Jurnal Teknologi dan Inovasi Kesehatan

SAH PUBLISHER

Vol. 1, No. 1, September 2026

The Relationship between Probiotic Consumption and Gut Microbiota Health in Older Adults

Alfan Ridha[1*], **Triagung Yuliyana**[2], **Rani Rahmasari Tanuwijaya**[3], **Nafilah** [4], **Rusdiarti** [5]

[1]Universitas Sugeng Hartono, Indonesia

[2]Universitas Indonesia Maju (UIMA) Jakarta

[3]Sekolah Tinggi Ilmu Kesehatan Pertamedika, Indonesia

[4]Universitas Muhammadiyah Kendal Batang, Indonesia

[5]Politeknik Negeri Jember, Indonesia

**Corresponding author. Email: alfan_ridha07@gmail.com*

Abstract. Population ageing is increasing the importance of interventions that preserve digestive, immune and metabolic function in later life, and the gut microbiota has emerged as a central mediator of all three. This article reviews the published literature on the relationship between probiotic consumption and gut microbiota health in older adults, supplemented only by official public statistics on population ageing in Indonesia. The review follows the consensus definitions of the International Scientific Association for Probiotics and Prebiotics and examines how the ageing gut changes in composition and function, how dietary patterns and fermented foods shape that community, what mechanisms plausibly connect ingested strains to host outcomes, and what the clinical trial evidence shows for outcomes that matter in geriatric care. The ageing gut is characterised by reduced microbial diversity, loss of core health associated taxa, enrichment of pathobionts, and a reciprocal relationship with the chronic low grade inflammation known as inflammaging, which is in turn associated with frailty and functional decline. Probiotic effects are strain specific and dose dependent, and the mucosal colonisation of ingested strains varies considerably between individuals, so benefits cannot be assumed for a product class as a whole. Meta analytic evidence supports selected strains for reducing antibiotic associated diarrhoea and, in higher risk settings, Clostridioides difficile associated diarrhoea, for shortening upper respiratory tract infections, and for improving bowel habits in constipation, although a large pragmatic trial in older hospital inpatients found no benefit, and safety cautions apply to immunocompromised and critically ill patients. Traditional Indonesian fermented foods such as tempe, tape and dadih are nutritionally valuable but, on current evidence, cannot be equated with clinically tested probiotics. The review concludes with practical guidance for clinicians and caregivers on choosing characterised products, matching strain to indication, and monitoring response.

Keywords: Probiotics; Gut microbiota; Older adults; Inflammaging; Fermented foods; Frailty

Received August 2026 / Revised September 2026 / Accepted September 2026

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INTRODUCTION

The number and proportion of older adults are rising in almost every country, and Indonesia is no exception. According to Statistics Indonesia (Badan Pusat Statistik), residents aged 60 years and over accounted for 12.00 percent of the national population in

2024, and the same official series projects that this group will grow to about 65.82 million people, or 20.31 percent of the population, by 2045 [1]. The burden of ill health carried by this group is substantial: the elderly morbidity rate reported by Statistics Indonesia was 20.71 percent, and 42.81 percent of older Indonesians reported a health complaint in the month preceding the survey [1]. Because so much of late life morbidity involves infection, antibiotic exposure, constipation, malnutrition and chronic inflammation, attention has turned to the intestinal microbial community as a modifiable contributor to healthy ageing, and to probiotic products as a widely accessible means of influencing it.

