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Intermittent Fasting and Skin Health: Molecular Mechanisms and Clinical Implications

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Abstract: Objective: Intermittent fasting (IF) is a dietary pattern alternating between periods of eating and fasting, known for its metabolic, hormonal, and anti-inflammatory benefits. This review aims to explore the molecular, physiological, and clinical links between IF and skin health. Methods: A comprehensive review of recent preclinical and clinical literature was conducted, focusing on the effects of IF on skin biology, including modulation of oxidative stress, induction of autophagy, hormonal regulation, and interactions between the gut and skin. Results: Evidence suggests that IF may enhance skin regeneration, delay ageing, and improve inflammatory skin disorders such as acne, psoriasis, and eczema. Mechanistically, IF downregulates IGF-1 and mTOR signalling, enhances antioxidant defences, promotes autophagy, and modulates the immune response. Preliminary clinical findings also indicate potential benefits in photoaging, wound healing, and tissue repair. However, most data are preclinical, with limited human trials. Conclusion: IF represents a promising non-pharmaceutical strategy for improving skin health and managing dermatological conditions. Future research should focus on developing standardised protocols, molecular profiling, and assessing long-term safety in diverse populations.

Keywords: Intermittent Fasting; Skin Health; Autophagy; Inflammation; Dermatology.

Abbreviations:
IF: Intermittent Fasting
TRF: Time-Restricted Feeding
ADF: Alternate-Day Fasting
AGEs: Advanced Glycation End-Products
TEWL: Trans Epidermal Water Loss
EFAs: Essential Fatty Acids
HGH: Human Growth Hormone
ROS: Reactive Oxygen Species
SOD: Superoxide Dismutase
SCFA: Short-Chain Fatty Acid
CR: Caloric Restriction

I. INTRODUCTION

As a barrier, the skin protects the body from physical, chemical, and even microbial threats, making it the most exposed and most significant organ in the body. Additionally, the skin plays a crucial role in the body's thermoregulation, Perception of stimuli, immune responses, and metabolism, including the synthesis of vitamin D, are influenced by the skin's layers, such as the epidermis, dermis, and hypodermis, which reflect an individual's health condition.

Moreover, the skin serves unique functions, as it is influenced by intrinsic factors such as age, genetics, and hormonal balance, as well as lifestyle and environmental exposure [1].

Among lifestyle habits, nutrition has become increasingly recognised as a key factor that directly impacts skin health, repair processes, and the ageing process. Recently, intermittent fasting (IF) has garnered considerable academic and clinical attention as a potent non-pharmacological intervention that influences metabolism throughout the body, immune system activity, oxidative stress balance, and various cellular repair processes. IF is an eating pattern that alternates between periods of fasting and feeding and has a simpler structure than continuous caloric restriction. It may facilitate adherence and allow for some flexible metabolic adjustments over a prolonged period [2].

Some of the most prevalent Intermittent Fasting (IF) methods include the following [3]:

A. Time-Restricted Feeding (TRF): This involves limiting daily food intake to a specific window period, for example, 8 hours of eating followed by fasting for 16 hours (16:8 model).
B. Alternate-Day Fasting (ADF): This method involves periodic fasting days with no or minimal calorie consumption and alternating those with eating days.
C. The 5:2 Diet: Under this diet, one maintains everyday dietary habits for five days a week and restricts calories on two non-consecutive days.

Research studies regarding IF show that fasting improves one's insulin sensitivity, body fat percentage, lipid profile, systemic inflammation, and other factors. These alterations not only enhance cardiovascular and metabolic functions but also benefit the skin, which is sensitive to hormonal and inflammatory changes [4].

Regardless of this increasing focus, the research burden investigating the impacts of intermittent fasting on the skin's biology, including the potential effects of skin ageing visage as fasting windows trigger metabolic shifts, and intermittent fasting (IF) on various skin functions such as wound repair, skin barrier maintenance, and modulating skin conditions, remains sparse. Nonetheless, some preclinical research and a few clinical trials indicate that intermittent fasting may have positive effects on the skin by [5]:

A. Targeting oxidative stress, one of the leading causes of skin ageing and inflammation.
B. Promoting Autophagy, a self-

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repairing cellular mechanism crucial for skin renewal.

