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Intermittent Fasting and Cognitive Performance in Adults: A Narrative Review

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ABSTRACT

Background: Intermittent fasting (IF) is widely promoted for metabolic and brain health, and mechanistic research suggests that fasting-induced metabolic switching could influence brain function. Human evidence on cognition is, however, limited and heterogeneous, and acute (single) fasting episodes differ from repeated IF regimens in their physiological and behavioural effects.

Objective: To review human evidence on IF and cognitive performance in adults, focusing on acute responses, repeated interventions, potential cognitive adaptation, and proposed mechanisms.

Methods: This narrative review considered human experimental studies, randomised controlled trials, observational studies, and systematic reviews identified through scholarly searches on IF, time-restricted eating, alternate-day fasting, experimental fasting, and cognitive performance. Animal and laboratory studies were included to explain potential mechanisms. Findings were synthesised by fasting approach and cognitive domain.

Results: Human studies suggest that cognitive performance is generally maintained during the acute fasting conditions investigated, although responses vary according to fasting duration, participant characteristics, and cognitive domains. Repeated IF interventions have generally shown no consistent cognitive advantage over control diets or continuous calorie restriction. Nevertheless, selected studies have reported improvements in specific cognitive outcomes, particularly among adults with obesity and older adults with mild cognitive impairment. Potential mechanisms include altered ketone utilisation, neurotrophic signalling, regulation of oxidative stress, and modulation of neuroinflammation. Although experimental findings support these biological pathways, their contribution to cognitive improvements in humans remains incompletely understood.

Conclusion: Intermittent fasting represents an emerging area of research in nutritional approaches to brain health and cognitive function. Current human findings suggest that cognitive performance is generally preserved under several studied fasting conditions, with preliminary evidence of improvements in selected cognitive domains. IF may be explored as a potential adjunctive lifestyle approach alongside appropriate

medical care; however, the relationship between fasting duration, repeated exposure, and cognitive adaptation remains an important area for further investigation. Larger, longer-duration clinical trials using standardised cognitive assessments are needed to clarify these effects and their potential relevance to cognitive health.

Keywords: Intermittent Fasting; Cognitive Performance; Naturopathy; Working Memory; Metabolic Switching.

1. INTRODUCTION

Cognitive performance underpins everyday functioning, including learning, memory, attention, decision-making and problem-solving, and its preservation is central to functional independence and quality of life, particularly with advancing age. Cognitive change is influenced by ageing, metabolic disturbance, chronic inflammation, oxidative stress and neurological disease, so modifiable lifestyle factors that may support cognitive health are of considerable clinical and scientific interest.

Among dietary approaches, intermittent fasting (IF), characterised by alternating periods of eating and fasting, has been widely investigated [1, 2]. Unlike continuous calorie restriction (CR), which lowers daily energy intake, IF primarily alters the timing and frequency of eating, although some protocols also reduce energy intake [2,3]. Common approaches are time-restricted eating (TRE), alternate-day fasting (ADF) and the 5:2 intermittent energy-restriction regimen, which differ in fasting duration, frequency and energy intake. During fasting, falling insulin and increased fat mobilisation promote ketone body production, giving the brain an alternative fuel; this transition is commonly described as the metabolic switch [4,5]. Intermittent metabolic switching has been proposed to influence brain-derived neurotrophic factor (BDNF) signalling, oxidative stress responses, neuroinflammation and synaptic plasticity, which provides a biological rationale for studying fasting and cognition. However, much of this mechanistic evidence comes from animal and preclinical research, and its relevance to measurable cognitive outcomes in humans remains to be established [6].

A key distinction is between acute and repeated fasting. Acute fasting is a single period of reduced or absent energy intake, during which performance may be influenced by hunger, mood, stress, glucose availability and ketone production. Repeated fasting means following a structured regimen for weeks or months, which may allow physiological and behavioural adaptation but is often accompanied by changes in body weight, energy balance, sleep and metabolic health [3, 4]. These concurrent changes make it difficult to determine whether a cognitive outcome reflects fasting itself or broader metabolic and lifestyle change. Previous investigations have reported findings ranging from no cognitive impairment to changes in selected domains, including executive function and working memory [7,8].

Earlier systematic have examined fasting in relation to cognition, brain health, BDNF and neurodegenerative conditions, and a meta-analysis of randomised trials of dietary restriction reported a small pooled cognitive benefit [9,10]. Nevertheless, the human evidence remains heterogeneous, findings from acute experiments may not generalise to commonly practised IF regimens, and the effects of IF should be distinguished from those of CR and accompanying metabolic change. This narrative review therefore critically examines the human evidence on IF and cognitive performance in adults. It separates acute fasting responses from outcomes of repeated interventions, evaluates findings across cognitive domains, discusses the proposed mechanisms while distinguishing human findings from preclinical

evidence, and considers methodological limitations, modifying factors, clinical implications and research priorities.

2. APPROACH TO THE NARRATIVE REVIEW

This article is a narrative review and does not follow systematic review methodology. Literature was identified through PubMed searches conducted in October 2026, using terms related to intermittent fasting, time-restricted eating, alternate-day fasting, experimental fasting, cognitive performance, attention, memory, working memory, executive function and metabolic switching. Relevant publications were explored through accessible journal websites, institutional repositories and open-access sources; reference lists of key reviews were also screened. Priority was given to systematic reviews and meta-analyses, randomised and controlled human trials, prospective cohort studies and controlled physiological studies, Animal and laboratory studies were included only where they helped explain potential biological mechanisms or clarify limitations in interpreting human findings.

Study selection was performed by a single reviewer on the basis of relevance to the review objectives. Major bibliographic databases were not directly searched, screening was not performed in duplicate, and formal risk-of-bias assessment, evidence grading and statistical pooling were not undertaken. The review therefore does not claim to be exhaustive. Reporting was informed by the Scale for the Assessment of Narrative Review Articles (SANRA) [27]. Human cognitive outcomes were considered separately from proposed mechanisms supported by preclinical evidence.

3. INTERMITTENT FASTING: CONCEPTS AND PHYSIOLOGICAL BASIS

3.1 Definition and Common Types of Intermittent Fasting

IF refers to dietary patterns in which periods of eating alternate with planned periods of fasting or substantial energy restriction; it has been defined broadly as abstinence from, or strong limitation of, energy intake for roughly 12 to 48 hours, alternated with periods of unrestricted intake [5]. Unlike conventional calorie-restricted diets, IF primarily modifies the timing and frequency of food consumption, although some protocols also reduce overall energy intake [21, 23]. The commonly studied approaches (TRE, ADF and the 5:2 regimen) differ in fasting duration, feeding window, energy consumed during restricted periods and frequency of fasting, so their physiological and cognitive effects should not be considered interchangeable [1, 2].

Time-Restricted Eating confines daily food intake to a consistent window, commonly with 12:12, 14:10 or 16:8 schedules (fasting hours: feeding hours). It is practised daily and does not usually involve a full day without food. TRE does not necessarily require a prescribed energy deficit, although spontaneous reductions in intake can occur, so its effects may reflect meal timing, energy intake and circadian influences together. Cognitive studies of TRE have been short, for example a four-week randomised pilot of 16:8 eating in adult women and a two-month study of daily 16-hour fasting [7, 8] (Table 2).