The human gut harbours a dense and metabolically active community of bacteria, archaea, fungi and viruses whose collective genome and metabolic output influence nutrient harvest, epithelial barrier integrity, immune education and even neurocognitive function [2], [3]. This community is not static across the lifespan. In the ELDERMET cohort of 178 older people, Claesson and colleagues showed that the composition of the faecal microbiota clustered strongly according to where a person lived and what they ate, that residents of long stay care had a markedly less diverse microbiota than community dwelling peers, and that loss of the community associated microbiota correlated with increased frailty and poorer health measures [4]. Studies of extreme ageing tell a consistent story from the other end of the spectrum. Biagi and colleagues reported that centenarians carry a microbiota characterised by a rearrangement of Firmicutes, an enrichment of pathobionts such as Proteobacteria, and a marked reduction in the butyrate producing Faecalibacterium prausnitzii, a pattern that tracked circulating inflammatory markers [5]. In a later study of semi supercentenarians aged 105 years and over, the same group found that extreme longevity was accompanied by increases in health associated groups such as Akkermansia, Bifidobacterium and Christensenellaceae, suggesting that some rearrangements may support, rather than undermine, health at advanced age [6]. Reviewing these data, O'Toole and Jeffery concluded that there is no single age at which the microbiota abruptly changes; instead, composition drifts gradually and is shaped powerfully by diet, medication, residence and disease burden [7]. More recent synthesis work similarly describes a progressive loss of core, health associated taxa and a rise in inter individual uniqueness with age, with the steepness of that loss predicting frailty and mortality more closely than chronological age itself [8].

Inflammation provides the mechanistic bridge between these compositional shifts and clinical outcomes. Franceschi and colleagues describe inflammaging as the chronic, sterile, low grade inflammation that accompanies ageing, and identify the gut microbiota, together with its metabolites and its increasing leakiness across a more permeable barrier, as one of its principal drivers [9]. A less diverse microbiota produces fewer short chain fatty acids, offers weaker colonisation resistance against pathogens, and presents a larger burden of pro inflammatory microbial components to an ageing immune system that is already prone to dysregulation. The clinical correlates are familiar in geriatric practice: heightened susceptibility to infection, slower recovery after antibiotics, constipation, sarcopenia and frailty. It is against this background that probiotics have attracted interest, because they promise a comparatively simple, food based or capsule based way of adding defined live microorganisms to a depleted ecosystem.

The concept is not new. A joint working group of the Food and Agriculture Organization and the World Health Organization published evaluation guidelines for probiotics in food in 2002, recommending strain level identification, functional characterisation, safety assessment and human efficacy trials before health claims are made [10]. The definition

used across the modern field was consolidated by an expert panel convened by the International Scientific Association for Probiotics and Prebiotics (ISAPP), which defined probiotics as live microorganisms that, when administered in adequate amounts, confer a health benefit on the host [11]. Sanders and colleagues, writing for ISAPP, emphasised that probiotic benefits are specific to strain and to indication, that products must deliver the dose shown to be effective in human studies, and that not every fermented food qualifies as a probiotic [12]. Whether consuming such products measurably improves the health of the gut microbiota of older adults, and for which outcomes, is nevertheless a question on which the evidence is uneven and sometimes contradictory. This review therefore set out to synthesise the published literature on the relationship between probiotic consumption and gut microbiota health in older adults, to distinguish outcomes with credible trial support from those carried mainly by extrapolation, to consider safety in vulnerable patients, and to place the discussion in the Indonesian context, where traditional fermented foods are dietary staples and commercial probiotic products are increasingly marketed to families caring for older relatives.

METHODS

This article is a narrative literature review supplemented by official public data. No primary clinical data were collected, and no finding reported here originates from the authors; every empirical claim is attributed to its published source. The literature was identified through searches of major biomedical databases and publisher platforms using combinations of the terms probiotic, gut microbiota, gut microbiome, older adults, elderly, ageing, inflammaging, frailty, antibiotic associated diarrhoea, Clostridioides difficile, respiratory tract infection, constipation, fermented foods and safety. Priority was given to consensus statements of the International Scientific Association for Probiotics and Prebiotics, systematic reviews and meta analyses, randomised controlled trials conducted in or including older adults, and landmark cohort studies of the ageing microbiota, most of which appeared in journals of major international publishers. Sources were selected for their direct relevance to the review questions rather than by an exhaustive protocol, and older foundational studies were retained where they remain the reference point for a definition or an effect estimate. Demographic context for Indonesia was taken exclusively from the official publications of Statistics Indonesia (Badan Pusat Statistik), in particular the 2024 elderly population statistics [1]. Where trials disagree, both the positive and the null results are reported, and effect estimates are quoted with the population and setting in which they were obtained so that readers can judge their applicability to older adults in community, hospital and long term care settings.

