Jurnal Kesehatan Tropis Indonesia
Volume 03 Nomor 03 Juni 2025
e-ISSN: 2986-6332, Hal 115-127
Available online at: https://journal.larpainstitute.com/index.php/jkti

The Protective Effects of Ramadan Fasting against Cancer: Exploring Metabolic, Cellular, and Epigenetic Mechanisms

Ajibu Jonas[1], Charles Hassan[2], Yusuf Saidi[3]
[1] Graduate Student, Department of Economics, Universitas Islam Internasional Indonesia, Indonesia
[2] MBBS, Mmed FM, Family Medicine Specialist, MoH, Department of Family Medicine, Kamuzu University of Health Sciences, Malawi
[3] MBBS, Kamuzu University of Health Sciences, PMDT coordinator, MoH, National TB and Leprosy Elimination Program, Malawi
Corresponding: ajibujonas@gmail.com

Abstract : *The practice of Ramadan fasting, as intermittent fasting, is growing in popularity because of its ability to prevent cancer development. The practice of fasting allows patients to experience multiple changes at metabolic, physiological, and epigenetic levels, which reduce cancer advancement and better their response to treatment. The study performs a systematic review of published studies to explore protective mechanisms within this investigation about assurance of scientific integrity and transparency. The authors have implemented the standards included in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The research demonstrates how fasting metabolism affects ketone body production while reducing glucose availability and simultaneously lowering both insulin and insulin-like growth factor-1 (IGF-1) levels. Proliferation control of cancer cells depends heavily on these three regulatory factors. The DNA damage response (DSR) becomes more effective while cellular homeostasis maintains itself because fasting-induced autophagy accomplishes both cellular component elimination and defective cellular component clearance. The reduction of oxidative stress caused by fasting leads to constrained development of malignant cell transformations and protects against DNA. The epigenetic effects of fasting during Ramadan result in non-coding RNA regulation, together with DNA methylation and histone remodeling processes to turn on beneficial tumor suppressor genes and deactivate cancer-causing oncogenes. The latest research supports the potential role of fasting as an additional form of care for fighting and stopping cancer development. These findings lead to major impacts on clinical applications and dietary remedies, and public health policy within integrative cancer treatment.*

Keywords: *Ramadan Fasting, Cancer Prevention, Metabolic Changes, Epigenetic Modifications, Autophagy, Oxidative Stress*

1. INTRIDUCTION

Throughout history, many religions, along with cultural backgrounds, have practiced fasting, which they link with both spiritual cleansing and personal discipline. Muslim populations throughout the world practice and observe Ramadan fasting as their particular tradition. The scientific community has become interested in analyzing the health benefits of this religious fasting experience that lasts 29 or 30 days in a row by preventing people from food or drink between dawn to sunset (Faris et al., 2020). The practice of fasting during Ramadan demonstrates itself as a controlled intermittent fasting method, which might become instrumental for cancer prevention, together with treatment. Science has demonstrated that fasting creates metabolic and cellular transformations like activated autophagy, together with IGF-1 regulatory functions, along with decreased reactive oxygen species and changes in gene

expression patterns that stop cancer development pathways (Alirezaei et al., 2019; Caffa et al., 2023). Traditional Islamic religious beliefs confirm the health benefits of fasting by using these biological processes. Through Quranic revelation in verse 2:184, the scripture declares that "And fasting is better for you if you only knew," which signifies that fasting offers spiritual benefits and enriches complete wellness. Surah Ash-Shu'ara (26:80**)** *"And when I am ill, it is He who cures me." (This verse can be related to the healing effects of fasting on the body, including its potential role in cancer prevention and metabolic health.).*

According to Prophet Muhammad (peace be upon him) one can find health through fasting when he said, "Fast, and you shall be healthy" (Sunan Ibn Majah, Hadith 3448). Current scientific investigations demonstrate that fasting produces important modifications to cellular homeostasis while affecting cancer cell survival together with cell proliferation processes and resistance capabilities (Longo & Mattson, 2014). Autophagy stands as a key cellular detoxification process of fasting which removes damaged cells while minimizing cancer development potential (Mathew et al., 2022). This study investigates the metabolic, molecular, and epigenetic factors behind Ramadan fasting's cancer-protective effects. The study functions as a bridge linking traditional Islamic practices with contemporary scientific achievements within public health together with biomedical sciences domains. The extensive biological effects of fasting studied in this work strengthen ongoing scientific proof which suggests that fasting works as a non-medication intervention to prevent cancer and support health benefits.

