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Epigenetic remodeling in response to intermittent fasting and its role in visceral fat reduction

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Abstract

A compelling regimen that has recently gained popularity is intermittent fasting (IF), which aims to improve metabolic function and promote weight loss. Recent research suggests that IF has complex effects, influencing metabolic regulators like insulin sensitivity and lipolytic pathways as well as causing epigenetic changes, which are thought to be responsible for a significant portion of its therapeutic efficacy. Epigenetic remodeling controls inflammatory responses, fat deposition, and energy homeostasis by modifying chromatin structure and transcription dynamics in a way that is reversible and inheritable while leaving the nucleotide sequence unchanged. Metabolic abnormalities are characterized by an increase in visceral adipose tissue, which is a risk factor for obesity and type 2 diabetes. This review focuses on the inhibitory effect of IF on this expansion and how it leads to epigenetic changes. This sudy examines IF and its influence on certain epigenetic changes regulating 'master' genes and 'core' metabolic pathways parameters involved in fat storage, mitochondrial bioenergetics, and inflammation related to oxidative stress. In light of recent studies on targeted epigenomic remodeling and visceral fat loss, this text examines how time-restricted fasting may provide clinically relevant cardiometabolic benefits and explores personalized approaches to energy restriction based on epigenetic predictive factors. Such prediction may represent a selective point of relevance to offer therapeutic intervention in a day-to-day manner. This article ends with a set of prioritized suggestions for longitudinal studies that delineate the overlapping time frame of epigenetic fasting and its influence on the genome in the context of obesity and its associated cardiovascular components.

Keywords: Intermittent fasting, epigenetic remodeling, visceral fat, metabolic health, gene expression.

Introduction

Overview of Epigenetic Remodeling

Epigenetic remodeling encompasses heritable modifications in gene expression that leave the primary DNA sequence intact. DNA methylation, covalent modifications of histones, and the activity of non-coding RNAs constitute the principal molecular components of this process. Through these modifications, specific genes can be either up-regulated or silenced, thereby influencing core intracellular programs and, at a larger scale, organismal physiology. Importantly, the marks imposed by these modifications are not fixed; they can adapt in response to a wide spectrum of environmental exposures, habitual behaviors, and dietary factors. As a result, the resultant epigenetic changes are characterized by dynamic and reversible properties. This plasticity enables cells to adjust gene expression patterns almost instantaneously in the face of fluctuations in nutrient supply, changes in metabolic demand, or encounters with pathogens **[1-7]**.

Continued inquiry is elucidating the role of epigenetic remodeling in the orchestration of metabolic circuits, the shaping of adaptive immune reactivity, and the maintenance of cellular homeostasis throughout

aging. The accumulating evidence foregrounds epigenetic pathways as decisive determinants in the prevention and management of a spectrum of diseases **[4,8-11]**.

Intermittent Fasting and Its Physiological Impact

Intermittent fasting (IF) is a nutritional paradigm that rhythmically alternates feeding and fasting intervals **[1,12-15]**. Recent literature has assigned it considerable visibility as a flexible, nondietary intervention with modest-to-large effects on body composition, metabolic maintenance, and, arguably, lifespan extension. The absence of ingesta under fasting conditions precipitates a suite of regulated responses that conserve biomass, hemodynamic stability, and cellular functionality **[16]**.

Noteworthy metabolic adaptations comprise enhanced peripheral and hepatic insulin sensitivity, an upregulated fatty-acyl CoA mobilization and oxidation, and a pronounced engagement of autophagic enzymology, operative in the proteolytic and organelle-recycling apparatus that renews cellular viability. Gradually, adherence to IF prompts an augmentation of metabolic flexibility, facilitating the seamless transition between glucose and lipid oxidation as the primary fuel substrate. Concurrently, IF demonstrates a suppressant effect on several circulating inflammatory mediators, mediators whose sustained elevation singly or cumulatively predisposes the onset of chronic pathologies including, but not limited to, diabetes and atherosclerotic cardiovascular disorders. The progressive adaptation of the organism to the IF protocol fuels these salutary alterations, which cumulatively reinforce homeostatic durability. Empirical data substantiate the hypothesis that sustained IF participation confers significant modulatory influence upon body weight and underlying metabolic architecture **[16,17]**.