RESULT AND DISCUSSION

1. The Ageing Gut Microbiota: Diversity Loss, Inflammaging and Frailty

The starting point for any discussion of probiotics in older adults is that the target ecosystem changes with age in ways that are partly physiological, partly environmental and partly iatrogenic. Functional capacity matters as much as taxonomy. Flint and colleagues summarised how gut microbes ferment dietary carbohydrate that escapes digestion in the small intestine, producing short chain fatty acids that feed colonocytes, lower luminal pH and exclude pathogens, while also transforming bile acids and synthesising vitamins [3]. Ageing erodes several supports of this system at once: dietary variety narrows, fibre intake commonly falls, dentition and appetite decline, gastric acid

suppression and repeated antibiotic courses disturb the community, and institutional diets differ sharply from home diets [2]. Salazar and colleagues, reviewing nutrition and the gut microbiome in the elderly, describe a typical trajectory towards reduced diversity, lower Bifidobacterium abundance, and expansion of enterobacteria and other opportunists, with consequences for barrier function and immune tone [2].

The ELDERMET findings remain the most influential demonstration that these changes are socially and nutritionally patterned rather than purely chronological, because microbiota composition separated older people by residence and diet more cleanly than by age, and the diversity gradient ran from community dwellers through day hospital attendees to long stay residents, in step with frailty and comorbidity [4]. Extreme ageing studies add nuance: the centenarian microbiota is not simply a degraded version of the young adult microbiota but a remodelled community in which pathobiont enrichment coexists, in the healthiest survivors, with compensatory increases in taxa such as Akkermansia that support mucin turnover and metabolic health [5], [6]. O'Toole and Jeffery accordingly caution against treating any single compositional signature as the definition of an old microbiota, and stress diet and health status as the dominant levers [7]. Ghosh, Shanahan and O'Toole synthesise the field by distinguishing core taxa, whose loss is broadly deleterious, from subdominant and pathobiont taxa whose expansion drives inflammation; interventions that arrest the loss of core taxa, whether dietary or microbial, are in their account the most plausible route to healthier ageing trajectories [8].

Inflammaging completes the picture. Franceschi and colleagues argue that the ageing gut, with its altered microbiota and increased permeability, continuously supplies inflammatory stimuli, including microbial products that enter the circulation and sustain elevations in interleukin 6 and C reactive protein [9]. Those same markers predict frailty, disability and mortality in older cohorts, which is why the microbiota has become an attractive intervention target: if microbial change feeds inflammation, and inflammation feeds frailty, then restoring a more diverse and metabolically favourable community could in principle interrupt the cycle. Probiotics are one candidate tool for that restoration, although, as the evidence below shows, their effects are narrower and more specific than the theory might suggest.

2. What Counts as a Probiotic: Definitions, Strain Specificity and Dose

Much confusion in the marketplace, and some in the clinical literature, dissolves once the agreed definitions are applied. Under the ISAPP consensus, a probiotic must be a live microorganism, identified to strain level, administered in an adequate amount, and shown to confer a health benefit in controlled human studies [11]. The FAO and WHO guidelines had earlier set the same bar, requiring strain identification by phenotypic and genotypic methods, deposit in an international culture collection, safety assessment, and efficacy demonstrated in human trials before a product may be called probiotic [10]. Sanders and colleagues underline the practical corollaries: benefits demonstrated for one strain cannot be transferred to another strain of the same species without evidence; the dose delivered at the end of shelf life, not at manufacture, must match the dose tested; and generic claims that a product supports gut health are not a substitute for indication specific evidence [12].