C. Modulating immune response alongside pro-inflammatory cytokines associated with psoriasis and eczema.

D. Regulating the gut–skin axis, as eating habits influence the makeup of the gut microbiome that has an impact on the skin through immune mediators.

With the increasing prevalence of chronic skin diseases and the more complex demands for lifestyle modifications, it is crucial to consider the impact of intermittent fasting on skin health. This review will focus on addressing this gap by:

A. Summarizing available molecular, cellular and clinical data regarding intermittent fasting and skin physiology;

B. Identifying biological pathways through which intermittent fasting may offer skin protective or therapeutic benefits;

C. Summarizing clinical literature on intermittent fasting and dermatological conditions including acne, psoriasis, eczema, and skin aging.

D. Reviewing potential risks and contraindications of prolonged fasting [6].

E. Formulating recommendations for further research aimed at incorporating intermittent fasting into dermatologic care and skin health policies.

F. Evaluating the relationship between nutrition timing and skin biology reveals how intermittent fasting may assist with skin health concerns and support healthy ageing.

G. The skin not only acts as the body's first line of defence, but also as a sophisticated organ system that indicates one's internal health and metabolism. From a structural standpoint, the skin consists of three primary layers—the epidermis, dermis, and hypodermis—all of which contribute to homeostasis, immune function, and other bodily systems essential to health. The epidermis, the outermost layer, is primarily composed of keratinocytes that undergo a tightly regulated process of proliferation, differentiation, and desquamation. It provides a crucial barrier against environmental insults such as pathogens, ultraviolet radiation, and mechanical injury. The epidermis is avascular and relies heavily on nutrient diffusion from the dermal capillaries to sustain cellular activities [7].

H. The dermis contains fibroblasts, immune cells, collagen, and elastin fibres, as well as blood vessels and lymphatic vessels. It is a fibrous connective tissue located underneath the epidermis. This layer contributes to the skin's elasticity, strength, and its ability to heal. Furthermore, it helps regulate the body's temperature and provides immune protection [8].

I. The hypodermis, or subcutaneous tissue, contains connective tissue, large blood vessels, and adipocytes. This layer serves as a structural reserve and thermal insulator, while also providing structural and functional support to the layers above [9].

A. Skin as a Nutrient-Sensitive Organ

Due to the continual shedding of skin cells and the oxidative and inflammatory stressors to which the skin is exposed, the systemic nutritional status has a significant impact on skin physiology. Various nutrients play a crucial role in maintaining skin health as structural building blocks, signalling molecules, enzymatic cofactors, or antioxidants [10].

i. The diet containing vitamins A, C, D and E is crucial for collagen modulation and triggers immune function while serving as an antioxidant protector. To illustrate, vitamin C serves as a cofactor for prolyl hydroxylase enzymes in collagen synthesis, and vitamin E prevents cellular membranes from lipid peroxidation [11].

ii. Zinc, selenium, copper, and iron are examples of trace elements that aid in the proliferation of keratinocytes and immune function, as well as antioxidant enzyme activity. Zinc plays a crucial role in tissue recovery and exhibits anti-inflammatory properties in wounds and skin diseases, including acne and eczema [12].

iii. Among the essential fatty acids (EFAs), omega-3 and omega-6 polyunsaturated fatty acids play a crucial role in sustaining skin moisture by minimizing trans epidermal water loss (TEWL) and stopping barrier dysfunction and skin dryness (xerosis) [13].

Deficiencies or imbalances in these nutrients can impair epidermal renewal, compromise barrier integrity, delay wound healing, and exacerbate dermatologic conditions. Conversely, optimal nutritional intake can enhance skin elasticity, hydration, immune resilience, and resistance to ageing-related changes [14].

