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THE ROLE OF AUTOPHAGY AND INTERMITTENT FASTING IN METABOLIC HEALTH : A SCOPING REVIEW

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ABSTRACT

Background: Intermittent fasting (IF) and autophagy have garnered significant attention in recent years due to their potential therapeutic effects on metabolic health and chronic diseases. Autophagy, a crucial cellular process for maintaining homeostasis, is closely linked with the metabolic changes induced by intermittent fasting. This scoping review aims to explore and summarize the existing literature on the relationship between IF and autophagy, particularly in the context of metabolic and medical research. **Methods:** A Scopus search identified 158 articles. After applying filters for publication year, article type, language, and medical relevance, 18 articles met the criteria for screening. Following title, abstract, and full-text assessment, 14 studies were included in this review. **Results:** Fourteen studies met the inclusion criteria. Most demonstrated that intermittent fasting (IF) enhances autophagy, although a few reported inconsistent effects. IF also produced several health benefits, including improved metabolic outcomes, weight reduction, anti-inflammatory and antioxidant activity, neuroprotection, and cardiovascular improvements. Some studies showed limited effectiveness of IF under specific conditions, while others compared IF with alternative interventions and reported mixed advantages. Overall, the evidence supports IF as a promising non-pharmacological strategy to modulate autophagy and improve metabolic and cellular health. **Conclusion:** The studies reviewed highlight the beneficial effects of IF on autophagy, particularly metabolic diseases and aging. This review emphasizes the need for more targeted studies to fully understand the role of IF in modulating autophagy and its broader health implications.

INTRODUCTION

Autophagy is a highly regulated process by which cells break down and remove damaged organelles, misfolded proteins, and other intracellular debris, allowing for cellular recycling and repair (1,2). This process plays a crucial role in maintaining cellular homeostasis (3,4) and is particularly important in tissues with high metabolic demands, such as the liver, heart, and skeletal muscles. Dysregulated autophagy has been implicated in the pathogenesis of various diseases, including neurodegenerative disorders, cardiovascular diseases, and metabolic conditions like obesity and type 2 diabetes (5,6). As such, stimulating autophagy has become a potential therapeutic target for preventing or mitigating the effects of these conditions.

Intermittent fasting (IF) has gained significant popularity not only as a weight loss strategy but also as a means to improve overall health (7). The cyclic pattern of eating and fasting creates metabolic stress that encourages the activation of autophagy (8), particularly during the fasting period when the body shifts from using glucose as a primary energy source to utilizing fatty acids and ketones. This metabolic switch enhances fat oxidation, reduces insulin resistance, and promotes the clearance of damaged

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cells through autophagy. As a result, IF has been linked to numerous health benefits, including improved cardiovascular health (9), reduced inflammation (10), and enhanced cognitive function (11,12).

Despite the growing body of evidence supporting the benefits of IF and autophagy activation, there are still many unanswered questions regarding the optimal fasting protocols, the long-term safety of these interventions, and their effects in different populations. For instance, while IF has shown promise in improving metabolic health in obese individuals, its effects on lean individuals or those with specific health conditions, such as diabetes or cardiovascular disease, are not fully understood. Moreover, the extent to which autophagy activation contributes to the health benefits of intermittent fasting, as opposed to other metabolic changes such as insulin sensitivity or fat oxidation, remains an area of active investigation.

In this review, we aim to bridge the gap between the molecular mechanisms of autophagy and the clinical applications of intermittent fasting. By analyzing recent research findings, we will explore how intermittent fasting-induced autophagy can be harnessed to improve metabolic health and prevent disease. Additionally, we will discuss the potential for combining IF with pharmacological agents that target autophagy pathways, such as rapamycin, to enhance therapeutic outcomes. Through this comprehensive exploration, we hope to provide insights that can inform future clinical trials and guide the development of personalized nutrition strategies aimed at optimizing metabolic health and extending lifespan.

METHODS

This study employed the scoping review method to map out the existing research on IF and autophagy, to provide a comprehensive overview of current findings and identify gaps in the literature. The scoping review approach is particularly suitable for exploring broad topics that have not yet been thoroughly reviewed or need clarification across various studies. In this review, we aimed to understand the effects of IF on autophagy and its health implications, particularly in the field of metabolic disorders and related conditions. This study utilizes secondary data and therefore does not require ethical clearance.

