Asian Ferments: Microbiota, Metabolic, Lipid, Inflammatory, Barrier, And Bile-Acid Pathways

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Abstract : Aim — This review synthesizes open-access evidence on Asian fermented foods, particularly Indonesian varieties, containing lactic acid bacteria (LAB) and Bacillus probiotics. Six health domains were considered: gut microbiota modulation, glucose metabolism, lipid regulation, bile-acid/energy signalling, anti-inflammatory activity, and gut-barrier integrity. Mechanistic pathways were mapped, notably SCFA→FFAR2/3→AMPK and BSH-mediated bile-acid remodelling→FXR/TGR5→GLP-1/energy, linking microbial activity to clinical outcomes. Methods — Systematic searches (2013–2025) were performed in PMC, ScienceDirect, Semantic Scholar, BioMed Central, PLOS, and MDPI for studies on tempeh, natto, kimchi, miso/doenjang, bekasam/peda, tempoyak, and isolated strains. Results — Evidence converged across domains: (1) gut microbiota shifted toward LAB/Bifidobacterium and SCFA producers; (2) modest glycaemic improvements (HbA1c, FBG, HOMA-IR) via SCFA–AMPK pathways; (3) lipid reductions (TC, LDL-C, TG) associated with BSH-driven bile-acid remodelling and FXR/TGR5–GLP-1 activity; (4) lower inflammation (CRP, cytokines) through NF-κB/MAPK suppression; (5) strengthened gut-barrier integrity (increased ZO-1/occludin/claudins). High-value microbe–food anchors included *Lactiplantibacillus plantarum* (kimchi/tempeh), *Bacillus subtilis* (natto), and *Pediococcus* spp. (bekasam/peda). Conclusion — Asian fermented foods provide culturally congruent dietary platforms that modestly improve glycaemia, lipid profiles, inflammation, and barrier health. Future priorities are head-to-head randomized trials of Southeast Asian ferments, strain-resolved reporting, matrix-aware dosing/duration, and shared biomarker endpoints.

Keywords: Asian fermented foods, Gut microbiota, SCFA–AMPK, BSH–FXR/TGR5 signalling

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1. Introduction

Metabolic risk is rising across Asia, making food-first strategies both practical and urgent. Yet the evidence is fragmented—scattered across strains, food matrices, and endpoints—so this review offers an integrative, Asia-focused synthesis across six domains: gut microbiota modulation, glucose metabolism, lipid profile regulation, bile-acid/energy signalling, anti-inflammatory effects, and gut-barrier integrity. Two mechanistic "highways" are tracked throughout: (1) SCFA→FFAR2/FFAR3→AMPK and (2) BSH-mediated bile-acid remodelling→FXR/TGR5→GLP-1/energy, both linking to changes in glycaemia, lipids, inflammation, and barrier function [3,13,84].

Everyday Asian ferments—tempeh, kimchi, natto, and fish–rice foods—map onto modern probiotic mechanisms and modest clinical gains. Populations across Asia already consume fermented vegetables, soy-based ferments, and soybean natto. Across recent reviews and trials, lactic acid bacteria (LAB) from these foods consistently shifted the gut ecosystem toward SCFA-producing taxa, alongside improvements in glycaemia (HbA1c, FBG, HOMA-IR), lipid profiles (TC, LDL-C, TG), inflammatory tone (CRP, cytokines), and gut-barrier markers [11,86,59,9,97,66,75,78,29]. Two routes help explain these signals: SCFAs engaging FFAR2/FFAR3 to activate AMPK and support insulin signalling, and LAB bile-salt hydrolase reshaping bile acids to stimulate FXR/TGR5, GLP-1 release, and energy balance [21].

However, the literature is still incomplete. Variability in strains, matrices, doses, and endpoints makes it difficult to translate mechanisms into practice. To address this, the review emphasizes Asian—particularly Indonesian—fermented foods, asking which microbe–food pairs line up with both mechanism and endpoint. Six domains structure the synthesis: microbiota, glucose, lipids, bile-acid/energy, inflammation, and barrier integrity. Mechanistic "highways" (SCFA→FFAR2/3→AMPK; BSH→FXR/TGR5→GLP-1/energy) are directly mapped to outcomes (HbA1c/FBG/HOMA-IR; TC/LDL-C/TG; CRP/cytokines; permeability) [86,59,9,97,75,29,21].

Real-world anchors are brought in early: *Lactiplantibacillus plantarum* in kimchi/tempeh, *Bacillus subtilis* in natto, and *Pediococcus* spp. in bekasam/peda. These microbes and foods are repeatedly linked to SCFA production, BSH activity, and barrier support [3,13,84,44,34,61,40,2,46,82,88]. Open-access evidence (2013–2025) was gathered from PMC, ScienceDirect, Semantic Scholar, BioMed Central, PLOS, and MDPI. Studies were screened with an Asia/Indonesia focus (tempeh, natto, kimchi, miso/doenjang, bekasam/peda, tempoyak). Extracted variables included strain/food, dose/duration, mechanistic readouts (SCFAs; bile-acid panels; GLP-1/PYY; NF-κB/MAPK; tight-junction markers), and outcomes in six domains. Sources of heterogeneity (strain identity, matrix, dose, duration, baseline diet) were noted [86,59,9,97,21,75,29].

The aims were fourfold: (1) synthesise evidence on Asian ferments and LAB across six domains; (2) map mechanisms to clinical endpoints via two primary pathways (SCFA→FFAR2/3→AMPK; BSH→FXR/TGR5→GLP-1/energy); (3) identify high-

leverage microbe–food pairs for use in Asia, especially Indonesia (*L. plantarum* in kimchi/tempeh; *B. subtilis* in natto; *Pediococcus* in bekasam); and (4) surface research gaps and practical priorities—strain reporting, matrix-aware dosing, shared biomarker cores, and head-to-head trials—to guide culturally congruent dietary interventions.

2. Systematic Review Method

2.1 Populating open-access articles

The review searched open-access databases—PMC, ScienceDirect, Semantic Scholar, BioMed Central, PLOS, and MDPI—using harmonized queries that targeted six domains: (1) gut microbiota modulation, (2) glucose metabolism, (3) lipid profile regulation, (4) bile-acid/energy signalling, (5) anti-inflammatory effects, and (6) gut-barrier integrity. When available, open-access and date filters were applied, and titles, abstracts, and keywords were searched; MeSH terms were used in PMC. All retrieved records were exported with metadata, abstracts, and identifiers to a single reference manager for screening.

2.2 Refining, de-duplicating, and categorizing

The corpus was narrowed to studies published from 2013 onward that involved Indonesian or Asian fermented foods—*tempeh, natto, kimchi, miso/doenjang, bekasam/peda, tempoyak*—or strains isolated from these foods. Duplicates were removed by matching DOI/PMID, titles, and author lists. Title/abstract screening was performed, followed by full-text assessment against predefined criteria: open-access availability, probiotic or fermented-food exposure, and at least one of the six domain outcomes. Editorials, non-primary reports without mechanistic relevance, and inaccessible full texts were excluded. Each included study was categorized into one or more domains to reflect overlapping mechanism.

2.3 Analysing contexts to extract mechanisms and roles of probiotics.

From each article, standardized variables were extracted, i.e. study type (human RCT/observational, animal, in vitro), population/model, microbes/strains and their food sources, and intervention details (dose or CFU, duration, matrix). Mechanistic readouts were captured (SCFAs and FFAR2/FFAR3; bile-acid pools and FXR/TGR5; BSH activity; GLP-1/PYY; NF-κB/MAPK; tight-junction proteins) and aligned to outcomes (HbA1c, fasting glucose, HOMA-IR; lipid fractions; inflammatory markers; permeability measures). Study quality was appraised with RoB 2 (RCTs) and SYRCLE (animal studies), and potential heterogeneity drivers were noted (dose, duration, matrix, baseline metabolic status, background diet).

2.4 Integrating results and develop the post-review discussion.

Finally, findings were synthesized within and across the six domains. The synthesis linked mechanisms to endpoints (SCFA→FFAR2/3→AMPK; BSH-mediated bile-acid remodelling→ FXR/TGR5→GLP-1/energy) and summarized results in evidence tables and logic diagrams that traced the path from probiotic intake and survival to clinical signals. Consistencies and discrepancies were interpreted by strain, matrix, dose, and duration, and evidence gaps were identified to guide future, region-specific trials.