Significance of Visceral Fat Reduction in Health

Visceral adipose tissue, located within the abdominal cavity in close proximity to key sites of metabolism including the liver, pancreas, and intestines serves as a central nexus in the pathogenesis of metabolic perturbation and a wide spectrum of chronic diseases **[12]**. In contrast to subcutaneous depots, which are situated just beneath the dermis, visceral fat is distinguished by its pronounced endocrine activity; the tissue secretes a constellation of peptides, free fatty acids, and inflammatory cytokines that collectively impair homeostatic circuits. Greater visceral fat accumulation is robustly correlated with a spectrum of obesity-pertinent pathophysiology, the most clinically pressing of which are type 2 diabetes, atherosclerotic cardiovascular syndromes, and selected malignancies.

Pathomechanistic investigation has substantiated that enlarged visceral depots potentiated the development of insulin resistance, heightened systemic vascular resistance, and elevated circulating lipoprotein cholesterol. Visceral adiposity has repeatedly been linked to an array of pathophysiological consequences, including impaired insulin sensitivity, heightened vascular tone, and dysregulated lipoprotein profiles, all of which serve as salient risk factors for cardiometabolic disorders. In light of this, the selective abatement of intra-abdominal fat has consolidated its status as a cornerstone for prophylactic and restorative strategies in clinical practice. Contemporary nutritional paradigms, notably restrictive temporal feeding (i.e., intermittent fasting), have surfaced as feasible regimens that recalibrate endocrine milieu and substrate flux, thereby facilitating lipolytic activity in the visceral depot and accentuating substrate mobilization during the fasting phase **[3,17]**.

Hence, the concomitant decrease in visceral mass engenders not merely a transient reduction in body weight, but invokes robust and durable shifts in silhouette, vascular dynamics, and lipid homeostasis, which singularly underscore the depot as a focal therapeutic target in the delineation and management of metabolic derangements.

Literature review

Epigenetic Mechanisms and Their Role in Metabolism

Epigenetic controls govern gene activity without any modification of the nucleotide sequence itself, relying upon the modification of DNA itself, its associated histones, and intermediary RNA **[18]**. Methylation of cytosine residues within promoter and enhancer territories generally represses transcription by recruiting repressors and by altering recruitment of the transcriptional machinery. Consolidating chromatin compaction into higher-order structures and controlling the transitory surfacing of critical cis-regulatory regions for transcription-factor recruitment are both regulated by covalent modifications of histones, such as methyl, acetyl, and phosphate additions.

Complementary sequences in target messenger RNAs bond with short RNAs, such as microRNAs, at the post-transcriptional stage, imposing translational silence and driving transcript destruction. All of these epigenetic investments act together to stabilizely rewire the basal metabolic network of eukaryotic cells in response to cues from the periphery. An important factor in maintaining metabolic homeostasis is

epigenetic remodelling, which involves fine-tuning gene transcription related to energy production, lipid storage, insulin sensitivity, and inflammation resolution **[17,18]**.

Macronutrient composition, habitual physical activity, and psychosocial stress, among other environmental perturbations, recalibrate epigenomic circuits to change energy expenditure, as needed, for the maintenance of homeostasis **[8,13]**. Epigenetic mechanisms also enable persistent metabolic reprogramming that constitutes real-time changes across generations, imprinting stress resilience, and creating lineage-specific predisposition to the metabolic disease spectrum, is also a fundamental reconfiguration of the transcriptional state of key metabolic genes spanning generations.

Effect of Intermittent Fasting on Genetic Expression and Epigenetic Modifications

A fascinating new dietary paradigm, IF causes systematic changes in epigenetic configuration and transcriptional patterns **[2,14]**. Feeding restrictions force the body to use lipids produced internally rather than glucose for energy, setting off a cascade of events that includes changes in hormone levels, enzyme activity, and gene expression. Accelerated free-fatty acid mobilization, increased hepatic ketogenic activity, improved insulin receptor signaling pathways, and up-regulation of cellular housekeeping mechanisms among which autophagy is a key component are all brought about by this change in metabolic substrate **[10]**. Intermittent fasting also functions as an important ecological signal that changes epigenetic processes: changes to the methylation patterns across the genome will functionally restructure the transcriptional value of the loci governing fatty acid β-oxidation and the anti-inflammatory response **[19]**.