Alternate-day fasting or markedly restricted-intake days with regular eating days. Fasting days may involve complete abstinence or a small meal (modified ADF), which influences hunger, tolerability, ketone production and weekly energy intake; the longer fasting intervals may alter fuel use more than daily fasting, depending on duration, physical activity and individual metabolism. A three-month randomised trial in adults with obesity compared ADF with Mediterranean and ketogenic diets, all prescribed as hypocaloric, and reported cognitive improvement in the ADF group [9]. The fasting-day energy allowance is not detailed in the source accessed, and concurrent weight loss limits attribution of

the improvement to fasting itself. The 5:2 approach involves usual eating on five days and substantial restriction on two non-consecutive days; it is better understood as intermittent energy restriction than as complete fasting. An eight-week study of women with overweight or obesity (N = 46) compared intermittent energy restriction with continuous restriction, both prescribed approximately 70% of calculated energy requirements [10]. This energy-matched design helps to separate the pattern of restriction from total intake. Direct cognitive evidence for the 5:2 approach was limited in the literature identified, so findings from TRE or ADF should not automatically be extrapolated to it.

Fasting duration does not necessarily indicate the degree of energy restriction. Some individuals compensate for a shorter feeding window by consuming their usual energy requirements, whereas other protocols deliberately reduce weekly intake; this matters when interpreting metabolic and cognitive outcomes.

The distinction between acute and repeated fasting is central to this review. **Acute fasting** is a single period of food abstinence, generally lasting several hours to approximately 24–48 hours in the experimental studies discussed here, with cognition assessed during or immediately after the fast; performance may be influenced by hunger, glucose and ketone availability, hydration, mood and stress. **Repeated fasting** involves a structured schedule (daily TRE, ADF or intermittent energy restriction) followed for weeks to months, with testing during the intervention, after an overnight fast or at the end of a designated fast, and with possible additional influences from weight change, energy intake, sleep and dietary quality. The physiological state at testing should therefore be considered separately from intervention duration: for example, an eight-week study also assessed cognition after a 24-hour fast in the IF group [10]. Acute studies address immediate responses, whereas repeated-intervention studies address outcomes of sustained dietary patterns and possible adaptation.

3.2 Metabolic Adaptation to Fasting

As the interval since the last meal lengthens, insulin concentrations generally fall, glycogen helps maintain blood glucose and lipolysis increases; with continued fasting, fatty acids become a major energy source and the liver produces ketone bodies [21, 22]. The brain normally relies substantially on glucose, but during prolonged starvation ketone bodies contribute to cerebral energy requirements and reduce glucose dependence [11, 12]. The extent of ketone use depends on fasting duration and metabolic conditions, and findings from prolonged starvation should not be extrapolated to shorter TRE schedules.

Repeated fasting has been proposed to produce physiological and behavioural adaptation, potentially involving substrate utilisation, hunger responses, sleep and metabolic regulation. However, evidence that such adaptation translates into improved cognitive performance in humans is insufficient: in one eight-week intervention, mood, sleep and cognitive outcomes did not differ between IF and continuous CR [10]. The mechanisms discussed in Section 4 provide a rationale, but much of the supporting evidence comes from animal experiments or indirect findings rather than adequately controlled human cognitive studies [14, 25]. Physiological adaptation should therefore be regarded as a plausible hypothesis, not an established explanation for cognitive change. Longitudinal studies are needed to determine whether repeated fasting alters human cognition independently of weight loss, energy restriction and other lifestyle changes.

4. BIOLOGICAL MECHANISMS LINKING FASTING AND COGNITION

IF has been proposed to influence cognition through changes in cerebral energy metabolism, ketone utilisation, neurotrophic signalling, oxidative stress responses, inflammation and circadian regulation

(Figure 1). The strength of evidence differs considerably between pathways. Metabolic changes during fasting and ketone utilisation have been demonstrated in humans, whereas several proposed neuroprotective mechanisms are supported predominantly by animal and preclinical studies, and evidence that these changes translate into better human cognitive performance is limited. Table 1 summarises the evidence source for each mechanism.

4.1 Brain Energy Metabolism and Ketone Bodies

The brain has a high energy requirement and normally relies primarily on glucose. During fasting, falling insulin and glucose concentrations, greater mobilisation of stored fat and hepatic gluconeogenesis help maintain energy availability [1, 2, 12]. The liver also produces ketone bodies, chiefly beta-hydroxybutyrate and acetoacetate, which cross the blood–brain barrier, can serve as alternative cerebral substrates and may also act as signalling molecules in metabolism, inflammation and stress responses [5, 11, 28]. The capacity to shift between substrates according to availability is described as metabolic flexibility. In classic catheterisation studies of obese patients fasted for 5–6 weeks, ketone bodies replaced glucose as the predominant fuel for brain metabolism [11].

The magnitude of ketosis depends on fasting duration and intensity. The response to a 16-hour TRE schedule may differ substantially from that during a 24- or 48-hour fast, so evidence from prolonged fasting should not be extrapolated to shorter protocols, and whether the modest ketone concentrations produced by commonly practised IF regimens are sufficient to improve human cognition remains uncertain.

Human experimental data suggest that reduced glucose availability does not necessarily cause immediate cognitive impairment. Two days of calorie deprivation combined with aerobic exercise lowered interstitial glucose in young adults without impairing measured cognitive performance, although mood deteriorated [13] (Section 6.1). That study involved acute, severe energy deprivation rather than a conventional IF regimen and cannot be generalised to daily TRE or ADF. IF should also be distinguished from a ketogenic diet: both may raise circulating ketones but differ in composition, energy intake and metabolic exposure, and in the randomised trial in adults with obesity the ketogenic group improved less on one visual-attention outcome than the other groups, showing that increased ketone availability alone cannot be assumed to enhance cognition [9]. Overall, human evidence supports the occurrence of metabolic change during fasting more clearly than a beneficial cognitive effect of that change.

4.2 Brain-derived neurotrophic factor [BDNF] and Neuroplasticity

Brain-derived neurotrophic factor [BDNF] is a neurotrophin involved in neuronal survival, synaptic function, learning, memory and neuroplasticity. IF has been proposed to influence BDNF-related signalling through metabolic switching and cellular stress-response pathways, which may support neuronal adaptation, synaptic plasticity and resistance to cellular stress [5, 6, 14].

The strongest supporting evidence comes from animal experiments. In adult mice, dietary restriction increased hippocampal BDNF protein, and BDNF contributed in part to the enhancement of hippocampal neurogenesis [29]. In old mice, cycles of a fasting-mimicking diet promoted hippocampal neurogenesis and improved cognitive performance, although this diet is not equivalent to IF [32]. A systematic review of the BDNF literature concluded that IF was consistently reported to upregulate BDNF and improve cognition in animal models, and that cognitive outcomes in humans require further study [25].

