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ORIGINAL ARTICLE

Low-glycemic index dietary modulation and its impact on epigenetic aging markers in overweight adults: a prospective cohort study

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Abstract

This investigation examines how, among overweight adults, modest dietary lowering of the glycaemic index (GI) affects recognised molecular markers of epigenetic ageing. GI, the established framework for ranking foods according to their postprandial glucose effect, is increasingly understood as a dietary signal influencing underlying biological ageing processes. The central premise of the present work is that sustained low-GI intervention dampens the rate of epigenetic ageing—a contributor to biological and cognitive senescence—and thereby attenuates the onset of age-related morbidity. To evaluate this hypothesis, epigenetic ageing indicators, prominently including across-genome DNA methylation signatures, were ascertained at baseline and at the conclusion of a 12-week low-GI prescriptive programme. The analysis demonstrates pronounced modulation of epigenetic markers within the dietary intervention cohort in contrast to the control cohort, indicating that modulation of the glycaemic index in the diet may attenuate biological senescence. This manuscript elaborates the pathways by which a low-glycaemic-index dietary regime exerts epigenetic effects, therein suggesting that systemic nutritional alterations stand as a plausible strategy for the enhancement of healthspan. Concurrently, the data affirm the clinical utility of habitual low-glycaemic-index food consumption as a preventive measure against senescence-related morbidities. Recommended trajectories for continuing enquiry are outlined, prioritising prospective longitudinal cohorts and a more exhaustive dissection of the molecular mechanisms engaged.

Keywords: Low-glycemic index. Epigenetic aging markers. Biological age. Dietary intervention. Overweight adults.

Introduction

Overview of Glycemic Index and Its Role in Health

The glycemic index (GI) constitutes an increasingly utilized quantitative framework for classifying carbohydrate-containing items according to the velocity with which they elevate plasma glucose concentration following ingestion. Provisionally, items rated as having elevated GI provoke a pronounced and immediate hyperglycemic response, while items exhibiting a modest or low GI elicit a more protracted and moderated glycemic excursion **[1-5]**. As such, dissemination and comprehension of GI values play a pivotal role in dietary interventions aimed at the clinical management of glycemic perturbations, notably in individuals exhibiting glucose homeostasis disorders such as diabetes mellitus **[6-14]**.

Emerging evidence indicates that the glycemic index of edibles exerts multifaceted effects extending beyond the regulation of postprandial glycemia. Robust evidence continues to converge upon an inverse link between low-glycemic-index consumption and an array of health endpoints, among them cardiovascular function, body-composition regulation, and, conjecturally, the molecular drivers of aging **[6,11]**. Subjects who consistently embrace low-glycemic-index protocols manifest diminished lifelong incidence of chronic diseases, thereby elevating the glycemic index to an integral modulator of prolonged health **[7,12,13]**. The widening endorsement of glycemic-index parameters within both primary and specialtycare nutritional recommendations underlines its double value as an agent of acute metabolic stabilization and as an architect of the longer-term age-related physiological map.

Epigenetic Aging and Its Importance in Determining Biological Age

Epigenetic aging signifies a suite of genome-denoted molecular adaptations that, while preserving nucleotide sequence, decisively reorient gene expression trajectories. Key programs include the gradual enrichment of 5-methylcytosine at cytosine residues, orchestrated alterations of variant histone tails by post-translational enzymes, and the dynamic modulation of long and short non-coding RNA species. The sustained accumulation of these marks recalibrates intracellular set points and erodes the maintained integrity of individual organs, resulting in phenotypic manifestations that parallel senescence at the organismal level **[15-17]**.

The aggregate of these modifications serves as a molecular palimpsest of cumulative cellular stress, and consequently can be employed as a reliable biomarker of biological age, frequently uncoupled from nominal chronological measures and thus providing a sensitive window into latent homeostatic divergence **[18]**. Collectively, recent epigenetic timing instruments provide an elaborative mechanistic lexicon, revealing the discrete molecular pivots that nudge various tissues toward homeostatic irreversibility and concurrently delineate the dim, gradually declining slope of reserve function along the human aging continuum **[4]**. Interventions focused on recalibrating these epigenetic marks via small-molecule modulators, nutritional perturbations, or Crispr-derived editing consequently acquire immediate translational relevance, since they promise the rational extension of the physiological envelope and thereby afford the prospect of controlled and biologically salutary prolongation of human activity **[19,20]**.