The differential histone marks will also have an effect by modifying the chromatin structure to make some genes more or less available to the transcriptional complex. Concurrently, non-coding RNAs, which exert control over expression subsequent to transcription, may be modulated upward or downward by the fasting state, thereby fine-tuning metabolic circuitry. The constellation of these nuclear alterations, both genetic and epigenetic, equips the organism to manage the fasting stress, enhancing energetic efficiency and reinforcing defenses against metabolic disorders. Consequently, protocols of IF not only produce acute effects on metabolic function but also induce stable epigenetic reprogramming that may underlie prolonged, favorable changes in health **[9]**.

Studies Linking Epigenetic Changes to Visceral Fat and Metabolic Health

Investigating the interplay between epigenetic alterations and the reduction of visceral adiposity is increasingly recognized as pivotal, given the pronounced effects of visceral fat on systemic metabolic profiles **[11]**. This particular depot, encasing intra-abdominal critical organs, serves as a powerful predictor of pathologies such as obesity, type 2 diabetes mellitus, and atherosclerosis. Molecular changes, principally DNA methylation and post-translational modifications of histones, are known to modulate transcriptional circuits that govern adipose tissue distribution and energy balance. Specifically, these modifications may direct the expression of genes implicated in pre-adipocyte differentiation, mitochondrial fatty acid utilization, and the cellular response to insulin. Controlled studies reveal that dietary regimens, including intermittent fasting, impose reversible epigenetic shifts that attenuate visceral fat accumulation and concurrently enhance several aspects of metabolic health **[5,20]**. For instance, alterations in DNA methylation profiles at loci governing lipid oxidation have been observed following fasting, resulting in an augmentation of lipid catabolism and a concurrent suppression of triglyceride deposition, with a pronounced effect on visceral adipose tissue.

Moreover, the coordinated shifts in histone acetylation and microRNA abundance reinforce transcriptional circuits that oversee the adaptogenic program and the insulin signaling axis. Together, these epigenetic modifications deliver a mechanistic underpinning to the reduced visceral adiposity that accompanies time-restricted eating regimens, thereby framing IF as a promising strategy in the clinical modulation of metabolic derangement and the mitigation of disorders linked to adipose-tissue expansion **[6]**.

Proposed Model

Conceptual Framework of Epigenetic Remodeling in Response to Intermittent Fasting

The proposed construct of fasting-induced epigenetic remodeling retains a tripartite character, encompassing metabolism, genetics, and epigenetics, and thereby elucidates the adaptive transformations concordant with IF. Within this architecture, engagements with IF appear to precipitate a sensed metabolic reorientation, the consequences of which are subsequently inscribed at molecular epigenetic sites, ultimately securing favorable measures of metabolic homeostasis. Foremost, the fasting state stimulates

lipid-derived combustion, selective autophagic responses, and heightened insulin reactivity, each of which reconfigures gene transcription at selective loci **[19,20]**.

Differential recruitment of post-translational histone markings, altered patterns of 5-methyl-cytosine deposition, and the modulation of regulatory non-coding RNAs with known metabolic and circadian relevance are the recognised epigenetic modalities that give rise to the newly activated transcriptional landscape. For instance, methylation of DNA affects the transcriptional commitment of genes that control lipid metabolism, and histone acetylation status may selectively enhance or suppress loci that are involved in energy balance. The framework presented elucidates an integrated circuitry in which epigenetic adjustments serve as conduits for translating the external cue of IF into intracellular programs that enhance lipid oxidative capacity and attenuate visceral adiposity. By meticulously tracing the underlying molecular cascades and cross-talk networks, the model endeavors to articulate a cohesive perspective on how sustained patterns of eating and subsequent fasting induce reversible and lasting remodeling of the epigenome, thereby securing enduring physiological sequalae and metabolic resilience **[20]**.

