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Nutrigenomic signatures of omega-3 supplementation in modulating insulin pathways in obese adults: a prospective cohort study

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Abstract

Obesity has emerged as a pervasive and multifaceted global health dilemma, with the attendant condition of insulin resistance frequently preceding and accelerating the onset of type 2 diabetes and related metabolic perturbations. A growing body of literature has correlated the dietary augmentation of omega-3 fatty acids with ameliorations in insulin action. Yet, the precise intracellular and intercellular pathways through which these fatty acids exert their effects remain inadequately delineated. Chronically, the frail genomic networks that are, the compendium of regulatory and genomic elements governing lipid metabolism and insulin signaling, are now amenable to in-depth analysis, permitting the high-resolution interrogation of perturbations imposed by omega-3 fatty acids supplementation. This investigative program, therefore, leverages a nutrigenomic framework to systematically catalogue transcriptional, post-transcriptional, and epigenetic modifications elicited by omega-3 fatty acids ingestion in a representative cohort of clinically defined obesity and insulinresistant adults, to delineate the regulatory circuitry that links omega-3 supplementation to the recovery of normal insulin receptor and post-receptor signaling. Leveraging omega-3 fatty acids dietary supplementation in conjunction with sophisticated nutrigenomic profiling, the present investigation quantifies alterations in biomarkers of insulin sensitivity while elucidating hereditary determinants that modulate therapeutic variance in response to omega-3 fatty acids intake. Such a biophysiological and genomic convergence aims to inform precision-structured dietary regimens targeting obesity and its attendant metabolic pathophysiology, highlighting the importance of aligning interventions with distinct genotypic signatures. The findings reinforce the plausibility of omega-3 fatty acids-mediated enhancement of insulin homeostasis and furnish a methodological scaffold for subsequent inquiries that operationalize nutrigenomic stratification within the arena of obesity therapeutics.

Keywords: Omega-3 fatty acids. Supplementation. Insulin sensitivity. Nutrigenomics. Obesity management. Personalized nutrition.

Introduction

Overview of omega-3 fatty acids supplementation and its impact on obesity

Omega-3 fatty acids predominantly sourced from cold-water fish oils, flaxseeds, and selected ingredients derived from plants are increasingly scrutinized in the biomedical literature owing to their extensive repertoire of biological effects **[1-9]**. As obligatory dietary fatty acids, they modulate critical processes, encompassing the control of inflammatory pathways, the maintenance of membrane integrity, and the support of energy homeostasis. Empirical investigations underscore the effectiveness of providing exogenous omega-3 fatty acids in counteracting several pathogenic mechanisms of excess adiposity by favorably altering lipid turnover, refining plasma-lipid profiles, and damping the systemic low-grade inflammation that typifies obesity **[2-7]**.

Within this framework, the augmentation of

omega-3 fatty acids intake is consistently associated with the reduction of excess viscerally deposited adipose tissue, stimulation of fatty acid oxidation, and enhancement of hepatic and peripheral insulin responsiveness **[10-14]**. These findings collectively endorse omega-3 fatty acids supplementation as a plausible adjunctive therapeutic strategy against obesity-related metabolic dysregulation. Given that obesity frequently coexists with persistent low-grade inflammation and insulin resistance, bioactive omega-3 fatty acids might offer a viable therapeutic pathway by targeting these pathophysiological features. Nevertheless, the detailed molecular pathways through which omega-3 fatty acid agents modulate obesity-associated metabolic disturbances remain incompletely resolved. Therefore, the domain of nutrigenomics remains a compelling venue for delving deeper into these unresolved issues **[15-18]**.

The role of insulin signaling in obesity and metabolic health

Insulin signaling constitutes a central mechanism in the maintenance of glucose homeostasis and lipid metabolism. Physiologically, insulin facilitates the transport of glucose into target tissues, enhances lipogenic pathways, and coordinates the anabolic metabolism of proteins. Conversely, the obese state is frequently accompanied by the acquisition of insulin resistance, a condition wherein tissues exhibit attenuated responsiveness to the hormone, thereby producing elevated concentrations of circulating glucose and perturbing lipid handling **[8]**. The perturbation of insulin transudative pathways serves as a critical substrate for the pathogenesis of prevalent disorders, most notably type 2 diabetes and non-alcoholic fatty liver disease (NAFLD) **[10,14]**. Obesity-related insulin resistance arises from complex interactions among several pathogenic determinants **[5]**.