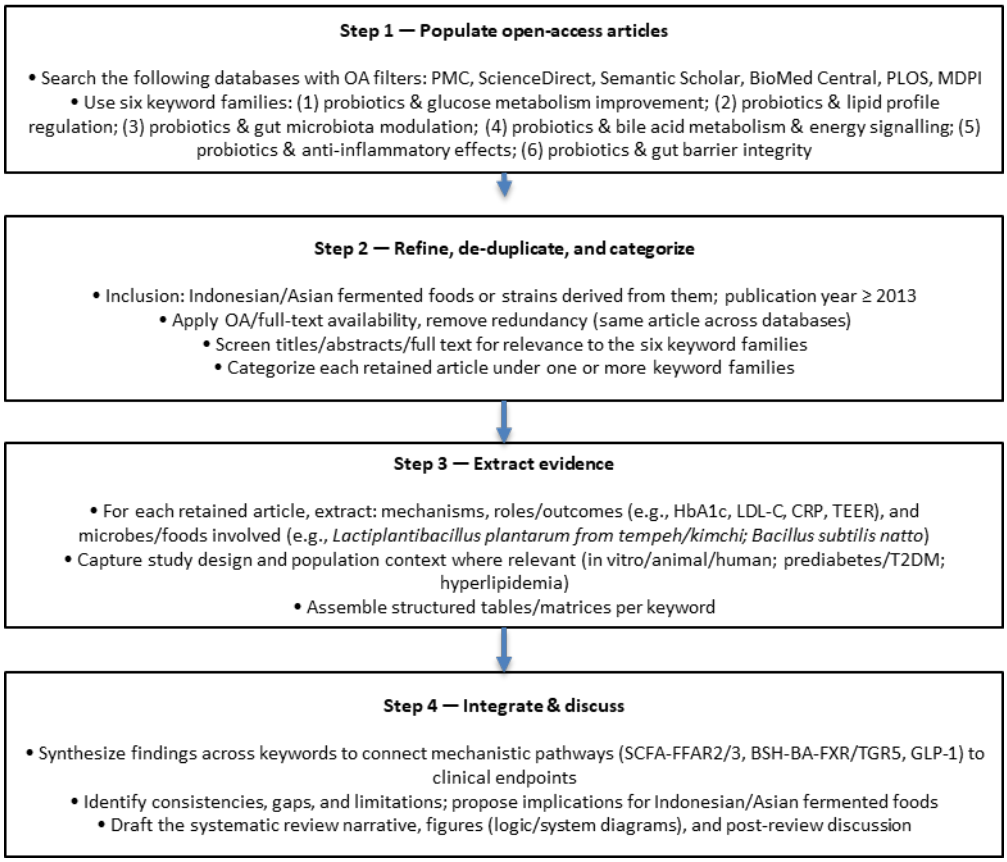

Figure1. Systematic review approach

3. Results

3.1 Database Search and Articles Curation

Table 1 shows the size and shape of the open-access landscape we screened since 2013. Anti-inflammatory queries returned the largest pool (e.g., ScienceDirect ≈9,526; PMC ≈2,829), followed by broad gut-microbiota modulation (ScienceDirect ≈4,891; PMC ≈1,789). Glucose and lipid endpoints each yielded several thousand hits on ScienceDirect (≈3,220 and ≈3,721) but far fewer on PMC and Semantic Scholar, while bile-acid/energy signalling was smaller across all platforms—consistent with a more mechanistic niche. Counts on PLOS were near-zero because publisher-level OA is high but search filtering for our multi-term strings was limited on that interface; MDPI contributed modestly in microbiota and barrier domains. These numbers represent the **pre-screening universe** prior to de-duplication and eligibility checks, and they justified a domain-structured, narrative synthesis: large anti-inflammatory and microbiota bodies supported broader overviews, whereas bile-acid signalling required deeper, mechanism-led reading. They also flagged platform bias (OA filters and indexing differences), which we mitigated by running harmonised strings across six sources and exporting everything for centralised de-duplication.

Table1. Number of open access articles related to probiotics improving human health

Keyword	Number of open access articles since 2013					
	PMC	**Science Direct**	**Semantic Scholar**	**Biomed Central**	**PLOS**	**MDPI**
probiotics and glucose metabolism improvement	699	3220	112	850	0	109
probiotics and lipid profile regulation	731	3721	80	1467	0	21
probiotics and gut microbiota modulation	1789	4891	1300	1578	0	754
probiotics and bile acid metabolism & energy signalling	154	1843	292	486	0	0
probiotics and Anti-inflammatory effects	2829	9526	1260	2515	0	348
probiotics and gut barrier integrity	1008	4407	404	1451	0	156

Table 2 narrows the view to Indonesian/Asian ferments and strains (2013–present) after de-duplication and full-text eligibility. Lipid profile regulation (n=20) emerged as the most populated category—driven by kimchi/soy/natto trials and animal work consistent with BSH-linked bile-acid remodelling—closely followed by glucose metabolism (n=16) and gut-microbiota modulation (n=15), where fermented vegetables and soy foods repeatedly shifted communities toward SCFA producers. Anti-inflammatory effects (n=12) and gut-barrier integrity (n=10) provided coherent mechanistic support (NF-κB dampening; tight-

junction gains), while bile-acid/energy signalling (n=10) anchored the link between BSH activity and FXR/TGR5 pathways. Because many papers span multiple domains (e.g., *kimchi* affecting lipids, inflammation, and barrier), each study was assigned to the most informative primary domain for counting, with cross-domain mechanisms captured in the narrative. This curated distribution validated the review's emphasis on three practical microbe–food anchors—*Lactiplantibacillus plantarum (kimchi/tempeh), Bacillus subtilis (natto)*, and *Pediococcus (bekasam)*—and highlighted where evidence is thinner (bile-acid signalling, barrier) and thus prioritised for future head-to-head trials.

Table 2. Curated number open access articles related to Indonesian and Asian probiotics improving human health

Domain	**Count**	**References (numeric style)**
Glucose metabolism	16	[84], [3], [19], [7], [8], [20], [76], [10], [60], [85], [70], [84], [102], [9], [86], [59]
Lipid profile	20	[84], [15], [27], [13], [14], [18], [43], [17], [37], [42], [1], [44], [16], [61], [84], [104], [9], [66], [82], [97]
Gut microbiota modulation	15	[95], [63], [2], [46], [100], [34], [54], [21], [24], [84], [11], [80], [82], [90], [103]
Bile acids & energy signalling	10	[21], [13], [39], [37], [96], [6], [24], [21], [102], [103]
Anti-inflammatory effects	12	[84], [49], [40], [32], [101], [79], [81], [33], [92], [69], [78], [75]
Gut barrier integrity	10	[50], [49], [95], [38], [74], [45], [87], [50], [69], [29]

3.2 Probiotics and Gut Microbiota Modulation

Probiotic-rich Asian ferments rebalance the gut microbiota toward SCFA-producing taxa, thereby reducing endotoxaemia and contributing to modest improvements in glycaemia, lipid metabolism, inflammation, and barrier integrity through SCFA and BSH-dependent pathways (Table 3). Supplementation with probiotics typically increases *Lactobacillus* and *Bifidobacterium*, enriches short-chain-fatty-acid producers, and decreases potentially pathogenic bacteria. This compositional shift lowers endotoxin exposure and stabilises metabolic regulation [11].

Pooled analyses in prediabetes and type 2 diabetes populations show small but clinically relevant improvements in HbA1c, fasting glucose, and insulin resistance, particularly when interventions involve multiple strains and sufficient duration [86,59].For lipid outcomes, evidence follows a similar trend. Though not dramatic, meta-analyses consistently report modest reductions in total cholesterol, LDL-C, and triglycerides after probiotic or fermented-food intake, with strain, dose, and food matrix determining the magnitude of benefit [9,97,66]. A mechanistic explanation lies in bile-salt hydrolase (BSH) activity: probiotics unhook bile acids, reducing cholesterol absorption and reshaping the bile-acid pool, which in turn activates FXR and TGR5 receptors influencing GLP-1 release, energy balance, and hepatic lipid handling [21].

Anti-inflammatory effects are also consistent. Reviews and clinical trials note reductions in CRP and pro-inflammatory cytokines, accompanied by downregulation of NF-κB and MAPK signalling [75,78]. Furthermore, meta-analyses indicate that probiotic

and synbiotic supplementation enhances gut-barrier function, reducing translocation of lipopolysaccharides (LPS) into circulation—an effect that may underpin improvements observed in both glycaemic control and lipid metabolism [29].

Table 3. Resume of probiotics influence to health outcomes.

Mechanism	Description	References
Gut microbiota modulation	Probiotics increase beneficial bacteria (*Lactobacillus*, *Bifidobacterium*, SCFA producers) and reduce pathobionts, improving gut barrier and reducing endotoxaemia.	[11]
Glucose metabolism improvement	Probiotics may improve HbA1c, fasting glucose, and insulin sensitivity (HOMA-IR), especially in prediabetes and T2DM with multi-strain formulations.	[86], [59]
Lipid profile regulation	Probiotics modestly reduce total cholesterol, LDL-C, and triglycerides, with mixed effects on HDL, via bile acid loss, cholesterol assimilation, and SCFA signalling.	[9], [97], [66]
Bile acid metabolism & energy signalling	Probiotics alter bile acid pools via bile-salt hydrolase, activating FXR/TGR5 pathways, enhancing GLP-1 secretion, energy homeostasis, and cholesterol metabolism.	[21]
Anti-inflammatory effects	Reduction of systemic inflammation through CRP and cytokines (e.g., IL-6, TNF-α) by improving intestinal barrier and downregulating NF-κB pathways.	[75], [78]
Gut barrier integrity	Strengthening of tight junctions and reduced intestinal permeability, lowering LPS translocation and systemic inflammation.	[29]

Probiotic intake—whether through Asian ferments such as kimchi, tempeh, natto, or bekasam, or via multi-strain supplements—activates a coordinated set of pathways that converge on metabolic health. The gut microbiota shifts toward SCFA-producing taxa and away from pathobionts, lowering endotoxaemia. This microbial reset strengthens gut-barrier function (↑ ZO-1, occludin, claudins; ↓ permeability and LPS translocation) and reduces inflammation via NF-κB/MAPK downregulation and lower cytokine levels.

In parallel, bile-salt hydrolase (BSH) activity remodels the bile-acid pool, activating FXR and TGR5, which enhances GLP-1 secretion, energy homeostasis, and cholesterol metabolism. These upstream processes translate into clinical signals: improved glycaemic control (lower HbA1c, fasting glucose, HOMA-IR) through SCFA→FFAR2/FFAR3→AMPK and GLP-1 signalling; and softer lipid profiles (reduced TC, LDL-C, TG) via bile-acid loss, cholesterol assimilation, and SCFA effects.

The interconnected arrows highlight feedback loops: microbiota shifts fortify the barrier; a stronger barrier reduces inflammation; and together these mechanisms reinforce improvements in glycaemia and lipid metabolism.

Asian ferments provide context-specific delivery of active microbes: *Tempeh* (*Rhizopus oligosporus* + *Lactobacillus* spp.) enhances soy breakdown, enriches SCFA-producing taxa, and raises microbial diversity, linked to better insulin sensitivity, lower inflammation, and improved lipid handling [100,34]. *Tempoyak* (fermented durian) contains *Lactiplantibacillus plantarum*, *L. fermentum*, and *Leuconostoc mesenteroides*; these strains show probiotic effects including cholesterol reduction, immune modulation, and anti-inflammatory activity via SCFA and BSH pathways [63,34]. ***Peda*** (fermented salted fish) carries *Lactobacillus* spp., *Pediococcus pentosaceus*, and *Weissella* spp., providing BSH-active LAB that support lower LDL-C, improved gut-barrier integrity, and reduced dysbiosis risk [47,34].

Together, these foods embody culturally relevant sources of SCFA- and BSH-active microbes that modulate microbiota and bile-acid pools, offering modest but meaningful improvements in metabolic health outcomes.