Key Genes and Pathways Involved in Visceral Fat Reduction

Several genes and corresponding metabolic pathways are instrumental in the reduction of visceral adipose tissue, and their activity is markedly governed by epigenetic modifications that occur in the context of IF. A prominent regulatory axis involves the transcription factors that orchestrate the suppression and supply of fatty acids. Within this framework, the peroxisome proliferator-activated receptor-γ (*PPARγ*) pathway functions as the lynchpin, directing the commitment of pre-adipocytes and the synthesis of triacylglycerol. Prolonged food deprivation imposes epigenetic remodeling that dampens *PPARγ* transcriptional potential, resulting in diminished differentiation and filling of visceral adipose depots. Simultaneously, the AMP-activated protein kinase (*AMPK*) circuit, operating as an intracellular fuel gauge, is promptly engaged by the low-AMP/ATP ratio induced by alimentary scarcity; this enhancement in *AMPK* activity promotes the mobilization and oxidation of stored lipids to sustain substrate-level energy provision **[20]**.

Moreover, AMPK-dependent pathways are fundamentally connected to the induction of mitochondrial biogenesis, an adjustment that elevates the capacity for fatty acid oxidation within the cell. Under regimes of intermittent fasting, the *SIRT1* locus an established orchestrator of stress response and lipid equilibrium becomes strongly up-regulated, thereby promoting the selective release of visceral adipose depots. Concurrently, fasting initiates epigenetic alterations that reposition the transcriptional landscapes of the insulin receptor substrate (*IRS*) and the peroxisome proliferatoractivated receptor coactivator 1-alpha (*PGC-1α)* genes; both factors are indispensable for enhancing insulin responsiveness and for the targeted enhancement of mitochondrial biogenesis. The *SIRT1IRS-PGC-1α* circuitry acting in concert, incorporates a cohesive molecular strategy that prioritizes visceral adipose tissue, in this case, epigenetic changes caused by fasting enact a permissive, longterm regulatory influence on gene expression and related metabolic endpoints, and the adaptation is enduring **[20]**.

Mechanisms through Which Epigenetic Changes Influence Fat Metabolism

Modifications to epigenetics influence lipid metabolism through regulating genes involved in adipose tissue accumulation, lipolysis, and metabolic energy expenditures. The addition of methyl groups to promoters that are rich in CpG usually decreases the transcription of loci that regulate lipid management, making DNA methylation one of the most consequential of these alterations. A pertinent illustration is inherited repression of the *PPARγ* locus, where promoter hypermethylation diminishes gene dosage and thereby curtails the hyperplastic enlargement of visceral adipose tissue. In concert, dynamic alterations to the histone code modulate transcript abundance; lysine acetylation of histone tails recruits bromodomain-containing transcriptional coactivators, relieving compact chromatin packaging and enabling requisite transcription factor binding to adipocyte and hepatocyte regulatory regions **[18-20]**.

Alternatively, the removal of acetyl groups from histones serves to downregulate gene expression while concurrently decreasing the accumulation of adipose tissue. Similarly, microRNAs, entities traditionally categorized as non-coding RNAs, exert a repressive effect on the translation of mRNAs that encode proteins responsible for the biosynthesis and storage of lipids. Periods of caloric restriction activate epigenetic alterations in loci such as *AMPK, SIRT1, and IRS*, processes that collectively intensify the rate of fat catabolism and enhance sensitivity to insulin. These modifications extend their impact to mitochondrial dynamics and global energy expenditure, ultimately reinforcing lipolytic pathways and curtailing the excess of visceral adipose tissue. By integrating histone

remodeling, microRNA-mediated silencing, and fasting-responsive methylation and acetylation shifts, the organism secures a continual mobilization of adipose reserves for energetic purposes, thereby promoting a decrease in visceral fat and favorably reprogramming metabolic homeostasis **[15]**.

Figure 1 presents a schematic integrating intermittent fasting, heritable chromatin alterations, and the selective shrinkage of visceral adipose reserves. Illustrated within are the principal epigenetic modalities, including cytosine methylation, histone remodeling, and downstream transcriptional outcomes, represented as interdependent fluxes governing lipid homeostasis. The illustration subsequently foregrounds the notion that fasting-triggered epigenetic rewiring preferentially promotes the catabolic oxidation of visceral fat while curtailing lipogenic expansion.