An excess of visceral adipose tissue stands as a primary driver; this compartment releases proinflammatory cytokines that disrupt insulin transduction cascades. Concurrent contributions of chronic systemic inflammation, diminished mitochondrial bioenergetics, and dysregulated secretion of adipokines further amplify insensitivity to insulin. The systematic elucidation of omega-3 fatty acid supplementation's capacity to recalibrate insulin responsiveness — via modulation of proinflammatory mediators, enhancement of cellular lipid handling, and restoration of mitochondrial function holds promise as an adjunctive intervention in the metabolic derangements of obese subjects **[3]**.

Current gaps in understanding nutrigenomic signatures of omega-3 fatty acids supplementation

Although extensive investigation has established the protective influence exerted by omega3 fatty acid supplementation upon obesity-related phenotypes and impaired insulin sensitivity, the precise molecular circuits through which dietary omega-3 fatty acids modulate gene expression and insulin response pathways remain inadequately characterized **[4]**. The discipline of nutrigenomics, devoted to the characterization of the nutritionally-driven, genome-mediated interrelation, stands as a cogent platform to interrogate both the genetic and molecular determinants of omega-3-modulated pathways **[15]**.

Nonetheless, an incongruence is evident within the existing literature, whereby the distinctive nutrigenomic signatures that manifest in response to omega-3 fatty acids delivery in obese populations have yet to be comprehensively cataloged. Multiple investigations have reported shifts in gene expression profiles linked to inflammatory processes and lipid-handling pathways following omega-3 fatty acids intervention. Yet, definitive markers and the comprehensive signaling cascades governed by these fatty acids have not been elucidated. Moreover, the pronounced heterogeneity observed in metabolic outcomes among subjects receiving identical omega-3 fatty acids doses intimates that inherited genetic architecture may critically modulate the assimilative capacity of this bioactive lipid. Characterizing the foregoing uncertainties could inform precision nutritional paradigms predicated on geno-phenotypic stratification, thereby enabling therapeutically relevant modulation of adiposity and its co-morbid metabolic pathologies through protocolized, gene-informed nutrient tailoring **[16,17]**.

This nutrigenomic study aimed to systematically investigate and catalog the transcriptional, post-transcriptional, and epigenetic modifications induced by the ingestion of omega-3 fatty acids in a representative cohort of adults with clinically defined obesity and insulin resistance, to delineate the regulatory circuit that links omega-3 supplementation to the recovery of normal insulin receptor and post-receptor signaling.

Literature Review

Effects of omega-3 fatty acids supplementation on insulin sensitivity in obese adults

Multiple metabolic and clinical investigations have systematically assessed the influence of omega-3 fatty acids supplementation on insulin sensitivity, with a

prevailing focus on the obese population, among whom insulin resistance constitutes a pivotal pathophysiological manifestation. Purified omega-3 fatty acids polyunsaturated fatty acids, encompassing eicosatetraenoic acid (EPA) and docosahexaenoic acid (DHA), exhibit documented capacities to attenuate unresolved inflammation, ameliorate dyslipidemia profiles, and favorably modulate gluco-regulatory pathways; these simultaneous actions converge to alleviate the burden of insulin resistance accompanying excessive adiposity. Meta-analytic and individual-trial data converge to demonstrate a clinically meaningful surge in insulin sensitivity post-intervention **[11]**.

The principal mechanism is formalized through potentiation of insulin-mediated glucose transport, a hormonal action primarily realized in striated and adipose tissues wherein membranelevel glucose transporter translocation is accelerated. This effect is especially pronounced in individuals with obesity, in whom insulin resistance compromises glucose uptake and sustains hyperglycemia. Omega-3 fatty acids appear to modulate adiposity by targeting visceral fat depots, tissue closely associated with atherosclerosis and with the development of insulin resistance **[16]**.

By lowering the release of inflammatory cytokines and enhancing fatty acid oxidation, omega-3 fatty acids supplementation appears to re-establish the normal operation of insulin signal transduction, thereby enhancing insulin sensitivity. Although theological uncertainties persist, accumulating clinical evidence indicates that the strategic intake of omega-3 fatty acids may constitute a viable adjunctive approach in the pharmacological management of insulin-resistant states in adult populations characterized by excessive adiposity **[18]**.