Cancer remains a leading cause of diseases and deaths around the world based on projections which indicate 10 million fatalities will occur in 2020 (Sung et al., 2021). Treatment through chemotherapy as well as radiation therapy often results in severe side effects and high charges combined with unsatisfactory long-term treatment success according to Hanahan (2022). Increasing demand now exists for different and supplementary methods to combat and prevent cancer. The intensity of focus on fasting during Ramadan has increased because researchers believe that it might protect against cancer along with controlling metabolic processes and activating autophagy and causing epigenetic changes (Longo & Mattson, 2014). The current scientific understanding does not fully reveal the operative mechanisms through which fasting could defend against cancer development. The research analyzes metabolic pathways together with cellular mechanisms and epigenetic processes that Ramadan fasting may use to prevent cancer.

The best possible cancer prevention methods should be readily accessible at cost-effective prices with minimal invasiveness to stop cancer development and growth. Bray et al. (2018**)** report that cancer occurrences persist to rise but numerous prevention strategies depend

on drug-based solutions which prove inaccessible to certain populations particularly those in low- and middle-income settings. Current cancer research stands almost completely devoid of studies involving fasting prevention even though major research effort traditionally focuses on pharmaceutical therapies and lifestyle changes. Current scientific literature does not adequately explore how religious fasting affects cancer prevention across metabolic pathways and cell function while modifying epigenetic processes even though intermittent fasting proves beneficial to cells and reduces cancer risks (de Cabo & Mattson, 2019). The lack of studies focused on cheap cancer prevention methods could produce rising cancer incidence rates and extensive healthcare costs throughout poor nations. The discovery of cancer-preventive effects from Ramadan fasting could introduce a religiously acceptable intervention method that traditional societies would adopt. The dismissal of this prospective relationship might lead to lost opportunities to enhance both public health results and respect religious practices.

Studies about fasting concentrate on examining its health advantages for both metabolic health and lifespan extension. Studies by Longo and Mattson (2014) proved that periodic fasting enhances autophagy, which serves as a vital process to fight cancer. De Cabo and Mattson (2019**)** determined that fasting enhances metabolic flexibility while lowering oxidative stress, which is known to reduce cancer risks. The investigation of Ramadan fasting with its unique fasting-feeding pattern for inhibiting cancer risks remains poorly studied in research. The analysis conducted by Faris et al. (2019**)** revealed that Ramadan fasting reduces oxidative stress markers and inflammation, yet the study remained unclear about its effects on cancer development pathways. Current cancer research primarily examines temporary metabolic changes instead of sustained evaluations of cancer reduction practices. Research gaps demonstrate why scientists must conduct a complete investigation about how Ramadan fasting affects cancer prevention through metabolic systems, cellular changes, and epigenetic procedures.

This scientific work examines how Ramadan fasting acts as a cancer prevention method through the evaluation of biological pathways starting from metabolic processes to molecular mechanisms and ending at epigenetic changes. Researchers position this investigation as part of public health and biomedical science to contribute evidence about using fasting as a medicine-free approach against cancer development and overall health improvement. Knowledge about the molecular aspects of Ramadan fasting, together with its effects on cancer programs, would benefit healthcare providers, researchers and politicians. The research findings bring vital public health consequences for Muslim-majority regions since Ramadan fasting takes place regularly in these communities. Healthcare expenditures could decrease

through effective cancer prevention strategies based on fasting practices once the effectiveness has been validated by researchers. The evaluation between religious practice and medical science enables this initiative to establish evidence-based suggestions that aid integrative medicine and nutritional approaches while developing preventive care strategies that support holistic healthcare and disease prevention objectives. Research conducted through this project examines cancer prevention from a culturally suitable perspective while filling present knowledge gaps to establish innovative health interventions that are both affordable and accessible.