B. Nutrition, Metabolism, and Cutaneous Health

The skin's physiology is adversely affected by metabolic and hormonal imbalances that come from a high-glycemic diet, as well as insulin resistance, obesity, and inflammation. The pathophysiological mechanisms include the formation of advanced glycation end-products (AGEs), which irreversibly bind to collagen fibres, leading to loss of elasticity and exacerbating the development of wrinkles. Moreover, chronic low-grade inflammation, along with oxidative stress, contributes to the inflammatory pathways of acne, psoriasis, and atopic dermatitis, which are common skin disorders [15].

The skin does not passively respond to metabolic signals; it interacts with insulin, IGF-1, and glucocorticoids and adapts to changes in their levels. These factors can also modulate the expression of specific genes, affect mitochondrial activity, and alter the skin's ability to rejuvenate and repair tissues [16].

II. INTERMITTENT FASTING: CONCEPTS AND MECHANISMS

A. Intermittent Fasting as a Metabolic Modulator of Skin Function

New research suggests that intermittent fasting (IF) may have a broader impact on more than just metabolism and immune function. IF alters metabolic and immune pathways, promotes stress adaptation at the cellular level, and modulates inflammation—all of which have effects on the skin [5].

One notable mechanism is the induction of hormetic stress, which is a form of low-level stress that triggers protective cellular mechanisms. Under IF regimens, this hormesis can lead to [17]:

i. Enhanced autophagy leading to the removal of damaged proteins and organelles in fibroblasts and basal keratinocytes.
ii. Stimulated antioxidant defences like glutathione peroxidase and superoxide dismutase.
iii. Reduced secretion of pro-inflammatory cytokines alleviates the inflamed surroundings that foster tissue injury, degradation, and disease flare exacerbation.

Additionally, IF could alter the skin microbiome and, simultaneously, affect it through the gut–skin axis. Changes in microbial composition resulting from fasting can influence systemic immune responses, potentially contributing to the improvement of inflammatory skin conditions.

B. A Basis for Therapeutic Exploration

The rationale for considering intermittent fasting (IF) as an adjunctive treatment in dermatology stems from the precise interplay of nutrient signalling, metabolic health, and skin biology. Fasting has been shown to aid in the management of numerous dermatological diseases by addressing systemic drivers of chronic inflammation, oxidative imbalance, and tissue repair deficits. These effects suggest IF could be an effective, low-cost, and easy means of improving skin health and preventing disease progression [6].

Fasting intermittently is one of several forms of dietary restrictions. Major differentiators stem from periods of caloric restriction, followed by periods of regular food intake. Unlike traditional calorie restriction, where a dieter is directed to reduce food intake, intermittent fasting focuses on the consumption windows. This method has garnered considerable attention due to its impactful metabolic, hormonal, and cellular changes, many of which are related to ageing. Notably, these alterations may also have a positive impact on skin health [5].

C. Patterns of Intermittent Fasting

Different IF schemes have been studied in clinical and preclinical research, each with varying implications for metabolism, endocrine function and cellular homeostasis:

i. *Time-Restricted Feeding (TRF):* This method restricts eating to a specific time within a 24-hour cycle (typically 6 - 10 hours) with fasting during the remaining hours. One of the most studied and widely adopted patterns is the 16:8 model, which consists of 16 hours fasting and 8 hours feeding. TRF facilitates aligning food consumption with circadian rhythms, which may improve efficiency and decrease inflammation after meals [18].
ii. *Alternate-Day Fasting (ADF):* In this model, there are alternating complete fasting/very low energy (<25% of daily energy requirements) days and unrestricted eating days. This approach is associated with improved lipid profiles, insulin sensitivity, and elevated pro-inflammatory markers [19].
iii. *The 5:2 Diet:* This diet consists of five normal eating days and two non-consecutive days of calorie restriction (500-600 kcal). It is perceived as easier to adhere to for individuals seeking moderate benefits over a sustained period.

Each of these patterns may produce unique physiological responses, but all share core benefits related to metabolic regulation, hormonal modulation, and inflammatory control [19].