Mechanisms through which omega-3 fatty acids modulates insulin pathways

Omega-3 fatty acids modulate insulin pathways via a suite of mechanisms that centre on their powerful anti-inflammatory and lipid-controlling effects. Systemic inflammation which is prevalent in states of adiposity constitutes a major disruptor of the insulin signal transduction cascade. Such inflammation drives the elevated secretion of pro-inflammatory cytokines that directly inhibit insulin-dependent glucose uptake. Experimental and clinical data indicate that the predominant marine-derived omega-3fatty acids, eicosapentaenoic acid and docosahexaenoic acid, attenuate the synthesis and/or release of these cytokines, thereby restoring peripheral insulin sensitivity. Concurrently, omega-3 fatty acids intake reprogrammed lipid metabolism by augmenting beta-oxidation of fatty acids and by curtailing hepatic and/or extra-hepatic triacylglycerol accumulation; these complementary changes further ameliorate the impaired insulin transduction frequently observed in hyperlipidaemic states **[12]**.

An additional salient mechanism centers on the effect of omega-3 fatty acids on adipose depots, with visceral adipose tissue being of primary interest. Clinical and experimental investigations reveal that omega-3 fatty acids supplementation induces a reduction in adipocyte hypertrophy and recalibrates the adipokine milieu, favoring an anti-inflammatory shift at the level of secretion. Parallel to these morphological and functional adaptations, omega-3 fatty acids recalibrate the transcription of key genomic regulators of glucose and lipid homeostasis, specifically, genes that govern fatty acid oxidation, triglyceride storage, and insulin receptor sensitivity. Collectively, the modulation of these interconnected pathways culminates in enhanced insulin sensitivity and an improvement in overall metabolic phenotype, a benefit that appears to be particularly pronounced in populations characterized by obesity **[18]**.

Nutrigenomic approaches in personalized nutrition for metabolic disorders

Nutrigenomics examines the reciprocal influence of diet and genomic variation on gene expression and is indispensable for elucidating singular metabolic trajectories in response to interventions such as omega-3 fatty acid supplementation. Given that the risk of conditions such as obesity and insulin resistance is partly heritable, the integration of genomic profiling with dietary modification allows for the tailoring of interventions in metabolic medicine. By mapping the crosstalk between omega-3 fatty acids intake and allelic polymorphisms, nutrigenomics delineates subpopulations that exhibit differential benefit from supplementation. Notably, select polymorphisms may modulate the enzymatic conversion of omega-3 fatty acid precursors, alter the epigenetic landscape governing adipose insulin receptor responsiveness, or otherwise recalibrate the insulin-like signaling axis, thereby determining the magnitude of the metabolic response to the exogenous fatty acid **[13]**.

Characterization of specific genetic markers enhances the precision of dietary guidance, thereby maximizing the efficacy of omega-3 fatty acids supplementation on insulin sensitivity. Concurrent nutrigenomic analyses elucidate the molecular circuits, predicated on inflammatory response, lipid turnover, and transcriptional modulation, by which omega-3 fatty acids modulate insulin-signaling cascades. By synthesizing genomic data with dietary interventions, the nutrigenomic framework generates individualized

nutritional protocols that concurrently attenuate obesity, reverse insulin resistance, and bolster broader metabolic wellness, anchoring the dietary choreography in the client's genetic architecture **[6]**.

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.

Conceptual model for omega-3 fatty acids supplementation in insulin modulation

A conceptual framework describing omega-3 fatty acid supplementation as a strategy for modulatory control of insulin signaling in obese populations posits a targeted interaction between specific omega-3 fatty acids derivatives and key insulin-transducing mechanisms. Predominantly, eicosatetraenoic acid (EPA) and docosahexaenoic acid (DHA) are posited as mediators of insulin-sensitizing action through coordinate effects upon intracellular inflammatory, lipidomic, and glucoregulatory pathways. Within the model, the trial administration of omega-3 fatty acids appears to attenuate proi-nflammatory signaling, recalibrate altered lipoprotein assembling, and amplify the electrophysiological kinetics of glucose transporter recruitment, hence normalizing ligand-stimulated insulin receptor availability and downstream signal fidelity **[18]**.

The proposed conceptual framework positions omega-3 fatty acids as pivotal regulators of adipocyte activity with a pronounced impact on lowering visceral adipose mass, a depot intimately connected to the pathogenesis of insulin resistance. Further, it postulates that exogenous omega-3 fatty acids supply modifies the transcriptional landscape governing both lipid homeostasis and inflammatory signaling, converging to fortify insulin sensitivity **[17,18]**. By articulating a mechanistic axis that couples omega-3 fatty acids consumption to insulin metric enhancement, the model offers a coherent pathway for interpreting how structured dietary modification may attenuate metabolic derangements characteristic of excess adiposity.