Translation of these findings to humans remains uncertain. Circulating BDNF does not necessarily reflect its activity within specific brain regions, and peripheral measurements alone cannot establish changes in neuronal plasticity. A systematic review of 16 human studies involving CR and IF regimens found that

five reported a significant increase in BDNF, five reported a decrease, and six found no significant change [30]. Interestingly, a three-month trial of 30% CR in healthy older adults reported improvements in verbal memory despite unchanged BDNF levels [31]. Experimental studies provide evidence that fasting-related metabolic adaptations may promote BDNF expression and neuroplasticity. However, human findings remain variable, highlighting the need for further research to establish whether BDNF-related changes contribute to measurable cognitive improvements

4.3 Oxidative Stress and Neuroinflammation

Oxidative stress arises when reactive oxygen species generation exceeds antioxidant capacity and can damage lipids, proteins and DNA; it has been implicated in neuronal ageing and neurodegeneration. Fasting has been proposed to activate adaptive stress responses that improve antioxidant defence, cellular maintenance and DNA repair [1, 14, 22]. Preclinical studies provide supporting evidence, but it remains uncertain whether such responses occur to a clinically meaningful extent in the human brain under commonly practised IF schedules. An observational study of older adults with mild cognitive impairment (MCI) examined IF in relation to cognition and biochemical markers, including indicators of oxidative stress and DNA damage [15]. Although group differences in these markers were reported alongside cognitive findings, the observational design does not show that oxidative-stress change mediated the cognitive outcomes; diet, metabolic health, physical activity and lifestyle may have contributed.

Neuroinflammation refers to inflammatory processes within the central nervous system, involving microglia and interactions with peripheral inflammatory pathways, and has been associated with neuronal dysfunction and neurodegenerative disease. IF has been proposed to influence inflammatory regulation through energy metabolism, systemic inflammatory signalling, cellular stress responses and gut microbiota–brain interactions [6,9]. In rodents, IF attenuated lipopolysaccharide-induced neuroinflammation and memory impairment [33]. In the randomised trial in adults with obesity, cognitive improvement following ADF was associated with changes in inflammatory markers and gut microbiota composition [9]. These findings are preliminary human evidence of an association, not of causation. The same report included mouse experiments in which microbiota transplanted from participants after different diets influenced cognitive performance and microglial activity, which supports a possible gut–brain pathway but cannot be taken as proof that the same causal pathway operates in humans [9].

Overall, regulation of oxidative stress and inflammation are plausible pathways linking fasting and brain health, but direct clinical evidence that they mediate cognitive change in humans remains limited. Studies combining inflammatory and oxidative biomarkers, microbiome analysis and validated cognitive assessment are needed.

4.4 Circadian Rhythm and Sleep Regulation

Circadian rhythms regulate sleep–wake patterns, hormone secretion, glucose metabolism and alertness over approximately 24 hours, and the timing of food intake is an important signal for peripheral clocks involved in metabolic regulation [24]. TRE may improve circadian alignment by establishing consistent feeding and fasting periods, and early versus late eating has been proposed to influence metabolic rhythms, sleep quality and alertness [5]. In mice, time-restricted feeding attenuated diet-induced metabolic disease, with benefits proportional to fasting duration [34]. In humans, such effects could affect cognition indirectly through attention, memory consolidation and daytime functioning, but they may depend on whether eating windows align with an individual's biological day, as well as sleep duration, chronotype, work schedule and light exposure.

The human evidence identified does not show a consistent cognitive benefit through this pathway. A two-month IF intervention with a daily 16-hour fast reported no significant group-by-time effects on sleep quality, mood or cognition [8], and an eight-week comparison of IF with continuous energy restriction found no significant difference in sleep quality [10]. Human studies directly comparing early and late TRE schedules for cognitive outcomes were not identified. Circadian and sleep effects therefore remain plausible but unestablished contributors to any cognitive effect of IF.

In summary, metabolic substrate change and cerebral ketone utilisation have been demonstrated in humans; BDNF-related neuroplasticity and several cellular stress pathways are supported predominantly by preclinical research; and human findings on oxidative stress, inflammation and sleep are preliminary and do not establish causal mechanisms.

Table 1. Potential Mechanisms Linking Intermittent Fasting and Cognitive Function

Proposed mechanism	Biological rationale	Evidence source	Important uncertainties
Brain energy metabolism	Falling glucose and insulin shift substrate use during fasting [12]; interstitial glucose lowered without cognitive impairment after 2 days of calorie deprivation [13]	Human	Mood and hunger confound; prolonged-fast data do not equal IF
Ketone bodies	Alternative fuel and signalling molecules [11, 14, 28]; brain ketone uptake shown in prolonged fasting [11]	Human (prolonged fast); animal	Level of ketosis in 16-hour or alternate-day regimens unclear
BDNF and neuroplasticity	Metabolic switching proposed to raise neurotrophic signalling [14]; rodent dietary-restriction and fasting-mimicking-diet data [29, 32]	Mainly animal; human BDNF findings mixed [30, 31]	No verified human trial linking a marker change to cognition
Oxidative stress responses	Adaptive stress responses proposed to increase antioxidant defence [22]	Human observational biomarker data in older adults with MCI [15]	Mediation not established; confounding likely
Neuroinflammation and gut–brain axis	Lower inflammation and microglial modulation proposed [9, 33]	Human (correlational) [9]; animal and ex vivo data [9, 33]	Causality in humans untested; applicability beyond obesity unknown
Circadian and sleep effects	Feeding time acts on peripheral clocks [5, 24]; mouse time-restricted feeding data [34]	Human (limited) [8, 10]; animal [34]	Timing of eating window rarely tested

BDNF, brain-derived neurotrophic factor; IF, intermittent fasting; MCI, mild cognitive impairment. Evidence source reflects the studies identified in this review and is not an exhaustive evaluation.

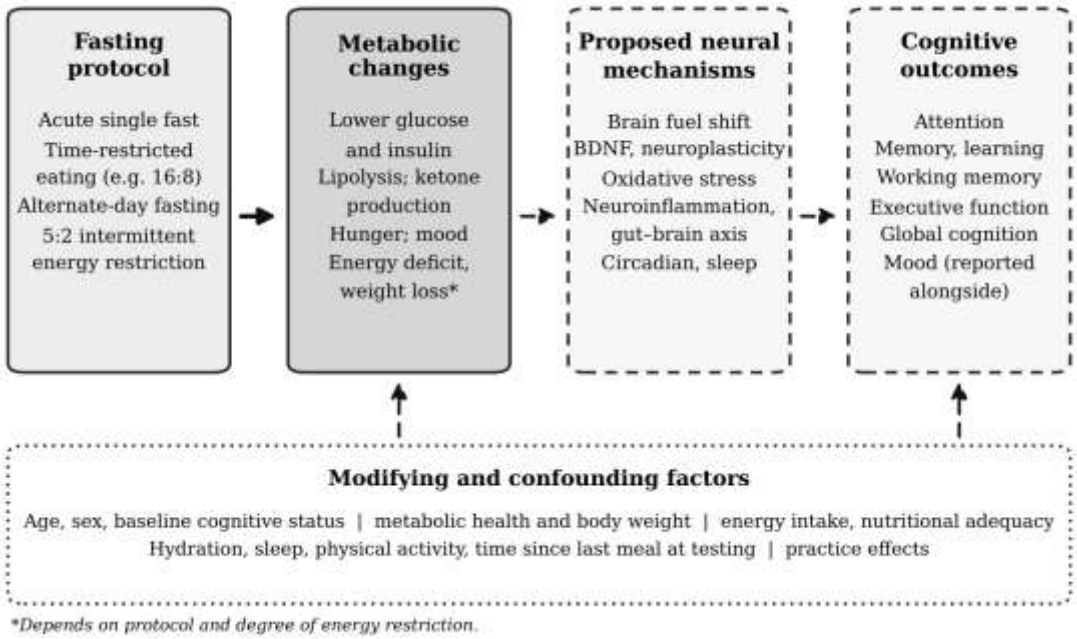


Figure 1. Proposed Pathways Linking Intermittent Fasting and Cognitive Performance. Conceptual model. Fasting protocol and duration (acute single fast, time-restricted eating, alternate-day fasting, 5:2) lead to metabolic changes (lower glucose and insulin, lipolysis, ketone production, hunger, energy deficit). These are linked to possible neural mechanisms (brain fuel shift, neurotrophic signalling, oxidative stress responses, neuroinflammation including gut–brain effects, circadian and sleep alignment) and to possible cognitive outcomes (attention, memory, working memory, executive function, global cognition, mood). The solid arrow marks the reasonably established link between fasting protocol and metabolic change; dashed arrows and outlines mark hypothetical or incompletely established pathways. Modifying factors are shown below. The figure does not demonstrate causality.

