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Review Article

A Review on Nutrigenomics: Understanding Diet-Gene Interactions and Their Implications for Chronic Disease Prevention

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ABSTRACT

Nutrigenomics investigates the complex interactions between dietary components and individual genetic makeup, influencing health outcomes and disease susceptibility. This review evaluates the role of nutrigenomics in understanding diet-gene interactions and their impact on chronic disease prevention and management. A systematic review was performed to identify relevant literature on nutrigenomics and its applications in chronic disease management. Peer-reviewed articles, books, and reports were sourced from the Scopus, PubMed, and Google Scholar databases. The search utilized the following keywords: "nutrigenomics," "diet-gene interactions," "genetic polymorphisms," "bioactive foods," and "personalized nutrition." Studies were included based on their relevance to the application of nutrigenomics in the prevention or management of chronic diseases. Selection criteria prioritized studies that provided insights into diet-gene interactions, genetic polymorphisms influencing nutritional outcomes, and the role of bioactive food components in personalized nutrition strategies. Genetic variations, particularly single nucleotide polymorphisms (SNPs) in genes such as MTHFR, CYP1A2, GSTs, and ACE, significantly influence individual responses to dietary components and disease risk including cardiovascular diseases, certain cancers, and neural tube defects. Bioactive foods and supplements, including fenugreek, curcumin, HCA-SX, and various vitamins, demonstrate therapeutic potential in modulating gene expression and improving metabolic health. The gut microbiome plays a crucial role in mediating nutrient-gene interactions, with examples such as *Akkermansia muciniphila* impacting Western diet-induced atherosclerosis. While promising, translating nutrigenomics research into personalized nutrition strategies faces challenges due to limited human studies and reliance on animal models.

Keywords: Bioactive foods; Chronic diseases; Diet-gene interactions; Genetic polymorphisms; Gut microbiome; Nutrigenomics; Personalized nutrition

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INTRODUCTION

Nutritional genomics, commonly referred to as nutrigenomics, represents an emerging scientific discipline that examines the intricate relationships between living organisms' genomes, nutrition, and health outcomes (Alrawi *et al*., 2024). This field explores how dietary components influence the body's homeostasis and aims to identify cellular interactions that affect inflammatory stress pathways contributing to diet-related diseases (Benton *et al*., 2015). Nutrigenomics illuminates the complex interplay between bioactive compounds in food and human genes, establishing pathways for utilizing nutritional systems biology to discover biomarkers associated with disease susceptibility (Benton *et al*., 2015).

The revolutionary approach of nutrigenomics bridges molecular biology and nutrition by applying advanced molecular technologies to analyze diverse responses elicited by specific diets in individuals and populations (Reddy *et al*., 2018). A fundamental aspect of this field involves investigating whether dietary components can affect cellular gene expression and ultimately influence an organism's phenotypic profile (Reddy *et al*., 2018).

Methods in nutrigenomics aim to characterize gene products, including their physiological functions and interactions (Gobard & Hurlimann, 2019).

Historical examples of nutrigenomics include well-established interactions between food and inherited genes, termed 'inborn errors of metabolism,' which have long been managed through dietary manipulation. Phenylketonuria (PKU) exemplifies this concept, where individuals with a single gene mutation must avoid foods containing phenylalanine (Pullakhandam *et al*., 2018). The lactase persistence polymorphism among northern Europeans approximately 10,000-12,000 years ago represents another significant example, enabling continued lactase gene expression into adulthood and providing nutritional advantages in regions with short growing seasons (Reddy *et al*., 2018).

The current nutritional environment differs substantially from those to which humans genetically adapted. Major dietary changes accompanied animal domestication and the agricultural revolution about 10,000 years ago, followed by the Industrial Revolution and food technology developments that further altered food composition, particularly regarding fatty acid quantity and quality (Srinivasarao *et al*., 2017).

A systematic review was performed to identify relevant literature on nutrigenomics and its applications in chronic disease management. Peer-reviewed articles, books, and reports were sourced from the Scopus, PubMed, and Google Scholar databases as shown in Table 1 below. The search utilized the following keywords: "nutrigenomics," "diet-gene interactions," "genetic polymorphisms," "bioactive foods," and "personalized nutrition." Studies were included based on their relevance to the application of nutrigenomics in the prevention or management of chronic diseases. Selection criteria prioritized studies that provided insights into diet-gene interactions, genetic polymorphisms influencing nutritional outcomes, and the role of bioactive food components in personalized nutrition strategies.