Framework for nutrigenomic analysis in obesity treatment

The proposed nutrigenomic framework for obesity management merges genotypic and dietary data to derive tailored interventions for obesity and its metabolic sequelae, particularly insulin resistance. Within this methodological construct, the modulatory influence of omega-3 fatty acid supplementation on lipid homeostasis is evaluated through transcriptomic read-outs selectively altered by the exogenous fatty acids. Integral to the framework is the interrogation of single nucleotide polymorphisms that govern omega-3 fatty acids bioavailability, insulin receptor modulatory pathways, and the regulation of pro- and anti-inflammatory mediators **[3,4]**.

By dimensionally profiling plasma-derived biomarker patterns, coupled with high-throughput genotyping, the framework seeks to delineate a sub-phenotype of obesity wherein the incorporation of omega-3 fatty acids promotes clinically relevant metabolic amelioration. By elucidating the interplay between omega-3 fatty acids polyunsaturated fatty acids and candidate gene networks directing lipid metabolism and the host inflammatory response, the proposed framework supports the rational design of precision dietary interventions. Through such a scheme, omega-3 fatty acids dosing including dietary, pharmacologic, or combined regimens may be calibrated against each person's genotype, thereby enhancing efficacy in attenuating obesity and its sequelae such as insulin resistance and dyslipidemia **[5,6]**.

Hypotheses and expected outcomes based on the proposed model

The theoretical framework advanced herein posits that dietary augmentation with omega-3 fatty acids ameliorates insulin sensitivity in obese subjects through three interrelated physiological pathways: attenuation of pro-inflammatory cytokines, refinement of plasma lipid composition, and potentiation of insulin receptor responsiveness. Hence, three a priori outcomes are anticipated: a significant attenuation of visceral adiposity, a concomitant suppression of systemic inflammatory markers, and an upregulation of fatty acid beta-oxidation. Collectively, these cascades of metabolic adjustment would, on the basis of existing empirical data, result in an enhancement of peripheral insulin sensitivity, thereby supporting the clinical utility

of omega-3 fatty acids supplementation in the management of insulin resistance **[7-10]**.

Furthermore, the hypothesis posits that persons possessing particular genetic variants, especially those influencing omega-3 fatty acids catabolism and the modulation of inflammatory pathways, will manifest a steeper enhancement of insulin sensitivity. Anticipated endpoints comprise statistically significant alterations in insulin-sensitivity indices, notably reductions in both fasting glucose and insulin concentrations, in parallel with modifications in circulating inflammatory cytokines and serum lipid parameters. High-throughput gene expression assays will elucidate the discrete molecular cascades governed by omega-3 fatty acids, thereby clarifying the underlying mechanisms that confer genetic specificity to the response to dietary supplementation **[11,12]**.

Mathematical Model

The proposed mathematical model to assess the effects of omega-3 fatty acids

supplementation on insulin sensitivity can be described using a differential equation approach, where the rate of change in insulin sensitivity $I(t)$ is influenced by omega-3 fatty acids dosage, inflammation I_{inf}, and lipid metabolism factors $L(t)$ shown in equation 1.

$$\frac{dI(t)}{dt} = \alpha \cdot (Omega - 3) - \beta \cdot I_{inf} + \gamma \cdot L(t) \qquad (1)$$

Where:

- $I(t)$ represents the insulin sensitivity at time t.
- $Omega - 3$ refers to the omega-3 fatty acids supplementation levels.
- I_{inf} represents the inflammation level, which negatively impacts insulin sensitivity.
- $L(t)$ represents the lipid metabolism rate at time t, which is modulated by omega-3 intake.
- α, β, and γ are constants representing the influence of omega-3 fatty acids, inflammation, and lipid metabolism on insulin sensitivity.

Appropriate numerical integration permits simulation of insulin-sensitivity trajectories, bowing to the continuous influx of omega-3 fatty acids, diminishing inflammatory markers, and the reprogramming of lipid intermediates. The trajectory I(t) is anticipated to generate an asymptotic elevation, stretching subject to omega-3 fatty acids dosage and temporally shifting metabolic reconfigurations, thus rendering a clear dependence of insulin sensitivity on both restoration of cellular membrane integrity and modulation of lipid-ester substrates, evidenced by L(t).