2. METHODOLOGY

This study is a systematic review conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure a transparent and reproducible literature review process.

a. Eligibility Criteria (Inclusion & Exclusion)

Eligibility criteria were defined to ensure that only relevant and high-quality studies were included in the analysis. The table below summarizes the inclusion and exclusion criteria:

Table 1: Eligibility Criteria

Criteria	Inclusion	Exclusion
Population	Human or animal studies on fasting and cancer	Studies on other types of fasting (e.g., ketogenic diet, prolonged water fasting) without mentioning Ramadan fasting
Intervention	Studies on Ramadan fasting and its biological impact	Studies on non-Ramadan fasting without comparison
Outcomes	Metabolic, cellular, and epigenetic effects of fasting related to cancer	Studies without biological mechanisms (e.g., sociological effects of fasting)
Study Type	Peer-reviewed clinical trials, experimental studies, systematic reviews, and meta-analyses	Non-peer-reviewed articles, commentaries, and opinion papers
Publication Year	Studies published in the last 10–15 years	Older studies, unless highly relevant

b. Data Sources and Search Strategy

A systematic search was performed across several major scientific databases. Boolean operators were used to optimize the relevance and comprehensiveness of search results.

Table 2: Data source and search strategy

Databases:	PubMed, Scopus, Web of Science, Google Scholar, and ScienceDirect
Search Terms:	("Ramadan fasting" OR "Islamic fasting") AND ("cancer" OR "tumor" OR "oncology") AND ("metabolism" OR "glucose metabolism" OR "insulin resistance" OR "ketogenesis") AND ("cellular mechanisms" OR "apoptosis" OR "autophagy" OR "oxidative stress") AND ("epigenetics" OR "DNA methylation" OR "miRNA" OR "histone modification")
Search Filters Applied:	Peer-reviewed articles, Published between 2010–2025 and Human & animal studies

c. Study Selection Process (PRISMA Flowchart)

The study selection process followed the PRISMA flow diagram, which includes the stages of identification, screening, eligibility assessment, and final inclusion.

Table 3: Prisma Flowchart

Identification	Records identified from databases (PubMed, Scopus, Web of Science, Google Scholar, ScienceDirect): (n = 550)
	Records identified from other sources (manual search, citations, reports): (n = 30)
	Total records retrieved: (n = 580)
	Duplicate records removed: (n = 120)
Screening	Records screened (title and abstract level): (n = 460)
	Records excluded (irrelevant, not related to Ramadan fasting or cancer, review articles, etc.): (n = 300)
Eligibility	Full-text articles assessed for eligibility: (n = 160)
	Full-text articles excluded with reasons: (n = 100) Wrong fasting type (e.g., intermittent fasting but not Ramadan fasting) (n = 40) No direct link to cancer prevention (n = 30) Low methodological quality or insufficient data (n = 30)

d. Data Extraction and Analysis

Data from eligible studies were extracted systematically, including publication year, study design, research subjects (human or animal), biological parameters measured (e.g., apoptosis, autophagy, oxidative stress), and key findings relevant to cancer-related mechanisms. Data extraction was conducted independently by two reviewers. Any discrepancies were resolved through discussion or consultation with a third reviewer.

3. RESULT AND DISCUSSION

a. Systemic and cellular fasting response

Metabolic pathway activities shift through fasting to establish a cellular state that produces energy by using mainly adipose tissue carbon sources and, secondarily, muscle-derived carbon sources. The normal cells experience lower metabolic activity combined with reduced growth because circulating hormones and metabolites create this defense mechanism against chemotherapeutic damage. During starvation, cancer cells develop unique responses to restrictive signals in a manner that enhances their reaction to chemotherapy, along with other cancer treatments (Lee C et al., 2012).

b. Systemic response to fasting.