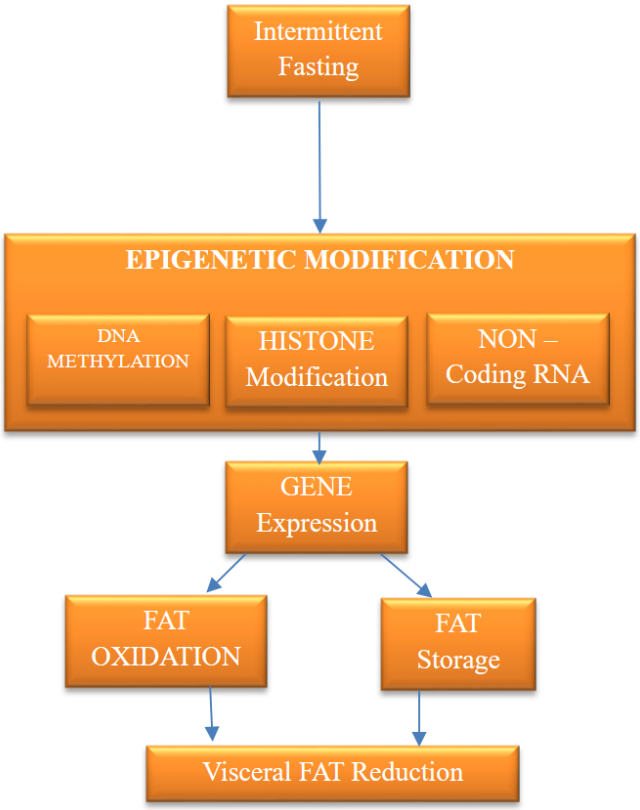

Figure 1. Model of Epigenetic Influence on Visceral Fat Reduction through Intermittent Fasting. Source: Own authorship.

Mathematical Model

The mathematical model used to quantify the effects of IF on visceral fat reduction through epigenetic changes can be represented by the following equation 1:

$$V_f = \frac{P_{oxidation} \times E_{adipocyte} \times G_{mod}}{T_{IF}} \quad (1)$$

Where:

- V_f = Visceral fat reduction
- $P_{oxidation}$ = Rate of fat oxidation (influenced by AMPK activation)
- $E_{adipocyte}$ = Efficiency of adipocyte differentiation and storage (influenced by PPARγ and SIRT1)
- G_{mod} = Gene modulation index, accounting for epigenetic changes (methylation and histone modifications)
- T_{IF} = Duration and frequency of IF cycles

The proposed computational architecture formalizes the interplay between epigenomic adaptation and the basal metabolic rate, providing a measurable annotation of diachronic responses explicable by intermittent fasting. Through systematic calibration against empirical metabolomic and transcriptomic records, parameter refinement facilitates the prospective evaluation of fasting regimens upon visceral lipid compartmentalization, thereby supplying a rational basis for the bespoke design of caloric restriction protocols.

Results and Discussion

Summary of Research Findings on Epigenetic Changes in Intermittent Fasting

Recent investigations affirm that IF induces substantial epigenetic alterations that modulate gene expression governing lipid metabolism and resilience to cellular stress. The epigenetic processes of interest comprise DNA methylation and histone modification, which mediate the organism's adaptation to periodic nutrient scarcity. Experimental evidence indicates that caloric deprivation stimulus culminates in the recruitment of pivotal metabolic pathways, foremost among which are those governing lipid oxidation, mitochondrial bioenergetics, and improvement of insulin responsiveness **[1-4]**.

Correlative analyses have documented that methylation cytosine residue alterations proximal to the *PPARγ* and *SIRT1* loci concomitantly attenuate lipogenic signaling and enhance β-oxidative capability, thereby favoring metabolic homeostasis in the fasting state. Histone acetylation, conversely, promotes the transcriptional activation of genes governing cellular maintenance and bioenergetic functionality. Concurrently, regulatory populations of non-coding RNA, notably microRNAs, selectively suppress transcripts central to metabolic homeostasis. Collectively, these regulatory embodiments articulate the capacity of epigenetic perturbation to transduce the adaptive responses elicited by IF and intimate the cellular pathways responsible for the mobilization of ectopic lipid stores, most notably visceral adipose depots **[5,6]**.