III. MOLECULAR EFFECTS OF IF ON SKIN HEALTH

A. Metabolic and Hormonal Effects of Intermittent Fasting

i. A variety of preclinical and clinical studies have shown that IF has a positive impact on many core metabolic pathways with significant implications for skin physiology, including damage from glycation, oxidation, and the integrity of collagen [5].
ii. *Insulin Sensitivity and Glucose Homeostasis:* IF has a positive impact on fasting insulin levels and peripheral insulin sensitivity through peripheral tissue glucose uptake and suppression of hepatic glucose output. These changes can help counterbalance hyperglycemia-induced damage, including the formation of Advanced Glycation End Products (AGEs) that impair collagen structure and promote skin ageing [20].
iii. *Lipid Metabolism:* IF increases lipolysis and fatty acid oxidation, thereby reducing triglyceride and LDL cholesterol levels. These improvements are advantageous not only to cardiovascular health but also help to lower systemic inflammation, which contributes to inflammatory dermatoses such as psoriasis and acne [21].
iv. *Hormonal Changes:* Intermittent fasting is linked to lower levels of insulin-like growth factor-1 (IGF-1). This hormone promotes keratinocyte proliferation and sebum secretion while augmenting human growth hormone (HGH) levels, which aids in dermal fibroblast activity and collagen synthesis. These changes in hormones help slow down skin ageing and improve repair mechanisms in the skin [21].
v. *Increased Autophagy:* One of the most interesting cellular responses to fasting is the initiation of autophagy, which is a conserved catabolic process allowing cells to destroy and recycle damaged organelles, protein aggregates, as well as lipid droplets. Increased autophagy promotes the maintenance of skin cell resilience, enhances resistance to oxidative stress, and supports the reversal of cellular ageing [22].

B. Systemic and Organ-Specific Impact of IF

i. Apart from its metabolic effects, intermittent fasting (IF) has far-reaching impacts that affect inflammation, oxidative stress, mitochondrial function, and immune system balance—all factors that may be directly or indirectly important for dermatological issues [5].

ii. *Anti-Inflammatory Actions:* IF blocks the pro-inflammatory signalling molecules NF-κB and TNF-α and stimulates the production of anti-inflammatory cytokines. Chronic low-grade inflammation ("inflammaging") contributes to systemic ageing and cutaneous ageing; hence, IF can be considered an effective anti-inflammatory therapy [23].

iii. *mTOR, SIRT1, and AMPK Pathways:* IF manages to suppress the activity of the mechanistic target of rapamycin (mTOR), a master regulator of cell growth and metabolism that is often overactivated in hyperproliferative skin disorders. At the same time, IF activates AMP-activated protein kinase (AMPK) and sirtuin 1 (SIRT1), which are associated with protective mitochondrial functions, cellular longevity, and defences against oxidative stress. The boost to dermal resiliency and protection from photoaging resulting from these pathways is substantial [24].

iv. *Gut Microbiota Modulation and the Gut-Skin Axis:* IF has the potential to modify the structure and function of the gut microbiome, increasing preexisting microbial diversity along with SCFA-producing bacteria. It helps reduce endotoxemia, improves intestinal barrier function, modulates systemic immunity, and strengthens the immune response. These changes via the gut-skin axis may help reduce inflammatory skin diseases, including atopic dermatitis, rosacea, and acne [25].

C. Molecular Mechanisms Linking Intermittent Fasting to Skin Health

Intermittent fasting (IF) affects integrated molecular and cellular networks that are crucial for maintaining skin health, controlling inflammation, promoting rejuvenation, and optimising wound healing. These mechanisms represent global metabolic changes elicited by fasting, providing plausible explanations for the dermatologic benefits associated with fasting [5].

IV. MOLECULAR MECHANISMS LINKING IF TO SKIN HEALTH

A. Regulation of Inflammation

Chronic low-intensity inflammation, also known as "inflammaging," plays a significant role in both intrinsic and extrinsic skin ageing. It also contributes to the development of inflammatory skin disorders, such as psoriasis, eczema, and rosacea. IF helps fight inflammation by acting on some of the major anti-inflammatory pathways [23].

i. Cytokines such as TNF-α and IL-6 are produced from inflammatory genes controlled by the transcription factor complex NF-κB. Fasting has been shown to inhibit NF-κB activation, which in turn decreases cytokine expression and immune cell activity, thereby improving tissue integrity [26].

ii. Persistent inflammation is associated with elevated circulating inflammatory biomarkers like CRP. Some studies of fasting have reported lower levels of these markers, which in turn reduce skin redness, irritation, and plaque formation [26].

