

# Traditional Asian Fermented Foods and Neuroprotection: A Narrative Review of Evidence on Neuroinflammation, Oxidative Stress, and the Microbiota–Gut–Brain Axis

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## ABSTRACT

**Introduction:** Neuroinflammation and oxidative stress contribute to the progression of neurodegenerative disorders. Traditional Asian fermented foods may influence these processes through microorganisms and fermentation-derived compounds acting along the microbiota–gut–brain axis (MGBA), although their clinical relevance remains uncertain. This review critically synthesised evidence concerning the potential effects of kimchi, miso, natto, and tempeh on neuroinflammation, oxidative stress, and MGBA-related pathways.

**Methods:** A structured narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, Embase, and Google Scholar. Peer-reviewed publications from January 2010 to June 2025 were considered. Twenty-seven publications were included, comprising 10 primary studies and 17 reviews. Mechanistic, animal, observational, and human-intervention evidence was synthesized thematically. Evidence from whole foods was distinguished from that concerning food-derived microorganisms and isolated bioactive components.

**Results:** Fermented-food-associated microorganisms, short-chain fatty acids, isoflavone aglycones, GABA, and bioactive peptides may influence intestinal-barrier function, inflammatory signaling, antioxidant responses, and neurotrophic pathways. However, evidence varied substantially among foods and was derived mainly from experimental models or isolated components. Human findings were limited, heterogeneous, and largely observational. Current evidence therefore supports biological plausibility more strongly than clinical neuroprotection.

**Conclusion:** Future research should evaluate standardized whole-food interventions using adequate sample sizes, longer follow-up, and clinically meaningful neurological outcomes. Current evidence is insufficient to recommend these foods specifically for preventing or treating neurodegenerative disease.

**Keywords:** Traditional Asian fermented foods; Microbiota–gut–brain axis; Neuroinflammation; Oxidative stress; Neuroprotection

## Introduction

Neurodegenerative disorders, particularly Alzheimer’s disease (AD) and Parkinson’s disease (PD), are major causes of progressive cognitive and functional impairment. Their pathogenesis is multifactorial, but neuroinflammation and oxidative stress are closely connected processes implicated in neuronal injury and disease progression. Persistent

activation of microglia and astrocytes increases the production of pro-inflammatory cytokines, including tumour necrosis factor-alpha and interleukin-6, while excessive reactive oxygen species contribute to mitochondrial dysfunction, lipid peroxidation, and neuronal damage [1–3]. Interactions between these pathways may therefore accelerate neurodegenerative changes.

The microbiota–gut–brain axis (MGBA) provides a framework for understanding how intestinal conditions may influence neurological function. It comprises bidirectional neural, endocrine, immune, and metabolic communication between the gastrointestinal tract and the central nervous system [1]. Gut dysbiosis and impaired intestinal-barrier integrity have been associated with systemic inflammation and oxidative stress, although their causal contribution to human neurodegenerative diseases remains uncertain [1,2]. Traditional Asian fermented foods, including kimchi, miso, natto, and tempeh, contain diverse microorganisms and fermentation-derived compounds. Depending on the food and production process, these may include lactic acid bacteria, *Bacillus* species, isoflavone aglycones, bioactive peptides, and  $\gamma$ -aminobutyric acid. Fermented foods may also modify intestinal microbial metabolism and the production of short-chain fatty acids [4,5]. Experimental evidence suggests that some of these microorganisms and compounds can influence inflammatory signaling, antioxidant responses, intestinal-barrier function, and neuroactive pathways [4,5]. Fermented soy products have received particular attention because fermentation can increase the availability of isoflavone aglycones and generate biologically active peptides [4]. However, the evidence must be interpreted cautiously. Much of the available research consists of *in vitro* experiments, animal studies, or investigations of isolated microorganisms and metabolites. Such findings provide mechanistic plausibility but do not demonstrate that consumption of a whole fermented food produces equivalent effects in humans. Direct clinical evidence concerning traditional Asian fermented foods, cognitive outcomes, and neurodegenerative diseases remains limited.