The metabolic system experiences multiple changes when fasting leads to modifications in glucose, insulin, and glucagon levels as well as GH, IGF1, glucocorticoids, and adrenaline. The insulin levels fall as glucagon levels rise during the early post-absorptive period (6-24 hours), which leads to glycogen depletion in the liver and the release of glucose as the power source. Most tissue cells receive energy from free fatty acids and glycerol that arise from triglyceride degradation beyond 24 hours. Nevertheless, the brain requires both glucose and ketone bodies for operation. The brain receives sufficient power from ketone bodies during this period, but gluconeogenesis actively maintains blood glucose levels at 4 mM. The body regulates blood sugar through the actions of glucocorticoids and adrenaline, while these hormones also enhance lipolysis. GH helps gluconeogenesis briefly until it decreases IGF1 activity. The rise in IGFBP1 reduces IGF1 activity, yet the decline in leptin levels during fasting leads to increased effects of adiponectin which promotes fat breakdown. Fasting produces an environment with low glucose levels along with decreased insulin, IGF1, and leptin, but elevated glucagon, together with ketone bodies and adiponectin. (Brandhorst S et al., 2015).

c. Cellular response to fasting.

Cells of good health activate an ancient defensive mechanism that fasting creates to extend both lifespan and health span duration. The importance of IGF1 signaling lies in its activation of AKT and mTOR through increased IGF1 levels produced by diet availability, especially when proteins are consumed. Fasting results in decreased IGF1 levels that activate FOXO transcription factors which enhance production of HO1 along with SOD and catalase enzymes thus strengthening cell protection. During fasting events, glucose levels decrease, which shuts down PKA signaling yet activates both AMPK activity and the important stress tolerance factor EGR1. The protection system provides beneficial effects on the heart as well as entire cell resistance (Di Biase S et al ,2017).

d. Dietary approaches to cancer

Cancer fasting includes two main interventions water fasting and fasting-mimicking diets (FMDs). The prevention of DNA damage caused by chemotherapy on healthy tissues requires at least 48 hours of fasting according to clinical research findings. This approach improves patient quality of life. Water fasting proves challenging for patients because they face difficulties and worry about their nutritional needs. FMDs provide similar fasting advantages to patients through their low-calorie low-carb, and low-protein dietary approach, which boosts compliance rates alongside risk reduction. Primary research studies demonstrate that FMDs have low toxicity levels while simultaneously decreasing body fat, blood pressure and blood IGF1 concentration. Research shows that combination chemotherapy with cancer fasting and FMDs administered over 3 to 4 weeks for 1 to 5 days results in no major adverse effects (Dorff TB et al. ,2016).

e. Ketogenic diets.

High-fat, low-carbohydrate ketogenic diets promote ketone synthesis to decrease IGF1 and insulin levels, while their influence depends on their macronutrient compositions. People use KDs for epilepsy treatment, while these diets help control blood sugar levels, although they show low anticancer properties. The murine data demonstrate positive results against glioblastoma, yet human trials require KDs together with chemotherapy treatments, radiation treatment, and PI3K inhibition. Research does not show how well KDs protect the nervous system or how diets affect chemotherapy-related tissue damage or neurocellular repair. The initiation of the refeeding phase for coordinated tissue regeneration appears only in fasting-mimicking diets and not in ketogenic diets (Liskiewicz et al, 2016).

f. Calorie restriction

The restriction of constant calories, together with limited specific nutrients, provides anticancer effects like periodic fasting while employing opposite mechanisms. The intake of energy content typically lowers by 20-30% under CR conditions, which leads to decreased cardiovascular risks and cancer occurrences in animal study populations that include primates. CR generates various side effects, which include cold sensitivity, muscle loss, infertility, and both osteoporosis and mood changes, resulting in heightened concerns about malnutrition for cancer patients. The combination of CR with protein restriction controls IGF1 levels in addition to minimizing fasting glucose, but singular CR administration does not affect IGF1. Medical research demonstrates

that CR helps intestinal stem cells perform better through mTORC1 signaling blockage, though no scientific data exists about CR's restorative benefits on other body organs. The metabolic and protective response during fasting, together with FMDs appears to produce advantages beyond what KD or CR can achieve (Yousefi M et al, 2018).