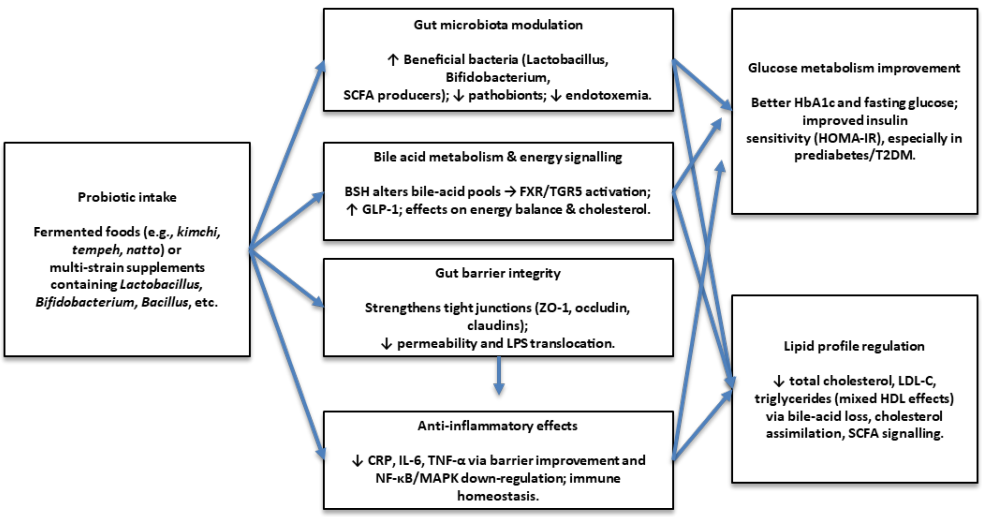

Figure 2. Logical diagram for probiotics influence to health outcomes

Bekasam, a fermented freshwater fish with rice, combines LAB such as *Lactobacillus* and *Leuconostoc* with native yeasts. The rice substrate together with LAB promotes higher gut microbial diversity, improved digestion, and immune modulation. These effects align with SCFA-mediated strengthening of the gut barrier and modulation of cytokine profiles, reducing inflammatory tone and supporting metabolic balance [34].

Other Asian staples further illustrate the convergence of LAB- and BSH-active microbes on health outcomes. **Kimchi** delivers *Lactobacillus kimchii* and related LAB strains that enrich SCFA producers and are linked to improvements in glycaemic control and immune balance. **Natto**, rich in *Bacillus subtilis*, contributes both probiotic activity and bioactive peptides, with evidence of lipid-lowering and cardioprotective effects. **Miso/doenjang**, fermented soybean pastes featuring *Leuconostoc* spp. and LAB consortia, provide antioxidant and immune-supportive activities while modulating bile acids through BSH pathways. Together, these foods illustrate how diverse Asian ferments converge on

similar probiotic mechanisms—SCFA production, bile-acid remodelling, gut-barrier strengthening, and inflammation reduction—ultimately complementing Indonesian ferments in supporting glucose regulation, immune homeostasis, and cardiovascular protection [95].

Table4. Gut microbiota modulation and health outcomes

Food	**Dominant Microbes**	**Gut Microbiota Modulation**	**Health Outcomes**	**References**
Tempeh (fermented soybean)	*Rhizopus oligosporus*, *Lactobacillus* spp.	Increases SCFA-producing bacteria, improves fibre digestibility, enhances microbial diversity.	Improved insulin sensitivity, reduced inflammation, better lipid metabolism.	[100], [34]
Tempoyak (fermented durian)	*Lactobacillus plantarum*, *L. fermentum*, *Leuconostoc mesenteroides*	LAB colonisation increases lactic acid and SCFA, suppresses pathogens, modulates bile-acid metabolism.	Cholesterol reduction, immune modulation, anti-inflammatory effects.	[63], [34]
Peda (fermented salted fish)	*Lactobacillus* spp., *Pediococcus pentosaceus*, *Weissella* spp.	Provides BSH-active LAB, modulates gut microbial composition.	Lower LDL-C, improved gut barrier, reduced dysbiosis risk.	[47], [34]
Bekasam (fermented freshwater fish with rice)	*Lactobacillus* spp., *Leuconostoc* spp., yeasts	Combination of LAB + rice substrate acts as synbiotic, enriching beneficial microbes.	Supports gut diversity, improves digestion, strengthens immunity.	[34]
Other Asian ferments (Kimchi, Natto, Miso)	*Lactobacillus kimchii*, *Bacillus subtilis*, *Leuconostoc* spp.	Rich in LAB and Bacillus, producing SCFAs and bioactive peptides.	Enhanced glucose regulation, immune balance, cardiovascular protection.	[95]

Probiotic Influence in Regulation of Glucose and Lipid Metabolism

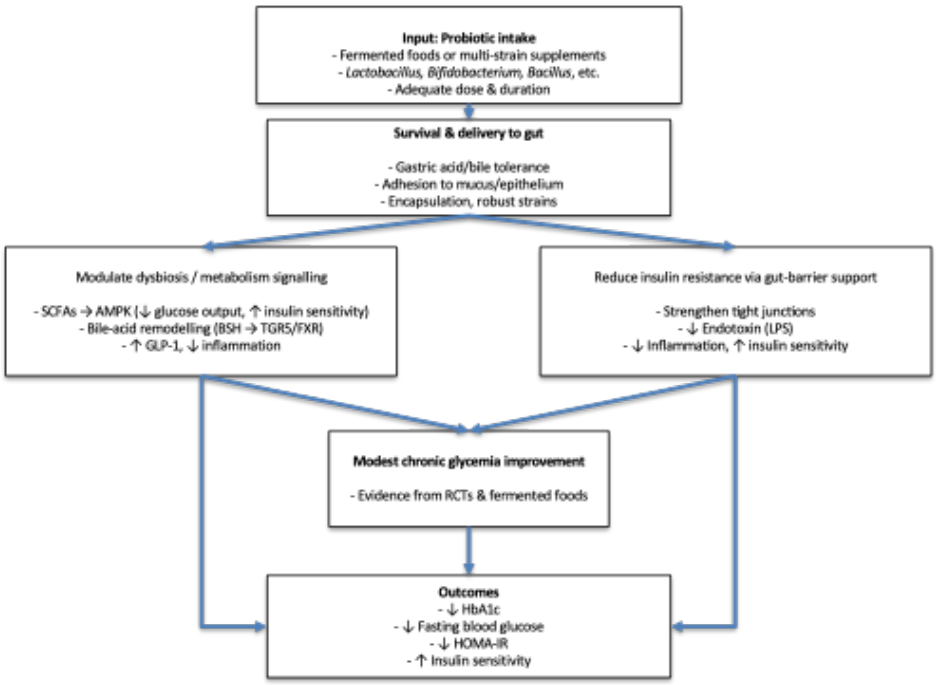

Figure 3. Logical diagram for probiotics role in glucose metabolism improvement

Figure 3 walks through how probiotics can nudge glucose control in everyday life. It starts with what goes in—fermented foods or multi-strain supplements at an adequate dose and duration—then reminds us that survival and delivery matter: strains need to tolerate stomach acid and bile, stick to the mucus/epithelium, and sometimes benefit from encapsulation. From there, two main routes do the work. On the left, probiotics help remodel dysbiosis and metabolism signalling: SCFAs engage FFAR2/FFAR3→AMPK to lower hepatic glucose output and raise insulin sensitivity, while BSH-driven bile-acid remodelling nudges TGR5/FXR, boosting GLP-1 and easing low-grade inflammation. On the right, they steady the gut barrier, tightening junctions, cutting LPS spill-over, and further improving insulin action. Those upstream nudges converge on the centre box—modest, chronic improvements in glycaemia—which is exactly the pattern seen across RCTs and fermented-food studies. The downstream outcomes are the familiar ones: slightly lower HbA1c, fasting glucose, and HOMA-IR, with better insulin sensitivity. The take-home is simple: when robust strains reach the intestine and are given enough time, these two pathways work together to deliver small but clinically meaningful gains in glucose control.

Multi-strain probiotics and Asian ferments (kimchi, tempeh) modestly improve HbA1c/FBG/HOMA-IR by restoring dysbiosis and barrier function while engaging SCFA→FFAR2/3→AMPK, GLP-1, and BSH-mediated FXR/TGR5 signalling (Table 5). Across randomized trials, multi-strain probiotics nudged glucose control in the right

direction. In medication-naïve adults with T2DM, a 12–13-week blend of *Bifidobacterium* and *Lactobacillus* species improved insulin resistance (HOMA-IR), consistent with a barrier-support/endotoxin-lowering route [76]. A similar 12-week, India-based formulation lowered HbA1c versus placebo (with nonsignificant trends for FBG and HOMA-IR when used alongside metformin), pointing to dysbiosis repair and SCFA/GLP-1–linked pathways [60]. In Malaysia, a multi-strain preparation modestly improved HbA1c (per-protocol) and reduced fasting insulin over 12 weeks in T2DM, reinforcing the "small but clinically relevant" pattern seen across studies [20].

Dietary ferments tell a consistent story, i.e. an 8-week fermented-kimchi diet improved insulin resistance/sensitivity compared with fresh kimchi in Korean adults with prediabetes [3]. Culture work describing typical kimchi microbiota—*Leuconostoc*, *Lactobacillus/Lactiplantibacillus*, *Weissella*—helps explain why fermented vegetables can drive these outcomes via SCFA production and immune-barrier signalling [54]. Beyond vegetables, tempeh-derived (fermented soy) capsules taken for three months reduced HbA1c in people with T2DM in an open-label setting, aligning Indonesian/Taiwanese fermented foods with glycaemic benefits [104], a view summarized in a recent review of tempeh's functional microbes and metabolic effects [80].

Mechanistically, probiotics and ferments can raise SCFAs that activate FFAR2/FFAR3 related to AMPK, boost GLP-1 secretion, and reduce low-grade inflammation—each supportive of better insulin action [70, 102]. In parallel, bile-salt hydrolase (BSH) activity remodels the bile-acid pool, tuning FXR/TGR5 signalling that feeds into glucose and energy balance [6]. Taken together, these linked pathways help contextualize the trial results above, i.e. modest improvements in HbA1c, FBG, and HOMA-IR are exactly what we would expect when dysbiosis is corrected, the barrier is steadied, and enteroendocrine and bile-acid circuits are re-engaged [76, 60, 20, 3, 104, 80].