5. EFFECTS OF INTERMITTENT FASTING ON COGNITIVE PERFORMANCE

Human studies have assessed attention, memory, working memory, executive function and global cognition, but they differ in participants, fasting protocols, duration and cognitive measures. Acute fasting experiments and repeated interventions assess different physiological and behavioural conditions and are distinguished throughout. Table 2 summarises the human studies for which key design details could be confirmed; CR and observational studies are included for context. Table 3 summarises the direction of findings by cognitive domain. The synthesis considers positive, neutral and negative findings without assuming that change on one task represents an overall improvement in cognition.

Table 2. Characteristics of Human Studies of Fasting, Calorie Restriction and Cognitive Performance

Study	Design and population	Protocol and duration	Cognitive outcomes	Principal findings	Limitations
Solianik et al., 2016 [16]	Experimental before-and-after study; 9 amateur male weight lifters aged 20–30 y	Zero-calorie fast, water ad libitum; 48 h	Prefrontal- and hippocampus-related tasks	Improved mental flexibility and set shifting; hippocampus-related functions unaffected; anger increased; resting frontal brain activity decreased	Acute fast; very small, specialised sample
Solianik and Sujeta, 2018 [17]	Randomised crossover; 11 women with overweight aged 20–30 y	2-day zero-calorie diet vs 2-day usual diet	Cognitive, psychomotor and motor performance	No effect on mood, brain activity or cognition; fasting evoked moderate stress	Acute fast; very small sample
Lieberman et al., 2017 [13]	Crossover study, reported as double-blind and placebo-controlled; 23 healthy young adults (17 men, 6 women)	Calorie deprivation (about 266 kcal/day) vs fed (about 3,935 kcal/day), each with about 4 h/day aerobic exercise; 51-h inpatient phase	Vigilance, reaction time, working memory, motor learning, reasoning	Cognition not impaired; mood worsened; interstitial glucose lowered	Acute severe underfeeding with exercise; not total fasting
Harder-Lauridsen et al., 2017 [18]	Non-randomised crossover; 10 healthy lean men	Ramadan model: 14 h daytime abstinence from food and drinking; 28 days, with a 28-day control period	Cognitive function tests (NR)	No major effect on cognition, body composition or glucose metabolism	Small; food and fluid restriction

Study	Design and population	Protocol and duration	Cognitive outcomes	Principal findings	Limitations
Teong et al., 2021 [10]	Prespecified secondary analysis of an open-label RCT; 46 women with overweight or obesity, mean age 50 y	IF vs continuous CR, each prescribed 70% of energy requirements; acute 24-h fast tested in IF group; 8 weeks	Cognitive performance after 12-h overnight fast (tasks NR)	No group difference; both groups improved on one task; 24-h fast did not change cognition	Short; women only
Batorek and Hromatko, 2024 [8]	Open-label, non-randomised controlled study (participants chose group); 105 completers: 76 fasting, 29 control	16-h daily fast (TRE); 2 months	Attention, memory, working memory, executive function	No significant group-by-time interactions; scores improved in both groups; BMI fell only in fasting group	Non-randomised; self-selected groups; substantial dropout; volunteers
McLeod et al., 2026 [7]	Randomised controlled pilot; 43 healthy women (23 fasting, 20 control)	16:8 TRE; 4 weeks	Symmetry Span; Stroop, Simon and Flanker Squared	Improvement over time in both groups, no group difference; BMI fell more with TRE	Pilot; small; women only; repeated testing
Mela et al., 2025 [9]	RCT substudy (NCT04453150); adults with obesity aged 18–65 y (BMI 30–45 kg/m²); 96 randomised, 84 completed (30 ADF, 28 ketogenic, 26 Mediterranean)	ADF vs ketogenic vs Mediterranean diet (all hypocaloric); 3 months	Stroop, letters-and-numbers span, Trail Making Test	Greatest improvement with ADF; working memory improved with ADF and ketogenic diet; ketogenic group worse on Trail Making Test A	Completers analysed (about 10–17 per test); weight loss confounds; open-label; microbiota and animal data indirect
Ooi et al., 2020 [15]	Longitudinal observational study; 99 adults aged ≥60 y with MCI	Self-reported regular IF (protocol NR); 36 months	MMSE, MoCA, RAVLT, Digit Span, Digit Symbol	Regular fasters had better scores and better cognitive status at 36 months	Observational; self-selection and confounding

Study	Design and population	Protocol and duration	Cognitive outcomes	Principal findings	Limitations
Currenti et al., 2021 [35]	Cross-sectional cohort; 883 adults aged ≥50 y, Sicily	Self-reported eating window <10 h vs longer	Short Portable Mental Status Questionnaire	Shorter eating window associated with lower odds of cognitive impairment (OR 0.28 reported)	Cross-sectional; dietary self-report; no causal inference
Leclerc et al., 2020 [19]	Secondary analysis of the CALERIE RCT; 220 non-obese adults (BMI 22–28)	25% CR vs ad libitum diet (not fasting); 2 years	Spatial working memory (CANTAB)	Working memory improved with CR vs control	Not fasting; cannot be extrapolated to IF
Witte et al., 2009 [31]	Prospective interventional study; 50 healthy older adults (CR group n = 20)	30% CR (not fasting); 3 months	Verbal memory	Memory score rose about 20% with restriction, correlating with falls in fasting insulin and C-reactive protein; BDNF unchanged	Not fasting; small sample; interventional design with stratified groups

ADF, alternate-day fasting; BDNF, brain-derived neurotrophic factor; BMI, body mass index; CANTAB, Cambridge Neuropsychological Test Automated Battery; CR, calorie restriction; IF, intermittent fasting; MCI, mild cognitive impairment; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; NR, not reported in the sources accessed; OR, odds ratio; RAVLT, Rey Auditory Verbal Learning Test; RCT, randomised controlled trial; TRE, time-restricted eating.

5.1 Attention, Vigilance and Working Memory

Evidence on attention is limited and mostly neutral. In the four-week randomised pilot of 16:8 TRE in 43 healthy women, attentional control (Stroop Squared, Simon Squared and Flanker Squared tasks) and working memory (Symmetry Span task) were assessed weekly. Performance improved over time in both groups with no significant between-group difference, which is more consistent with practice or familiarisation than a TRE-specific effect; body mass index fell more in the fasting group [7]. In the two-month open-label study of daily 16-hour fasting, in which participants chose their group and 105 completed the study, attention, memory, working memory and executive function showed no significant group-by-time interactions; scores improved over time in both groups, which the authors attributed to practice effects [8].