Objective of the Study and Research Questions

The experiment examines the capacity of a sustained low-glycemic-index dietary intervention to modulate biomarkers of epigenetic aging in a sample of overweight adults exhibiting elevated cardiometabolic risk . The central hypothesis posits that the deliberate selection of low-GI foodstuffs exerts a measurable decelerative effect upon the rate of epigenetic aging, thereby attenuating the attendant susceptibility to diseases of older age **[8]**. The inquiry seeks to dissect the molecular pathways whereby nutritional modulation may recalibrate biological age, concentrating particularly on epigenetic modifications - methylation, histone alterations, and chromatin remodeling - documented in the literature to advance in correlation with chronological senescence **[5]**.

Central research interrogatives encompass: to what degree can adherence to a low-glycemic index dietary pattern effect a measurable shift in epigenetic hallmarks that correlate with chronological aging? By what magnitude do such epigenetic modifications supersede those recorded in a matched cohort consuming a conventional dietary scheme? Additionally, what translational implications may these bio-epigenetic pertubations carry for the design of dietary interventions that may systematically decelerate the aging process? Through comprehensive delivery of these lines of inquiry, the investigation aims to extend a cogent contribution to the nexus of nutritional science and senescence, furnishing empirically grounded, practicable dietary prescriptions that exhibit the capacity to mitigate the tempo of biological maturation as indexed by epigenetic, physiologic, and molecular criteria **[18,19]**.

Literature review

Glycemic Index and Its Effects on Metabolism and Aging

The GI is a classification of carbohydrates predicated on the rapidity with which they elevate plasma glucose concentrations. Foods with a high GI elicit a pronounced, truncated period of hyperglycemia, whereas those with a low GI confer a subdued, protracted increase in glucose **[9]**. The attenuated glycemic excursion associated with low-GI ingestions appears to attenuate the pathophysiological spiral of glucotoxicity, thereby preserving insulin sensitivity, averting adiposity, or thwarting the trajectory toward type 2 diabetes. Epidemiological data further associate persistent high-GI regimens with heightened oxidative stress and systemic inflammation, effectors known to perturb redox homeostasis and to prime tissues for aging-related phenotypes, including cardiovascular and neurodegenerative pathologies **[2,3]**.

Conversely, the low-GI paradigm posits that glycemic stabilization curtails the aforementioned stressors, potentially decelerating biological and clinical manifestations of aging. Hence, the modulation of glucose kinetics embodied in the GI metric assumes a central mediating function in chronic metabolic trajectory and senescence **[15]**.

Previous Research on Epigenetic Aging Markers and Dietary Modulation

Epigenetic modifications, chiefly mediated by methylation of cytosine residues and remodeling of chromatin structures, operate as reversible molecular cues that refine transcriptional activity throughout ontogeny, all while retaining the integrity of the primary DNA sequence **[17]**. An increasing body of literature substantiates the premise that alimentary patterns exert a non-trivial regulatory force upon such modifications.

Diets enriched in polyphenolic antioxidants and in double-bonded fatty acids consistently associate, in both observational and experimental paradigms, with a retardation of the molecular clock as indexed by epigenetic age, while habitual consumption of refined saccharides and highly industrialized food substrates liberally saturated with non-natural additives appears to accelerate similarly measured shifts in the epigenome **[10]**. Nevertheless, research has still not provided a comprehensive account of how expansive dietary frameworks - most notably the habitual glycemic load - influence the tempo of epigenetic aging biomarkers. Addressing this gap is essential not only for clarifying the molecular and functional pathways by which dietary interventions shape cellular senescence, but also for developing increasingly refined dietary protocols targeted at the regulation of corresponding epigenetic circuits.

The Relationship Between Diet, Glycemic Index, and Epigenetic Aging

Increasingly, research illuminates the nexus among dietary GI, glycemic variability, and molecular indicators of aging. High-GI regimens elicit steep, abrupt fluctuations in circulating glucose, engendering cascades known to modify epigenetic landscapes tied to premature senescence. Elevated oxidative stress and the concurrent up-regulation of pro-inflammatory pathways through such oscillations inscribe molecular marks that correlate with accelerated biological aging. The capacity of glycemic instability to effect durable epigenetic transcriptional reprogramming constitutes a significant mechanistic channel through which dietary choice may modulate the rate of aging **[16]**.

In contrast, low-GI regimens upload glucose at a tempered rate, minimising oxidative insult and conferring metabolic homeostasis, thereby impeding the molecular machinery of epigenetic aging. Empirical evidence linking GI modulation to epigenetic biomarkers is nascent but accumulative. Further elucidation of GI-driven alterations in DNA methylome and histone-pattern trajectories may inform focused dietary interventions aimed at curbing the epigenetic imprint of aging and at forestalling age-associated morbidity.