Table 1: Synthesis of Literature on Nutrigenomics and Diet Gene Interaction

Reference	Focus Area	Key Findings	Implications for Chronic Disease Prevention
Alrawi *et al.,* 2024	Nutrigenomics Overview	Examines relationships between genomes, nutrition, and health outcomes.	Provides a framework for personalized nutrition to manage chronic diseases.
Benton et al., 2015	Diet-Gene Interactions	Dietary components like indole-3-carbinol influence CYP1A2 expression, affecting inflammatory stress pathways.	Highlights dietary modulation of gene expression to reduce cancer risk.
Bray *et al.,* 2004	Obesity Genetics	Genes regulate energy intake, lipid metabolism, and thermogenesis.	Genetic profiling can guide obesity prevention strategies.
Ferrannini *et al*., 2004	Type II Diabetes	High-sugar/fat diets cause glucolipotoxicity, impairing insulin secretion.	Dietary interventions can prevent diabetes progression.
Chobanian *et al*., 2003	Hypertension	Lifestyle changes modify hypertension risk.	Supports dietary and lifestyle interventions for cardiovascular health.
Karaman *et al.,* 2006	ACE Polymorphisms	ACE GG genotype linked to hypertension with high-saturated fat diets.	Supports genotype-specific dietary recommendations for hypertension.
Vijayakumar *et al*., 2015	Fenugreek Bioactives	Fenugreek extracts stimulate insulin signaling.	Potential for fenugreek in diabetes and dyslipidemia management.
Mrudula *et al*., 2017	Curcumin Benefits	Curcumin inhibits VEGF in diabetic retinopathy.	Supports curcumin use in preventing diabetic complications.
Roy *et al*., 2019	HCA-SX	HCA-SX aids weight management and reduces leptin expression.	Indicates potential for HCA-SX in obesity treatment.
Thompkinson *et al*., 2012	Omega-3 Fatty Acids	Omega-3 and antioxidants benefit cardiovascular health.	Supports inclusion of omega-3-rich foods in heart-healthy diets.
Yusuf & Sarin, 2016	Genetic Engineering	Fortified *Brassica juncea* increases α-tocopherol intake.	Demonstrates genetic engineering for nutritional enhancement.
Rojas *et al.,* 2011	AMPK Regulation	Low AMPK activity linked to high-fat diets and reduced glucose uptake.	Supports low-glycemic diets to enhance AMPK-mediated metabolic health.

Nutrigenomics and Microbiome Interactions

The human microbiome, comprising approximately 40 trillion microorganisms including bacteria, viruses, yeast, and fungi, significantly influences various physiological processes including metabolism, hematopoiesis, and immune function (Cho & Blaser, 2016). While relatively stable in adulthood, the microbiome can be altered by infections, antibiotic use, lifestyle factors, surgical procedures, diet, and various disease states (Cho & Blaser, 2016).

Research demonstrates that specific microbiota can provide health benefits, while others are associated with clinical pathologies. The atherosclerotic metabolic syndrome exemplifies this concept, where DNA from oral microbiota *Veillonella* and *Streptococcus* was identified in atherosclerotic plaques of individuals with

high abundances of these microbes in their digestive systems (Vatanen *et al*., 2016).

Conversely, certain microbes demonstrate protective effects against atherosclerosis. Studies show that *Akkermansia muciniphila* reversed Western diet-induced atherosclerosis in mice (Huo *et al*., 2013). Additionally, alterations in the balance between Bacteroidetes and Firmicutes, two major gut bacterial families, have been linked to obesity (Ley *et al*., 2006).

Probiotics, defined as live bioactive compounds recognized as safe by regulatory authorities, consist of beneficial bacteria and yeasts that naturally reside in the body. These organisms require regular consumption through diet or supplements due to their limited colonization ability. Probiotics offer benefits in conditions including lactose intolerance, irritable bowel syndrome, colorectal cancer risk reduction, and gastric ulcers, with additional potential benefits for osteoporosis, obesity, and type 2 diabetes prevention.