In the three-month randomised trial in adults with obesity, the ketogenic group performed less favourably on Trail Making Test A than the ADF and Mediterranean groups [9]. This should not be read as evidence that ADF enhances vigilance, since the test indexes visual attention and processing speed rather than

sustained vigilance. The eight-week energy-matched trial in women found no group difference in cognition tested after a 12-hour overnight fast, and an additional 24-hour fast in the IF group did not alter performance [10]. Sustained (psychomotor) vigilance across fasting durations has not been adequately examined, so it is uncertain whether fasting affects vigilance during prolonged or demanding tasks.

Working memory findings are mixed, with most studies reporting no fasting-specific benefit. Besides the null results above [7, 8], a 48-hour fast in nine male weight lifters produced no change in working memory [16]. In contrast, the ADF and ketogenic groups improved on a letters-and-numbers span task in the obesity trial [9]. Because body weight, energy intake and metabolic health changed alongside the diets and no non-dietary control group was included, this does not show that fasting alone was responsible. CR research provides context: in a secondary analysis of the CALERIE trial, two years of 25% CR improved spatial working memory in healthy non-obese adults relative to an ad libitum diet [19]. This cannot be used as evidence of an IF effect, but it underlines the need to separate fasting pattern from energy restriction. Larger studies are needed to establish whether any benefit relates to fasting frequency, energy deficit, weight loss or metabolic change.

5.2 Memory and Learning

Human evidence on memory and learning is limited. In the two-month TRE study, memory was assessed but no significant group-by-time interaction was reported [8]. In a 48-hour zero-calorie fast in nine amateur male weight lifters, hippocampus-related functions were unchanged [16]; these tasks do not equate to a comprehensive assessment of verbal learning or consolidation, but they do not indicate an acute improvement.

A potentially positive association comes from a three-year longitudinal study of 99 older adults aged 60 years or above with MCI, classified by reported IF practice. Cognition was assessed with the Rey Auditory Verbal Learning Test (RAVLT), Digit Span, Digit Symbol, Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA), and regular fasters showed more favourable changes in cognitive scores, including verbal learning, than those who did not fast regularly [15]. The observational design, self-reported fasting and possible lifestyle confounding preclude a causal interpretation. As context, in a prospective study of healthy older adults three months of 30% CR was followed by an increase of about 20% in verbal memory scores, correlating with reductions in fasting insulin and C-reactive protein, with no significant change in the other two groups [31]; CR is not equivalent to IF, but this finding shows that energy restriction itself may influence memory. The letters-and-numbers span task in the obesity trial assesses working memory and is discussed in Section 5.1, not as evidence of improved long-term memory [9].

Taken together, the evidence does not establish that IF improves memory or learning in healthy adults. The favourable findings in older adults with MCI require confirmation in randomised trials, and learning acquisition, delayed recall and memory consolidation remain insufficiently investigated.

5.3 Executive Function and Global Cognitive Performance

Executive function (cognitive flexibility, inhibitory control, planning, set shifting) has produced some of the most contrasting findings in acute fasting research. After a 48-hour zero-calorie fast, nine amateur male weight lifters showed significant improvement in mental flexibility and set shifting, but other cognitive functions did not improve and anger increased [16]. In contrast, a randomised crossover study of 11 women with overweight, aged 20–30 years, found that a two-day zero-calorie fast did not significantly affect cognitive or psychomotor performance, although it evoked physiological stress and a shift towards sympathetic activity [17]; the positive findings in trained men were therefore not reproduced.

A systematic review of experimental fasting studies concluded that short-term fasting was more likely to impair than improve higher-order cognitive functions, although the included studies were heterogeneous [4]. Repeated-fasting evidence is largely neutral: the four-week and two-month TRE studies found no fasting-specific improvement in inhibitory control or executive measures [7, 8], whereas in the obesity trial improvement across dietary groups was greatest with ADF [9], without any confident separation of fasting from concurrent weight and metabolic change. Current evidence does not establish a consistent benefit of IF on executive function, and the improvement in mental flexibility after a 48-hour fast in a small group of trained men should not be generalised.

Global cognitive performance has been examined mainly in older adults with MCI and in short interventions in otherwise healthy participants. The most notable positive finding is the three-year study of 99 older adults with MCI: regular fasters had more favourable scores on the MMSE, MoCA, RAVLT, Digit Span and Digit Symbol tests, and a greater proportion improved in cognitive status at follow-up [15]. However, participants were not randomised, and differences in education, lifestyle, health status or diet quality cannot be excluded. In a cross-sectional analysis of 883 adults aged 50 years or older in Sicily, those with an eating window shorter than 10 hours had lower odds of cognitive impairment (odds ratio 0.28) than those without eating-time restriction [35]; the cross-sectional design, dietary self-report and brief screening questionnaire prevent the conclusion that a shorter window protects cognition.

In healthy adults, interventional findings have generally been neutral. A 28-day Ramadan-model intervention in ten healthy lean men, involving about 14 hours of daytime abstinence from food and drinking, produced no significant change in cognition relative to a control period [18]. The eight-week trial comparing IF with continuous CR in women with overweight or obesity found no between-group difference; both groups improved on one task, and a 24-hour fast at the end did not change performance within the IF group [10]. The three-month trial in adults with obesity reported more favourable overall cognitive change with ADF than with the comparison diets [9], but this specific population, weight loss and metabolic change preclude generalisation to healthy adults or the conclusion that ADF improves global cognition.

Pooled randomised evidence points modestly in a favourable direction for dietary restriction as a whole. A meta-analysis of 11 randomised trials of CR and IF reported a small overall benefit for cognitive function (standardised mean difference 0.22; 95% CI 0.09–0.34), with effects for global function (0.35; 95% CI 0.04–0.65) and a borderline effect for memory (0.18; 95% CI 0.00–0.35), the largest effect in people with MCI (0.36; 95% CI 0.05–0.68), and no significant difference between IF and CR; the authors cautioned against generalising to other populations [26]. Because it combined two types of dietary restriction from a small number of trials, it does not show that IF specifically improves cognition. Global cognitive findings therefore remain uncertain: positive associations in older adults with MCI and in cross-sectional data require confirmation in well-designed randomised trials, and short-term studies in healthy adults have not shown consistent improvement. There is currently insufficient evidence to recommend IF to improve global cognition or prevent cognitive decline.

Table 3. Summary of Cognitive Outcomes Associated with Intermittent Fasting

Cognitive domain	Relevant evidence and designs	Overall direction and consistency	Key references
Attention and vigilance	Pilot RCT, open-label controlled study, RCT in obesity, energy-matched RCT	Mostly neutral; one adverse signal with ketogenic diet; vigilance rarely measured	[7–10]
Memory and learning	Acute fasting study, controlled study, observational cohort	Neutral in healthy adults; positive association in an MCI cohort	[8, 15, 16]
Working memory	Pilot RCT, controlled study, RCT in obesity, calorie-restriction trial	Inconsistent; improvement possible in obesity or under CR	[7–9, 16, 19]
Executive function	Acute fasts, repeated protocols, systematic review	Inconsistent; acute impairment, null and improvement all reported	[4, 7–9, 16, 17]
Global cognition	Observational cohort in MCI, cross-sectional study, short RCTs, meta-analysis of dietary restriction	Uncertain; positive associations in observational data, null in healthy adults; small pooled benefit for dietary restriction overall	[10, 15, 18, 26, 35]

CR, calorie restriction; MCI, mild cognitive impairment; RCT, randomised controlled trial. Directions reflect the studies identified in this review and are not formal evidence grades.