Table 5. The role of probiotics in glucose metabolism improvement

Mechanism	Role (clinical endpoint & context)	Microbes involved	References
Reduce insulin resistance via gut-barrier support & endotoxin lowering	12–13 weeks multi-strain probiotics improved HOMA-IR in medication-naïve T2DM (Saudi cohort).	*B. bifidum* W23, *B. lactis* W52, *L. acidophilus* W37, *L. brevis* W63, *L. casei* W56, *L. salivarius* W24, *Lactococcus lactis* W19/W58	[76]
Modulate dysbiosis/host metabolism (SCFA/GLP-1–linked pathways) show lower chronic glycemia	12 weeks multi-strain formulation (India) reduced HbA1c vs placebo; FBG & HOMA-IR trends not significant (adjunct to metformin).	*L. salivarius* UBLS22, *L. casei* UBLC42, *L. plantarum* UBLP40, *L. acidophilus* UBLA34, *B. breve* UBBr01, *B. coagulans* Unique IS2 (+FOS)	[60]
Modestly improve	12 weeks multi-	Mixed *Lactobacillus* +	[20]

chronic glycemia	strain probiotics (Malaysia) modestly improved HbA1c (per-protocol) and reduced fasting insulin in T2DM.	*Bifidobacterium* spp. (multi-strain MCP)	
Enhance insulin sensitivity with fermented vegetables	8-week fermented kimchi diet improved insulin resistance/sensitivity in prediabetes vs fresh kimchi (Korean adults).	Naturally fermented LAB typical of kimchi: *Leuconostoc*, *Lactobacillus* (*Lactiplantibacillus*), *Weissella* spp.	[3], [54]
Fermented-food–derived intervention (Indonesia/Taiwan) lowering HbA1c	3-month tempeh (fermented soy) capsules reduced HbA1c in T2DM (open-label). Supports relevance of Indonesian fermented foods to glycaemic endpoints.	Tempeh microbes include *Rhizopus* spp. (starter) with co-occurring LAB (e.g., *L. plantarum*)	[104], [80]
Mechanistic backdrop (multi-strain & fermented foods): SCFA–FFAR2/3, BA–TGR5/FXR, BSH activity, GLP-1 increase, low-grade inflammation reduce	Explains observed HbA1c/FBG/HOMA-IR improvements across trials; links common LAB from Asian ferments (kimchi/tempeh) to host energy & glucose signalling.	Typical probiotic genera from Asian ferments: *Lactobacillus/Lactiplantibacillus*, *Bifidobacterium*, *Leuconostoc*, *Weissella*, *Lactococcus*	[102], [6], [70]

Figure 4 lays out a straightforward route from what we eat to friendlier blood lipids. It begins with probiotic intake—typically fermented foods such as *kimchi, tempeh*, or *natto*, or multi-strain capsules containing *Lactiplantibacillus/Lactobacillus, Bifidobacterium, Bacillus, Leuconostoc, or Weissella*—taken long enough for an effect (about 8–12 weeks in many trials). Those microbes then have to survive the journey (acid and bile), reach the intestine, and briefly adhere and act; taking them with food or using microencapsulation helps. Once there, several mechanisms push lipids in the right direction: bile-salt hydrolase (BSH) deconjugates bile acids, which reduces micellar cholesterol solubilisation and increases faecal loss of bile acids and cholesterol; cells from some strains bind or co-precipitate cholesterol directly; and SCFA signalling (acetate/propionate/butyrate) engages AMPK to curb lipogenesis, support fatty-acid oxidation, and ease low-grade inflammation. The downstream picture is the one seen in trials—lower total cholesterol, LDL-C, and triglycerides, a reduced atherogenic index, and more faecal lipid/bile-acid excretion—

illustrating how routine, food-based probiotics can translate microbial chemistry into clinically relevant improvements in lipid profiles.

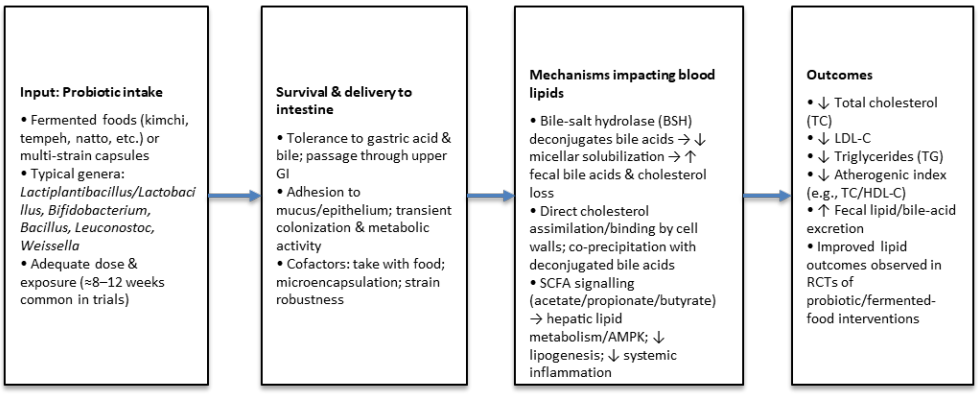

Figure 4. Logical diagram for probiotics role in lipid profile regulation

Asian ferments—kimchi, natto, fermented soy and fish—deliver BSH-active LAB and lipid bioactives that lower TC/LDL-C/TG via bile-acid deconjugation, cholesterol assimilation, and gut–liver signalling, with supportive human trial data (Table 6). A consistent pattern across the kimchi literature is that lactic-acid bacteria with bile-salt hydrolase (BSH) activity make it harder for the gut to solubilize and reabsorb cholesterol, which shows up as lower total cholesterol, LDL-C, and triglycerides in preclinical work and improvements in human trials using fermented vegetables. Kimchi starters dominated by *Lactobacillus* and *Leuconostoc* fit this picture, with clinical signals for lipid improvement alongside the expected rise in faecal bile acids [15, 43, 45]. When researchers focused on a single strain isolated from kimchi—*Lactobacillus plantarum* EM—they also saw lipid lowering that likely combined direct cholesterol binding/assimilation with co-precipitation by deconjugated bile acids, and even higher faecal lipid excretion in animal models [13, 42]. A similar BSH-plus-assimilation mechanism has been demonstrated with Indonesian fermented fish (*bekasam*), where *Pediococcus acidilactici* BK01 reduced serum TC and TG in vivo [61].

Fermenting soy does more than change flavour. Bioactive peptides and isoflavones formed during fermentation, together with modest microbiome shifts, have been linked to lower TC and LDL-C in a human RCT of a fermented soy product [44]. On the grain side, red-yeast rice made with *Monascus* offers some of the strongest human evidence in this space. Pooled and large trials show LDL-C reductions of roughly 15–34%, along with drops in TC and TG and even fewer cardiovascular events in secondary prevention settings [58, 16]. Traditional natto adds another pathway—*Bacillus subtilis*–driven effects on gut and hepatic signalling (e.g., LXR/FXR)—with animal studies reporting lower TC, TG, and LDL-C and higher HDL-C, alongside anti-obesity effects [98].

LAB from many Asian ferments—notably *L. plantarum* strains common to pickles and kimchi—have repeatedly shown cholesterol-lowering potential across human and animal

studies, consistent with their high BSH activity [27, 17]. Indonesia's tempeh gembus offers a local example. Small trials reported reductions in TC and LDL-C (with mixed changes in TG), which aligns with the broader BSH/deconjugation story and the ability of fermented-food microbes to bind or co-precipitate cholesterol in the gut [1, 104].

Table 6. The role of probiotics in lipid profile regulation

Mechanism	**Roles (lipid endpoints)**	**Microbes/foods involved**	**References**
Bile-salt hydrolase (BSH) deconjugates bile acids → less micellar cholesterol absorption; ↑ faecal bile acids	Reduce TC, LDL-C, TG in animals; improvements in human RCTs with kimchi	LAB from kimchi (*Lactobacillus* spp., *Leuconostoc* spp.)	[15], [42], [43]
Direct cholesterol assimilation/binding by bacterial cell wall; co-precipitation with deconjugated bile acids	Reduce TC, LDL-C, TG; reduce atherogenic index; ↑ faecal lipid excretion (animal models)	*Lactobacillus plantarum* EM (kimchi)	[13], [42]
Probiotic LAB from Indonesian fermented fish (bekasam): BSH + assimilation	In vivo reduce TC and TG (mice)	*Pediococcus acidilactici* BK01 (bekasam)	[61]
Fermented soy—bioactive peptides/isoflavones; possible microbiome modulation	Human RCT: reduce TC and LDL-C	Fermented soy product (Korea, *Bacillus subtilis*)	[44]
Red yeast rice (RYR/Xuezhikang)—monacolin K inhibits HMG-CoA reductase	Robust human data: reduce LDL-C (≈15–34%), reduce TC and TG; ↓ CV events	*Monascus*-fermented rice (China)	[58], [16]
Natto-derived effects via gut microbiota & hepatic pathways (e.g., LXR/FXR)	Animal models: ↓ TC, TG, LDL-C; ↑ HDL-C; anti-obesity effects	*Bacillus subtilis natto* (Japan)	[90], [92], [56]
LAB from Asian ferments (general) with high BSH activity	Human/animal: cholesterol-lowering with *L. plantarum* strains	*L. plantarum* (pickles/kimchi)	[27], [17]
Indonesian fermented soy (tempeh gembus)	Small human trials: ↓ TC and LDL-C (TG mixed)	Tempeh gembus (Indonesia)	[1], [104]

Figure 5 follows the bile-acid highway from food to physiology. It starts with probiotic intake—typically fermented foods or multi-strain capsules—whose strains must survive digestion and briefly work in the intestine, often tipping the microbiota toward *Lactobacillus*. That shift brings more bile-salt hydrolase (BSH) activity. In Mechanism A, extra BSH deconjugates bile acids, making micelles poorer at dissolving cholesterol; more

bile acids and cholesterol are then lost in faeces, and the bile-acid pool shifts in ways that talk to FXR/TGR5 receptors. Mechanism B adds a second lever, alongside deconjugation, some strains can bind/assimilate cholesterol, further altering gut–liver signalling and lipid homeostasis. These upstream steps feed into Mechanism C (in vivo), where studies report lower serum total and LDL cholesterol, higher hepatic CYP7A1 (more bile-acid synthesis), and greater faecal bile-acid/cholesterol excretion—the fingerprints of bile-acid remodelling. The diagram also flags a mechanistic caveat: effects on receptor signalling are context- and strain-dependent. Putting it together, the expected outcomes are friendlier lipids (reduced TC, LDL-C, sometimes TG), plus altered FXR/TGR5 activity with downstream changes in GLP-1 and energy balance—a clear, food-first route from routine fermentation microbes to systemic metabolic gains.

