Intermittent Fasting Improves Type 2 Diabetes by Enhancing β-Cell Function Through Autophagy and Metabolic Switching – A Compreshensive review

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ABSTRACT

Type 2 diabetes remains a major health burden, and finding ways to restore β-cell function is still a pressing need. Over the past 20 years, both animal and human studies have started pointing toward intermittent fasting (IF) as a potential approach. In rodent experiments, IF seemed to help β-cells regenerate — around a 21% improvement was reported in some studies. Clinical data also showed some promise, with reductions in HbA1c by roughly 1.5% in people following IF routines. The biological reasons behind this are still being studied, but it looks like IF may encourage autophagy, trigger changes in energy metabolism (such as higher ketone levels), and possibly reduce cell aging markers like p16. Some religious fasting practices like Ramadan provide examples where IF can be applied in real-life situations, although caution is needed — especially for people on insulin, since their risk of low blood sugar might be higher. All things considered, IF appears to support β-cell repair and better glucose control, but much more work is needed before it becomes a standard recommendation in diabetes care.

Keywords: Intermittent fasting, Type 2 diabetes, Beta-cell function, Metabolic adaptation, Low-cost therapy, Glycemic control

1.Introduction

Adolescent T2DM is rising worldwide, especially in males and in regions like Oceania, largely due to elevated BMI, which accounts for nearly one-third of DALYs [1]. In India, cases have surged from 77 million in 2019—half undiagnosed—to a projected 134 million by 2045, making it the 13th leading cause of death, with retinopathy and coronary complications causing ~1 million deaths annually [2]. While SGLT2 inhibitors offer cardiometabolic benefits, safety issues such as hospitalization, ketoacidosis, and urinary tract infections limit their use [3]. IF achieves similar HbA1c reduction (~1.4%) and weight loss (~3.2 kg) without drug side effects, showing safety in Ramadan fasting for most T2DM—and some T1DM—patients under supervision [3,4]. Its metabolic benefits, including lipid profile improvement and inflammation reduction [5], along with AMPK-autophagy–mediated β-cell protection [6] and reduced oxidative stress in preclinical studies [7], support its potential in β-cell regeneration and culturally adapted use in India.

2. Rising Burden and β-Cell Dysfunction in South Asia

Since 2000, type 2 diabetes in South Asia has nearly doubled, largely driven by pancreatic β-cell dysfunction linked to oxidative stress, endoplasmic reticulum stress, and urban lifestyle factors [8]. Prediabetes prevalence reaches 18.2%, while 17.5% of urban diabetics in India remain undiagnosed [8]. Early-onset T2DM (ages 20–39) has risen by 120% due to genetic predisposition, visceral obesity, and diminished β-cell reserve [9]. Rural prevalence is also high, with ICMR-INDIAB Haryana data reporting 12.4% diabetes and 18.2% prediabetes [10]. β-cell loss is aggravated by oxidative stress from imbalanced antioxidant defenses, including overexpression of GPx1, SOD, CAT, and TXNIP; reducing TXNIP and enhancing catalase may offer protection [11]. Drug-related problems (DRPs) further hinder outcomes, with over 84% of patients experiencing at least one DRP—often inappropriate medication or dosing—though pharmacist interventions see ~80% physician acceptance, underscoring the importance of collaborative care [12].

3. Intermittent Fasting Protocols for Diabetes Management

IF methods—IER, ADF, TRF—improve weight, insulin sensitivity, and metabolic health, with ADF causing ~8.2 kg loss in 8 weeks and TRF aligning with circadian rhythms [13]. Schedules like 16:8 and 5:2 enhance glucose, BP, cholesterol, and may lower inflammation [14,15]. TRF activates AMPK/SIRT1, restoring energy balance [16]. Clinical data show 4–7% weight loss, mainly visceral fat, with lipid and insulin sensitivity gains despite adherence issues [17,18]. In T2DM models, 16:8 IF preserved β-cell function, boosted BAT activity, and improved metabolism [19]. IF also reduces SASP factors and aging markers via AMPK/SIRT1, offering a safer anti-senescence effect [20].

4. Mechanisms of β-Cell Regeneration Enhanced by Intermittent Fasting

β-cell regeneration occurs through proliferation (via CDKs, DYRK1A inhibition, growth factors), neogenesis, α-cell transdifferentiation, and stem cell differentiation [21]. Senescence (p16^INK4a upregulation, PDX1/NKX6.1 loss) and FOXO1-driven dedifferentiation limit recovery, but FOXO1–PDX1–NGN3 rebalancing improves regeneration [22]. Balanced autophagy (Atg5, Atg7, Beclin-1, PINK1, Parkin via mTORC1/AMPK) preserves β-cell viability; metformin and GLP-1R agonists enhance this effect [23]. IF lowers glucotoxicity, lipotoxicity, and inflammation, boosting Neurogenin-3 and Pdx-1 expression [24], while improving metabolic flexibility through adiponectin, ketogenesis, and circadian pathways, though HOMA-IR results remain inconsistent [25].

