to anticancer therapies.[19]

Mechanistically, fasting reduces circulating growth factors and nutrient availability, shifting cellular priorities from growth toward maintenance and repair. Autophagy relies on ATG genes to recycle cellular components, but its regulation differs in cancer cells: oncogenes such as Akt, PI3K, and Bcl-2 suppress autophagy, while tumor suppressor genes like PTEN and DAPK1 promote it. This differential regulation enables fasting-induced autophagy to protect normal cells while leaving cancer cells vulnerable to therapy.[20]

Mechanistically, fasting activates autophagy primarily through the AMPK–mTORC1–ULK1 signalling axis. During nutrient deprivation, falling glucose and amino acid levels cause a rise in the intracellular AMP:ATP ratio, which activates AMP-activated protein kinase (AMPK). AMPK upregulation in turn inhibits mechanistic target of rapamycin complex 1 (mTORC1) — a major suppressor of autophagy — and this inhibition permits activation of the UNC-51-like kinase 1 (ULK1) complex, thereby initiating the autophagy cascade.[21]

Metabolic Reprogramming (Warburg Effect)

Metabolic reprogramming is a hallmark of cancer, enabling tumor cells to sustain rapid proliferation and adapt to environmental stress. A key feature of this reprogramming is the Warburg effect, in which cancer cells preferentially use aerobic glycolysis rather than oxidative phosphorylation, even in the presence of oxygen. Although glycolysis is less efficient in ATP production, its rapid energy generation meets the high proliferative demands of tumor cells. Lactate accumulation from glycolysis also promotes metabolic cooperation within the tumor microenvironment and contributes to immune evasion and tumor progression.[22]

Fasting and fasting-mimicking diets (FMDs) can exploit these metabolic vulnerabilities. By reducing systemic glucose and growth-promoting factors, such as IGF-1, fasting imposes metabolic stress on tumor cells and downregulates key growth pathways, including PI3K/AKT/mTOR. Cancer cells, reliant on continuous nutrient supply, become more susceptible to oxidative stress, DNA damage, and treatment-induced cytotoxicity. In contrast, normal cells adapt by shifting toward fatty acid oxidation and oxidative phosphorylation, preserving their survival. These mechanisms suggest that fasting may enhance the efficacy of chemotherapy and radiotherapy by selectively targeting tumor metabolism.[23]

At the molecular level, the Warburg effect in cancer cells is sustained by oncogenic activation of the PI3K/AKT/mTOR pathway, which drives persistent expression of hypoxia-inducible factor 1-alpha (HIF-1α) even under normoxic conditions. In tumour cells, high AKT and mTOR activity promote HIF-1α expression regardless of oxygen availability, leading to persistent transcription of glycolytic enzymes and continuous lactate production.[24] HIF-1α in turn upregulates key downstream effectors including glucose transporter 1 (GLUT1) — which increases glucose import — and hexokinase 2 (HK2), which catalyses the first committed step of glycolysis, locking cancer cells into a proliferative, glucose-dependent programme they cannot escape under nutrient stress. Fasting and FMD directly disrupt this axis. By reducing systemic glucose availability and inhibiting glycolysis, fasting and FMD suppress the constitutively active PI3K-AKT-mTORC1 cascade that cancer cells depend on for growth and survival, an effect that has been exploited therapeutically by combining FMD with PI3K, AKT, and mTOR inhibitors to enhance antitumour outcomes.[25]

Oxidative Stress and DNA Protection

The term ROS encompasses oxygen free radicals, such as superoxide anion radical ($O_2^{\cdot-}$) and hydroxyl radical ($^{\cdot}OH$), and nonradical oxidants, such as hydrogen peroxide (H_2O_2) and singlet oxygen ($^1O_2$). ROS can be interconverted from one to another (depending on ΔG of relevant processes) by enzymatic and nonenzymatic mechanisms.[26] Reactive oxygen species are generated in cells primarily as by-products of mitochondrial respiration and through the active production of superoxide by NADPH oxidase enzymes, especially in neutrophils and phagosomes.[27]

Fasting and fasting-mimicking diets (FMDs) help regulate oxidative stress by reducing mitochondrial overactivity, lowering ROS and H2O2 production, and enhancing cellular antioxidant defences. These effects are partly mediated by gut microbiota derived metabolites, which support epithelial integrity and limit inflammation.[28] By modulating oxidative stress and activating stress-response pathways, fasting may protect normal cells while rendering cancer cells more vulnerable to therapy-induced damage, thereby improving the efficacy of anticancer treatments in preclinical models.[29]