6. ACUTE VERSUS REPEATED FASTING: COGNITIVE RESPONSES AND ADAPTATION

Acute fasting studies assess cognition during or immediately after a single period of food restriction, whereas repeated-fasting studies examine the effects of following a regimen for weeks or months. They should be interpreted separately because acute responses may reflect hunger, stress and energy availability, while repeated interventions may involve changes in body weight, metabolic health, sleep and eating behaviour. Whether the body and brain adapt to repeated fasting, leading to stable or improved cognition, is frequently proposed as an explanation for differences between acute and repeated studies, but current human evidence is insufficient to establish this.

6.1 Cognitive Performance During Acute Fasting

Acute studies have examined fasts of approximately 12 to 48 hours, with mixed findings: short-term fasting does not consistently change cognitive performance, although certain higher-order functions and mood may be affected in some individuals. In a double-blind crossover study of 23 healthy young adults (17 men, 6 women), about two days of calorie deprivation (approximately 266 kcal/day, compared with approximately 3,935 kcal/day when fed, each with the same aerobic exercise load) lowered interstitial glucose and worsened mood but had no meaningful effect on measures including vigilance, reaction time, working memory, motor learning and reasoning [13]. Because the exercise load was identical in both conditions, the comparison isolates calorie deprivation, but the result applies to physically active young adults under severe underfeeding rather than to fasting in general.

In a 48-hour zero-calorie fast, nine amateur male weight lifters showed improved mental flexibility and set shifting, unchanged hippocampus-related functions, increased anger and decreased resting frontal brain activity [16]. In contrast, a two-day fast in 11 women with overweight did not significantly alter mood,

cognitive, psychomotor or motor performance, although a stress response with sympathetic shift was observed [17], and a 24-hour fast in the IF arm of an eight-week trial in women did not alter cognition [10]. Acute fasting therefore does not produce uniform cognitive responses across populations.

Systematic reviews give a broader view. A review by Benau et al. of studies published between 2013 and 2020 (19 articles representing 21 studies) found that fasting was more frequently associated with cognitive deficits than with improvements, although evidence was heterogeneous and limited by differences in protocols, populations and assessment methods [4]. A review of CR and fasting similarly concluded that the severity of restriction most likely predicts whether cognition improves or is impaired, with fasting linked to poorer cognitive flexibility and psychomotor performance [40]. More recently, Bamberg and Moreau meta-analysed experimental studies in healthy individuals (71 studies; 222 effect sizes; 3,484 participants; median fasting duration 12 hours), mostly adults with a small proportion of children. The hierarchical model showed no meaningful average difference between fasted and satiated participants (Hedges' $g = 0.02$; 95% credible interval −0.05 to 0.10), with low between-study heterogeneity, but modest reductions in performance were associated with longer fasting intervals and with younger participants, and differences across cognitive domains did not reliably explain the variation in effect sizes [20]. Acute fasting thus does not appear to produce a consistent improvement or a substantial impairment in healthy adults, although prolonged fasting may affect mood, stress and selected functions, and individual tolerance should be considered.

6.2 Cognitive Outcomes During Repeated Fasting

Repeated-fasting studies have generally lasted between four weeks and three months. In the four-week randomised pilot of 16:8 TRE in healthy women, cognitive scores improved over time but did not differ between the fasting and control groups [7]. The two-month study of 16-hour daily fasting found no significant group-by-time interactions for attention, memory, working memory or executive function [8]. The eight-week randomised trial comparing IF with continuous CR in women with overweight or obesity found no between-group difference; since both arms were energy restricted, it is particularly relevant to separating fasting schedules from reduced intake [10]. A 28-day Ramadan-model intervention in healthy lean men reported no major change in cognition [18], although daytime abstinence from food and drinking is not equivalent to water-permitted TRE. One notable positive finding was reported in the three-month trial in adults with obesity, in which ADF produced greater overall cognitive improvement than the ketogenic or Mediterranean diets [9]; concurrent weight loss and metabolic change may have contributed, and confirmation in other populations is needed. Taken together, repeated-fasting studies of up to three months have mostly reported neutral outcomes in healthy adults, and a positive signal in adults with obesity cannot yet be attributed to fasting itself, weight reduction or other metabolic change.

Adaptation to repeated fasting, involving changes in hunger perception, substrate use, metabolic flexibility and tolerance of fasting periods, has been proposed as an explanation for maintained cognition, but the existence of such changes does not demonstrate improved cognitive function. In the four-week pilot, weekly cognitive scores improved similarly in both groups, indicating that practice effects may have contributed [7], and the two-month study did not show a distinct improvement trajectory in the fasting group [8]. Most studies compared baseline with post-intervention performance rather than investigating adaptation. Demonstrating adaptation would require repeated assessments during early and later phases of fasting, at standardised intervals relative to meals, to determine whether an initial change diminishes or performance remains stable. The meta-analysis of acute fasting [20] was not designed to test whether responses change with repeated exposure. Current human evidence therefore does not establish that

repeated IF produces cognitive adaptation or progressive enhancement, and maintained cognition during repeated fasting should not automatically be interpreted as physiological adaptation.

6.3 Influence of Fasting Duration and Timing

A 12–16-hour overnight or daily fast differs considerably from a continuous 24–48-hour fast in energy availability, hunger, metabolic change and physiological stress. Short daily fasting periods have generally produced no significant cognitive differences from control or CR conditions [7, 8, 10], whereas 48-hour fasting has produced both positive and neutral findings depending on population and domain [16, 17], and two-day calorie deprivation worsened mood despite preserved test performance [13]. The acute-fasting meta-analysis found an overall effect close to zero but modest reductions in performance with longer fasting intervals [20], supporting the distinction between overnight fasting and prolonged experimental fasting.

The timing of cognitive testing relative to the last meal is another important consideration, yet the available studies do not consistently standardise or report this interval. The placement of the daily eating window may also influence circadian rhythm, sleep and alertness, so early and late TRE could create different physiological conditions; however, direct human comparisons of early versus late eating windows with cognition as an outcome were not identified. Ramadan-style fasting involves abstinence from both food and fluids during daylight, and changes in sleep timing, hydration and routine may influence cognition independently of fasting duration [18, 36], so Ramadan-model findings should be interpreted separately from water-permitted protocols. Direct comparisons of fasting durations and eating-window timing remain insufficient.

7. FACTORS INFLUENCING COGNITIVE OUTCOMES

Cognitive responses to IF may vary with participant characteristics, the fasting protocol and the conditions of assessment. However, most studies were not designed to investigate subgroup differences, so the factors below should be viewed as potential sources of variation, not established predictors.