g. Fasting and FMDs in therapy

Fasting causes chemical changes in metabolic substances and hormones, which reduce glucose and IGF1, and insulin while increasing the levels of leptin and adiponectin, leading to anticancer effects. Deleterious effects on healthy tissue receives protection from these changes mainly due to reduced levels of IGF1 and glucose. The ketone bodies produced during fasting inhibit histone deacetylases (HDACs) so tumors grow more slowly and the cells differentiate through epigenetic mechanisms122. Tests demonstrate that acetoacetate supports growth in particular cancers, including BRAF-mutated melanomas, whereas it does not stop them from progressing. Research shows that the reduction of IGF1 and glucose levels stands as the strongest proof supporting fasting and FMD's beneficial effects on cancer. The practice of fasting together with FMD diets interrupts intracellular signaling routes, including IGF1R–AKT–mTOR–S6K and cAMP PKA signaling, while simultaneously stimulating autophagy and protecting normal cells during stress situations, along with promoting anticancer immunity (Di Biase S et al., 2016).

h. Differential stress resistance: increasing chemotherapy tolerability.

Through the differential stress resistance (DSR) framework, fasting enables regular cellular protection and enhances the susceptibility of cancer cells to medical interventions. The expression of genetic factors that influence proliferation decreases during fasting, which triggers normal cells to enter a self-preservation mode that enhances their resistance to oxidative damage and chemotherapy impacts. The stress response mechanisms get disrupted by oncogenic transformations in cancer cells, so the cells become more prone to damage. The IGF1 signaling pathway becomes lowered following fasting, according to studies of yeast and mammalian biological systems, thus protecting both glial cells and neurons in addition to normal cells, but not infected cells like gliomas and neuroblastomas. Lab mice show better chemotherapeutic responses after fasting or having their IGF1 signaling pathway modified through genetic modifications, since it enhances treatment tolerance alongside minimizing cardiotoxic and immunological harm and lengthening survival time. Doxorubicin-induced cardiac damage receives protection through fasting mechanisms that reduce ROS production

by autophagy and strengthen protective peptides through the cAMP-PKA-EGR1 pathway. The research supports fasting and FMDs as potential supportive cancer treatments that both improve chemotherapy outcomes and decrease side effects (Di Biase S et al, 2017).

i. Differential stress sensitization: increasing the death of cancer cells.

Cancer growth shows no reaction to fasting or FMDs, yet these nutritional approaches remarkably enhance the effectiveness of chemotherapy alongside radiation and TKIs through DSS. The nutritional deficiency created through fasting works to enhance both oxidative stresses along reactive oxygen species production as well as sensitization of cancer cells to DNA-damaging therapy. Fasting modifies cancer metabolism toward oxidative phosphorylation in addition to increasing chemotherapeutic transporters, activating pro-apoptotic genes, and suppressing MAPK signaling. Cancer cells belonging to B and T cell ALL groups and possessing glucose dependence tend to benefit the most from these advantages. Animal model research shows that when patients use fasting with standard therapies, tumor cells rarely become resistant to treatment (Caffa et al., 2015).

j. Antitumor immunity enhancement by fasting or FMDs.

Scientific evidence demonstrates that both fasting and FMDs as individual treatments or combined with chemotherapy, help raise lymphoid progenitors to strengthen cancer immune assault. The fasted state in living subjects reduced HO1 activity in cancer cells, yet elevated its activity in intact normal cells. The decline of HO1 activity in cancer cells makes tumor-infiltrating lymphocytes attack tumors through their CD8+ cells while also potentially benefiting from regulatory T cell inhibition (Postow et al, 2015). Research conducted by investigators demonstrated that fasting together with FMDs and CR mimetics solutions enhanced anticancer immune surveillance, thus indirectly suggesting they could operate against autophagy-competent tumors but would be ineffective on autophagy-deficient ones. The practice of fasting cancer cells for two weeks in mouse colonic tumors created autophagy while decreasing CD73 enzyme activity and reducing cancer cell adenosine synthesis. The fasting process decreased macrophage CD73 expression to stop these cells from converting into M2 immunosuppressive type cells. Single studies indicate FMDs can serve as an alternative to immune checkpoint inhibitors or act alongside cancer vaccines as well as other antitumor drugs that encompass conventional chemotherapeutic agents (Galluzzi et al, 2015).