The literature search was conducted using the Scopus database, one of the most extensive and authoritative sources of scientific literature. The search was initiated using the keyword “intermittent fasting and autophagy”. A total of 158 articles were initially identified from Scopus. After applying the inclusion and exclusion criteria, limiting to English-language publications, the year range 2018–2024, experimental study designs, and relevant medical scope, 140 articles were excluded, leaving 18 articles for title and abstract screening. Two articles were excluded at this stage. The remaining 16 articles underwent full-text eligibility assessment, resulting in the exclusion of 2 articles that were not relevant. Ultimately, 14 articles were included in the final review.

After identifying the eligible studies, data from each article were systematically extracted using a standardized charting framework. Each study was summarized according to three core components: (1) Title, (2) Purpose, and (3) Results/Findings, as presented in Table 1. This extraction process enabled the reviewers to clearly compare study objectives, methodological approaches, and key outcomes related to the relationship between intermittent fasting (IF) and autophagy.

The structured synthesis allowed for the identification of consistent patterns, variations in experimental findings, and the overall strength of evidence regarding the influence of IF on autophagy and its potential health impacts. In addition, this process facilitated the detection of gaps in the existing literature, highlighting areas where further experimental work is needed to deepen mechanistic understanding or address unresolved questions in the field.

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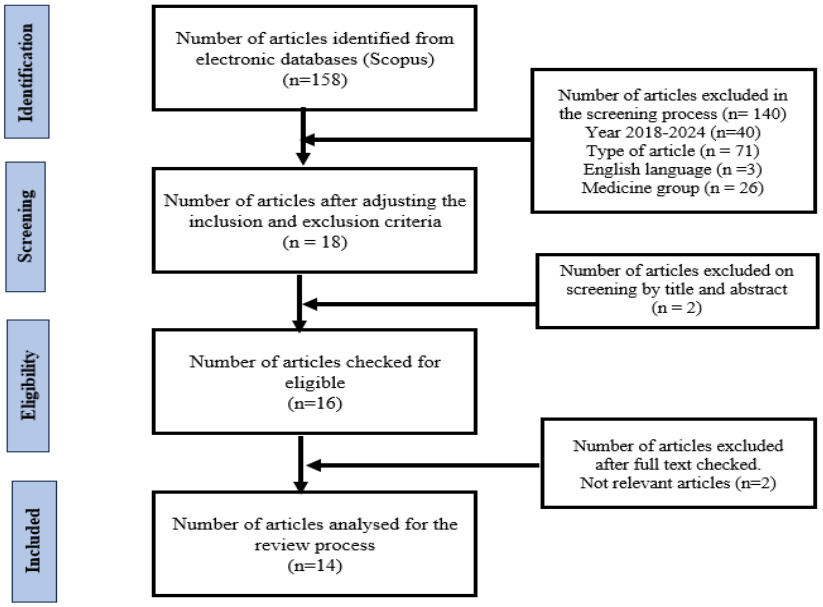