This systemic downregulation of inflammation may alleviate symptoms in inflammatory skin disorders and improve skin texture and appearance in ageing individuals. The mechanism by which intermittent fasting suppresses inflammatory cytokine activity is illustrated in Figure 1.

B. Oxidative Stress and Antioxidant Defence

The skin is exposed to oxidative stress from various sources, including UV radiation, pollution, and metabolic processes. Reactive oxygen species (ROS) promote the deterioration of collagen, elastin, and lipids, thereby weakening the skin barrier and accelerating skin ageing [27].

Some studies on IF have shown that it enhances the skin's natural defence mechanisms by [28]:

i. Stimulating the expression and activity of key metabolic enzymes like superoxide dismutase (SOD), catalase, and glutathione peroxidase.

ii. Inducing the Nrf2 signalling pathway (nuclear factor erythroid 2-related factor 2), which controls the cellular antioxidant defence system and is responsible for upregulating protective genes, hence bolstering the defences against oxidative damage.

These responses improve dermal resilience, reduce wrinkle formation, and protect against photoaging.

C. Autophagy and Cellular Repair

Autophagy is a catabolic process through which cells degrade and recycle damaged proteins, organelles, and cytoplasmic debris via lysosomes. In the skin, autophagy maintains the health of keratinocytes and fibroblasts, supports epidermal turnover, and protects against genotoxic stress [29].

Fasting induces autophagy through:

i. AMP-activated protein kinase (AMPK) activation, which enhances energy sensing and promotes autophagy.

ii. mTOR (mechanistic target of rapamycin) inhibition, which removes a key brake on autophagic flux.

Enhanced autophagy in dermal cells leads to improved mitochondrial function, protein homeostasis, and DNA repair. These changes help delay skin ageing, support barrier recovery, and may accelerate wound healing and scar remodelling [29].

D. Hormonal and Growth Factor Modulation

Hormonal signalling plays a crucial role in both the normal functioning and disease processes of the skin. Fasting has been shown to induce some hormonal alterations, which might have better cutaneous results [30]:

i. *Reduced Insulin and IGF-1 levels:* Chronic hyperinsulinemic and hyper-IGF-1 conditions are associated with escalated sebum production, follicular hyperkeratinization, and keratinocyte proliferation. These are the hallmark features of acne vulgaris and other hyperproliferative skin disorders. Fasting tends to downregulate the

IGF-1/PI3K/mTOR axis, leading to reduced cell turnover and enhanced barrier function.

ii. *Increased Human Growth Hormone (HGH):* HGH levels increase during fasting periods. This hormone is crucial for the activation of fibroblasts, collagen production, and dermal regeneration, thereby improving elasticity, enhancing skin tone, and promoting wound repair.

Such hormonal changes help in the modulation of anti-inflammatory and rejuvenative processes in the skin [30].

E. Influence on the Skin and Gut Microbiomes

Both the cutaneous and gastrointestinal microbiomes are essential in immune system functions, maintaining barrier integrity, and resisting pathogens. IF has been shown to alter the gut microbiota by [21]:

i. Elevating microbial diversity along with short-chain fatty acid (SCFA)-producing bacteria like Faecalibacterium prausnitzii [31].
ii. Lowering gut permeability and systemic endotoxemia, which reduces inflammatory responses that could potentially occur in the skin [21].

These microbiome alterations, through the gut-skin axis, may help reduce the risk or manage conditions such as eczema, rosacea, and seborrheic dermatitis. Moreover, evidence is scarce to suggest that fasting may directly impact the skin microbiota, although this is still an early-stage area of research [31].

