

From Diet To Dementia Risk: Nutritional Epigenomics In Precision Alzheimer's Prevention

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Diet-sensitive epigenetic regulation may help explain how long-term nutritional exposure shapes Alzheimer's disease vulnerability. A precision-prevention model can combine diet quality, APOE-related risk, cardiometabolic profile, blood biomarkers, and multi-omic data to identify individuals most likely to benefit from targeted nutritional strategies.

INTRODUCTION	METHODS	RESULTS	CONCLUSIONS	REFERENCES
Alzheimer's disease develops gradually through interacting genetic, vascular, metabolic, inflammatory, and lifestyle-related pathways. Current prevention science increasingly recognizes that dementia risk can be modified before clinical symptoms become established. Diet is a major modifiable exposure because nutrients and dietary patterns can influence systemic metabolism, oxidative balance, vascular health, gut-brain signalling, and neuroimmune function. Nutritional epigenomics provides a mechanistic bridge between diet and brain ageing. Bioactive nutrients may affect DNA methylation, histone modifications, chromatin regulation, and non-coding RNA networks that regulate synaptic plasticity, mitochondrial function, lipid handling, inflammation, and proteostasis. This makes nutrition relevant not only as a lifestyle factor, but also as a biologically measurable component of precision Alzheimer's prevention.	This work presents a focused narrative synthesis of evidence linking nutrition, epigenetic regulation, and Alzheimer's disease prevention. The review mapped four connected domains: diet-sensitive epigenetic mechanisms, AD-related molecular pathways, biomarker-based risk stratification, and systems-level precision prevention. Evidence was organized around Mediterranean and MIND-style dietary patterns, omega-3 fatty acids, polyphenols, B-vitamins, antioxidants, choline, and fibre-derived metabolites. Key mechanisms included DNA methylation, histone acetylation and deacetylation, microRNA regulation, APOE-related lipid biology, neuroinflammation, oxidative stress, amyloid-tau biology, vascular-metabolic risk, and plasma biomarkers such as phosphorylated tau, neurofilament light chain, and glial markers.	Current evidence suggests that diet may modify dementia risk through epigenetic, metabolic, vascular, and inflammatory pathways. Brain-supportive diets may improve methyl-donor availability, lipid balance, mitochondrial function, antioxidant defence, and anti-inflammatory signalling. Nutrients such as omega-3 fatty acids, polyphenols, B-vitamins, antioxidants, and fibre-derived metabolites may influence gene regulation linked to synaptic health, amyloid-tau stress, oxidative injury, and neuroinflammation. Precision prevention becomes stronger when dietary data are combined with APOE genotype, cardiometabolic status, cognitive profile, plasma biomarkers, and longitudinal risk models. However, clinical translation needs standardized diet assessment, diverse cohorts, and validated predictive tools.	Nutritional epigenomics reframes Alzheimer's prevention as a dynamic interaction between diet, genes, metabolism, ageing, and brain vulnerability. The most useful future approach is targeted prevention: identifying high-risk individuals early, measuring biological change, and matching nutritional strategies to molecular and metabolic profiles. Future studies should integrate detailed dietary phenotyping, epigenomic markers, plasma biomarkers, neuroimaging, cognitive trajectories, and ancestry-aware genetic data. This approach may clarify who benefits from nutritional intervention, when prevention should begin, and which dietary components produce measurable brain-relevant effects.	Livingston et al., 2024, The Lancet. DOI: 10.1016/S0140-6736(24)01296-0. Palmqvist et al., 2025, Alzheimer's & Dementia. DOI: 10.1002/alz.70535. Barnes et al., 2023, New England Journal of Medicine. DOI: 10.1056/NEJMoa2302368. Castillo-Ordoñez et al., 2024, Neural Regeneration Research. DOI: 10.4103/1673-5374.382232. Bekdash, 2024, International Journal of Molecular Sciences. DOI: 10.3390/ijms25074036.
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