Figure 1. PRISMA Flowchart

Table 1. Extraction Data from Eligible Articles

Title	Purpose	Results/Findings
The effect of prolonged Intermittent Fasting on autophagy, inflammasome, and senescence gene expressions: An exploratory study in healthy young males (13)	Investigate the effects of prolonged IF on autophagy, inflammasome, and senescence gene expressions in healthy young males.	Increased autophagy markers (ATG5, ULK1, BECN1), upregulation of inflammasome (NLRP3, IL-1β), and reduced TNF-α levels over time.
Intermittent Fasting reduces mouse body fat while maintaining muscle mass by regulating protein synthesis and autophagy (14)	To investigate the effect of IF on the regulation of skeletal muscle protein metabolism in response to nutrient supplementation during fasting.	IF group had better insulin sensitivity, lower body and fat weights, maintained muscle mass, increased protein synthesis markers, and reduced autophagy markers compared to ad libitum.
Intermittent Fasting activates markers of autophagy in mouse liver, but not muscle from mice or humans (15)	To examine the effect of IF on markers of autophagy in liver and skeletal muscle in mice and humans.	Fasting increased autophagy markers in mouse liver but not skeletal muscle. In humans, autophagy markers in muscle were reduced, likely due to weight loss rather than fasting.
Alternate-day fasting and time-restricted feeding may confer similar neuroprotective effects during aging in male rats (16)	The study examined how ADF and TRF protect against aging in male rats by assessing oxidative stress, mitochondrial function, and autophagy markers.	ADF and TRF reduce oxidative stress, enhance mitochondrial function, activate autophagy, and protect neurons from age-related degeneration, making them promising anti-aging dietary strategies
Intermittent fasting, exercise, and dietary modification induce unique transcriptomic signatures of multiple tissues governing metabolic homeostasis during weight loss and rebound weight gain (17)	To investigate the transcriptomic changes in multiple tissues during weight loss and rebound weight gain in response to interventions such as IF, exercise (EX), and dietary modifications in obese mice.	EX and IF improved metabolism and reduced inflammation, with IF also altering key genes in fat tissue. DR aided metabolic balance but had minimal gene impact. Genes like Gdf15 and Tfc were linked to obesity resistance and metabolic traits
Ketone body β-hydroxybutyrate is an autophagy-dependent vasodilator (18)	To explore how β-hydroxybutyrate (βHB), a ketone body, is linked to autophagy and its vasodilatory effects, particularly in the context of high-salt diets.	βHB promotes vasodilation via potassium channels, independent of Gpr109a. High-salt diets reduce βHB and autophagy, impairing vascular function, but restoring βHB prevents endothelial dysfunction.
Chronic activation of cardiac Atg-5 and pancreatic Atg-7 by IF alleviates acute myocardial infarction in old rats (19)	To investigate the protective effects of IF against acute myocardial infarction (AMI) in old rats, focusing on autophagy activation in the heart and pancreas.	IF increased cardiac and pancreatic autophagy gene expression, reduced heart injury markers, lowered visceral fat, improved insulin resistance, and decreased heart inflammation and oxidative stress.
Autophagy induction contributes to the neuroprotective impact of intermittent fasting on the acutely injured spinal cord (20)	To investigate the therapeutic effect of IF on neuronal survival after spinal cord injury (SCI) in rats, focusing on the regulation of autophagy.	IF reduced apoptosis-related markers (e.g., cleaved caspase 3) and enhanced autophagy markers (e.g., LC3-II, beclin 1) in SCI rats. Autophagy inhibition reversed these effects, while IF activated the AMPK/mTOR pathway, enhancing lysosome function.

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Towards a fasting-mimicking diet for critically ill patients: the pilot randomized crossover ICU-FM-1 study (21)	The study examined if a 12-hour fast triggers metabolic responses in critically ill patients and assessed the feasibility of tracking autophagy via blood samples.	12-hour fasting increased serum bilirubin and beta-hydroxybutyrate (BOH), reduced insulin requirements, and decreased insulin-like growth factor I (IGF-I). Autophagic markers were largely unaffected in blood samples.
Intermittent Fasting Attenuates High-Fat Diet-Induced Cerebellar Changes in Rats: Involvement of TNF-α, Autophagy, and Oxidative Stress (22)	To investigate the effects of IF on cerebellar changes in high-fat diet (HFD)--fed rats and its relation to TNF-α, autophagy, and oxidative stress.	IF lowered cerebellar TNF-α, improved oxidative stress and autophagy markers, reduced body weight, enhanced metabolic profiles, and balanced AMPK–mTOR signaling.
Philippine Consensus Statement on the Use of Ketogenic Diet and Intermittent Fasting Diet on Adults for Weight Reduction (23)	To provide a consensus statement on the use of the ketogenic diet (KD) and IF for weight reduction based on available evidence.	KD and IF were found to be effective in weight reduction, with IF being associated with improved cardiovascular status and autophagy activation. However, adverse effects such as headaches and reduced energy levels were noted, particularly for IF in normal-weight individuals.
Dysfunction of aged liver of male albino rats and the effect of intermitted fasting; Biochemical, histological, and immunohistochemical study (24)	To investigate the protective effects of IF on age-related degenerative changes in the liver.	IF reduced liver injury and oxidative stress, boosted autophagy, lowered apoptosis, and improved liver histology by reducing fibrosis and inflammation.
Dietary and Pharmacological Interventions That Inhibit Mammalian Target of Rapamycin Activity Alter the Brain Expression Levels of Neurogenic and Glial Markers in an Age- and Treatment-Dependent Manner (25)	To investigate how short-term IF and rapamycin affect cellular and molecular changes in the brains of young and old zebrafish, focusing on age-dependent effects.	IF improved glucose levels and boosted neurogenic markers across ages, while rapamycin reduced neurogenesis in older animals. Effects on autophagy and mTOR were age-dependent, with IF more effective in older animals and rapamycin in younger ones
Therapeutic effect of autophagy induced by rapamycin versus Intermittent Fasting in animal model of fatty liver (26)	To compare the effects of rapamycin and IF on inducing autophagy in rats with experimentally induced non-alcoholic fatty liver disease (NAFLD).	Rapamycin and IF both improved liver health via autophagy, with rapamycin showing greater effects than IF in reducing liver damage