Key Genetic Polymorphisms in Nutrigenomics

Cytochrome P450 Enzymes and Genes

Cytochrome P450 (CYP) enzymes constitute a superfamily of heme-containing monooxygenases critical to the metabolism of a wide array of endogenous and exogenous compounds. These enzymes are predominantly membrane-bound proteins localized in the endoplasmic reticulum of hepatocytes, though they are also expressed in other tissues such as the intestines, lungs, and kidneys. Their primary function involves catalyzing oxidative reactions, facilitating the biotransformation of xenobiotics (e.g., drugs, toxins, and environmental pollutants) and endogenous molecules (steroids, fatty acids, and vitamins) through the transfer of oxygen atoms in a multi-step process (Guengerich *et al*., 2011). This oxidative metabolism often enhances the solubility of these compounds, aiding their elimination from the body.

CYP enzymes are encoded by a corresponding set of P450 genes, which exhibit significant genetic variability across individuals. These genetic polymorphisms can influence enzyme activity, leading to inter-individual differences in drug metabolism, toxin clearance, and susceptibility to environmental carcinogens. For example, single nucleotide polymorphisms (SNPs) in CYP genes can result in altered enzyme expression or function, categorizing individuals as poor, intermediate, extensive, or ultra-rapid metabolizers of specific substrates. The regulation of CYP gene expression is complex, involving transcriptional, post-transcriptional, and environmental factors. Nuclear receptors, such as the aryl hydrocarbon receptor (AhR), pregnane X receptor (PXR), and constitutive androstane receptor (CAR), play pivotal roles in modulating CYP gene transcription in response to environmental and dietary cues. Among the CYP enzymes, CYP1A2 is particularly significant due to its role in metabolizing a wide range of substrates, including drugs (e.g., caffeine, theophylline), environmental toxins, and dietary carcinogens such as heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs). The CYP1A2 gene, located on chromosome 15, is highly inducible, meaning its expression can be upregulated by various external factors.

CYP1A2 expression is induced by several dietary and environmental compounds such as; indole-3-carbinol, found in cruciferous vegetables like broccoli, cabbage, and Brussels sprouts, indole-3-carbinol activates the AhR pathway, leading to increased transcription of the CYP1A2 gene (Watkins *et al*., 2001). This induction enhances the metabolism of substrates processed by CYP1A2. Heterocyclic Amines (HCAs), formed during high-temperature cooking of meats, HCAs are both substrates and inducers of CYP1A2. Their presence in the diet, particularly in well-done or charred meats, can upregulate CYP1A2 expression, potentially increasing the bioactivation of these procarcinogens into reactive intermediates that may contribute to carcinogenesis (Benton *et al*., 2015). Polycyclic Aromatic Hydrocarbons (PAHs) also found in grilled or smoked foods and environmental pollutants like cigarette smoke, PAHs also induce CYP1A2 via the AhR pathway, amplifying the enzyme's activity in metabolizing these compounds. Conversely, certain dietary components inhibit CYP1A2 activity, thereby reducing its metabolic capacity. A notable example is naringenin, a flavonoid abundant in grapefruit and citrus fruits. Naringenin competitively inhibits CYP1A2, potentially slowing the metabolism of drugs and toxins processed by this enzyme (Watkins *et al*., 2001). This inhibition can lead to altered pharmacokinetics, such as increased bioavailability of drugs metabolized by CYP1A2, which may have clinical implications for drug dosing and toxicity.

The metabolic activity of CYP1A2 is governed by a dynamic interplay between genetic factors and dietary influences. Genetic variants of the CYP1A2 gene, such as the *1F allele (rs762551), are associated with altered enzyme inducibility and activity. For instance, individuals with the CYP1A2*1F variant may exhibit enhanced inducibility in response to environmental inducers like PAHs, leading to faster metabolism of substrates. Dietary habits further modulate CYP1A2 activity. Regular consumption of cruciferous vegetables may chronically upregulate CYP1A2 expression, while frequent intake of grapefruit or its juice could suppress its activity. This variability complicates the prediction of metabolic outcomes, as the metabolism of substrates

like HCAs depends on both an individual's genetic profile and their dietary patterns (Benton *et al*., 2015). For example, individuals with high CYP1A2 activity (due to genetic predisposition or dietary induction) may metabolize HCAs into reactive intermediates more rapidly, potentially increasing the risk of DNA damage and carcinogenesis, particularly in the context of high meat consumption.