7.1 Age, Sex and Baseline Cognitive Status

Studies in healthy younger adults have generally reported no consistent impairment in cognition during short-term fasting; for example, cognition was maintained in young adults despite reduced interstitial glucose during two days of calorie deprivation, although mood deteriorated [13]. In the acute-fasting meta-analysis, modest performance reductions were associated with younger age, and children appeared more vulnerable than adults [20]. Evidence in older adults is more limited: more favourable cognitive outcomes and biochemical changes were reported in older adults with MCI who practised IF over three years of observation [15], and a shorter eating window was associated with lower odds of cognitive impairment in a cross-sectional study [35]. These designs do not show that fasting caused the differences, and participant selection and lifestyle confounding may have contributed. A 2026 systematic review of IF in adults aged 60 years or older, whose cognitive findings rest on narrative synthesis of individual studies, reported associations between very restrictive eating windows (10 hours or less) or prolonged fasting and lower cognitive scores [37]. This contrasts with the lower odds of impairment reported for windows shorter than 10 hours in the cross-sectional study [35], and shows that observational signals are inconsistent and sensitive to how fasting is defined. Because studies involve different age groups, cognitive profiles and designs, direct comparison between younger and older adults is not possible.

Regarding sex, available studies include women only [7, 10] and men [16, 18], but none compared responses between sexes, and differences in populations, protocols, duration and assessments prevent

conclusions. Individual predictors of response cannot be identified from the existing evidence. Future studies should include adequately powered analyses by sex and other participant characteristics.

7.2 Metabolic Health and Nutritional Adequacy

Several IF studies involved participants with overweight or obesity. In the three-month trial by Mela et al. [9], adults with obesity assigned to ADF, a ketogenic diet or a Mediterranean diet showed cognitive improvements in some domains, with favourable changes in the ADF group. Body weight, metabolic status and inflammation changed alongside the diets, and the study does not establish that baseline metabolic health modifies the cognitive response to fasting or that the improvements resulted directly from fasting. Evidence from healthy adults and adults with obesity should not be considered interchangeable, and future trials should report baseline metabolic characteristics and examine whether changes in body composition or metabolic markers relate to cognition.

Energy intake is also important because protocols differ in the extent of energy restriction. TRE may alter meal timing without a prescribed deficit, whereas ADF and some modified regimens substantially reduce intake on designated days. The weight-loss literature shows how much of an apparent fasting effect may be due to energy intake: in a 12-week randomised trial of 116 adults with overweight or obesity, 16:8 TRE was not more effective for weight loss than eating throughout the day [38], and in a 12-month trial of 139 adults with obesity, adding TRE to CR was not more beneficial than CR alone, which the authors interpreted as indicating that CR accounted for most of the benefit [39]. Neither trial assessed cognition, but both support separating meal timing from energy deficit when interpreting cognitive outcomes. In the energy-matched comparison by Teong et al. [10], both groups were prescribed approximately 70% of energy requirements and cognition did not differ between them, suggesting that cognitive outcomes should be interpreted in relation to the overall dietary intervention, not fasting duration alone; a review concluded that the severity of restriction most likely determines whether cognition improves or is impaired [40]. Preserved test performance also does not mean that the experience of fasting is unaffected, as mood deteriorated in the calorie-deprivation study [13]. The available studies do not establish an optimal energy intake or nutritional composition for maintaining cognition during IF.

7.3 Sleep, Circadian Timing and Physical Activity

Sleep, feeding schedule and physical activity may influence the interpretation of cognitive outcomes, but the studies provide limited evidence that they independently modify cognitive response. Neither the two-month IF study, which assessed cognition, mood and sleep quality [8], nor the eight-week comparison with continuous CR [10] showed a consistent cognitive benefit or impairment. Direct comparisons of early and late eating windows are insufficient to establish whether meal timing independently affects cognition. Physical activity complicates interpretation of acute studies: in the calorie-deprivation study, both the deprived and fed conditions included the same aerobic exercise, so findings relate to exercising individuals and cannot be extrapolated to sedentary fasting [13]. Future studies should standardise or document sleep, meal timing, physical activity and the timing of cognitive assessment to distinguish the effects of fasting from other behavioural and physiological influences.

8. CRITICAL APPRAISAL, RESEARCH GAPS AND FUTURE DIRECTIONS

8.1 Evidence from Human Studies

Randomised and controlled trials. Randomised trials permit comparison of fasting with control or alternative diets, but those identified here are small, short and heterogeneous. McLeod et al. [7] studied 43 healthy women for four weeks; cognition improved in both groups, so no intervention-specific

advantage was shown, and the sample and duration limit inference about longer-term change. Batorek and Hromatko [8] studied healthy volunteers for two months, but participants chose their group (non-randomised) and only 105 completers (76 fasting, 29 control) were analysed after substantial dropout, so unequal groups, self-selection and attrition limit interpretation. Mela et al. [9] randomised 96 adults with obesity (BMI 30–45 kg/m²) to ADF, a ketogenic diet or a Mediterranean diet for three months, with 84 completing (30, 28 and 26 respectively; dropout was lowest with ADF, 6.25%, compared with 12.5% and 18.75% in the other arms). Weight loss was included as a covariate, but cognitive analyses involved only about 10–17 participants per test, all diets were hypocaloric, there was no non-dietary control, and analysing completers can inflate apparent benefit when dropout differs between arms. Teong et al. [10] compared eight weeks of IF with continuous CR in women with overweight or obesity; both arms were energy restricted and cognition did not differ, which usefully tests the pattern of restriction but does not show whether either intervention differs from unrestricted eating. Blinding is not feasible for dietary interventions, so expectation effects on effortful outcomes are possible. These trials are best interpreted individually, not as evidence for one uniform effect of IF.

Observational studies. Observational studies provide longer-term associations but cannot establish causality because fasting is not randomly assigned. Ooi et al. [15] reported more favourable cognitive outcomes in older adults with MCI who regularly practised IF over three years, with associated biochemical changes, and a cross-sectional study in Sicily reported lower odds of impairment with a shorter eating window [35]. Participants who fast regularly may differ in diet, physical activity, health awareness, medication use and other characteristics, and self-selection, reverse causation and residual confounding limit interpretation. Longer follow-up is valuable but does not remove the limitations of non-randomised designs, and these findings are complementary to, not confirmation of, the trial results.

Long-term evidence and generalisability. Randomised human studies lasted four weeks to three months [7–10], so none can show whether IF affects long-term cognitive ageing, the development of cognitive impairment or neurodegenerative disease. Long-term CR studies have reported improvements in selected domains such as working memory [19], but CR is not IF. Populations studied also differ (healthy adults, women with overweight or obesity, older adults with MCI, small groups of physically active participants), and findings from one group cannot automatically be applied to another, nor can Ramadan-based fasting, with daytime food and fluid restriction, be equated with water-permitted TRE.

8.2 Methodological Limitations and Conflicting Findings

Findings across studies are not consistent. The trials by McLeod et al. [7], Batorek and Hromatko [8] and Teong et al. [10] showed no cognitive advantage attributable to IF, whereas Mela et al. [9] reported favourable changes in selected outcomes with ADF in adults with obesity and Ooi et al. [15] reported more favourable outcomes in older adults with MCI, though observational. A meta-analysis pooled CR and IF and found a small overall benefit with no significant difference between them [26]. Acute studies add variation: calorie deprivation did not impair cognition in young adults although mood worsened [13], mental flexibility improved after a 48-hour fast in a small group of male weight lifters [16], and no significant impairment occurred after two days of fasting in women with overweight [17]. The acute-fasting meta-analysis found no meaningful average difference between fasted and satiated states [20], but this concerns acute fasting and should not be extrapolated to repeated IF. Differences may reflect fasting duration, energy restriction, participants, cognitive domains, timing of testing and the distinction between short-term responses and change after repeated intervention, so the literature does not support a single conclusion that IF consistently improves or impairs cognition.