Table 2. Thematic Grouping Based on Result or /Findings

Main Findings	References
Autophagy increased	(13), (15), (19), (20), (22), (27), (25), (26), (17)
Autophagy did not increase / inconsistent	(15), (21) (17)
Metabolic effects and weight reduction	(14), (22), (19), (23)
Anti-inflammatory & antioxidant effects	(22), (19), (16), (24)
Neuroprotective effects	(20), (16), (25), (27)
Cardiovascular & vascular effects	(18), (19)
IF not effective in certain conditions	(21), (15)
Comparison of interventions (IF vs others)	(26), (17)

The thematic synthesis of all articles in Table 2 shows that research on intermittent fasting (IF) yields diverse findings, which can be organized into several major themes: enhanced autophagy, inconsistent autophagy responses, metabolic and weight-related benefits, anti-inflammatory and antioxidant effects, neuroprotective mechanisms, cardiovascular outcomes, limited effectiveness under certain conditions, and comparisons with other interventions.

The following discussion summarizes these themes and integrates evidence across studies.

1. Increased Autophagy ((13), (15), (19), (20), (22), (24), (25), (26), (17))

A wide range of studies demonstrated that intermittent fasting (IF) robustly induces autophagy in multiple tissues and experimental contexts. In healthy adults (13), prolonged IF upregulated key autophagy-related genes (ATG5, ULK1, BECN1), reflecting enhanced cellular turnover and reduced inflammation. In experimental models, IF increased autophagy in liver but not skeletal muscle in mice (15), while other animal studies consistently showed autophagy activation in the liver and pancreas (19) and enhanced neuronal autophagy and survival in spinal cord injury models (20).

Further evidence shows that IF stimulates autophagy under metabolic stress conditions, such as in fatty liver disease models where IF improved liver histology and increased autophagy markers (17). Studies on high-fat diet–induced metabolic dysfunction demonstrated that IF reduced inflammatory cytokines and boosted autophagy activation (22). Age-related studies also confirmed increased autophagy in aging liver tissues (24) and

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enhanced mitochondrial and oxidative stress regulation (25).

Enhanced autophagy effects were amplified when IF was compared or combined with other metabolic interventions. In particular, IF demonstrated autophagy induction comparable to rapamycin in fatty liver disease models (26), further supporting the role of IF as a potent physiological autophagy trigger across tissues, ages, and metabolic states.

2. Autophagy Did Not Increase / Inconsistent ((15), (21))

Two studies revealed contexts in which autophagy activation was limited or inconsistent. Study (15) showed that although IF increased autophagy markers in liver tissue, it did not induce autophagy in skeletal muscle of mice, and in humans, autophagy markers were reduced, likely due to weight loss rather than enhanced autophagy signaling.

In critically ill patients (21), a 12-hour fasting window did not lead to measurable increases in autophagy markers. This lack of responsiveness is likely due to severe physiological dysregulation, the catabolic state of critical illness, and the overriding influence of inflammatory and metabolic disturbances that may suppress normal fasting responses. These findings highlight tissue-specific and condition-specific variability in autophagy induction under IF.

3. Metabolic Effects and Weight Reduction ((14), (22), (19), (23), (17))

IF produced consistent metabolic improvements across human and animal studies. In healthy young adults (14), IF enhanced insulin sensitivity, reduced body fat, improved protein metabolism, and lowered inflammatory markers. In high-fat diet models (22), IF reduced oxidative stress, improved metabolic profiles, and enhanced autophagy, demonstrating interplay between metabolic and cellular stress pathways.

Cardiometabolic benefits were also evident in models of cardiac injury and pancreatic dysfunction (19), where IF reduced systemic inflammation, lowered visceral fat, and improved overall metabolic homeostasis. In models assessing long-term metabolic adaptations (23), IF demonstrated age-dependent improvements in glucose levels and neurogenic markers.