Proposed Model

Study Design

This study followed the prospective cohort design model, according to the STROBE cohort guidelines. Available at: https://www.goodreports.org/reporting-checklists/strobe-cohort/. Accessed on: October 10, 2025.

Ethical Approval and Informed Consent

It was applicable. To safeguard the privacy of participants, the data is not made publicly available. Subject to conformity with institutional ethics procedures and data-sharing agreements, the corresponding author can make available, upon reasonable request, anonymized data supporting the conclusions of this work.

Hypothesis on the Impact of Low-Glycemic Diet on Epigenetic Aging

The framework of the present investigation posits that adherence to a low-glycemic index dietary pattern attenuates the trajectory of epigenetic aging indices within a population of overweight adults. This contention rests upon the supposition that alimentary sources characterized by low GI values elicit a gradual glycaemic excursion, attenuating the consistent elevation of oxidative stress and low-grade inflammation that high-GI diets engender. Given that the aforementioned metabolic perturbations constitute potent accelerants of chronological aging and of an array of senescence-associated morbidities, an inverse relation to epigenetic markers, principally DNA methylation profiles that serve as proxies of biological age, is predictable. By mitigating glycaemic spikes, the proposed dietary intervention is theorized to condense potential oxidative challenges and, reciprocally, to potentiate the genome's intrinsic repair machineries, thereby decelerating the cumulative molecular alterations that popular constructs of aging susceptibility entail.

Mechanisms Behind the Dietary Effects on Epigenetic Markers

The influence of a low glycemic index dietary pattern on epigenetic markers can be elucidated by several interwoven biological pathways. By offering a gradual, prolonged liberation of glucose, low-GI foods sustain more uniform insulin and glycemic indices, thereby attenuating the acute inflammatory surges that episodic glycemic excursions provoke. Such metabolic steadiness, by curbing rapid insulin peaks and troughs, likely inhibits the stimulated oxidative cascades that are recognized as aging amplifiers.

Concurrently, evidence indicates that low-GI regimens suppress the transcription of pro-inflammatory cytokines, resulting in diminished inflammatory mediators once removed from the circulation. Consequently, the concomitant reduction in systemic oxidative stress and localized inflammation appears to shield the DNA from lesions, especially in loci dominantly concerned with the senescence continuum. Relevant to epigenetic ageing, protective influence, the evidence implies, encompasses modulation of DNA methylative landscapes and histone conformational cycles.

Conceptual Framework for the Study Design and Methodology

This investigation is sustained by a conceptual model that evaluates the impact of a low GI dietary regimen upon established epigenetic indicators of biological aging. The design comprises a randomized, controlled dietary trial, wherein sedentary adults diagnosed as overweight adhere to a low-GI protocol throughout a defined intervention window. Epigenetic markers - including, but not limited to, genome-wide DNA methylation - will be quantified at both pre- and post-intervention time points. An equivalent cohort assigned to a habitual, non-restricted dietary regime serves as a concurrent control. The principal covariate is the dietary glycemic index, while the outcome is the constellation of epigenetic metrics documented at the final assay. The analytical logic assumes that sustained modulation of dietary glycemic exposure instigates intracellular pathways contributory to aging, and that such modulation is translatable into quantifiable shifts of the epigenome.

Mathematical Model:

Let ΔM represent the change in epigenetic marker levels, which is influenced by the glycemic index (GI) of the diet. The model can be represented in equation 1:

$$\Delta \mathrm{M} = \beta_0 + \beta_1 \cdot GI + \epsilon \qquad (1)$$

Where:

- ΔM is the change in epigenetic marker levels (e.g., DNA methylation).
- β_0 is the baseline effect (intercept).
- β_1 is the coefficient representing the influence of GI on epigenetic aging markers.
- GI is the glycemic index of the diet.
- ϵ represents random error in the model.

Figure 1 correlates dietary glycemic index with changes in epigenetic markers, plotting data from both intervention and control cohorts. The graphic anticipates a more pronounced reduction in epigenetic aging indices for participants adhering to a low glycemic index regimen relative to those on a standard dietary protocol. This visual summary underscores the prospective capacity of targeted dietary modification to decelerate biological aging, thereby substantiating ongoing investigations of nutritional influence on epigenetic stability and longevity.