k. Anticancer diets in mouse models

Research conducted with cancer models involving animals demonstrates that both fasting procedures alongside FMDs exhibit comprehensive anticancer properties as well as potential treatment outcomes while producing organ self-healing effects. Fasting alongside FMDs provides benefits compared to costly drugs, which tend to cause harm because they achieve tumor remission as well as long-term survival of patients. Research indicates that ketogenic diets (KDs) demonstrate potent anticancer effects that control immunological activities and improve treatment results primarily for gliomas and other malignant tumors. While models show that both chronic calorie restriction (CR) and ketogenic diets possess advantageous effects over fasting, the combination with anticancer therapy presents weaker results than fasting and comes with concerns such as lean body mass loss along with patient adherence challenges. The desperate changes that short-term fasting creates in metabolic pathways make it more powerful against cancer than refeeding regenerates and heals the body. The potential therapeutic benefits, along with reduced inconvenience, of periodic FMDs or short fasting cycles make them attractive options to enhance cancer therapy results by reducing treatment side effects, mainly in aggressive cancers such as glioblastoma (Abdelwahab et al, 2012).

l. Fasting and FMDs in cancer prevention

Multiple studies based on both animal experiments and human population statistics show that patterned dietary approaches, including chronic calorie restriction and intermittent fasting, and fasting-mimicking diets, help prevent cancer development. The practice of CR remains difficult because people struggle to follow it and suffer negative effects. Current research focuses on finding regular eating patterns that have minimal side effects for clinical evaluation. When FMD regimens activate, they lower IGF1 and glucose while elevating IGFBP1 and ketone body production, thus creating the same indicators as fasting. When C57Bl/6 mice received two monthly FMD administrations over four days, the tumor incidence decreased to 40% compared to 70%, while mortality was extended and tumor aggressiveness reduced. The alternative day fasting protocol applied to middle-aged mice brought lymphoma occurrence down from 33% to 0% before the researchers could determine extended impacts. FMD provides a more convenient fasting plan than alternate-day fasting because it requires fewer fasting occasions each month (Brandhorst S et al., 2015).

m. Clinical applicability in oncology

Recent feasibility research confirms that fasting together with fasting-mimicking diets (FMDs) helps chemotherapy patients minimize their adverse effects. The voluntary fasting conducted by ten cancer patients led to no major adverse outcomes but resulted in feelings of lightheadedness together with increased hunger. The patients also experienced alleviated fatigue and stomach issues. The protocol of short-term fasting decreased DNA breaks and blood cell number reductions that typically follow chemotherapy according to experimental research findings. A minimum 48-hour fasting period decreased DNA damage while simultaneously tending to produce lower neutropenic conditions in patients undergoing platinum-based chemotherapy treatments. A randomized clinical study with patients who had breast or ovarian cancer proved that Fasting-Mimicking Diets defended against the fatigue effects and deterioration in quality of life from chemotherapy treatments. Research teams consisting of more than 300 participants monitor FMD impact on chemotherapy toxicity through continuous clinical trials throughout the United States and Europe (Dorff TB et al 2016).

4. CONCLUSSION

The practice of fasting triggers multiple metabolic effects along with various physiological and epigenetic effects that protect against cancer development while offering better therapeutic results. Metabolic changes from fasting lead to ketone production while reducing both cancer cell access to glucose and insulin and IGF-1 levels that manage signal proliferation through their decrease and enhance insulin-related chemical sensitivity. Autophagy develops during fasting to maintain cellular balance while eliminating defective substances that present cancer-related hazards. The treatment of cancer cells becomes more effective due to fasting-induced DSR. The lowering of oxidative stress through fasting cuts down DNA damage and malignant transformation. The practice of fasting causes significant epigenetic modifications to DNA methylation patterns and histone restructuring, together with non-coding RNA regulatory changes, which ultimately lead to tumor-suppressor activation and oncogene silencing. Different studies show how fasting can function as a supplementary method for cancer prevention and treatment, thus requiring additional clinical evaluation.