Furthermore, in metabolic disease models (17), IF markedly improved liver structure, reduced fibrosis, and enhanced mitochondrial and metabolic function. Collectively, these studies support IF as an effective metabolic intervention across species, ages, and disease conditions.

4. Anti-Inflammatory & Antioxidant Effects ((22), (19), (16), (24))

Several studies highlighted the strong anti-inflammatory and antioxidant effects of IF. In models with high inflammatory burden (22), IF reduced TNF-α and oxidative stress markers while boosting autophagy. Study (19) showed similar reductions in systemic and pancreatic inflammation, decreased visceral fat, and lowered markers of metabolic stress.

IF also produced antioxidant and mitochondrial protective effects in aging rat models (16), where it mitigated oxidative damage and supported autophagic turnover. Study (24) demonstrated that IF reduced oxidative stress in aged livers, decreased apoptosis, and improved histological structure through enhanced autophagy and reduced inflammation. These findings confirm that IF exerts broad anti-inflammatory and antioxidant actions across tissues.

5. Neuroprotective Effects ((20), (16), (25), (27))

Neuroprotection emerged as a key theme across multiple studies. In spinal cord injury models (20), IF reduced apoptosis, increased neuronal survival, and modulated autophagic pathways such as LC3-II and beclin-1. IF also protected neurons from age-related degeneration in rodent models (16), with benefits linked to mitochondrial improvement and enhanced autophagy.

Study (25) further showed that IF improved markers of oxidative stress and promoted autophagy in both older rats and zebrafish, with age-dependent variations in response. In a comparative study (27), ketone diet (KD) and IF both offered neuroprotective effects by modulating NF-κB signaling and oxidative stress; however, the magnitude of response varied depending on the model and intervention. These studies collectively illustrate that IF supports neural

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resilience and may modulate pathways relevant to aging and neurodegeneration.

6. Cardiovascular & Vascular Effects ((18), (19))

Two studies specifically investigated cardiovascular outcomes. Study (18) showed that β-hydroxybutyrate metabolic product of fasting, improved vasodilation, enhanced vascular channel activity, and protected against salt-induced endothelial dysfunction, linking IF to vascular homeostasis.

In cardiac injury models (19), IF reduced inflammation in both heart and pancreas, mitigated visceral fat accumulation, and improved metabolic markers, demonstrating systemic cardioprotective effects. Together, these findings indicate that IF promotes cardiovascular health through metabolic, inflammatory, and vascular pathways.

7. IF Not Effective in Certain Conditions ((21), (15))

Two studies documented limited effectiveness of IF. In critically ill patients (21), a fasting-mimicking protocol did not significantly change autophagy pathways or metabolic markers, indicating that the disease state may overwhelm normal fasting physiology.

Similarly, study (15) reported that IF did not induce autophagy in skeletal muscle of mice or in human subjects, likely because caloric restriction and weight loss can reduce autophagy markers independent of metabolic stress pathways. These findings emphasize that IF may not be universally applicable, particularly in acute illness or tissues with low fasting responsiveness.

8. Comparison of Interventions ((26), (17))

Comparative studies provided insight into how IF performs relative to other metabolic interventions. In fatty liver models (26), IF increased autophagy and improved liver health, but rapamycin produced even stronger effects, especially in older animals, suggesting age-dependent differences in pathway activation.

Meanwhile, study (17) compared IF with other models of liver injury and demonstrated that IF offered significant benefits in reducing fibrosis and improving metabolic function, indicating that IF may serve as an effective standalone or complementary intervention. These comparisons highlight that although IF is beneficial, its relative effectiveness varies depending on the comparator, tissue type, and physiological context.

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CONCLUSION

Overall, the included studies indicate that intermittent fasting (IF) has meaningful potential to induce autophagy and improve metabolic, cardiovascular, and neuroprotective outcomes. The evidence also shows that autophagy responses to IF are both tissue-specific and age-dependent, with mTOR playing a central regulatory role. While IF demonstrates promising biological effects, inconsistencies across studies highlight the need for further research to refine fasting protocols, evaluate long-term safety, and determine individual factors influencing responsiveness. IF, alone or in combination with autophagy-modulating agents such as rapamycin, remains a promising non-pharmacological approach for enhancing autophagy and supporting metabolic health.

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