Glutathione S-transferases (GSTs)

GSTs represent a major family of cytosolic enzymes catalyzing the conjugation of reduced glutathione to numerous electrophilic compounds generated by cytochrome P450 enzymes. These enzymes protect cells from cytotoxic and mutagenic effects of reactive compounds by preventing DNA adduct formation (Hayes & Flanagan, 2005). GSTs are classified into four major classes: alpha (GSTA), pi (GSTP), mu (GSTM), and theta (GSTT), with tissue-specific distribution patterns (Hayes & Flanagan, 2005).

Methylenetetrahydrofolate Reductase (MTHFR)

MTHFR exemplifies nutrient-gene interactions influencing cancer and chronic disease risk. This enzyme produces 5-methyltetrahydrofolate, a coenzyme necessary for converting homocysteine to methionine (Bailey & Gregory, 1999). The common C677T polymorphism results in the MTHFR 677TT genotype with reduced enzyme function. Studies suggest individuals with the TT genotype may require higher folate intake, and this variation modulates risk for vascular diseases, neoplastic diseases, and neural tube defects (Grønbaek *et al*., 2001).

Research demonstrates that men with the MTHFR 677TT genotype and adequate folate intake had a 55% lower colorectal cancer risk compared to those with other genotypes, though this protective effect was lost with insufficient folate intake (Giovannucci *et al*., 2020).

Nutrigenomics in Chronic Disease Management

Obesity

Genes influencing weight regulation are categorized based on genetically controlled processes contributing to body weight homeostasis, including physical activity, appetite, adipocyte differentiation, insulin signaling, mitochondrial functions, lipid turnover, thermogenesis, and energy efficiency (Bray *et al*., 2004). These genes regulate metabolic functions through various pathways:

- Energy intake regulation (MC3R, MC4R, POMC, LEP, LEPR, FTO) (Zhang *et al*., 2005)
- Lipid metabolism and adipogenesis (PLIN1, APOA5, LIPC, FABP2) (Sacks, 2006)
- Thermogenesis (ADBRs, UCPs) (Cinti, 2005)
- Adipocytokine synthesis (ADIPOQ, IL6) (Hotamisligil & Spiegelman, 1994)
- Transcription factors (PPARG, TCF7L2, CLOCK) (Rosen & Spiegelman, 2000)

Type II Diabetes Mellitus

Insulin secretion regulation is crucial for both glucose and fat metabolism, with impaired regulation characteristic of obesity and type II diabetes mellitus. High-sugar and saturated fatty acid diets induce "glucolipotoxicity," negatively impacting β-cell insulin secretion ability and resulting in hyperglycemia and hyperlipidemia (Ferrannini *et al*., 2004). Many effects of sugars and fats are mediated through transcriptional regulation of β-cell gene expression (Ohlsson *et al*., 2005).

Cardiovascular System Diseases

Hypertension, associated with heart failure, renal disease, stroke, and cardiovascular death, is highly modifiable through lifestyle changes (Chobanian *et al*., 2003). Angiotensin-converting enzyme (ACE) polymorphisms have been implicated in hypertension and cardiovascular disease risk (Danesh *et al*., 2004). The NUGAT study demonstrated that the GG genotype of rs4343 of ACE was associated with increased hypertension and cardiovascular disease risk in individuals consuming high-saturated fat diets (Karaman *et al*., 2006).

Salt sensitivity represents another important genetic factor, with variants in estrogen receptor (ESR2) and epithelial sodium channel (ENaC) genes associated with salt-sensitive blood pressure changes (Ji *et al*., 2010; Lifton *et al*., 2006).

Therapeutic Applications of Bioactive Foods

Fenugreek

Dialyzed aqueous extract of fenugreek seeds demonstrates hypoglycemic properties and stimulates insulin signaling pathways in adipocytes and liver cells (Vijayakumar *et al*., 2015). Thermostable extract of fenugreek seeds (TEFS) shows hypolipidemic effects in vitro, suggesting potential applications in dyslipidemia management (Vijayakumar *et al*., 2010).