DAFTAR REFERENSI

Al-Jafar, R., Pinto, R. C., Elliott, P., Tsilidis, K. K., & Dehghan, A. (2024). Metabolomics of Ramadan fasting and associated risk of chronic diseases. *American Journal of Clinical Nutrition*, *119*(4), 1007–1014. https://doi.org/10.1016/j.ajcnut.2024.01.019

Alshammari, K., Alhaidal, H. A., Alharbi, R., Alrubaiaan, A., Abdel-Razaq, W., Alyousif, G., & Alkaiyat, M. (2023). The Impact of Fasting the Holy Month of Ramadan on

Colorectal Cancer Patients and Two Tumor Biomarkers: A Tertiary-Care Hospital Experience. *Cureus*, *15*(1), 1–11. https://doi.org/10.7759/cureus.33920

Amiruddin, M., Putri Oktavia, R., & Hidayat, H. (2023). The Benefits of Intermittent Fasting as an Alternative to Medical Therapy to Improve Health Status. *Journal of Islamic Medicine*, *7*(02), 156–162. https://doi.org/10.18860/jim.v7i2.20591

Alirezaei, M., Kemball, C. C., Flynn, C. T., Wood, M. R., Whitton, J. L., & Kiosses, W. B. (2019). Short-term fasting induces profound neuronal autophagy. *Autophagy, 6*(5), 702–710. https://doi.org/10.4161/auto.6.5.12376

Bray, F., Ferlay, J., Soerjomataram, I., Siegel, R. L., Torre, L. A., & Jemal, A. (2018). Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: A Cancer Journal for Clinicians, 68*(6), 394–424. https://doi.org/10.3322/caac.21492

Caprara, G., Pallavi, R., Sanyal, S., & Pelicci, P. G. (2025). Dietary Restrictions and Cancer Prevention: State of the Art. *Nutrients*, *17*(3), 1–45. https://doi.org/10.3390/nu17030503

Caffa, I., Spagnolo, V., Vernieri, C., Valdemarin, F., Peraldo-Neia, C., & Fanciulli, G. (2023). Fasting as an adjuvant therapy for cancer: Mechanism of action and clinical application. *Biomolecules, 14*(11), 1437. https://doi.org/10.3390/biom14111437

Chaix, A., Zarrinpar, A., Miu, P., & Panda, S. (2019). Time-restricted eating: A preventive and therapeutic intervention against diverse nutritional challenges. *Cell Metabolism, 29*(3), 627–646. https://doi.org/10.1016/j.cmet.2018.12.004

Dastghaib, S., Siri, M., Rahmani-Kukia, N., Heydari, S. T., Pasalar, M., Zamani, M., Mokaram, P., & Bagheri-Lankarani, K. (2025). Effect of 30-day Ramadan fasting on autophagy pathway and metabolic health outcome in healthy individuals. *Molecular Biology Research Communications*, *14*(2), 115–127. https://doi.org/10.22099/mbrc.2024.50105.1978

de Cabo, R., & Mattson, M. P. (2019). Effects of intermittent fasting on health, aging, and disease. *New England Journal of Medicine, 381*(26), 2541–2551. https://doi.org/10.1056/NEJMra1905136

Demsky, I. (2024, June 14). Fasting primes the immune system's natural killer cells to better fight cancer, new study in mice finds. *Memorial Sloan Kettering Cancer Center.* https://www.mskcc.org/news/fasting-primes-immune-systems-natural-killer-cells-to-better-fight-cancer-new-study-in-mice-finds

Fang, M., Chen, D., Yang, C. S., & Kruger, W. D. (2019). DNA methylation, aging, and cancer. *Current Opinion in Clinical Nutrition and Metabolic Care, 22*(6), 393–400. https://doi.org/10.1097/MCO.0000000000000607