Traditional fermented foods are complex dietary matrices whose biological properties cannot be inferred solely from studies of isolated probiotic strains or purified metabolites. Differences in ingredients, starter cultures, fermentation conditions, storage, and preparation can substantially alter their microbial and chemical composition [4,5]. A critical synthesis is therefore needed to determine which proposed neuroprotective mechanisms are supported by whole-food studies, which depend on component-based evidence, and whether findings from experimental models are consistent with observations in human populations.

Several limitations prevent firm conclusions. First, evidence from whole fermented foods has not always been clearly distinguished from evidence concerning food-derived microorganisms or isolated compounds. Second, mechanistic, animal, observational, and interventional studies differ considerably in their ability to support causal inference. Third, heterogeneity in fermented-food composition, dose, intervention duration, study population, and

outcome measurement limits comparisons across studies. Fourth, the bioavailability and physiological relevance of several proposed neuroactive compounds remain uncertain, including whether dietary concentrations correspond to those used in experimental models. Finally, adequately powered and long-term human studies examining clinically meaningful neurological outcomes are scarce. Consequently, current evidence supports biological plausibility more strongly than clinical effectiveness.

This narrative review aims to critically synthesize evidence on the potential roles of selected traditional Asian fermented foods—principally kimchi, miso, natto, and tempeh—in modulating neuroinflammation, oxidative stress, and MGBA-related pathways. It distinguishes evidence derived from whole foods from that concerning food-derived microorganisms and isolated bioactive compounds, compares mechanistic, preclinical, observational, and interventional findings, and identifies priorities for future clinical research.

## Methods

### *Study design and scope*

This study was conducted as a structured narrative review to critically synthesize evidence on the potential roles of traditional Asian fermented foods in modulating neuroinflammation, oxidative stress, and microbiota–gut–brain axis (MGBA) pathways. A narrative approach was selected because the available literature comprised heterogeneous *in vitro* experiments, animal studies, observational studies, human interventions, and previous reviews. These publications differed considerably in food products, microorganisms, bioactive components, study populations, exposures, and outcome measurements, making quantitative synthesis inappropriate.

For this review, a traditional Asian fermented food was defined as a food originating from an identifiable Asian culinary tradition and produced through intentional microbial fermentation. The review focused principally on kimchi, miso, natto, and tempeh because these foods represented the main food categories identified in the literature relevant to the review objective. Evidence concerning other Asian fermented products, including doenjang and related fermented soy foods, was considered when it contributed to understanding shared biological mechanisms. The evidence was classified as whole-food evidence, microorganism-based evidence, or isolated-component evidence. Whole-food evidence referred to studies in which the complete fermented food was consumed or administered. Microorganism-based evidence included studies of microorganisms isolated from, or explicitly associated with, an eligible fermented food. Isolated-component evidence comprised studies of fermentation-derived metabolites or compounds,

such as short-chain fatty acids,  $\gamma$ -aminobutyric acid, bioactive peptides, nattokinase, and isoflavone aglycones. This classification was applied to prevent component-based findings from being interpreted as direct evidence of the effects of whole-food consumption. A record-level audit trail was not retained during the original literature search; therefore, complete retrieval, deduplication, and exclusion counts could not be reconstructed reliably. This limits the reproducibility and comprehensiveness of the review.

### **Literature search strategy**

The literature search was conducted between 1 May and 30 June 2025 and covered publications published from January 2010 to June 2025. PubMed/MEDLINE, Scopus, Web of Science, Embase, and Google Scholar were searched. Reference lists of relevant primary studies and previous reviews were also examined to identify additional publications. Previous systematic and narrative reviews were used to provide contextual information and locate potentially relevant primary studies; they were not treated as equivalent to primary experimental or clinical evidence. Reports from governmental agencies and international organizations were used only to support contextual or public-health information and were not included in the principal evidence synthesis.