Impact of Epigenetic Remodeling on Visceral Fat and Health Outcomes

Epigenetic remodeling considerably influences the reduction of visceral adipose tissue and exemplifies the human organism's capacity to recalibrate metabolic operations under a regimen of intermittent fasting. The structural modification of chromatin gives rise to altered transcriptional profiles of genes governing adipocyte differentiation, lipid catabolism, and insulin sensitivity. Rigorous investigation has documented that fasting-responsive variations in DNA methylation result in transcriptional repression of the *PARγ* locus, a pivotal determinant of preadipocyte activation, thereby curtailing the accrual of intra-abdominal lipid. Complementarily, the fasting-mediated enhancement of histone acetylation at the *AMPK* locus has been correlated with augmented fatty-acid turnover and elevated functionality of the mitochondrial respiratory chain. The observed epigenetic remodeling induced by IF achieves a multifaceted metabolic benefit: attenuation of visceral adiposity is paralleled by heightened insulin sensitivity, diminished systemic inflammation, and ameliorated dyslipidemia **[7,8]**.

Visceral adipose tissue, as a major endocrine organ, releases bioactive mediators that perpetuate metabolic derangement; therefore, its selective reduction markedly lowers the attributable hazards of type 2 diabetes, cardiovascular pathologies, and non-alcoholic fatty liver disease. Collectively, these compelling mechanistic and clinical observations position IF as a precise intervention that harnesses epigenetic modulation to mitigate obesity-related comorbidities **[9,10]**.

Implications for Personalized Nutrition and Weight Management

Compelling evidence connecting the epigenetic reprogramming induced by IF to the refinement of personalized nutritional protocols marks a decisive advance in behavioral weight management. These epigenetic modifications display a high degree of inter-subject heterogeneity, mediated by a confluence of heritable polymorphisms, habitual behavioral patterns, and a spectrum of environogenic stressors. Consequently, fasting regimens may be tailored to align with the individual's epigenomic signature, yielding enhanced pharmacodynamic and behavioral outcomes **[11]**.

A pragmatic translational model involves the systematic acquisition of epigenetic biomarkers to establish evidence-informed fasting durations and frequencies, along with precise macronutrient targets. Such a stratified approach may preferentially mitigate visceral fat depots and restore energetic equilibrium. Concurrently, precise weight-control protocols may exploit gene-environment interaction frameworks to anticipate and calibrate the responsiveness to intermittent fasting, thereby delivering sustained therapeutic value and fostering adherence. The convergence of epigenetic research with nutritional science and caloric modulation heralds a paradigm of precision medicine for the prevention and management of obesity and attendant metabolic dysfunction. Continued inquiry is warranted to dissect the range of epigenetic adaptations to intermittent energy restriction, as resolution of this variance may undergird the development of tailored, evidence-based interventions that confer sustained metabolic improvement and weight control **[12]**.

Table 1 summarizes the progressive rise of three epigenetic markers DNA methylation, histone acetylation, and microRNA expression across progressive intervals of intermittent fasting. This offers quantitative evidence of their combined role in reducing visceral adipose tissue and precisely regulating lipid flow.

Gene/ Pathway	Effect of Intermittent Fasting	Role in Fat Metabolism
PPARγ	Downregulated through DNA methylation	Reduces adipogenesis and fat storage in visceral fat
AMPK	Activated through histone modification	Promotes fat oxidation and mitochondrial biogenesis
SIRT1	Upregulated through histone acetylation	Enhances fat metabolism and reduces inflammation
IRS	Modulated by fasting-induced epigenetic changes	Improves insulin sensitivity and fat utilization

Table 1. Changes in Gene Expression and Metabolic Pathways Induced by Intermittent Fasting. Source: Own authorship.

Figure 2 illustrates the relationship between fasting impacts, epigenetic changes principally through DNA methylation and histone acetylation, and the corresponding reduction of visceral adipose tissue mass, as outlined in the empirical data. The fasting-period, represented along the x-axis, is correlated with the proportional reduction in visceral fat, as reflected on the y-axis. The plotted results collectively reinforce the contention that these fasting-driven epigenomic adaptations critically fine-tune lipid metabolism and storage pathways.