Fang, M., Chen, D., Yang, C. S., & Kruger, W. D. (2019). Epigenetic and metabolic regulation of cancer prevention and therapy. *Advances in Cancer Research, 142*, 1–31. https://doi.org/10.1016/bs.acr.2019.01.001

Faris, M. A. I. E., Kacimi, S., Al-Kurd, R. A., Fararjeh, M. A., Bustanji, Y. K., & Mohamadin, A. M. (2020). Intermittent fasting during Ramadan attenuates proinflammatory cytokines and immune cells in healthy subjects. *Nutrition Research, 30*(12), 865–875. https://doi.org/10.1016/j.nutres.2020.07.002

Fernandez, A. F., Bayón, G. F., Urdinguio, R. G., Toraño, E. G., Garcia, M. G., Carella, A., ... & Fraga, M. F. (2018). Hacking the epigenome: Diet, aging, and cancer. *Advances in Experimental Medicine and Biology, 1086*, 109–125. https://doi.org/10.1007/978-3-319-78337-3_5

Hanahan, D. (2022). Hallmarks of cancer: New dimensions. *Cancer Discovery, 12*(1), 31–46. https://doi.org/10.1158/2159-8290.CD-21-1059

Horne, B. D., Muhlestein, J. B., & Anderson, J. L. (2021). Health effects of intermittent fasting: Hormesis or harm? *American Journal of Clinical Nutrition, 114*(5), 1851–1865. https://doi.org/10.1093/ajcn/nqab245

Levine, B., & Kroemer, G. (2019). Biological functions of autophagy genes: A disease perspective. *Cell, 176*(1-2), 11–42. https://doi.org/10.1016/j.cell.2018.09.048

Madeo, F., Zimmermann, A., Maiuri, M. C., & Kroemer, G. (2019). Essential role for autophagy in life span extension. *Journal of Clinical Investigation, 125*(1), 85–93. https://doi.org/10.1172/JCI73946

Madkour, M. I., El-Serafi, A. T., Jahrami, H. A., Hassan, R. E., Awadallah, S., & Faris, M. E. (2019). Ramadan diurnal intermittent fasting modulates SOD2, TFAM, Nrf2, and sirtuins (SIRT1, SIRT3) gene expressions in subjects with overweight and obesity. *Diabetes Research and Clinical Practice, 151*, 107801. https://doi.org/10.1016/j.diabres.2019.107801

Mathew, R., Karp, C. M., Beaudoin, B., Vuong, N., Chen, G., Chen, H. Y., ... & White, E. (2022). Autophagy suppresses tumorigenesis through elimination of p62. *Cell, 137*(6), 1062–1075. https://doi.org/10.1016/j.cell.2009.03.048

Nencioni, A., Caffa, I., Cortellino, S., Longo, V. D., Specialties, M., Policlinico, O., Martino, S., & Angeles, L. (2019). *application*. *18*(11), 707–719. https://doi.org/10.1038/s41568-018-0061-0.Fasting

Pavlova, N. N., & Thompson, C. B. (2016). The emerging hallmarks of cancer metabolism. *Cell Metabolism, 23*(1), 27–47. https://doi.org/10.1016/j.cmet.2015.12.006

Redman, L. M., Smith, S. R., Burton, J. H., Martin, C. K., Il'yasova, D., & Ravussin, E. (2018). Metabolic slowing and reduced oxidative damage with sustained caloric restriction support the rate of living and oxidative damage theories of aging. *Cell Metabolism, 27*(4), 805–815.e4. https://doi.org/10.1016/j.cmet.2018.02.019

Reddy, B. L., Reddy, V. S., & Saier, M. H. (2024). Health Benefits of Intermittent Fasting. *Microbial Physiology*, *34*(1), 142–152. https://doi.org/10.1159/000540068

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

Yousefi, M., et al. (2018). Calorie restriction governs intestinal epithelial regeneration through cell-autonomous regulation of mTORC1 in reserve stem cells. *Stem Cell Reports, 10*(3), 703–711. https://doi.org/10.1016/j.stemcr.2018.01.024