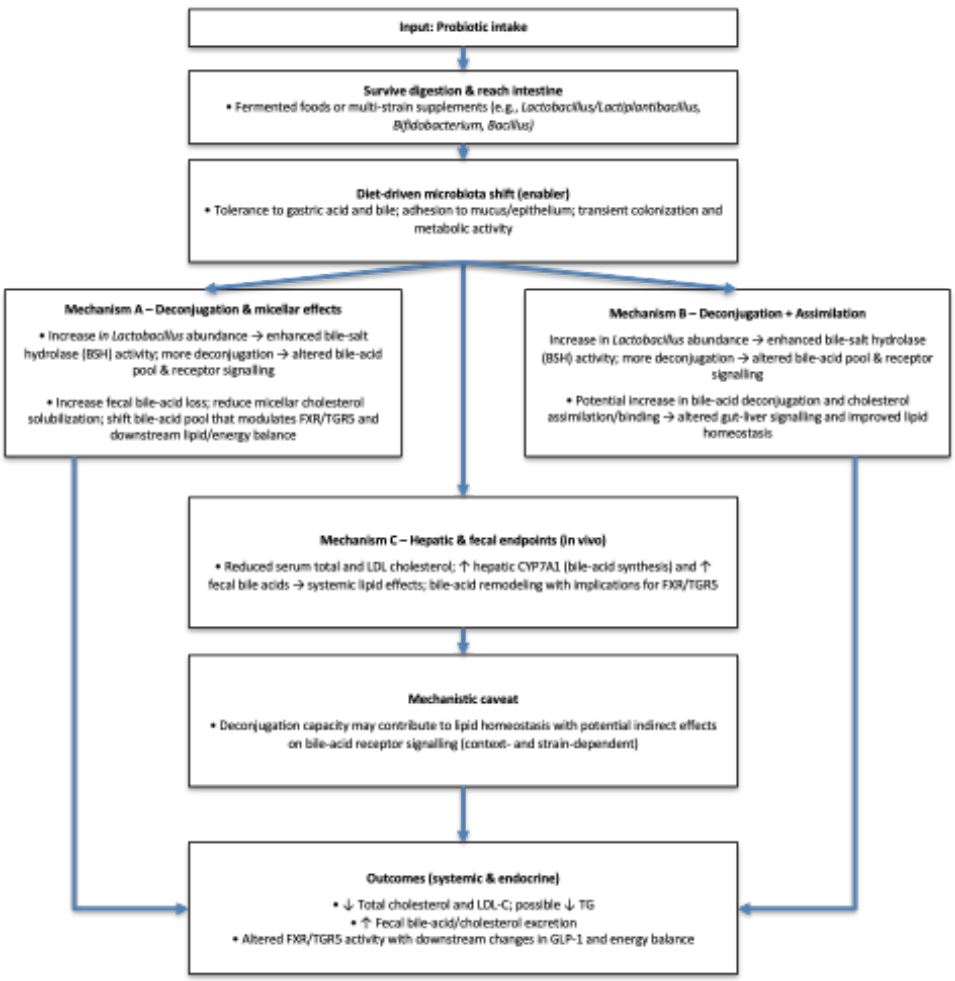

Figure 5. Logical diagram for probiotics role in bile acid metabolism & energy signalling

Many Asian ferments tap the same bile-acid playbook. Across kimchi, tempoyak, bekasam, natto and soy ferments, BSH-active microbes remodel the bile-acid pool—boosting faecal bile-acid loss, reducing micellar cholesterol and engaging FXR/TGR5→GLP-1/energy pathways—to deliver small but meaningful improvements in lipid homeostasis (Table 7). Kimchi LAB—including *Lactobacillus plantarum* strains such as TCI378 and EM—show classic bile-salt hydrolase (BSH) activity that increases faecal bile-acid loss, reduces micellar cholesterol, and shifts the bile-acid pool in ways that can nudge FXR/TGR5 and downstream GLP-1/energy balance [37, 13, 21]. A very similar pattern appears in tempoyak (fermented durian). Its LAB (*L. plantarum*, *L. fermentum*, *L. reuteri*, *L. pentosus*) tolerate bile and likely increase deconjugation and cholesterol assimilation, which together can alter FXR/TGR5 signalling and support lipid homeostasis [2, 46, 21]. In bekasam-related fish ferments, a BSH-positive *Pediococcus pentosaceus* KID7 lowered serum total and LDL cholesterol in vivo while raising hepatic CYP7A1 expression and faecal bile acids—clear hallmarks of bile-acid remodelling with implications for FXR/TGR5 [18, 80, 21].

Natto contributes *Bacillus subtilis* with measurable BSH capacity—e.g., DG101 and other BSH-positive isolates (SOM1–8)—suggesting a route to better lipid balance and indirect tuning of bile-acid–receptor pathways [56, 103, 21]. More broadly, soy ferments such as tempeh enrich BSH-bearing *Lactobacillus*, so diet-driven rises in these taxa are expected to increase deconjugation, reshape the bile-acid pool, and influence FXR/TGR5 with knock-on effects on GLP-1 and energy regulation [39, 21, 5]. Ordinary fermentation microbes leverage BSH-mediated bile-acid chemistry to deliver small but meaningful shifts in lipid and energy signalling.

Table 7. The role of probiotics in **bile acid metabolism & energy signalling**

Mechanism	**Roles (energy & lipid signalling)**	**Microbes involved (from the food)**	**References**
BSH-mediated deconjugation of bile acids by kimchi LAB (e.g., *Lactobacillus plantarum* TCI378; *L. plantarum* EM)	↑ Faecal bile acid loss; ↓ micellar cholesterol; shifts bile-acid pool that can modulate FXR/TGR5 with downstream effects on GLP-1 and energy balance	*L. plantarum* isolated from kimchi	[37], [13], [21]
BSH activity/tolerance in LAB from tempoyak (fermented durian)	Potential ↑ bile-acid deconjugation and cholesterol assimilation; altered FXR/TGR5 signalling and lipid homeostasis	*L. plantarum*, *L. fermentum*, *L. reuteri*, *L. pentosus* from tempoyak	[2], [46], [21]
BSH-positive *Pediococcus* from fermented fish (bekasam/related)	In vivo ↓ serum TC and LDL-C; ↑ hepatic CYP7A1 & faecal bile acids; bile-acid	*Pediococcus pentosaceus* (KID7); LAB from bekasam	[18], [80], [21]

	remodelling with implications for FXR/TGR5		
Bacillus from nattō; some strains exhibit BSH	Deconjugation capacity may contribute to lipid homeostasis; potential indirect effects on BA-receptor signalling	*Bacillus subtilis* (DG101 from nattō); BSH-positive *B. subtilis* strains (SOM1–8)	[56], [103], [21]
Soy ferments (tempeh/soy foods) enrich BSH-bearing LAB	Diet-driven ↑ *Lactobacillus* can enhance BSH activity; more deconjugation, altered BA pool and receptor signalling	LAB from tempeh/soy ferments (e.g., *Lactobacillus* spp.)	[39], [21], [5]

Probiotic Influence in Production of short-chain fatty acids (SCFAs)

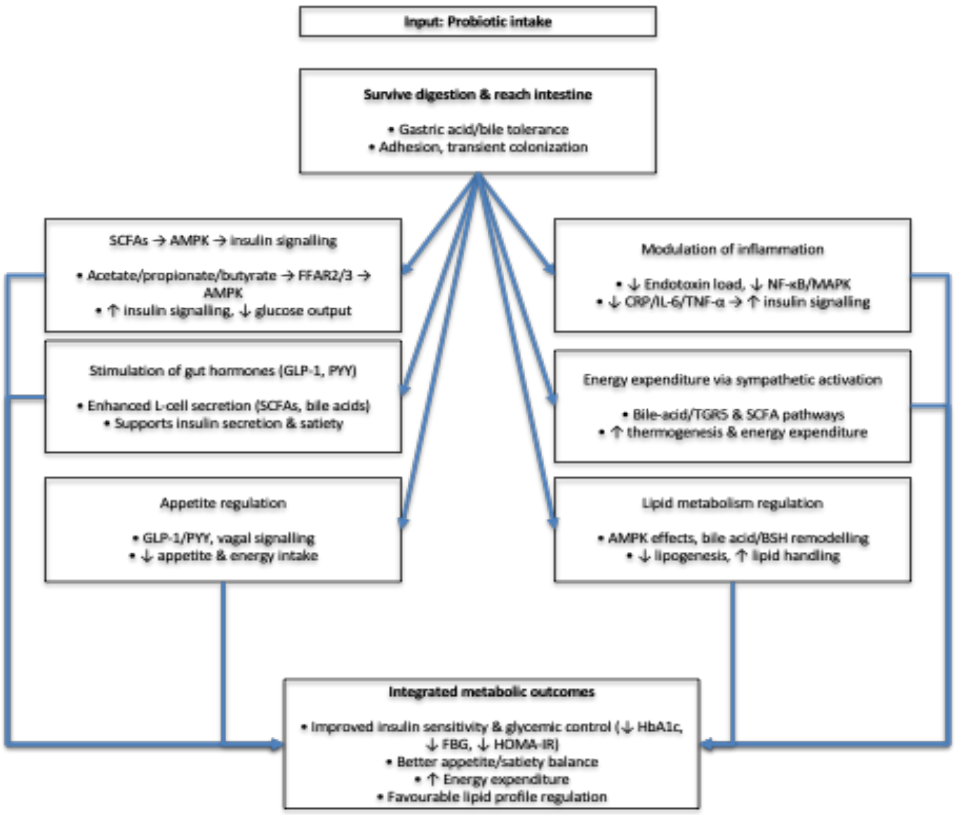

Figure 6. Logical diagram for probiotics role in enhancing insulin sensitivity and energy homeostasis

Figure 6 shows how ordinary probiotic intake can translate into better insulin sensitivity and steadier energy balance through several converging levers. Once strains survive

stomach acid and bile, briefly adhere, and become metabolically active, they generate SCFAs that signal through FFAR2/FFAR3 → AMPK to improve insulin signalling and curb hepatic glucose output. At the same time, they stimulate L-cells to release GLP-1 and PYY, which supports insulin secretion and meal-to-meal satiety, and they calm inflammation by lowering endotoxin load and dialling down NF-κB/MAPK, further easing insulin resistance. A bile-acid/TGR5 and SCFA axis nudges the sympathetic system, raising thermogenesis and energy expenditure, while AMPK activation plus BSH-mediated bile-acid remodelling improves lipid handling and reduces lipogenesis. These pathways also feed into appetite regulation via GLP-1/PYY and vagal signalling. The result, when given adequate dose and duration, is an integrated metabolic lift, modestly lower HbA1c, fasting glucose, and HOMA-IR, a better appetite/satiety balance, higher energy expenditure, and a more favourable lipid profile—all from food-first, culturally familiar probiotic sources.