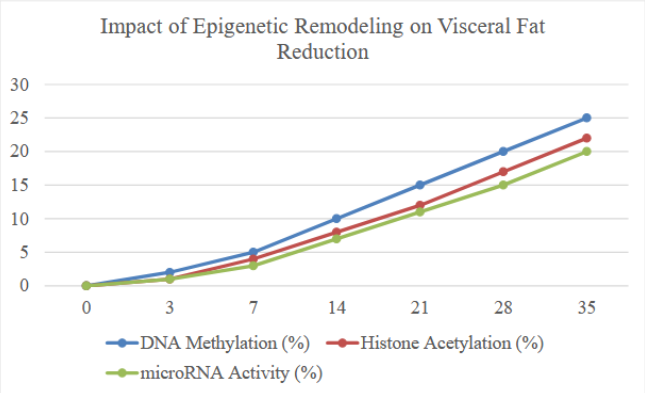

Figure 2. Impact of Epigenetic Remodeling on Visceral Fat Reduction. Source: Own authorship.

Conclusion

Epigenetic processes mediate the metabolic changes linked to intermittent fasting, especially in the management of visceral fat, according to the current work. Recent research provides evidence that changes in DNA methylation, modifications to histones, and regulation of non-coding RNA all work together to impact lipid metabolism and energy balance. The results of these processes are particularly noticeable in visceral adipose tissues and include better insulin sensitivity, regulated triglyceride storage, and accelerated fatty acid oxidation. The findings reviewed show beneficial effects on metabolism, although these effects vary in degrees and durations. This is due to differences in fasting regimen, genetics, and environmental influences. Because of these factors, IF can be viewed as a promising, but context-dependent, option for metabolic improvement. More longitudinal and mechanistic studies are necessary to address the persistence of epigenetic fasting changes and to define the characteristics of specific populations that predict a response. Such information is crucial for developing individualized, evidence-based dietary plans tailored to the fasting mechanisms on a molecular level.

Study Limitations

Relying on results from short-term and diverse research limits the causal interpretation of fasting-induced epigenetic modifications, which is a limitation of this study. It is possible that variations in fasting protocols, participant characteristics, and analytical methodologies introduced bias and limited comparability across results. Further clouding the issue of direct gene-metabolism links is the fact that the majority of the evidence is associative rather than mechanistic. To confirm and expand upon these findings, future studies should use standardized, longitudinal, and multi-omics methodologies.

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Data Sharing Statement

The data supporting the findings of this study are derived from previously published studies and publicly available datasets cited in the References section. No new experimental or human participant data were generated for this review. All referenced sources are accessible through the respective journals or public repositories.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

References

1. Vo N, Zhang Q, Sung HK. From fasting to fat reshaping: exploring the molecular pathways of

intermittent fasting-induced adipose tissue remodeling. Journal of Pharmacy & Pharmaceutical Sciences. 2024 Jul 22;27:13062. https://doi.org/10.3389/jpps.2024.13062