The search combined terms relating to fermented foods, the MGBA, neuroinflammatory and oxidative pathways, and neurological outcomes. The principal search terms included “fermented food,” “Asian fermented food,” “kimchi,” “miso,” “natto,” “tempeh,” “doenjang,” “fermented soy,” and “fermented soybean.” These terms were combined using the Boolean operator AND with terms including “gut microbiota,” “microbiome,” “gut–brain axis,” “microbiota–gut–brain axis,” “dysbiosis,” “intestinal barrier,” “short-chain fatty acids,” “SCFAs,” “GABA,” “ $\gamma$ -aminobutyric acid,” “bioactive peptides,” and “isoflavones.” A third group of terms included “neuroinflammation,” “oxidative stress,” “neuroprotection,” “cognition,” “cognitive decline,” “dementia,” “Alzheimer’s disease,” “Parkinson’s disease,” “NF- $\kappa$ B,” “Nrf2,” “brain-derived neurotrophic factor,” and “BDNF.” The syntax was adapted to the indexing requirements of each database. No study-design filter was applied because the review aimed to compare evidence across mechanistic, preclinical, observational, and interventional levels. Searches were restricted to English-language articles because the reviewers had sufficient language proficiency to assess full texts. Studies published in other languages were excluded during screening.

### **Eligibility criteria**

Studies were eligible if they investigated a traditional Asian fermented food, a microorganism explicitly derived from or associated with an eligible

food, or an identified fermentation-derived component. Eligible studies were required to examine at least one outcome related to gut microbial composition, intestinal-barrier function, neuroinflammation, oxidative stress, neuroactive signaling, cognitive function, neurological function, or a neurodegenerative disorder. In vitro, animal, observational, and human-intervention studies published as full-text, peer-reviewed articles between January 2010 and June 2025 were considered. Publications were also required to provide sufficient methodological and outcome information to permit interpretation of their findings. Eligible studies were required to be published as full-text, peer-reviewed articles in English.

Studies of isolated microorganisms or metabolites were included only when their origin or relevance to an eligible fermented food was clearly established. These studies were classified as indirect or component-based evidence and were not interpreted as demonstrating the effects of consuming the whole food. Studies of non-Asian probiotic interventions were excluded from the food-specific synthesis unless the investigated microorganism had a clearly documented connection to an eligible traditional Asian fermented food. Where such studies were required to explain a relevant biological pathway, they were used only as indirect contextual evidence and were identified accordingly.

Studies were excluded if they focused exclusively on non-Asian fermented foods without a clear relationship to the review scope. Studies were also excluded if they examined food processing, sensory characteristics, microbial composition, or nutritional properties without reporting a relevant biological or health outcome. Publications reporting gastrointestinal outcomes without an MGBA-related, inflammatory, oxidative, cognitive, or neurological outcome were not eligible. Conference abstracts, editorials, letters, commentaries, protocols, preprints, theses, and other non-peer-reviewed publications were excluded. Where multiple publications reported substantially overlapping data, the most complete primary report was retained. Review articles were used for contextual interpretation and reference searching but were not cited as primary evidence for specific experimental or clinical outcomes when an original study was available. Articles published in languages other than English were excluded.

### **Study selection and data extraction**

Search records were entered into Microsoft Excel, where duplicates were identified by title, author, year, and DOI and removed manually. Two reviewers (SS and ST) independently screened titles, abstracts, and full texts against the eligibility criteria. Disagreements were resolved by discussion or consultation with a third reviewer (PR).

Using a standardized Excel form, SS extracted study design, population or model, sample size, food or component, exposure, comparator, dose, duration, outcomes, findings, and limitations. ST verified all entries against the original publications. Sources were classified by study design and as whole-food, microorganism-based, or component-based evidence. Excel was also used to document selection numbers and reasons for full-text exclusion.

### ***Evidence appraisal***

Because this study was designed as a narrative review, formal risk-of-bias tools were not applied. Nevertheless, the methodological credibility of each publication was considered according to its study design, directness of evidence, sample size, appropriateness of controls or comparators, duration of exposure, validity of outcome measurements, consistency with related studies, and major sources of bias or confounding. Greater interpretive weight was given to primary studies that directly examined whole fermented foods, employed appropriate controls, reported clearly defined neurological or mechanistic outcomes, and provided sufficient methodological information.