Probiotic-derived SCFAs switch on AMPK, dampen NF-κB/LPS, and trigger GLP-1/PYY via FFAR2/3—raising energy expenditure and refining appetite and lipid handling—so glucose control becomes smoother and overall cardiometabolic balance improves (Table 8). Probiotic-driven short-chain fatty acids (SCFAs) help the body handle sugar more smoothly. In plain terms, butyrate, acetate, and propionate turn on AMPK, a cellular "energy switch" that improves insulin signalling in muscle and liver—so glucose is used rather than left to linger in the blood [8, 28]. At the same time, SCFAs calm slow-burn inflammation by dialling down NF-κB activity and cutting endotoxin (LPS) spillover, both of which are tied to insulin resistance [19, 10]. They also nudge the gut's L-cells to release GLP-1 and PYY, hormones that support insulin secretion and make meals more satiating [8, 7].

SCFAs reach beyond glucose. They can raise energy expenditure—partly by signalling through GPR41/43 and boosting sympathetic tone and mitochondrial function [28, 19]. On the appetite side, acetate and propionate talk to brain satiety circuits to help curb intake in a natural, meal-to-meal way [7]. Finally, SCFAs tune lipid metabolism, tending to reduce liver triglyceride synthesis, promote fatty-acid oxidation, and support cholesterol efflux—mechanisms that add up to friendlier lipid profiles [10, 19].

Table 8. The role of probiotics in **production of** short-chain fatty acids (SCFAs)

Mechanism	**Description**	**References**
SCFAs activate AMPK and improve insulin signalling	Butyrate, acetate, and propionate enhance insulin sensitivity by activating AMPK in muscle and liver, improving glucose uptake and reducing insulin resistance.	[8], [28]
Modulation of inflammation	SCFAs reduce chronic low-grade inflammation by inhibiting NF-κB and lowering endotoxemia (LPS), which protects against insulin resistance.	[19], [10]
Stimulation of gut hormones (GLP-1, PYY)	SCFAs activate GPR41/43, stimulating GLP-1 and PYY secretion, which enhance β-cell function, improve insulin secretion, and promote satiety.	[8], [7]

Energy expenditure via sympathetic activation	SCFAs regulate energy expenditure by stimulating sympathetic nervous system activity through GPR41 and improving mitochondrial function.	[28], [19]
Appetite regulation	Acetate and propionate influence hypothalamic satiety centres, reducing food intake and helping maintain energy balance.	[7]
Lipid metabolism regulation	SCFAs reduce hepatic triglyceride synthesis, promote fatty acid oxidation, and support cholesterol efflux.	[10], [19]

3.3 Probiotic Influence in Anti-inflammatory Regulation

Figure 7 pulls together how probiotics dial down inflammation through several coordinated routes. After hardy strains from fermented foods or multi-strain capsules survive the upper gut and become metabolically active, they start working on two fronts: directly suppressing NF-κB/MAPK, which lowers iNOS/COX-2 and pro-inflammatory cytokines like IL-6 and TNF-α, and reducing the LPS burden by steadying the barrier and shifting the microbiota—so there is less TLR4-NF-κB signalling to begin with. At the same time, SCFAs (acetate, propionate, butyrate) shape cytokine balance via FFAR2/3 and HDAC inhibition and strengthen tight junctions (ZO-1/occludin/claudins), while some probiotic cues skew immunity toward IL-10-rich, regulatory responses. These levers loop into one another—better barrier means less endotoxin, which makes NF-κB easier to quiet—so the integrated outcome is lower systemic inflammation (e.g., CRP, IL-6, TNF-α), reduced NF-κB/MAPK activity, and a healthier epithelial barrier. The message is pragmatic, given enough dose and time, everyday probiotic foods can nudge the immune–epithelial axis toward a calmer, more resilient state.

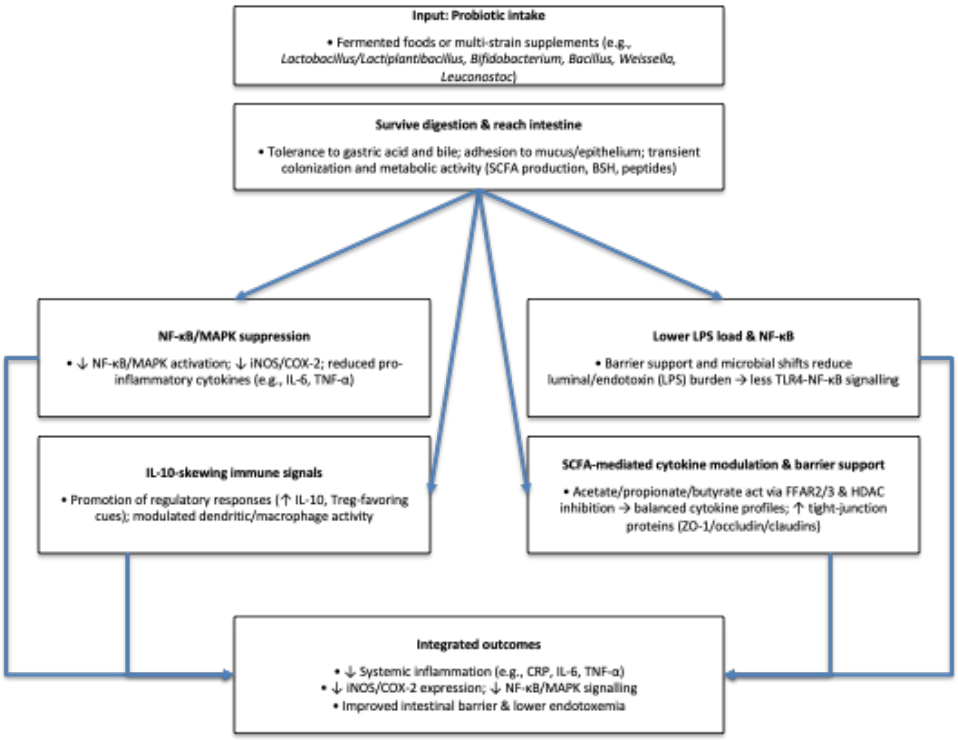

Figure 7. Logical diagram for probiotics role in **anti-inflammatory effects**

From kimchi to tempeh, doenjang, natto and bekasam, strains such as *L. plantarum* (incl. Dad-13), *L. reuteri* EFEL6901 and *B. subtilis* blunt NF-κB/MAPK and TLR4–LPS signals, lift IL-10/IgA and SCFAs, and tighten junctions—defining a coherent, food-first anti-inflammatory signature. Kimchi-derived *Lactiplantibacillus plantarum* strains repeatedly turned down the cell's inflammatory "volume knob" (Table 9). They suppressed NF-κB/MAPK signalling in LPS-challenged macrophages and lowered NO, iNOS/COX-2, and cytokines such as TNF-α and IL-6, with notable antioxidant co-effects [32, 33]. Building on that logic, a purpose-built kimchi starter—*Limosilactobacillus reuteri* EFEL6901—was selected and validated for anti-inflammatory activity in cell and animal tests [79]. Beyond vegetables, a traditional soybean paste (doenjang) eased TNBS-induced colitis by reducing gut-microbial LPS and blocking NF-κB activation, illustrating how fermented foods can act upstream on endotoxin burden as well as on host pathways [49].

In the natto model, *Bacillus subtilis* secreted GroEL, which drove IL-10/IL-12 production in human dendritic cells—an IL-10-skewing signature that helps explain natto's immunoregulatory reputation [Uesugi, 2023]. An indigenous Indonesian probiotic, *Lactobacillus plantarum* Dad-13, protected mice from TNBS colitis by raising SCFAs (acetate/propionate/butyrate), reducing TNF-α/IL-6/IL-1β, increasing IL-10, and supporting barrier integrity—an elegant example of SCFA-mediated cytokine balancing plus epithelial support [69].

Extracts from tempeh (nutrient-enriched fermented soy) reduced NO, IL-1β, and TNF-α in RAW264.7 cells and showed in-vivo anti-inflammatory/analgesic activity, suggesting that both the microbes and the soy bioactive contribute [101]. From fermented fish, bekasam-derived *Lactobacillus* increased intestinal IgA and shifted the cytokine milieu toward a more regulatory tone (increase IL-4/IL-10), supporting mucosal immunity [40]. LAB communities—*Lactiplantibacillus*, *Leuconostoc*, *Weissella*—tend to down-regulate TLR4/NF-κB and modulate B- and T-cell responses, with clinical reports noting cytokine reductions in some settings [Shahbazi, 2021; 88].

Table 9. The role of probiotics in **anti-inflammatory effects**

Mechanism	**Description**	**Microbes involved**	**References**
NF-κB/MAPK suppression; reduce iNOS/COX-2 & cytokines	Kimchi-derived *L. plantarum* strains suppressed NF-κB/MAPK signalling in LPS-stimulated macrophages, lowering NO, iNOS/COX-2, TNF-α, IL-1β, IL-6; strong antioxidant co-effects.	*Lactiplantibacillus plantarum* (from kimchi)	[32], [33]
Anti-inflammatory starter culture in kimchi	A kimchi starter (*Limosilactobacillus reuteri* EFEL6901) was developed for anti-inflammatory activity, reducing inflammatory mediators in cell/animal models.	*L. reuteri*	[79]
Reduce LPS load & NF-κB in colitis model	Doenjang (Korean fermented soybean paste) ameliorated TNBS-colitis by suppressing gut microbial LPS and NF-κB activation.	Mixed meju/soy paste microbiota (incl. *Bacillus*, *Aspergillus*)	[49]
IL-10–skewing immune signalling	*Bacillus subtilis* natto secretes GroEL that drives IL-10/IL-12 production in human THP-1 dendritic cells—mechanistic basis for natto's immunoregulatory effects.	*B. subtilis* (natto)	[54]
SCFA-mediated cytokine modulation; barrier support	Indonesian strain *L. plantarum* Dad-13 prevented TNBS-colitis in mice by ↑ SCFAs (acetate/propionate/butyrate), ↓ TNF-α/IL-6/IL-1β, ↑ IL-10.	*L. plantarum* Dad-13 (Indonesia, from dadih/fermented foods)	[69]
Direct anti-inflammatory effects of tempeh extract	Nutrient-enriched tempeh (fermented soybean) extracts reduced NO, IL-1β, TNF-α in RAW264.7 cells and showed in-vivo anti-inflammatory/analgesic activity.	Primarily *Rhizopus* spp.; co-occurring LAB	[101]
Mucosal immune modulation	Bekasam-derived *Lactobacillus* enhanced intestinal IgA and shifted cytokine milieu (↑ IL-4/IL-10	*Lactobacillus* spp. (bekasam)	[40]

from fermented fish	alongside innate responses), supporting anti-inflammatory tone.		
Overview of anti-inflammatory effects in Asian fermented veg/soy	Reviews summarize LAB from cabbage/soy ferments down-regulate TLR4/NF-κB pathways and modulate B/T-cell responses; clinical kimchi trials report cytokine reductions.	LAB: *Lactiplantibacillus*, *Leuconostoc*, *Weissella*	[88]