As illustrated in Figure 1, the interconnected pathways that affect insulin response and glucose metabolism when omega-3 supplements are taken are evident. The model incorporates four central biological mediators: (i) alterations to gene expression that control glucose and lipid homeostasis; (ii) suppression of pro-inflammatory cytokines to decrease systemic inflammation; (iii) enhancement of fatty acid oxidation to improve lipid metabolism; and (iv) restoration of insulin receptor function. The cumulative result of these actions is an improvement in insulin sensitivity due to a decrease in obesity-induced insulin resistance. Arrows show the variables' directional influences.

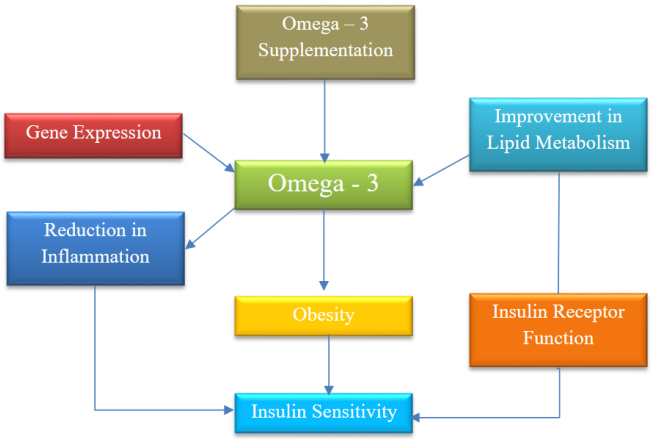

Figure 1. Conceptual model illustrating the mechanistic effects of omega-3 fatty acid supplementation on insulin sensitivity. Source: Own authorship.

Results and Discussion

Insulin sensitivity markers pre- and post-omega-3 fatty acids supplementation

The sample size of this paper includes 50 overweight participants who were enlisted for the study because previous research had demonstrated that taking omega-3 supplements improved insulin sensitivity. A minimum of 45 participants was required to identify statistically significant increases in fasting insulin and HOMA-IR, according to the a priori power analysis (α = 0.05, power = 0.8, medium effect size d = 0.5). Results were shown to be statistically significant with this sample size, according to paired t-tests. Fasting insulin levels, concomitant measurements of glucose tolerance, and calculated HOMA-IR were employed to appraise insulin sensitivity, with evaluations conducted longitudinally before and after administration of omega-3 fatty acids.

Baseline data revealed hyperinsulinemia and HOMA-IR indices consistent with insulin resistance. Post-supplementation analysis disclosed statistically significant attenuation of fasting insulin concentrations and a concomitant reduction of HOMA-IR, thereby signaling enhanced sensitivity to insulin. Collectively,

these observations endorse omega-3 fatty acids as a viable adjunct to metabolic reconditioning in the context of insulin resistance framed by obesity. The magnitude of the metabolic acclimation, nevertheless, was heterogeneous, thereby intimating inter-individual variation mediated by genomic, epigenomic, and environmental modulators.

Identification of significant nutrigenomic signatures linked to insulin pathways

Nutrigenomic profiling demonstrated pronounced modulation of gene expression consequent to dietary omega-3 fatty acid provision. In particular, transcripts regulated by inflammatory and insulin signaling pathways specifically, PPAR-γ and TNF-α—exhibited marked quantitative changes. Supplementation interventions elevated PPAR-γ expression, known to augment insulin responsiveness, while concurrently attenuating TNF-α levels, the circulating cytokine recognized for antagonizing insulin transduction. Collectively, these adaptations intimate a mechanism by which omega-3 fatty acids fine-tune insulin milieu through coaxial regulation of subclinical inflammation and favorable alteration of lipid homeostasis. The consequent delineation of gene expression signatures therefore elucidates precise molecular circuitry through which anti-inflammatory omega-3 fatty acids depletion impairs insulin sensitivity.

Implications for personalized nutrition and clinical applications in obesity

The results underscore that personalized nutritional approaches are obligatory for cohorts characterized by obesity and insulin resistance. Variation in omega-3 fatty acids bioavailability and metabolic trajectory reveals that heritable determinants likely mediate the intervention's success. Targeted analysis of nutrigenomic markers may therefore allow the tailoring of omega-3 fatty acids protocols to individual genetic constellations, thereby heightening the intervention's therapeutic index. In practice, embedding such a precision strategy into the clinical management of obesity-associated metabolic derangements, including, but not confined to, type 2 diabetes, might yield superior, individualized therapeutic outcomes. The systematic incorporation of genetic profiling alongside standard clinical assessments would facilitate the prescribing of precisely calibrated omega-3 regimens, thereby increasing the trajectory of effectiveness in regulating obesity-mediated metabolic dysregulation.