The key molecular mediator through which fasting modulates oxidative stress in normal versus cancer cells is the KEAP1–NRF2–ARE signalling axis. Under basal conditions, the transcription factor NRF2 (encoded by NFE2L2) is bound to its inhibitor KEAP1 and targeted for proteasomal degradation. Upon oxidative stress, sensor cysteines in KEAP1 are modified by ROS, leading to NRF2 stabilisation, nuclear accumulation, and binding to antioxidant response elements (ARE) to activate transcription of a broad panel of cytoprotective and antioxidant genes.[30] In normal cells, fasting activates this pathway appropriately, upregulating enzymes such as glutathione peroxidase, superoxide dismutase, and haem oxygenase-1, thereby protecting healthy tissue from therapy-induced oxidative injury. Cancer cells, however, frequently harbour gain-of-function mutations in NFE2L2 or loss-of-function mutations in KEAP1 that result in constitutive NRF2 hyperactivation independent of redox status. This aberrant NRF2 activation enhances expression of antioxidant enzymes that mitigate ROS levels and protect cancer cells from oxidative damage, thereby contributing to resistance to platinum-based chemotherapy and other oxidative anticancer agents.[31]

Clinical Evidence and Implications

Fasting-based interventions, including short-term fasting and fasting-mimicking diets, are currently under investigation as adjuncts to standard cancer therapies, with early-phase clinical trials reporting promising biological effects.[32] Early-phase clinical trials suggest that fasting-mimicking diets can reduce circulating IGF-1 levels in adults.[33]

Emerging evidence indicates that fasting and fasting-mimicking diets are safe and feasible in cancer patients. Early clinical studies suggest potential modulation of metabolic and immune biomarkers and possible improvement in patient-reported outcomes, including fatigue and quality of life, although findings remain inconsistent.[34] This Phase 2 randomized trial (BREAKFAST) demonstrated that a severely calorie-restricted, 5-day FMD regimen is safe and produces

excellent outcomes when combined with preoperative chemotherapy in early-stage TNBC patients, with early down-regulation of glycolytic pathways associated with higher pathologic complete response rates.[35]

Limited randomized evidence is encouraging. In the multicentre DIRECT phase 2 trial, patients with HER2-negative breast cancer receiving cyclic FMD alongside neoadjuvant chemotherapy showed improved tumor responses without increased toxicity.[36] Additionally, clinical trials investigating the safety and metabolic effects of FMD in cancer patients have demonstrated profound reductions in systemic glucose and growth factor levels, along with favourable shifts in immune cell populations within the tumor microenvironment, suggesting potential synergy with standard antitumor therapies.[37]

Limitations

Although early clinical trials of fasting and fasting-mimicking diets (FMDs) in cancer patients report safety and metabolic effects, many studies involve heterogeneous patient cohorts, varied cancer types, and concurrent therapies, which prevents clear conclusions about their impact on tumor response or survival outcomes. Additionally, small sample sizes and differences in fasting protocols make it difficult to standardize results and assess true clinical benefit across populations.[38]

Concerns remain regarding the nutritional risks and practical feasibility of fasting-mimicking diets (FMDs) in cancer patients. Prolonged calorie restriction may lead to weight loss, malnutrition, or impaired immune function, particularly in older adults or patients with poor baseline nutritional status. These issues underscore the need for larger, well-controlled randomized trials with standardized fasting interventions to determine long-term safety, adherence, and clinical efficacy.[39]

Conclusion

Fasting and fasting-mimicking diets (FMDs) represent promising adjunctive strategies in cancer therapy. By selectively stressing cancer cells while activating protective mechanisms in normal tissues, these interventions can enhance sensitivity to chemotherapy and other anticancer treatments. Mechanistically, fasting promotes autophagy, modulates cellular metabolism, and reduces oxidative stress, creating a favourable environment for therapeutic efficacy. Early clinical studies indicate that fasting is feasible, safe, and may improve treatment tolerance and outcomes.

Future Directions

To establish stronger clinical evidence for fasting and fasting-mimicking diets (FMDs) in cancer therapy, future studies must conduct larger, well-designed randomized trials that assess standardized fasting protocols (timing, duration, calories) in combination with chemotherapy, immunotherapy, and targeted therapies across diverse cancer types. Recent clinical evidence suggests that adding FMD to first-line carboplatin-based chemotherapy may improve overall survival in advanced triple-negative breast cancer patients, but these findings need validation in larger Phase II/III trials before clinical recommendations can be made.[40]

Mechanistic and translational research should explore how FMD influences treatment resistance and immune-metabolic signalling to enhance anticancer efficacy. New studies show that FMD can potentiate the effects of targeted inhibitors (eg, CDK4/6 inhibitors) by modulating signalling pathways involved in tumor growth and resistance, offering a rationale for combining dietary interventions with systemic cancer therapies in future clinical investigations.[41]

Data Sharing Statement

All data and materials supporting the findings of this review are derived from previously published studies, which are cited in the References below.

Funding

No funding was received for this study.

Disclosure

The author declares no conflicts of interest in this work.

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