In vitro and animal findings were interpreted as mechanistic or preclinical evidence and were not considered proof of clinical neuroprotection. Observational associations were interpreted cautiously because they could be affected by residual confounding, dietary measurement error, reverse causation, and correlated lifestyle factors. Findings from isolated microorganisms or metabolites were treated as indirect evidence unless they were supported by studies of the corresponding whole food. No study was described as providing the “highest level of evidence” solely on the basis of its design. The interpretation also considered methodological quality, consistency, directness, biological relevance, and applicability to customary food consumption.

### ***Evidence synthesis***

A meta-analysis was not conducted because the included studies varied substantially in their designs, fermented-food preparations, microbial composition, isolated components, doses, intervention periods, populations, and outcomes. Evidence was therefore synthesised using a structured thematic narrative approach. The synthesis examined MGBA-related mechanisms of neuroinflammation and oxidative stress, fermentation-derived microorganisms and bioactive components, and evidence relating to kimchi, miso, natto, and tempeh. Mechanistic, animal, observational human, and controlled human-intervention findings were presented separately to clarify the strength and directness of the available evidence.

The MGBA was used as the conceptual framework for examining relationships among fermented-food exposure, intestinal microbial ecology, intestinal-barrier integrity, microbial metabolites, immune and endocrine signalling, oxidative stress, and neurological outcomes. The synthesis considered the direction and consistency of findings, principal methodological limitations, and relevance of experimental exposures to customary dietary consumption. Particular attention was given to uncertainties concerning the bioavailability of fermentation-derived compounds, the limited or uncertain blood–brain barrier permeability of GABA and bioactive peptides, and the extent to which findings from isolated components or experimental models could be translated to whole-food consumption in humans. Safety considerations, including sodium content, product variability, microbial contamination, and potential food–medication interactions, were also examined.

### ***Methodological considerations***

This review used a structured narrative design and was not intended to provide a systematic estimate of intervention effects. Its principal limitations included the absence of protocol registration, duplicate independent screening, formal risk-of-bias assessment, and quantitative synthesis. The breadth and heterogeneity of the evidence also required interpretive judgement when comparing mechanistic, animal, observational, and clinical findings. Potential language, publication, and selection biases could not be excluded. Consequently, the findings should be interpreted as a critical synthesis of the biological plausibility, consistency, directness, and limitations of the available evidence rather than definitive proof that traditional Asian fermented foods prevent or treat neurodegenerative disease. The restriction to English-language publications may have introduced language bias and excluded relevant evidence published in Asian languages, particularly Korean, Japanese, Chinese, Indonesian, and Thai. Consequently, the review may not represent all regional research on traditional Asian fermented foods.

## **Results**

### ***Study selection and characteristics of included sources***

Twenty-seven publications were included in the narrative synthesis. These comprised three in vitro or mechanistic studies, one animal study, three observational human studies, three controlled human-intervention studies, one systematic review, and 16 narrative, mechanistic, or general reviews. Thus, 10 sources were primary studies and 17 were reviews. The evidence base was dominated by review-level and indirect mechanistic evidence, whereas direct human research on whole traditional fermented foods was limited (Table 1).

The original searches produced 27 publications that met the review scope and were included in the narrative synthesis. Because this review was initially conducted as a narrative rather than a systematic review, record-level counts for database retrieval, deduplication, title and abstract screening, and full-text exclusion were

not retained. The included sources comprised three in vitro or mechanistic studies, one animal study, three observational studies, three controlled human-intervention studies, one systematic review, and 16 narrative or mechanistic reviews (Table 1).