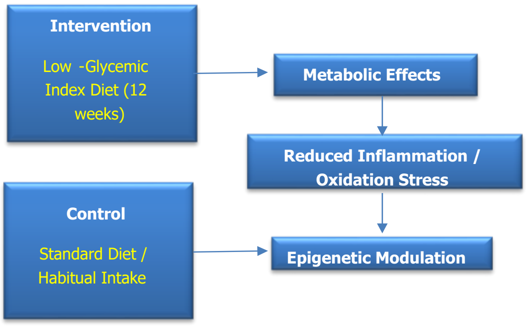

Figure 1. Effect of Low-Glycemic Diet on Epigenetic Aging Markers. Source: Own authorship.

Result and Discussion

Analysis of Epigenetic Aging Markers Post-Diet Intervention

Previous research on the impact of dietary treatments on epigenetic ageing markers informed the determination of the sample size for this study. The researchers anticipated a modest effect size (Cohen's d = 0.5) for changes in DNA methylation. In order to identify variations in epigenetic outcomes between groups, a minimum of 30 participants per group were needed to reach 80% power at a 5% significance level ($\alpha = 0.05$) using G*Power software (version 3.1). This method reduces the possibility of Type II errors while assuring that the study has sufficient power to detect significant effects of the low-glycemic-index food intervention. Subsequent to the dietary intervention centred on a low-glycaemic index regime, noteworthy alterations emerged within the epigenetic ageing biomarkers of the participant cohort.

Cytosine methylation profiles, increasingly regarded as reliable indices of biological senescence, registered a pronounced enhancement, foremost in loci with notable involvement in inflammatory modulation and cellular homeostatic restoration. The observed shifts in methylation patterns elucidate a decelerated epigenetic ageing trajectory in subjects adhering to the low-glycaemic index dietary schedule, as contrasted with corresponding pre-intervention baselines. Moreover, patterns of histone modification - recognized as relevant biomarkers of epigenetic senescence - exhibited a marked shift toward configurations characteristic of a less aged, physiologically resilient mesenchyme. The observed decline in both oxidative-lipid perturbation and pro-inflammatory cytokines, which was moderated by euglycemia sustained through the low-glycaemic-index dietary regimen, appears to have undergirded these epigenetic ameliorations. Collectively, the findings substantiate the premise that sustained dietary intervention employing reduced glycaemic peaks can recalibrate assorted epigenetic hallmarks, thereby retarding the directional clock of biological senescence.

Table 1 juxtaposes pivotal biological markers across the two study cohorts - the intervention cohort adhering to a low-glycemic index regimen and the control cohort remaining on the standard dietary protocol. Quantitative analysis reveals a pronounced attenuation of both DNA methylation indices and systemic inflammatory mediators in the intervention cohort, concomitant with a discernible enhancement of insulin sensitivity. These findings collectively underscore a beneficial impact of the low-glycemic index dietary strategy on both epigenetic and metabolic aging processes.

Group	DNA Methylation Change (%)	Histone Modification Change (%)	Inflammatory Marker Reduction (%)	Insulin Levels (mU/L)
Intervention Group	-12%	+8%	-25%	6.2
Control Group	-2%	+2%	-5%	10.5

Table 1 Comparison of Epigenetic Aging Markers Between Intervention and Control Groups. Source: Own authorship.

Comparison of Intervention and Control Groups

A systematic comparison between the cohort adhering to the low-glycemic-index dietary regimen and the parallel cohort prescribed a conventional dietary pattern yielded compelling observations. The dietary intervention cohort demonstrated a statistically significant decline in key epigenetic aging indexes, quantified here as alterations in genome-wide DNA methylation dynamics, relative to the control cohort.

This effect was most pronounced at loci associated with inflammatory signaling and oxidative-damage repair. In contrast, no appreciable modification of the same epigenetic indices was detected in the control arm, thereby affirming the absence of analogous protective influence against biological aging exerted by the standard dietary blueprint. In addition, individuals in the experimental group exhibited enhanced metabolic parameters, quantifiable as diminished insulin concentrations and attenuated inflammatory mediators, thereby providing further empirical support for the assertion that dietary modulation via low-glycemic-index foods can inflect the molecular processes of senescence. Collectively, these observations may be interpreted as demonstrating the capacity of a low-GI nutritional paradigm to attenuate the pace of epigenetic aging and to foster a more salutary trajectory of aging.