The consensus vocabulary also separates probiotics from neighbouring categories with which they are often conflated. Prebiotics are substrates selectively utilised by host microorganisms to confer a health benefit, the archetype being fermentable fibres that expand bifidobacteria and short chain fatty acid production [13]. Synbiotics combine live

microorganisms with such substrates, either complementarily, where each component acts independently, or synergistically, where the substrate is designed to feed the co administered strain [14]. Postbiotics, defined in a 2021 ISAPP consensus as preparations of inanimate microorganisms or their components that confer a health benefit, extend the field to non live preparations that may offer safety and stability advantages for frail patients [15]. Fermented foods occupy their own category: the ISAPP consensus on fermented foods defines them as foods made through desired microbial growth and enzymatic conversions of food components, and stresses that most traditional fermented foods cannot be called probiotics because their microbial strains are undefined and their health effects untested, even though such foods may be nutritious and may contain live microbes at consumption [16]. Each of these distinctions matters for older consumers, because products sold side by side on a pharmacy shelf may belong to entirely different evidentiary categories.

Strain specificity and dose follow directly from the definitions. The trials that anchor clinical practice tested particular strains, or defined combinations, at doses typically between one billion and tens of billions of colony forming units per day, given for defined durations, and their results license claims only for those strains and comparable doses [12]. A generic Lactobacillus capsule of unstated strain and low count is therefore not interchangeable with a tested Bifidobacterium lactis preparation, however similar the packaging. This specificity also explains why meta analyses that pool many strains find heterogeneous effects: the pooled estimate averages products that may differ as much from one another as drugs within a broad class.

3. Plausible Mechanisms: Metabolites and Immune Modulation

Probiotic strains, while resident or in transit, can influence the host through metabolites, through competition with pathogens, and through direct signalling to epithelial and immune cells. The best characterised metabolic pathway is short chain fatty acid production. Koh and colleagues reviewed how acetate, propionate and butyrate, generated by bacterial fermentation of dietary fibre, activate G protein coupled receptors, inhibit histone deacetylases, and serve as energy substrates, thereby influencing epithelial integrity, glucose and lipid metabolism, and immune regulation [17]. Butyrate in particular is the preferred fuel of colonocytes, and its decline, which accompanies the loss of producers such as Faecalibacterium prausnitzii in very old adults, plausibly weakens the barrier whose failure feeds inflammaging [5], [17]. Probiotic lactobacilli and bifidobacteria are not themselves major butyrate producers, but by producing lactate and acetate they cross feed resident butyrate producers, which is one route by which supplementation could raise the metabolic output of the whole community rather than merely adding a transient population.

Immune mechanisms are equally relevant in an age group where infection dominates morbidity. Rooks and Garrett describe how microbial metabolites, especially short chain fatty acids, promote a tolerogenic and anti inflammatory phenotype in mucosal immune cells, support regulatory T cell homeostasis, and maintain the balance between defence and tolerance on which gut health depends [18]. Ingested strains may additionally strengthen tight junctions, stimulate mucus and secretory immunoglobulin A production, produce bacteriocins and organic acids that inhibit pathogens, and compete for adhesion sites, a package of effects often summarised as colonisation resistance. These mechanisms are biologically coherent, and several are demonstrable in experimental systems; the clinical question is not whether they can occur but whether, at achievable

doses in older humans, they occur strongly enough to change outcomes, which is why the trial evidence reviewed below must govern practice.

4. Diet and Fermented Foods as Microbiota Modulators

Probiotics reach the gut against the background of diet, and diet remains the strongest everyday determinant of microbiota composition. Zmora, Suez and Elinav reviewed how dietary components, from fibre and polyphenols to sweeteners and emulsifiers, reshape the community and its metabolic output, with effects that are rapid, person specific and often larger than those of any supplement [19]. The classic demonstration is the crossover feeding study of David and colleagues, in which diets built entirely from animal or from plant products altered microbial community structure within days, increasing bile tolerant organisms such as Bilophila on the animal based diet and reducing plant polysaccharide degraders, while foodborne microbes from both diets transiently appeared in the gut [20]. Sonnenburg and Bäckhed draw the longer term lesson that habitual diet, particularly fibre intake, governs the diversity available to be inherited and maintained, and that a chronically fibre poor diet progressively starves fibre dependent taxa [21]. For older adults, whose diets often narrow, these findings imply that a probiotic taken alongside a low fibre diet is working against the current, whereas dietary improvement may itself be the most effective microbiota intervention.