3.4 Gut Barrier Integrity

Across Asian ferments (Table 10), probiotic strains tighten the intestinal barrier—raising ZO-1/occludin/claudin-1, restoring TEER and brush-border structure, and suppressing LPS–TLR4–NF-κB—via kimchi LAB, *Weissella* spp., natto *B. subtilis*, doenjang microbial consortia, and Indonesian ferments *L. plantarum*. Kimchi starter LAB protected Caco-2 monolayers from LPS/H_2O_2 leak and preserved ZO-1/occludin, showing a direct, cell-layer benefit [48]. A kimchi-derived *Weissella koreensis* strain then pushed this further in vivo, boosting ZO-1/occludin/claudins and attenuating TNBS colitis while lowering TNF-α/IL-1β/IL-6 [50]. From natto, *Bacillus subtilis* JLCC513 strengthened tight junctions (↑ ZO-1/occludin/claudin-1), dampened TLR4–NF-κB–NLRP3 signalling, and improved gut histology in high-fat-diet rats [93]. A mixed microbiota from doenjang (fermented soybean paste) worked upstream by reducing microbial LPS and suppressing NF-κB, easing TNBS colitis [49]. In an LPS-challenge model, *Weissella cibaria* MW01 restored TEER, lowered FITC-dextran flux, and up-regulated occludin/claudin-1/TJP1 via NF-κB–MLCK–pMLC inhibition [38]. Finally, an Indonesian *Lactiplantibacillus plantarum* (IS-20506) repaired brush-border structure, raising galectin-4 and myosin-1a across small-intestinal segments after LPS injury—another tangible sign of barrier recovery [72]

Table 10. The role of probiotics in gut barrier integrity

Mechanism	**Roles (what was shown)**	**Microbes involved / food source**	**References**
Tight-junction protection in epithelial monolayers	LAB from kimchi starter cultures prevented LPS/H_2O_2-induced permeability increases; preserved ZO-1/occludin in Caco-2 cells	*Lactococcus lactis*; *Companilactobacillus allii*; *Leuconostoc mesenteroides* — kimchi	[48]
Increased TJ proteins & anti-inflammation in colitis	↑ ZO-1, occludin, claudins; ↓ TNF-α, IL-1β, IL-6; attenuated TNBS-colitis	*Weissella koreensis* KJ — isolated from kimchi	[50]
Strengthened barrier & dampened TLR4/NF-κB/NLRP3	↑ ZO-1/occludin/claudin-1; ↓ TLR4–NF-κB–NLRP3 signalling; improved histology in HFD rats	*Bacillus subtilis* natto JLCC513 — natto	[93]

Lower microbial LPS production & NF-κB activation	Reduced gut microbial LPS production; suppressed NF-κB; ameliorated TNBS colitis	Mixed microbiota of doenjang (fermented soybean paste; typically *Bacillus*/LAB consortia)	[49]
Barrier preservation under LPS challenge	Recovered TEER; ↓ FITC-dextran flux; ↑ occludin/claudin-1/TJP1 via NF-κB–MLCK–pMLC inhibition	*Weissella cibaria* MW01 — isolated from Chinese sauerkraut (fermented cabbage)	[38]
Brush-border structural repair after LPS injury	↑ galectin-4 & myosin-1a in duodenum/jejunum/ileum; indicates restoration of epithelial brush border integrity	*Lactiplantibacillus plantarum* IS-20506 — from Indonesian dadih (fermented buffalo milk)	[72]

3.5 Probiotic and its bioactive compounds for human health

Tempeh, kimchi, natto, bekasam, tempoyak and similar ferments act like smart delivery vehicles for beneficial microbes and helpful fats. Core residents—such as *Lactiplantibacillus plantarum* and *Bifidobacterium*—can convert plant omega-3 (ALA) into conjugated linolenic acids (CLnA) during fermentation, so the food already carries these bioactives before it is eaten [54, 31, 25, 3]. After a meal, CLnA is absorbed and partly converted to CLA, which in cells and animals has supported cholesterol handling and energy signalling [71, 97, 59]. Many of these microbes also express bile-salt hydrolase (BSH), subtly reshaping the bile-acid pool and engaging FXR/TGR5 pathways that link to GLP-1 release, energy balance, and cholesterol metabolism [12, 21]. Consistent with those mechanisms, open-access reviews and trials have reported modest but meaningful improvements in glycaemia (HbA1c/FBG/HOMA-IR), lipid profiles (TC/LDL-C/TG), inflammatory tone (CRP/cytokines), and gut-barrier integrity [94, 57, 11, 100, 84, 29, 13, 4]. These foods also help with vitamins in a practical way. LAB truly synthesise vitamin K_2 (menaquinones), explaining why natto is consistently rich in MK-7, while kimchi contributes mostly plant vitamin K_1 with smaller amounts of K_2 [62, 102, 96, 47]. Although LAB do not make vitamins D or E, ferments are excellent carriers. UV-treated mushrooms or baker's yeast supply bioavailable vitamin D_2, and encapsulation keeps D_3 or E stable in yoghurt-style drinks without harming probiotic viability [55, 36, 53, 41, 64, 73]. On the lipid side, simple formulation levers—co-fermenting ALA-rich seeds/oils and using nano emulsions—can raise delivery of CLnA/CLA and other lipophilic nutrients in familiar, everyday foods [67, 68, 25, 3].

3.6 LAB and CLnA

CLnA-enriched Asian ferments—kimchi, fermented soy, bekasam and natto—combine LAB conversion of ALA to CLnA/CLA with BSH-driven bile-acid remodelling (FXR/TGR5) to lower atherogenic lipids and reinforce gut-barrier, anti-inflammatory defences. During fermentation on ALA-rich substrates, *L. plantarum* or selected

Bifidobacterium can generate CLnA in-product [54, 31, 25, 3]. Once ingested, CLnA is absorbed and partly converted to CLA, feeding into hepatic lipid handling and enteroendocrine signalling [71, 97, 59]. In parallel, LAB BSH activity remodels bile acids and nudges FXR/TGR5—providing a second lever for cholesterol and energy balance [12, 21]. These mechanisms surface in real foods, i.e. kimchi LAB lowered atherogenic lipids in models and small trials [13, 42], fermented soy reduced LDL-C in an RCT [44], *Pediococcus* from bekasam improved serum lipids in vivo [18, 61], and natto improved lipid profiles via gut–liver signalling [90]. Because CLnA such as α-eleostearic acid is absorbed and rapidly converted to CLA, CLnA-enriched ferments—or co-formulated oils—offer a realistic, food-first route to active lipid signalling [97, 59, 81].

CLnA/CLA can also complement the anti-inflammatory and barrier actions already shown for Asian ferments. Kimchi-derived LAB suppressed NF-κB/MAPK and lowered iNOS/COX-2 and cytokines, and purpose-built starters preserved that effect in cell and animal models [32, 33, 79]. Doenjang reduced microbial LPS and blocked NF-κB in colitis models [49], while *Weissella* and kimchi starters preserved ZO-1/occludin/claudins and tightened Caco-2 and in-vivo barriers; natto (*Bacillus subtilis*) strengthened junctions and damped TLR4–NF-κB–NLRP3 [38, 48, 50, 93]. In short, CLnA-fortified, region-familiar ferments can sit atop the BSH/bile-acid pathway and plausibly amplify diet-level lipid and barrier benefits.

3.7 Glucose metabolism improvement (food-first route)

Multi-strain probiotics and fermented vegetables modestly improved HbA1c/FBG/HOMA-IR in adults with prediabetes or type 2 diabetes, especially with adequate duration [3, 20, 76, 60, 44]. Mechanistically, SCFAs from these ferments engage FFAR2/FFAR3→AMPK and boost GLP-1—precisely the levers that pair well with CLnA/CLA's lipid-centric actions [8, 102]. Practically, Indonesian/Asian platforms already carrying *L. plantarum*, *L. fermentum*, and *Bifidobacterium*—tempeh, dadih, bekasam, tempoyak, kimchi—can be strain-selected and ALA-fortified to produce CLnA in situ, while nano emulsions or encapsulation improve fatty-acid delivery [2, 46, 67, 68].

3.8 LAB and lipid-soluble vitamins

Among D, E, and K, LAB truly synthesise only vitamin K_2 (menaquinones); Asian ferments are the key foods. Natto routinely carries very high MK-7, whereas kimchi provides mostly K_1 with minor K_2 [64, 47, 96, 48]. These matrices also co-deliver LAB or *Bacillus* with BSH activity, linking to bile-acid remodelling, FXR/TGR5 engagement, and downstream cholesterol/energy effects [6, 21]. Industrial optimisation of *Bacillus subtilis* natto has further increased MK-7 yields, explaining why soybean ferments dominate K_2 delivery regionally [57, 12, 103].

Although LAB do not synthesise vitamins D or E, food formats make them easy to co-deliver. UV-irradiated mushrooms and UV-treated baker's yeast supply bioavailable D_2 for

incorporation into fermented dishes [53, 35, 41, 36]. In probiotic beverages, encapsulation keeps D_3 stable during fermentation and storage without harming culture viability [65]; similarly, encapsulated vitamin E remains stable and bio-accessible in yoghurt-style matrices [72]. Fermentation of legumes/vegetables (e.g., tempeh/kimchi systems) also reduces antinutrients such as phytates, indirectly improving fat-soluble-vitamin uptake—useful for barrier and epithelial health when these vitamins are co-formulated [52, 77]. Set alongside SCFA-linked signalling and BSH-mediated bile-acid remodelling, these vitamin-enabled matrices offer a culturally congruent way to support microbiota modulation, barrier function, and downstream metabolic endpoints [6, 21, 53, 65, 72].

3.9 Synergistic effects of probiotics and bioactive lipids in disease prevention

Probiotic LAB and lipophilic bioactives work together on a few well-mapped levers. First, many strains raise short-chain fatty acids (SCFAs), which engage FFAR2/FFAR3 and activate AMPK—supporting insulin signalling and damping low-grade inflammation tied to diabetes and cardiovascular risk [8, 28, 19, 10]. Second, bile-salt hydrolase (BSH) reshapes the bile-acid pool and nudges FXR/TGR5, with downstream effects on GLP-1 release, energy balance, and cholesterol handling [6, 21]. Third, several LAB convert dietary PUFAs into conjugated linoleic/linolenic acids (CLA/CLnA), which are absorbed and partly converted in vivo, providing anti-inflammatory and cardiometabolic signals [51, 30, 71, 62]. Finally, some Asian ferments contribute vitamin K_2 (MK-7), with natto consistently rich and potentially supportive of cardiometabolic health [47, 96].