2. Yang Y. Fasting and autophagy and its effect on health. 2023 June 13. https://doi.org/10.5772/intechopen.1008728
3. Verma N, Thakkar N, Phillips J, Ealey K, Sung HK. Dynamic remodeling of white adipose tissue by intermittent fasting. Current Opinion in Food Science. 2020 Aug 1;34:21-9. https://doi.org/10.1016/j.cofs.2020.10.023
4. Asif S, Morrow NM, Mulvihill EE, Kim KH. Understanding dietary intervention-mediated epigenetic modifications in metabolic diseases. Frontiers in Genetics. 2020 Oct 15;11:590369. https://doi.org/10.3389/fgene.2020.590369
5. Yuliyanasari N, Rejeki PS, Hidayati HB, Subsomwong P, Miftahussurur M. The effect of intermittent fasting on preventing obesity-related early aging from a molecular and cellular perspective. Journal of medicine and life. 2024 Mar;17(3):261. https://doi.org/10.25122/jml2023-0370
6. Karim A, Raji Z, Habibi Y, Khalloufi S. A review on the hydration properties of dietary fibers derived from food waste and their interactions with other ingredients: Opportunities and challenges for their application in the food industry. Critical reviews in food science and nutrition. 2024 Dec 20;64(32):11722-56. https://doi.org/10.1080/10408398.2023.2243510
7. Akrami R, Chitsaz H, Ahmadi Z. Effect of dietary dehydrated sour lemon peel (Citrus limon) powder on metabolic enzymes, serum biochemistry and stress status of rainbow trout (Oncorhynchus mykiss) juvenile. International Journal of Aquatic Research and Environmental Studies. 2024 May 10;4(1):91-9.
8. Mustika S, Sofia ER, Sari NA, Poetri LN, Yudhanto HS, Handayani D. The Effects of Traditional Asian Diet on Metabolism, Gut Microbiota, and Liver Tissue in NASH Rats. Natural and Engineering Sciences. 2024 Sep 1;9(2):309-25. https://doi.org/10.28978/nesciences.1574444
9. Abulail RN. Enhanced Fitness Proportionate Selection Algorithm for Parent Selection in Genetic Algorithms. J. Internet Serv. Inf. Secur. 2025;15(1):257-70. https://doi.org/10.58346/JISIS.2025.I1.016
10. Nazarova J, Bobomuratov T. Evaluating the Clinical Utility of Genetic Testing in Guiding Medication Selection. Clinical Journal for Medicine, Health and Pharmacy. 2023 Oct 9;1(1):64-72.
11. Menon K, Patil S. Assessing Terminology Gaps in Global Health Guidelines: AWHO Terminology Audit. Global Journal of Medical Terminology Research and Informatics. 2023 Dec 29;1(1):5-8.
12. Kapoor R, Iyer S. Renewable Energy Integration in Sustainable Healthcare Systems. International Journal of SDG's Prospects and Breakthroughs. 2024 Dec 30:7-12.
13. Hossain DS, Bakhshi DS, Raihan DM, Zaffar H. Gastrointestinal impact of flatulence-causing compounds in foods: A scientometric study. Indian Journal of Information Sources and Services. 2024 Sep 30;14(3):110-4. https://doi.org/10.51983/ijiss-2024.14.3.15
14. Pedroso JA, Wasinski F, Donato Jr J. Prolonged fasting induces long-lasting metabolic consequences in mice. The Journal of Nutritional Biochemistry. 2020 Oct 1;84:108457. https://doi.org/10.1016/j.jnutbio.2020.108457
15. Malešević Z, Govedarica-Lučić A, Bošković I, Petković M, Đukić D, Đurović V. Influence of different nutrient sources and genotypes on the chemical quality and yield of lettuce. http://dx.doi.org/10.59456/afts.2023.1529.049M
16. Endo S, Uto A, Miyashita K, Sato M, Inoue H, Fujii K, Hagiwara A, Ryuzaki M, Oshida T, Kinouchi K, Itoh H. Intermittent fasting sustainably improves glucose tolerance in normal weight male mice through histone hyperacetylation. Journal of the Endocrine Society. 2023 Jul;7(7):bvad082.
17. Wilson RA, Stathis CG, Hayes A, Cooke MB. Intermittent fasting and high-intensity exercise elicit sexual-dimorphic and tissue-specific adaptations in diet-induced obese mice. Nutrients. 2020 Jun 12;12(6):1764. https://doi.org/10.1210/jendso/bvad082
18. Masi S, Ambrosini S, Mohammed SA, Sciarretta S, Luescher TF, Paneni F, Costantino S. Epigenetic remodeling in obesity-related vascular disease. Antioxidants & redox signaling. 2021 May 20;34(15):1165-99. https://doi.org/10.1089/ars.2020.8040
19. Surugiu R, Iancu MA, Vintilescu ȘB, Stepan MD, Burdusel D, Genunche-Dumitrescu AV, Dogaru CA, Dumitra GG. Molecular mechanisms of healthy aging: the role of caloric restriction,

intermittent fasting, mediterranean diet, and ketogenic diet—a scoping review. Nutrients. 2024 Aug 28;16(17):2878. https://doi.org/10.3390/nu16172878.

20. Herzog CM, Vavourakis CD, Theeuwes B, Redl E, Watschinger C, Knoll G, Hagen M, Haider A, Platzer HP, Kumar U, Zollner-Kiechl S. Systemic multi-omic remodelling underlies health benefits of intermittent fasting. bioRxiv. 2025 Sep 4:2025-08. https://doi.org/10.1101/2025.08.30.673138.