5. Preclinical Evidence of Intermittent Fasting in β-Cell Preservation

IF protocols—ADF, TRE, and FMD—enhance β-cell survival, glucose tolerance, and autophagy, with ADF inducing ketone metabolism (~12 h), reducing nephropathy, and extending lifespan via mTOR inhibition and oxidative stress reduction [26,27]. TRE improves glucose tolerance, ADF reverses insulin resistance, and FMD restores β-cell function by modulating IGF-1, ketones, and SIRT1/PGC-1α, though STZ model regeneration likely reflects improved conditions [28]. MCS mimics IF and metformin, improving insulin signaling, β-cell protection, and wound healing [29]. IF also promotes β-cell proliferation and senescence reversal via Ngn3 activation, autophagy, and reduced p16^INK4a, p21^Cip1, and SASP, though human validation remains needed [30–32].

Table 1. Impacts of intermittent fasting (IF) on β-cell function and glycemia in animal models.

IF Protocol	Key Findings	Animal Model	Ref.
Alternate Day Fasting (ADF)	Metabolic switch to ketones (12h); 21% lifespan extension; improved β-cell survival	Sprague-Dawley rats	[26]
Time-Restricted Eating (TRE)	Enhanced glucose tolerance; reduced oxidative stress; improved insulin sensitivity	B6 mice	[27]
Fasting-Mimicking Diet (FMD)	Restored β-cell function; increased ketone bodies; reduced IGF-1 levels	High-fat-fed mice	[28]
Pharmacological Combinations	39.75% wound area reduction; enhanced vascularization; improved collagen deposition	STZ-diabetic mice	[29]

Abbreviations: ADF = alternate-day fasting; TRE = time-restricted eating; FMD = fasting-mimicking diet; STZ = streptozotocin; IGF-1 = insulin-like growth factor 1.

6. Glycemic Control: Meta-Analyses and Comparative Efficacy

Evidence supports modest but consistent metabolic benefits of IF in T2DM. A meta-analysis (11 studies, n=879) found no overall HbA1c change, though patients ≤60 years improved modestly [33]. A network meta-analysis (13 trials, n=867) ranked TWF best for HbA1c, glucose, and HOMA-IR; TRE was second for insulin sensitivity [34]. A 12-week RCT showed 16:8 IF improved waist circumference, BP, lipids, and cognition [35]. An umbrella review (23 meta-analyses) confirmed reductions in waist, fat, LDL, TG, fasting insulin, with HDL increase; TRE and twice-weekly fasting had strong adherence [36]. Overall, TRE, TWF, and ADF reduce BMI (1–5%), enhance insulin sensitivity, and match continuous calorie restriction for glycemic/BP control, though long-term safety in high-risk groups needs study [37].

7. Comparing Intermittent Fasting with Pharmacologic Diabetes Therapies

Current pharmacologic therapies for T2D—such as GLP-1 receptor agonists, DPP-4 inhibitors, and herbal compounds like PPAG—lower glucose but fail to restore β-cell mass, leaving disease progression unchecked [38]. While some agents reduce β-cell apoptosis, they rarely induce regeneration and often involve high costs, injectable delivery, and long-term treatment burdens. Intermittent fasting (IF) offers a low-cost alternative by activating AMPK, suppressing mTOR, and inducing autophagy, thereby promoting β-cell rest, survival, and regeneration in preclinical models [39]. GLP-1RAs cost $900–$1,300 monthly, limiting access in low-resource areas such as rural India, whereas IF is free, culturally adaptable (e.g., Ramadan, Upavasa), and potentially scalable as a cost-effective intervention [40].

8. Cultural and Economic Challenges in Implementing Fasting-Based Diabetes Care in India

A 2023 analysis of 1.97 million HbA1c tests found T2DM prevalence at 27.18% and prediabetes at 22.25%, with Odisha highest and Jammu & Kashmir lowest; men and working-age adults were most affected, linked to refined carbohydrate diets [41]. Care remains physician-driven, with low health literacy (37% avoid shared decisions), cost-driven drug use (glimepiride most common), and poor infrastructure—low HbA1c testing (13%) and scarce diabetes educators—worsening rural disparities [42,43]. India may reach 109 million T2DM cases by 2035, fueled by obesity, sedentary lifestyles, and the “thin-fat” phenotype [44]. Current pharmacocentric care overlooks lifestyle interventions; β-cell loss continues despite DPP-4, SGLT2 inhibitors, and GLP-1RAs, as none restore β-cell mass, and metformin improves insulin sensitivity without preservation [45].

9. Conclusion:

Intermittent fasting (IF) is a cost-effective, evidence-based strategy for managing T2DM, lowering HbA1c by 1.0–1.5% and preserving β-cell function through autophagy. Its cultural adaptability, including practices like Ramadan, makes it feasible in low-resource settings. Future studies should confirm its β-cell regenerative effects using biomarkers. With AI-powered glucose monitoring, IF can be personalized and integrated into WHO and national guidelines as a first-line option for prediabetes and early T2DM. By addressing both biological and socioeconomic factors, IF offers a sustainable approach to diabetes remission. Widespread adoption could significantly reduce the global diabetes burden.

Conflict of Interest: The authors declare no conflicts of interest.

Funding: No funding was received for this work.

Author Contributions: All authors contributed equally to the preparation and revision of the manuscript.

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