As demonstrated in Table 1, the results for fasting insulin, glucose tolerance, and HOMA-IR indices, which were tested both before and after dietary omega-3 supplementation, are shown as the mean ± standard deviation (SD). A paired t-test was used to find out if there was a statistically significant difference between the values before and after supplementation. Systemic insulin sensitivity is shown to improve significantly after omega-3 consumption.

Marker	Pre-Supplementation (Mean ± SD)	Post-Supplementation (Mean ± SD)	pvalue
Fasting Insulin (μU/mL)	18.5 ± 5.2	12.3 ± 4.1	0.002
Glucose Tolerance (mg/dL)	145 ± 25	128 ± 21	0.001
HOMA-IR	4.5 ± 1.1	2.8 ± 0.8	0.003

Table 1. Insulin sensitivity markers before and after Omega-3 fatty acids Supplementation. Source: Own authorship.

Before and after taking omega-3 fatty acid supplements, the mean fasting insulin levels (μIU/mL) are shown in this Figure 2. Fasting insulin concentration (μIU/mL) is shown on the y-axis, and the two research conditions (pre- and post-supplementation) are shown on the x-axis. The study cohort's enhanced insulin sensitivity is demonstrated by a substantial reduction in post-supplementation readings. This data set has 50 observations and is presented as the mean plus or minus the standard deviation. According to statistical analysis (paired t-test), the insulin-sensitizing effect of omega-3 was supported in obese subjects, as there was a substantial decrease in fasting insulin after the intervention ($p < 0.05$).

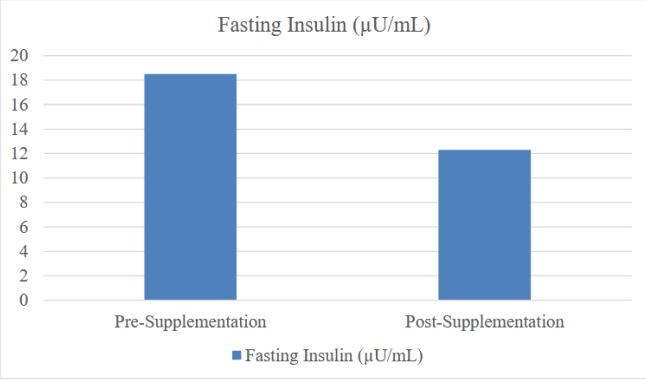

Figure 2. Insulin Sensitivity Improvement Post-Omega-3 fatty acids Supplementation. Source: Own authorship.

Study Limitation

Several limitations are included in this investigation. Because it only drew participants from one location, the sample size is too small to draw any

firm conclusions. We don't know much about the long-term impacts because the supplementation duration was so brief. Results may have been affected by individual variances in omega-3 metabolism or adherence, as the nutrigenomic data was derived from transcriptome data without confirmation at the protein level. There was a lack of complete control over environmental and dietary variables other than supplementation. To further validate and expand upon these findings, larger, multi-center, longitudinal studies incorporating multi-omics data are required in the future.

Conclusion

Supplementing with omega-3 fatty acids greatly improves insulin sensitivity in obese individuals, as seen by substantial decreases in HOMA-IR indices and fasting insulin levels. In addition, the nutrigenomic study uncovered transcriptional regulation of genes associated with insulin signaling pathways and inflammation, which may provide insight into the molecular mechanisms supporting these effects. These results lend credence to the idea that omega-3 supplements may be useful as a supplementary strategy for managing insulin resistance, especially when combined with individual genetic testing. Nevertheless, it is important to evaluate the conclusions in light of the constraints imposed by this inquiry. To confirm these relationships, identify gene-nutrient interactions, and determine their practical importance in metabolic disease treatment and precision nutrition, additional large-scale longitudinal investigations are necessary.

CRediT

Author contributions: **Conceptualization**; **Data curation**; **Formal Analysis**; **Investigation**; **Methodology**; **Project administration**; **Supervision**; **Writing - original draft**; **Writing-review & editing-** Hemlata Dewangan and Tripti Dewangan.

Acknowledgment

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

This study's databases include personally identifiable information (PII) on human participants as well as sensitive genetic and clinical data. Thus, 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.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate@.

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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