**Table 1** Evidence profile of sources included in the narrative review

| Evidence category                     | Sources n (%)     | Main contribution                                                      | Directness to whole-food effects                                  | References                   |
|---------------------------------------|-------------------|------------------------------------------------------------------------|-------------------------------------------------------------------|------------------------------|
| In vitro or mechanistic studies       | 3 (11.1)          | Cellular pathways, GABA production, and metabolite characterization    | Low                                                               | [6,7,24]                     |
| Animal studies                        | 1 (3.7)           | Cognitive outcomes and neurotrophic signaling following natto exposure | Moderate for food exposure; indirect for humans                   | [13]                         |
| Observational human studies           | 3 (11.1)          | Associations between soy or natto intake and dementia-related outcomes | Moderate for dietary exposure; insufficient for causality         | [12,14,15]                   |
| Controlled human-intervention studies | 3 (11.1)          | Cognitive, oxidative-stress, equol-related, or stress outcomes         | Variable; some interventions were supplements or isolated strains | [16,19,20]                   |
| Systematic reviews                    | 1 (3.7)           | Synthesis of neuroprotection-related evidence in older populations     | Indirect                                                          | [21]                         |
| Narrative or mechanistic reviews      | 16 (59.3)         | Conceptual pathways, fermented-food components, and research context   | Low to moderate                                                   | [1–5,8–11,17,18,22,23,25–27] |
| <b>Total</b>                          | <b>27 (100.0)</b> | —                                                                      | —                                                                 | <b>[1–27]</b>                |

### MGBA-Related Mechanisms

The microbiota–gut–brain axis (MGBA) comprises neural, endocrine, immune, and metabolic communication between the gastrointestinal tract and the central nervous system [1]. Experimental evidence suggests that altered gut microbial composition and intestinal-barrier function may contribute to systemic inflammation and oxidative stress, although their causal roles in human neurodegenerative diseases remain uncertain [1,2].

Microbial metabolites may influence inflammatory and antioxidant pathways. SCFAs have been associated with intestinal-barrier maintenance and regulation of NF-κB- and Nrf2-related signaling [1,3]. Fermented-food-associated microorganisms may also affect immune, neuroactive, and hypothalamic–pituitary–adrenal pathways [1,5]. However, these findings arise mainly from experimental models or component-based studies and therefore provide mechanistic rather than clinical evidence.

Traditional Asian fermented foods contain variable combinations of lactic acid bacteria, *Bacillus* species, yeasts, isoflavone aglycones, GABA, and

bioactive peptides [4,5]. Fermented-soy isoflavones have been associated experimentally with antioxidant, anti-inflammatory, and neurotrophic pathways [4,6]. GABA-producing bacteria have also been identified among *Lactobacillus* and *Bifidobacterium* strains [7], while fermentation-derived peptides have demonstrated antioxidant and immunomodulatory activity in experimental systems [8]. The direct neurological relevance of these components remains uncertain because their digestion, absorption, metabolism, and tissue availability were not consistently assessed.

### Evidence According to Fermented-Food Type

#### Kimchi

Kimchi contains diverse lactic acid bacteria, particularly *Lactobacillus* and *Leuconostoc* species, although its microbial composition varies according to ingredients and fermentation stage [5]. Experimental and review-level evidence has linked kimchi-derived microorganisms with changes in gut microbial composition, inflammatory signalling, and oxidative-stress markers [10,11]. However, most findings concern isolated bacterial strains rather than whole-kimchi consumption. Direct human evidence relating kimchi

intake to cognitive or neurodegenerative outcomes was limited (Table 2).

#### Miso

Evidence concerning miso was mainly indirect. Fermentation increases the proportion of isoflavone aglycones in soy products, providing a possible basis for antioxidant and anti-inflammatory activity [4]. Observational research in Japan has examined associations between soy-containing dietary patterns and dementia-related outcomes [12]. However, these studies did not isolate the contribution of miso from that of the overall diet (Table 2).

#### Natto

Natto contains nattokinase, vitamin K<sub>2</sub>, poly- $\gamma$ -glutamic acid, peptides, and fermented-soy isoflavones. In an animal model, natto exposure was associated with cognitive changes and regulation of the hippocampal TAAR1–CaMKII/CREB/BDNF pathway [13].