3.10 Evidence across the six domains and translation to foods

Across open-access reviews and trials, Asian ferments generally pushed multiple levers in the same direction, including microbiota shifted towards *Lactobacillus/Bifidobacterium* and other SCFA producers [11]; glycaemia improved modestly in prediabetes/T2DM [79, 59]; lipids softened (TC/LDL-C/TG) with heterogeneity by strain, dose, and matrix [9, 66]; inflammation eased (CRP, cytokines) [75, 78]; and barrier integrity improved, consistent with reduced permeability [29]. These signals align with SCFA→FFAR2/3→AMPK for glycaemia [3, 79, 59] and BSH→FXR/TGR5→GLP-1/energy for lipids and weight-related endpoints [21, 13, 9, 66, 102]. Kimchi LAB—especially *Lactiplantibacillus/Lactobacillus plantarum*—lowered lipids in models and small trials, consistent with BSH-linked bile-acid loss and cholesterol assimilation [13, 42, 79]; natto (*Bacillus subtilis*) reduced TC/TG/LDL-C and increased HDL-C in animals [90]; Indonesian fish ferments such as bekasam carry *Pediococcus* with BSH activity and lipid benefits [18, 61]; tempeh and fermented soy add modest LDL-C reductions and provide LAB “workhorses” capable of CLA/CLnA formation when given an ALA substrate [44, 51, 4, 25]. CLnA such as α-eleostearic acid is absorbed and rapidly converted to CLA, supporting a food-first route via CLnA-enriched ferments or co-formulated oils [62, 62, 84]. Barrier and anti-inflammatory gains—lower LPS/NF-κB/MAPK and higher tight-junction proteins—have been shown with LAB-rich vegetable and soybean ferments [49, 45].

3.11 Gaps, priorities, and near-term directions

The main limitations are familiar, i.e. heterogeneous strains, doses, durations, matrices, and outcome panels that blur effect sizes and slow translation [79, 59, 9, 66]. Further research priorities are clear, including head-to-head randomised controlled trials of Southeast Asian ferments, standardised strain reporting (genus–species–strain), matrix-aware dosing/duration, and a shared biomarker core—SCFAs; BSH/bile-acid panels; GLP-1; tight-junction markers; LPS—paired with clinical endpoints (HbA1c/FBG/HOMA-IR; TC/LDL-C/TG; CRP/cytokines; permeability) [21, 29]. On formulation, two practical levers deserve testing at scale: (1) co-fermenting ALA-rich oils/seeds with *L. plantarum/Bifidobacterium* to generate CLnA in familiar matrices (e.g., tempeh beverages, cultured soy), and (2) leveraging natto's MK-7 as a nutrient–microbe synergy within food-based programmes [30, 25, 96]. The preventive signal is modest—not pharmacological—but it is meaningful and culturally congruent when these foods are eaten routinely [90, 44].

4. Conclusion

This review synthesised open-access evidence showing that Asian fermented foods and their probiotic LAB nudge multiple cardiometabolic levers in the same, favourable direction across six domains, including microbiota composition shifts towards SCFA producers; glycaemia improves modestly (HbA1c/FBG/HOMA-IR); lipid profiles soften (TC/LDL-C/TG); inflammatory markers decline (CRP/cytokines); and intestinal permeability tightens. These clinical signals coherently map to two primary pathways—SCFA→FFAR2/FFAR3→AMPK and BSH-mediated bile-acidremodelling→FXR/TGR5→GLP-1/energy—linking microbial activity to improved insulin action, cholesterol handling, and barrier integrity. For real-world use, microbe–food pairs already embedded in regional diets offer practicable vehicles, i.e. *Lactiplantibacillus plantarum* in *kimchi/tempeh*, *Bacillus subtilis* in *natto*, and *Pediococcus spp.* in *bekasam*, aligning with these pathways and cultural eating patterns. Priorities, anchored to the aims and findings, are fourfold: (1) conduct head-to-head randomised controlled trials of Southeast-Asian ferments to quantify effect sizes in comparable settings; (2) require strain-resolved reporting (genus–species–strain) to enable replication and strain–mechanism mapping; (3) calibrate matrix-aware dosing and duration (viable CFU, food format) to enhance consistency of outcomes; and (4) adopt a shared biomarker core—SCFAs; bile-acid panels/BSH activity; GLP-1; tight-junction markers; LPS—paired with clinical endpoints (HbA1c/FBG/HOMA-IR; TC/LDL-C/TG; CRP/cytokines; permeability). With these steps, food-first, culturally congruent programmes can move from promising patterns to confident, scalable practice.

Glossaries

Table 11. Glossaries

Term	Plain-English definition
ALA (α-linolenic acid)	Plant omega-3; substrate for microbial CLnA formation.
AMPK	Cellular energy sensor; activation improves insulin action and fat oxidation.
Atherogenic index	Composite lipid risk score; higher values suggest greater risk.
Bacillus subtilis (*natto*)	Spore-former in *natto*; source of MK-7 and bioactive enzymes.
Bekasam / *Peda*	Indonesian fermented fish; sources of *Pediococcus/Lactobacillus*.
Bile salt hydrolase (BSH)	Microbial enzyme deconjugating bile acids; alters lipid uptake and signalling.
CLA (conjugated linoleic acids)	Conjugated linoleic isomers; anti-inflammatory/lipid-modulating candidates.
CLnA (conjugated linolenic acids)	Conjugated ALA isomers; partly converted to CLA in vivo.
CRP / IL-6 / TNF-α	Common blood markers of systemic inflammation.
CYP7A1	Liver enzyme catalysing the rate-limiting step of bile-acid synthesis.

Term	Plain-English definition
Dose / Duration	Amount of microbes or food and length of intake; key drivers of effect size.
Dysbiosis	Disrupted microbiota composition/function linked to disease.
Endotoxemia (metabolic)	Circulating LPS causing low-grade systemic inflammation.
FBG (fasting blood glucose)	Blood sugar after an overnight fast.
Fermented foods	Foods made by microbial growth/enzymes (e.g., *kimchi, tempeh, natto, bekasam, tempoyak, miso/doenjang*).
FFAR2 (GPR43) / FFAR3 (GPR41)	SCFA receptors on gut, immune, and metabolic cells.
FXR (NR1H4)	Nuclear bile-acid receptor regulating bile acids, lipids, and glucose.
GLP-1	Gut hormone that enhances insulin secretion and satiety.
Gut microbiota	Microbial community inhabiting the gastrointestinal tract.
HbA1c	Three-month average blood glucose marker.
HOMA-IR	Index estimating insulin resistance from fasting glucose and insulin.
IgA (secretory)	Antibody supporting mucosal defence in the gut.
iNOS / COX-2	Enzymes producing nitric oxide/prostaglandins during inflammation.
Kimchi	Korean fermented vegetables; rich LAB ecosystem with metabolic signals.
Lactic Acid Bacteria (LAB)	Gram-positive fermenters (e.g., *Lactiplantibacillus/Lactobacillus, Lactococcus, Leuconostoc, Pediococcus, Weissella*) used as probiotics/starters.
Lactiplantibacillus (Lactobacillus) plantarum	Versatile LAB in *kimchi/tempeh*; contributor to SCFA/BSH activity.
LPS	Endotoxin from Gram-negative bacteria; activates TLR4.
MAPK (p38/ERK/JNK)	Kinase cascades regulating inflammation and stress responses.
Matrix effects	Influence of the food vehicle (soy/dairy/vegetable/fish) on probiotic performance.
Mechanism→endpoint mapping	Linking biology (e.g., SCFA→AMPK; BSH→FXR/TGR5) to clinical measures.
Miso / Doenjang	Fermented soybean pastes; complex consortia affecting LPS/NF-κB pathways.
Nanoemulsion / Encapsulation	Techniques improving solubility, stability, and delivery of lipophilic actives.
Natto	Japanese fermented soybean; very high MK-7 (vitamin K_2).
NF-κB	Transcription factor driving inflammatory gene expression.
NLRP3 inflammasome	Intracellular complex activating IL-1β/IL-18; linked to metabolic inflammation.
Omega-3 / Omega-6 PUFAs	Essential polyunsaturated fatty acids for heart/metabolic health.
Open-access (OA)	Free-to-read research included in the synthesis.
Pathobionts	Normally low-abundance microbes that can promote disease under stress.
Pediococcus spp.	LAB in fermented fish (*bekasam*); some strains show BSH

Term	Plain-English definition
	activity.
Probiotic	Live microbes that, in adequate amounts, benefit the host.
PYY	Gut hormone promoting satiety and slower gastric emptying.
RCT (randomized controlled trial)	Intervention design that minimizes bias via randomization.
Reverse cholesterol transport	HDL-mediated removal of cholesterol from tissues to the liver.
RoB 2	Cochrane risk-of-bias tool for randomized trials.
Short-chain fatty acids (SCFAs)	Microbial acetate/propionate/butyrate that signal to host tissues.
Strain-resolved reporting	Naming genus–species–strain to ensure reproducibility.
SYRCLE	Risk-of-bias tool adapted for animal studies.
TC / LDL-C / HDL-C / TG	Total, "bad," "good" cholesterol, and triglycerides.
TEER	In-vitro electrical measure of epithelial barrier integrity.
Tempeh	Indonesian fermented soybean cake; mold-led with LAB present.
Tempoyak	Fermented durian (Indonesia/Malaysia); hosts *L. plantarum* and allies.
TGR5 (GPBAR1)	Bile-acid GPCR; activation boosts GLP-1 and energy expenditure.
TLR4	Innate immune receptor sensing LPS; triggers inflammation.
Tight junctions	Protein complexes sealing spaces between epithelial cells.
Vitamin D_2 / D_3	Ergocalciferol (UV-fungi/yeast) and cholecalciferol; carried via fermented matrices.
Vitamin E	Tocopherols/tocotrienols; fat-soluble antioxidants.
Vitamin K_2 (menaquinones/MK-n)	Microbially made fat-soluble vitamins; MK-7 abundant in *natto*.
Weissella / Leuconostoc / Lactococcus	LAB genera common in vegetable/soy ferments.
ZO-1 / Occludin / Claudins	Core tight-junction proteins; higher levels indicate a stronger barrier.
Zonulin	Protein associated with regulation of intestinal permeability.

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