V. CLINICAL EVIDENCE AND APPLICATIONS

The clinical evidence is sparse, though the promise shown by certain studies, trials, and case reports regarding diets with fasting windows to promote skin health is certainly intriguing. Some research indicates that certain dermatological disorders may benefit from fasting diets [5].

A. Acne Vulgaris

Acne can be triggered and developed due to several different factors, including inflammation, bacteria, and hormonal disorders related to androgens and IGF-1. Intermittent fasting (IF) has been shown to lower both insulin and IGF-1 levels, which, in turn, reduces activity of the sebaceous glands and decreases androgen sensitivity. There are unverified but suggestive reports of the positive impact of fasting diets on acne, which are more pronounced during Ramadan fasting. Further research is essential to establish fasting guidelines based on confirmed results [32].

B. Psoriasis

Psoriasis is a long-lasting skin condition that affects the immune system, causing the excessive proliferation of skin cells known as keratinocytes, inflammation throughout the body, and increased activity of Th17 cells. Psoriasis appears to benefit from IF by:

i. Reducing pro-inflammatory markers like TNF-α and IL-17.
ii. As noted in small interventional trials and during religious fasting, psoriasis also benefits from reduced erythema, scaling, and the thickness of plaques.

During Ramadan, a fasting clinical study demonstrated significant decreases in PASI scores for psoriasis among fasting subjects compared to non-fasting controls, thus endorsing the use of intermittent fasting as a complementary approach to psoriasis management [33].

C. Eczema (Atopic Dermatitis)

Atopic dermatitis includes barrier dysfunction, type 2 inflammation, and microbial dysbiosis. Although direct data are sparse, IF's anti-inflammatory effects, along with its microbiome modulation, could benefit eczema patients by:

i. Improving lipid profile and ceramide synthesis, leading to better skin barrier restoration.
ii. Gut–immune modulation leading to decreased inflammation and allergic response on a systemic level.

Further investigations are needed to assess the impact of IF on the prevention and management of eczema [34].

D. Skin Ageing and Photoaging

A decline in elastin and collagen marks the skin's ageing process, reduced dermal elasticity, and oxidative stress. Additionally, ultraviolet radiation and inflammaging contribute to the development of wrinkles. IF may help with:

i. Fortifying protective dermal fibroblasts through autophagy and antioxidant defences
ii. HGH and collagen synthesis are increased
iii. AGE build-up, which contributes to skin stasis and discolouration, is reduced.

Fasting has been shown to delay the signs of ageing in the skin of animals, including thinning of the epidermis and the development of wrinkles. Although limited, human data do support enhancement in skin tone and texture during IF programs [35].

E. Wound Healing and Tissue Regeneration

Heal wounds timely and efficiently by integrating all phases of inflammation, proliferation, and remodelling. This structured process may be intensified through fasting due to:

i. Activation of stem cells and greater regenerative capacity during fasting-induced autophagy [36].
ii. Decreased pro-inflammatory cytokines and reduced oxidative damage at the site of the wound [37].
iii. Enhanced angiogenesis and fibroblast function. [36].

Fasting has been associated with quicker epithelialization and decreased scarring in rodents. The distinction between fasting and continuous caloric restriction in humans remains a gap in research focus regarding wound healing [37].

Although traditional CR has proven useful, IF shows superior molecular and clinical benefits for dermatological applications (see Table 1). Other benefits include more potent triggers of autophagy, better responsiveness to inflammation, and greater patient compliance [36].

VI. CONCLUSION

Intermittent fasting (IF) emerges as a novel, non-drug approach to improve skin health and manage dermatological issues. By altering metabolic processes, regulating hormones, suppressing inflammation, enhancing antioxidant activity, and promoting autophagy, fasting has the potential to affect numerous skin functions, including barrier maintenance, senescence, immune function, and healing.

Preclinical studies appear to confirm IF's potential for dermatologic conditions, and early clinical feedback suggests its potential for treating acne, psoriasis, eczema, and age-related skin changes. However, the lack of uniform fasting guidelines and rigorous human clinical trials hinders progress.

Thus, there is a pressing need for clinical trials, combined with molecular profiling, to establish the optimal IF protocols, identify frameworks for targeted mechanisms, and assess safety in diverse populations over time.