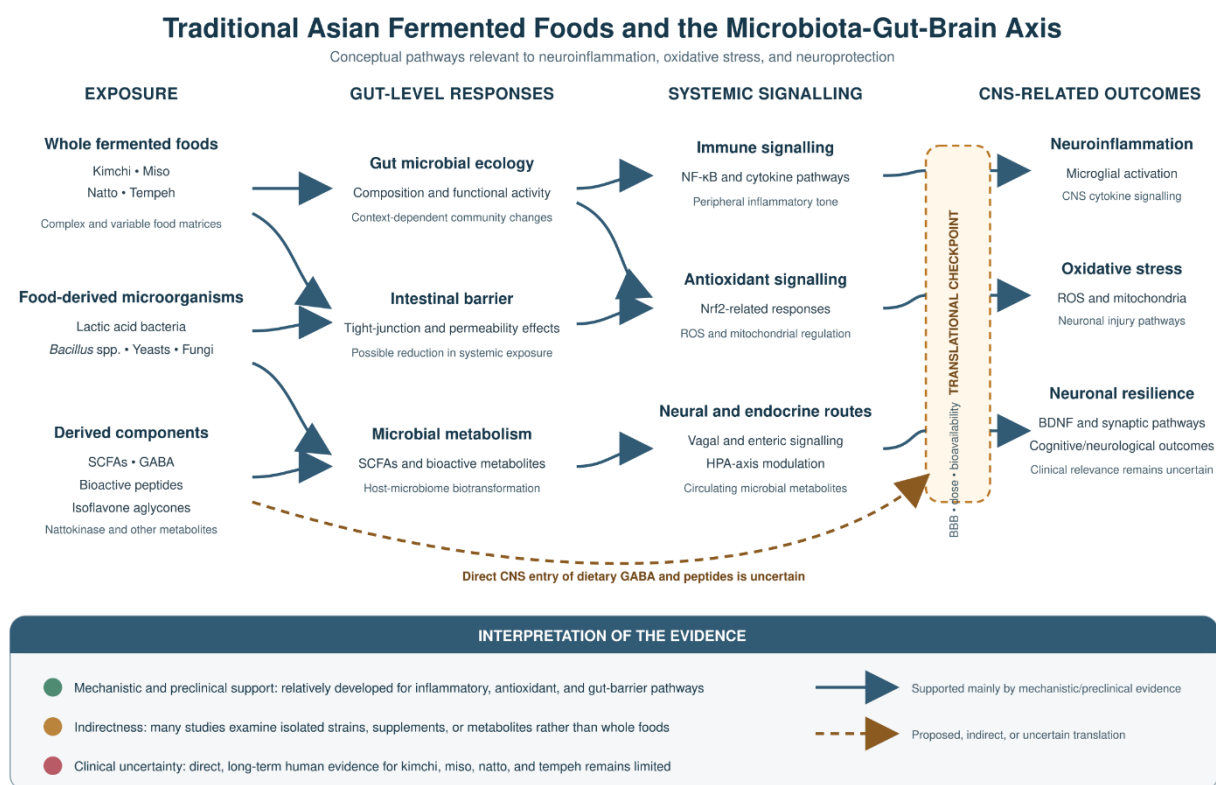
Prospective cohort studies also examined associations between fermented-soy or natto intake and disabling dementia, with possible differences according to sex [14,15]. Controlled human evidence concerning whole-natto consumption and neurological outcomes remained limited (Table 2).

#### Tempeh

Evidence concerning tempeh was predominantly mechanistic or preclinical. Fermentation by *Rhizopus* species can improve protein digestibility and increase the availability of peptides and isoflavone aglycones. Studies of fermented-legume components have reported antioxidant, intestinal-barrier, and inflammatory effects [17,18]. However, direct human studies examining whole-tempeh consumption and neurological outcomes were not identified in the current evidence set (Table 2).