Curcumin and Turmeric

Curcumin and turmeric are important for preventing and treating diabetic retinopathy. Studies show that curcumin can inhibit vascular endothelial growth factor (VEGF) expression in streptozotocin-induced diabetic rat retina (Mrudula *et al*., 2017). Additionally, curcumin demonstrates antiglycating properties that may delay cataract formation in diabetic conditions (Kumar *et al*., 2019).

HCA-SX

HCA-SX (Super Citrimax), derived from *Garcinia cambogia* fruit rind, serves as a unique source of hydroxycitric acid (HCA). Under experimental conditions, HCA-SX supplementation has been observed

to be conditionally effective in weight management and reduced abdominal fat leptin expression in both experimental animals and humans (Roy *et al*., 2019).

Omega-3 Fatty Acids and Antioxidants

Foods rich in omega-3 fatty acids, antioxidant vitamins, and fibers may benefit cardiovascular health (Thompkinson *et al*., 2012). Fish oils and nutraceuticals in vegetable fat-free diets with restricted lifestyle enhance cardio-protection and play major roles in positive gene regulation (Kamra *et al*., 2015).

Genetic Engineering Applications

Successful fortification of human diets with natural α-tocopherol through genetic engineering of *Brassica juncea* has been described, with α-tocopherol intakes exceeding recommended daily allowances associated with decreased cardiovascular disease risk and improved immune function (Yusuf & Sarin, 2016).

AMPK and Metabolic Regulation

AMP-activated protein kinase (AMPK) has emerged as a crucial element in energy control, appetite regulation, myogenesis, adipocyte differentiation, and cellular stress management. High-fat diets correlate with decreased AMPK-α2 isoform mRNA expression and AMPK phosphorylation, leading to decreased enzyme activity in skeletal muscle and reduced glucose uptake (Rojas *et al*., 2011).

Foods with low glycemic index may benefit body weight regulation through promoting satiety and increasing fatty acid oxidation, both mediated by AMPK action. High-protein diets control food intake through enhanced proopiomelanocortin (POMC) expression and neuropeptide Y (NPY) repression in the hypothalamus via mTOR activation and low AMPK phosphorylation rates (Rojas *et al*., 2011).

Factors Influencing Gene-Diet Interactions

Gene-diet interactions, a cornerstone of nutrigenomics, describe how dietary components interact with an individual's genetic makeup to influence health outcomes. These interactions are modulated by various factors, including ethnicity, geography, and environmental conditions, which shape both genetic profiles and dietary exposures. Understanding these factors is crucial for developing personalized nutrition strategies to prevent or manage chronic diseases such as obesity, type 2 diabetes, and cardiovascular disease.

Ethnicity

Ethnicity significantly influences gene-diet interactions due to genetic variations that differ across populations. These variations, often single nucleotide polymorphisms (SNPs), can affect how individuals metabolize nutrients, respond to dietary components, and are predisposed to certain diseases. For example, the *FTO* gene, associated with obesity and type 2 diabetes risk, shows varying effects across ethnic groups. In Asian populations, specific *FTO* variants have been linked to increased type 2 diabetes risk, with dietary factors like high-fat diets amplifying this predisposition (Lee *et al*., 2011). Similarly, African-specific haplotypes in the *FADS* gene cluster influence the metabolism of long-chain polyunsaturated fatty acids (PUFAs), such as omega-3 and omega-6, affecting inflammatory responses and cardiovascular health outcomes in response to dietary fats (Mathias *et al*., 2014). These ethnic-specific genetic profiles highlight the need for tailored dietary recommendations, as the same diet may yield different health outcomes in individuals of African, Asian, or European descent.

Ethnic differences also manifest in the prevalence of genetic polymorphisms affecting nutrient metabolism. For instance, the *MTHFR* 677TT genotype, which reduces enzyme activity and increases folate requirements, is more common in certain ethnic groups, such as those of European or Hispanic descent, compared to African populations (Bailey & Gregory, 1999). This polymorphism influences the risk of colorectal cancer and vascular diseases, with adequate folate intake reducing risk in individuals with the TT genotype by up to 55% (Giovannucci *et al*., 2020). Ethnic diversity in allele frequencies underscores the importance of considering genetic ancestry when designing nutritional interventions.