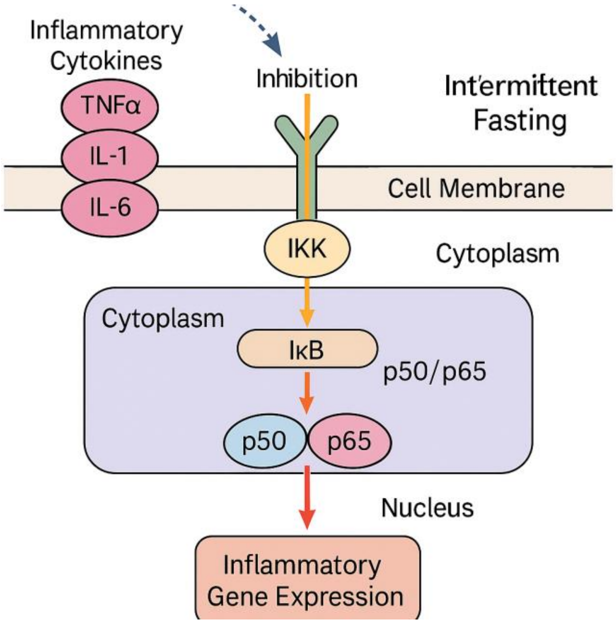
With the rise in demand for integrative and lifestyle options in dermatology, intermittent fasting could become a valuable complement to more traditional approaches, enabling more tailored and comprehensive skin care planning.

Table I: Comparison Between Intermittent Fasting and Continuous Caloric Restriction in Dermatologic Contexts

Feature	Intermittent Fasting (IF)	Caloric Restriction (CR)
Nutritional Pattern	Alternating periods of fasting and eating (e.g., 16:8, ADF)	Constant reduction in daily caloric intake (e.g., 20–40%)
Metabolic Adaptation	Enhances metabolic flexibility and ketogenesis	Maintains glucose-dependent metabolism
Insulin & IGF-1 Reduction	Significant reduction due to fasting windows	Moderate reduction over time
Autophagy Activation	Strongly activated during fasting phases	Mild or delayed activation
Impact on Inflammation	Rapid suppression of NF-κB, TNF-α, and IL-6	Gradual modulation of inflammatory markers
Effect on Oxidative Stress	Boosts endogenous antioxidants (e.g., Nrf2)	Moderate impact on oxidative pathways
Skin Ageing Delay	↑ Collagen preservation, ↓ glycation end-products	Also beneficial, but slower effect
Clinical Skin Benefits	Promising effects on acne, psoriasis, and wound healing	Limited dermatologic data available
Compliance & Practicality	Higher patient adherence due to flexibility	Lower long-term adherence
Risks	Dehydration and nutrient deficiency in vulnerable groups	Muscle loss, nutritional deficiencies, and poor planning

Table II: Summary Table: Clinical Effects of Intermittent Fasting on Skin Conditions

Skin Condition	Proposed Mechanism	Evidence Level
Acne Vulgaris	Reduced IGF-1 and insulin levels	Preliminary clinical
Psoriasis	Anti-inflammatory cytokine modulation	Controlled trials
Eczema	Inflammation reduction, microbiome modulation	Limited data
Skin Aging	Enhanced autophagy, antioxidation	Animal & observational
Wound Healing	Stem cell activation, reduced inflammation	Animal studies



[Fig.1: Intermittent Fasting Inhibits the NF-κB Signalling Pathway by Suppressing IKK Activation, Thereby Reducing the Transcription of Inflammatory Cytokines, Including TNF-α, IL-1, and IL-6]

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I must verify the accuracy of the following information as the article's author.

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Ms. Shereen Taha is a dedicated and motivated pharmacist with a strong background in pharmaceutical sciences and academic supervision. I have several years of experience in practical laboratory training and scientific representation. I am passionate about teaching, continuous learning, and contributing to the fields of medicine and pharmaceuticals. I possess excellent communication skills, flexibility, and the ability to work both independently and as part of a team