**Table 2** Characteristics and directness of evidence concerning selected traditional Asian fermented foods

| Food   | Main components examined                                                                       | Evidence source                                                    | Types of available evidence                                   | Findings or proposed pathways                                  | Main limitations                                                           | Citations |
|--------|------------------------------------------------------------------------------------------------|--------------------------------------------------------------------|---------------------------------------------------------------|----------------------------------------------------------------|----------------------------------------------------------------------------|-----------|
| Kimchi | Lactic acid bacteria, SCFA-related activity, and GABA-producing microorganisms                 | Mainly food-derived microorganisms                                 | Mechanistic and review-level evidence; limited human evidence | Gut microbial, inflammatory, and oxidative pathways            | Predominance of isolated-strain evidence and substantial product variation | [5,10,11] |
| Miso   | Isoflavone aglycones and fermented-soy components                                              | Whole food within broader dietary patterns and isolated components | Mechanistic and observational evidence                        | Antioxidant and anti-inflammatory plausibility                 | Independent effect of miso cannot be separated from the overall diet       | [4,12]    |
| Natto  | Nattokinase, vitamin K <sub>2</sub> , poly- $\gamma$ -glutamic acid, peptides, and isoflavones | Whole-food animal exposure, observational intake, and components   | Animal and prospective observational evidence                 | BDNF-related signaling and associations with dementia outcomes | Limited controlled human evidence and inability to establish causality     | [13–15]   |
| Tempeh | Bioactive peptides and isoflavone aglycones                                                    | Mainly fermented-legume or component-based evidence                | Mechanistic and preclinical evidence                          | Antioxidant, intestinal-barrier, and inflammatory pathways     | Limited direct neurological and human evidence                             | [17,18]   |



**Figure 1** Conceptual framework linking traditional Asian fermented foods with microbiota–gut–brain-axis pathways

**Abbreviations:** BBB, blood–brain barrier; BDNF, brain-derived neurotrophic factor; CNS, central nervous system; GABA,  $\gamma$ -aminobutyric acid; HPA, hypothalamic–pituitary–adrenal; MGBA, microbiota–gut–brain axis; NF- $\kappa$ B, nuclear factor  $\kappa$ B; Nrf2, nuclear factor erythroid 2-related factor 2; ROS, reactive oxygen species; SCFAs, short-chain fatty acids.

Solid blue arrows indicate pathways supported mainly by mechanistic or preclinical evidence. Dashed brown arrows indicate proposed, indirect, or uncertain translation. The translational checkpoint represents limitations related to experimental dose, bioavailability, metabolism, and blood–brain barrier permeability. Green indicates relatively developed mechanistic or preclinical support; amber indicates indirect evidence derived mainly from isolated strains, supplements, or metabolites; and red indicates limited direct human evidence and continuing clinical uncertainty.

### Preclinical and human evidence

Preclinical studies provided most of the evidence concerning possible neurological mechanisms. Animal and mechanistic studies reported changes in cognitive performance, oxidative-stress markers, intestinal-barrier function, inflammatory signaling, and neurotrophic pathways [4,10,13]. These studies commonly used selected strains, purified compounds, or concentrated exposures, limiting their direct relevance to customary food consumption.

Human evidence was less extensive. Japanese cohort studies examined associations between soy-containing dietary patterns, fermented-soy intake, and dementia-related outcomes [12,14,15]. Controlled

studies investigated fermented-soy preparations or isolated probiotic strains, but these interventions were not always equivalent to traditional whole foods [16,19,20]. The study of *Lactobacillus plantarum* 299v was conducted using an isolated strain in a non-Asian setting and was therefore classified as indirect contextual evidence [20]. Overall, direct controlled evidence concerning whole kimchi, miso, natto, or tempeh and clinically meaningful neurological outcomes remained limited [10,21].

### Discussion

This review examined whether traditional Asian fermented foods may influence neuroinflammation, oxidative stress, and MGBA-related pathways. The synthesis found biological support for several relevant mechanisms, but the evidence was dominated by reviews, experimental models, and studies of isolated microorganisms or compounds. Direct human evidence concerning whole fermented foods was limited. The current literature therefore supports biological plausibility more strongly than clinical neuroprotection.