Geography

Geographic location shapes gene-diet interactions by determining dietary patterns and the availability of specific foods, which interact with local population genetics. Historical dietary adaptations, such as the lactase persistence polymorphism prevalent in northern European populations, illustrate how geography influences genetic adaptations to diet. Approximately 10,000–12,000 years ago, populations in regions with short growing seasons developed persistent lactase gene expression into adulthood, enabling lactose digestion and providing a nutritional advantage through dairy consumption (Reddy *et al*., 2018). In contrast, populations in regions with limited dairy availability, such as parts of East Asia or sub-Saharan Africa, have lower frequencies of this polymorphism, leading to lactose intolerance and different dietary practices.

Geographic variations also affect exposure to bioactive compounds. For example, populations in Mediterranean regions, where diets are rich in olive oil, fish, and vegetables, benefit from high intakes of omega-3 fatty acids and antioxidants, which interact with genes like *APOA5* to reduce cardiovascular disease risk (Thompkinson *et al*., 2012). Conversely, populations in areas with high consumption of grilled or charred

meats, such as parts of North America or Europe, are exposed to heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs), which induce *CYP1A2* expression and may increase cancer risk in individuals with certain genetic variants (Benton *et al*., 2015). These geographic dietary patterns, shaped by local agriculture and food availability, interact with genetic predispositions to influence health outcomes.

Environmental Conditions

Environmental conditions, including lifestyle factors, climate, and exposure to pollutants, further modulate gene-diet interactions. Environmental pollutants like PAHs, found in cigarette smoke or industrial areas, induce *CYP1A2* gene expression, enhancing the metabolism of dietary carcinogens and potentially increasing cancer risk in genetically susceptible individuals (Watkins *et al*., 2001). Urban environments with high pollution levels may thus exacerbate the effects of dietary components on gene expression compared to rural settings.

Lifestyle factors, such as physical activity and stress, also interact with diet and genetics. For instance, high-fat diets in sedentary environments reduce *AMPK* activity, impairing glucose uptake and increasing obesity risk, particularly in individuals with specific genetic variants like those in the *FTO* or *MC4R* genes (Rojas *et al*., 2011). In contrast, active lifestyles in rural or less industrialized regions may mitigate these effects by enhancing metabolic efficiency. Climate can also influence dietary choices; for example, colder climates may lead to higher consumption of energy-dense foods, which interact with genes regulating lipid metabolism (*PLIN1*, *LIPC*) and contribute to obesity or cardiovascular risk (Sacks, 2006).

The microbiome, shaped by environmental conditions such as sanitation, antibiotic use, and dietary habits, plays a critical role in gene-diet interactions. For example, an imbalance in *Bacteroidetes* and *Firmicutes* in the gut microbiome, often influenced by high-fat Western diets, is associated with obesity, while *Akkermansia muciniphila* has protective effects against atherosclerosis (Ley *et al*., 2006; Huo *et al*., 2013). Environmental factors like antibiotic exposure can alter microbiome composition, affecting how dietary components are metabolized and their subsequent interaction with host genes.

CONCLUSION

Nutrigenomics represents a promising frontier in personalized medicine and nutrition, offering potential for preventing and managing nutrition-related and non-communicable disorders. The field's evolution from understanding simple gene-diet interactions to complex multi-factorial relationships demonstrates its growing sophistication and clinical relevance.

While evidence supporting nutrient-gene interactions continues to develop, the conceptual framework of nutrigenomics provides valuable insights for future healthcare approaches. Early identification of at-risk individuals through genetic screening, combined with appropriate interventions including dietary modifications, weight management, and increased physical activity, could significantly prevent or delay the onset of chronic diseases.

The integration of microbiome research, bioactive food compounds, and genetic engineering applications further expands nutrigenomics' therapeutic potential. However, successful clinical implementation requires continued research, standardized protocols, and addressing ethical and accessibility concerns to ensure equitable benefits for diverse populations.

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Author Contributions

IMS: Conceptualization, methodology, writing original draft preparation, writing review and editing. **IM:** Data validation, analysis, writing review and editing. UUE: Data validation, writing review and editing. All authors have read and agreed to the published version of the manuscript.

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