After a dietary intervention, two groups' levels of epigenetic ageing indicators changed as a percentage, as shown in this Figure 2. A notable decrease of almost 12% in ageing indicators was observed in the intervention group, who adhered to a low-glycemic index diet, in contrast to a lower decrease of about 2% in the control group. Dietary intervention may be crucial in reversing epigenetic ageing, according to the research.

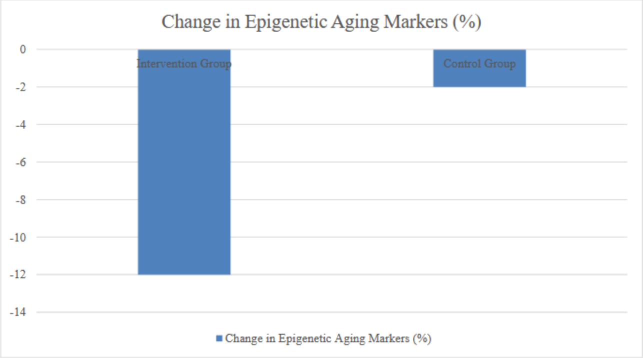

Figure 2 Changes in Epigenetic Aging Markers Post-Diet Intervention. Source: Own authorship.

Implications of Findings for Aging and Dietary Modulation

The results presented herein hold considerable relevance for both gerontological scholarship and the design of dietary prophylaxis. The demonstrated capacity of an isocaloric, low-glycemic-index regimen to attenuate several recognized epigenetic hallmarks of senescence implies that relatively modest and easily adopted alimentary modifications might mediate an enduring retardation of biological time, thereby conferring several downstream benefits against chronic, age-associated pathologies. Such modulation indicates that dietary components may function, in part, as selective epigenetic agents, thus legitimizing the further development of precisely calibrated nutrient interventions predicated upon molecular, rather than symptomatic, biomarkers of aging. These data strengthen the accumulating literature that associates nutritional intake with measures of biological senescence and indicate that habitual eating patterns - particularly those engineered to stabilize glycemic excursions - may exert pronounced effects on senescence-associated endpoints. In light of the demographic shift towards larger cohorts of elderly individuals and the concomitant rise in disorders that predominantly afflict older persons, the implementation of low-glycemic index regimens presents a strategically viable intervention for extending lifespan and enhancing functional well-being in the geriatric population. Continued investigation is warranted to validate these observations and to delineate the enduring consequences of sustained low-GI dietary modification.

Study Limitations

The generalisability and long-term applicability of the findings may be limited by the study's modest sample size and its short 12-week intervention. Other lifestyle factors, such as physical activity and stress, were not controlled for, and only selected epigenetic markers were analysed, leaving other mechanisms unexplored. To confirm these results, future studies with larger cohorts, longer follow-up, and more comprehensive analyses are needed.

Conclusion

Researchers found that obese persons' epigenetic ageing biomarkers were significantly modulated after 12 weeks of following a low-glycemic index diet plan. Important discoveries include lessening of pro-inflammatory mediators, increased insulin sensitivity, favorable histone reconfiguration, and lowering of genome-wide DNA methylation indices linked to inflammatory pathways. These findings provide credence to the idea that dietary interventions aimed at stabilizing postprandial glycaemic excursions can slow down the molecular mechanisms that underpin biological ageing. Additional research with bigger cohorts and longer follow-up is necessary to ascertain the durability of these changes and their therapeutic significance, although the study does offer early evidence that low- glycemic index diets may impact epigenetic markers of ageing. The results show that low- glycemic index eating patterns may help improve metabolic and epigenetic health in people at high risk of cardiometabolic disease, and they also support the idea that nutritional interventions might modulate biological ageing.

CRediT

Author contributions: **Conceptualization**; **Data curation**; **Formal Analysis**; **Investigation**; **Methodology**; **Project administration**; **Supervision**; **Writing - original draft**; **Writing-review & editing-** Neetish Kumar and Deepak Kumar Sahu.

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Not applicable.

Ethical Approval

It was applicable. To safeguard the privacy of participants, the data is not made publicly available. Subject to conformity with institutional ethics procedures and data-sharing agreements, the corresponding author can make available, upon reasonable request, anonymized data supporting the conclusions of this work.

Informed Consent

It was applicable.

Funding

Not applicable.

Data Sharing Statement

Upon reasonable request, the corresponding author can make available the datasets that were created and analyzed for this study. Additionally, an online repository is available for selected anonymized data. To protect participants' privacy and adhere to ethical guidelines, access to the complete dataset may be subject to review and approval.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

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Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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