Several methodological issues limit comparison and inference. **Sample size:** several studies were small, for example 43 women [7] and nine male weight lifters [16], limiting power to detect modest effects and to perform subgroup analyses. **Duration:** most interventions lasted weeks to months, which cannot evaluate sustained outcomes or long-term adaptation. **Protocol heterogeneity:** IF covers TRE, ADF, modified fasting and Ramadan-based fasting, which differ in fasting duration, meal timing, energy intake and fluid restriction, so findings for one protocol cannot be generalised to another. **Adherence:** differences between prescribed and achieved fasting patterns may influence results, but adherence reporting is not uniform. **Comparators:** usual diet, non-fasting controls and continuous CR address different questions, and the absence of a common comparator limits cross-trial comparison. **Confounding:** weight loss, energy intake, metabolic markers, physical activity and sleep change alongside fasting and can influence cognition independently, particularly when fasting is accompanied by substantial dietary or metabolic change [9, 15]. **Outcome measurement and reporting:** studies used different cognitive tests and schedules, and several assessed multiple outcomes without clearly designated primary outcomes, raising the risk of chance findings and selective reporting.

Cognition is multidimensional and cannot be captured by a single test. McLeod et al. [7] used working-memory and attentional-control tasks, Mela et al. [9] used the Stroop task, span tasks and the Trail Making Test, and Ooi et al. [15] used broader instruments including the MMSE, MoCA and tests of memory and processing speed. Differences in sensitivity and purpose complicate comparison, and change on one executive task does not indicate improvement in overall cognition. Global screening tools also differ in sensitivity: in the original validation of the MoCA, the MMSE detected only 18% of participants with MCI at a cut-off of 26, compared with 90% for the MoCA [42]. Repeated administration may produce practice effects, which vary with the test, retest interval, participant age and use of alternate forms and must be considered when interpreting change over time [41]; this is relevant where control groups also improved [7, 8]. Testing during a fast, shortly after eating or after an overnight fast creates different conditions, yet reporting of these conditions varies.

Why the evidence is insufficient. Taken together, current evidence does not establish IF as a cognitive-enhancing intervention. Randomised evidence is limited to small, short studies that are mostly neutral in healthy adults; the favourable signals arise in adults with obesity, where weight loss and diet composition confound the result, and in observational studies of older adults, where selection and confounding preclude causal inference. Energy restriction, not fasting pattern, may explain much of any benefit, long-term and clinically meaningful outcomes have not been studied, and mechanistic support is largely preclinical. This review has its own limitations: the search was non-systematic; selection was by a single reviewer.

8.3 Research Priorities and Future Directions

Future research should prioritise:

- **Adequately powered, longer randomised trials** with pre-registered primary cognitive outcomes, defined intervention periods and follow-up beyond three months, to determine whether cognitive outcomes remain stable or change.
- **Standardised cognitive assessment** using validated batteries covering attention, memory, working memory, executive function and global cognition, with alternate test forms or other strategies to account for practice effects.
- **Direct comparison of acute and repeated fasting** with assessment at baseline, during initial exposure, after repeated exposure and at follow-up, to determine whether responses change with repeated exposure and whether adaptation occurs.

- **Comparison of clearly defined protocols** (TRE, ADF and others), described precisely in terms of eating-window duration, fasting-day energy intake, meal composition, fluid allowance and adherence.
- **Energy-matched control groups** and measurement of body weight, body composition and metabolic markers, to separate fasting effects from calorie restriction and weight loss.
- **Systematic measurement of sleep, circadian timing** (including early versus late eating windows), physical activity, nutritional intake and the interval between the last meal and testing.
- **Assessment of adverse effects, tolerability and nutritional adequacy**, as mood and stress responses have been reported even when cognition was maintained [13, 17].
- **Carefully designed studies in older adults and other vulnerable groups** (existing cognitive impairment, metabolic disorders), with clinical safeguards and clearly defined eligibility criteria, and long-term clinical outcomes such as the rate of cognitive decline.

9. CLINICAL IMPLICATIONS

Current human evidence suggests that IF does not consistently impair cognitive performance in the adult populations studied, but it does not establish a reliable cognitive-enhancing effect. Short-term studies have reported no significant differences compared with control or continuous CR groups [7, 8, 10], and the favourable changes reported in selected domains require cautious interpretation because of differences in populations and protocols and accompanying metabolic changes [9, 15]. IF should therefore be considered a dietary pattern, not an established intervention for improving cognition, and its suitability depends on overall dietary intake, medical history, nutritional requirements, lifestyle and ability to follow the schedule.

In nutrition counselling, TRE without prescribed calorie restriction should be distinguished from fasting approaches involving substantial reductions in energy intake, which may have different nutritional and physiological consequences [40]. Counselling should consider whether the chosen pattern allows adequate energy and nutrient intake and whether it is appropriate given diabetes, pregnancy, medications and any history of disordered eating. Cognitive performance should be considered together with sleep, hydration, physical activity, meal timing and diet quality, and test performance alone should not be taken as evidence that a fasting regimen is suitable, since mood may change while performance is maintained [13].

The available evidence does not justify recommending IF as a treatment for MCI, dementia, Alzheimer's disease or other neurological conditions; the observational findings in older adults with MCI [15] are insufficient to establish efficacy, and controlled clinical research is required before fasting can be considered a strategy for preventing cognitive decline or treating neurological disease. Decisions should be individualised, with attention to nutritional adequacy, tolerability, medical circumstances and the risks of prolonged or restrictive fasting, and IF should not replace established diagnostic assessments or evidence-based treatments for cognitive or neurological disorders. However, it may be considered a potential adjunctive (add-on) lifestyle intervention alongside standard medical care, where appropriate, to support metabolic health and potentially contribute to cognitive health. Its safety and suitability should be evaluated individually, and its clinical benefits require further investigation.

10. CONCLUSION

The relationship between intermittent fasting (IF) and cognitive performance is complex and influenced by fasting protocols, population characteristics, intervention duration and cognitive domains. Current human evidence suggests that acute fasting generally preserves cognitive performance, as supported by

the meta-analysis of Bamberg and Moreau [20]. Repeated IF interventions show promising but inconsistent effects, with selected cognitive improvements reported in some studies involving adults with obesity and older adults with mild cognitive impairment [7, 8, 10]

Potential mechanisms involving brain energy metabolism, ketone utilisation, neuroplasticity, oxidative stress and neuroinflammation provide a biological basis for these findings, although further human evidence is needed to establish their clinical significance. Overall, IF represents a promising area of nutritional research with potential implications for metabolic and cognitive health. While it is not yet established as a cognitive-enhancing or neuroprotective intervention, it may be considered a potential adjunctive lifestyle approach alongside appropriate medical care. Larger, long-term clinical trials with standardised cognitive assessments are essential to clarify its benefits, underlying mechanisms and long-term relevance to brain health.

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