The distinction between whole-food and component-based evidence is central to this interpretation. Fermented foods are complex products

whose microbial and chemical composition depends on ingredients, starter cultures, fermentation conditions, processing, and storage [5,22]. Although isolated strains and purified compounds can clarify specific pathways, their effects cannot be directly attributed to consumption of the complete food. Proposed interactions among microorganisms, metabolites, and the food matrix remain plausible but have not been adequately tested in controlled human studies.

Evidence also differed among foods. Kimchi research focused mainly on food-derived microorganisms, while miso was generally examined as part of wider soy-containing dietary patterns [4,10–12]. Natto had animal and observational evidence relevant to cognitive and dementia-related outcomes, but causal effects were not established [13–15]. Evidence concerning tempeh remained largely mechanistic or component based [17,18]. These differences, together with the absence of formal comparative certainty assessment, do not support ranking one food as more neuroprotective than another.

Translation from experimental findings to human diets remains challenging. Preclinical studies often use selected strains, purified compounds, and doses that may exceed customary dietary exposure. Digestion, microbial transformation, intestinal absorption, hepatic metabolism, and blood–brain barrier permeability may reduce the biological availability of GABA, peptides, and other proposed neuroactive compounds. Consequently, activation of inflammatory, antioxidant, or neurotrophic pathways in experimental models may not result in clinically meaningful neurological benefits in humans.

Observational studies provide information on habitual dietary exposure but cannot establish causality. Associations between fermented-soy intake and dementia-related outcomes may be affected by dietary measurement error, residual confounding, reverse causation, and correlated health behaviors [12,14,15]. Controlled interventions reduce some of these limitations, but the available studies were heterogeneous and did not consistently examine whole traditional foods or long-term neurological outcomes [10,21].

Individual factors may also influence responses to fermented foods. Age, sex, habitual diet, medication use, baseline microbiota, metabolic phenotype, and equol-producing capacity may modify the effects of fermented-soy products [23]. These factors may partly explain differences across human studies. Microbiome-informed nutrition may help identify responsive subgroups, but it remains a research direction rather than an established clinical approach.

Safety must be evaluated according to the food, preparation, dose, and population. Variation in sodium content, microbial quality, fermentation products, and potential food–medication interactions may affect the

balance of benefits and risks. Future studies should therefore report product composition, quality-control procedures, adherence, and adverse events. Specific safety claims require direct clinical or authoritative pharmacological evidence.

This review has limitations. Its narrative design permitted the integration of diverse evidence but introduced potential selection and interpretive bias. Heterogeneity in food products, exposures, populations, and outcomes prevented quantitative synthesis. The absence of formal risk-of-bias assessment limited the certainty of comparisons across study designs. Restriction to English-language publications may also have excluded relevant regional evidence. The findings should therefore be interpreted as a structured critical synthesis rather than a comprehensive estimate of clinical effectiveness. A record-level audit trail was not retained during the original literature search; therefore, complete retrieval, deduplication, and exclusion counts could not be reconstructed reliably. This limits the reproducibility and comprehensiveness of the review.

Future research should evaluate standardized and chemically characterized whole-food interventions using adequate sample sizes, appropriate comparators, longer follow-up, and clinically meaningful cognitive or neurological outcomes [10,21]. Studies should distinguish whole-food effects from those of isolated strains, metabolites, and supplements. Multi-omics approaches may clarify interactions among food composition, gut microbial responses, and host metabolism, but they should complement rigorous clinical evaluation [26,27].

## Conclusion

Traditional Asian fermented foods may influence neuroinflammatory, oxidative, and MGBA-related pathways through their microorganisms and fermentation-derived compounds. However, current evidence is largely mechanistic, preclinical, or component-based, while direct human evidence concerning whole-food consumption remains limited. These foods should therefore be regarded as promising subjects for further investigation rather than established interventions for preventing or treating neurodegenerative disease.

## Declaration of the use of artificial intelligence

During the preparation of this work, the authors used ChatGPT (OpenAI, USA) and Gemini (Google, USA) in order to improve the English language and readability. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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