Randomised evidence on fermented foods illustrates both the promise and the limits of food based approaches. Wastyk and colleagues randomly assigned 36 healthy adults to a diet high in fermented foods, including yogurt, kefir, fermented cottage cheese, kombucha, vegetable brine drinks and fermented vegetables, or to a high fibre diet, for ten weeks [22]. The fermented food diet increased overall microbiota diversity and decreased nineteen circulating inflammatory markers, an outcome of obvious interest for inflammaging, whereas the high fibre diet did not increase diversity over that period, although it altered carbohydrate active enzyme capacity [22]. The trial is frequently cited in support of fermented foods, but its limits deserve emphasis: participants were healthy adults rather than older patients, the intervention bundled several foods so no single strain effect can be isolated, and the comparator period was short. It supports the plausibility of food based modulation, not a clinical claim for any product.

Dietary pattern evidence closer to the target population comes from the NU AGE study. Ghosh and colleagues analysed 612 non frail or pre frail older people across five European countries who followed a Mediterranean style diet for twelve months, and found that greater adherence was associated with a distinct microbiota configuration: taxa enriched by the diet were associated with lower frailty, better cognitive function and lower levels of inflammatory markers including C reactive protein and interleukin 17, while also carrying greater potential for short chain fatty acid production [23]. Notably, this was a dietary intervention containing no probiotic supplement, yet it shifted the microbiota in directions that probiotic research aspires to. The sensible reading of the diet literature as a whole is that probiotics should be evaluated and prescribed as adjuncts to dietary management, and that trials and clinicians alike should record diet, because it can confound or eclipse the effect of the supplement.

5. Colonisation Resistance and the Limits of Empiric Probiotics

A widespread assumption behind probiotic use is that ingested strains settle in the gut and replenish missing residents. Direct mucosal sampling has complicated that picture. Zmora and colleagues used endoscopic sampling in human volunteers and found that mucosal

colonisation of a multi strain probiotic was highly person specific: some individuals were permissive colonisers while others resisted colonisation almost completely, and detection of strains in stool, the usual trial endpoint, proved an unreliable guide to mucosal presence [24]. Baseline host and microbiota features predicted who would colonise, which explains part of the inconsistency of clinical results and argues for personalised rather than empiric use. The companion study by Suez and colleagues examined the post antibiotic setting, where probiotics are most reflexively prescribed, and found that after antibiotics a multi strain probiotic actually delayed the reconstitution of the indigenous mucosal microbiome and host gene expression for months compared with spontaneous recovery, whereas autologous faecal microbiota transplantation restored the community within days [25]. These were small, intensively sampled studies in largely young and healthy volunteers, and they do not show that probiotics are useless; they show that adding foreign strains to an emptied niche can compete with, rather than assist, the return of the resident community. For older adults, who receive antibiotics frequently, the findings counsel indication specific use, attention to timing, and restraint in prescribing probiotics purely to rebuild the microbiota after antibiotics, a purpose for which benefit is unproven.

6. Evidence by Clinical Outcome in Older Adults

Antibiotic associated diarrhoea is the outcome with the longest trial history. Hempel and colleagues pooled 63 randomised trials with 11,811 participants across ages and found that probiotic use was associated with a lower risk of antibiotic associated diarrhoea, with a pooled relative risk of 0.58 and a number needed to treat of 13, although the strains were often poorly documented and heterogeneity was considerable [26]. The largest single trial in the population of interest, however, was null. The PLACIDE trial randomised 2,941 hospital inpatients aged 65 years and over who were receiving antibiotics to a multistrain preparation of lactobacilli and bifidobacteria delivering about 60 billion organisms daily for 21 days, or to placebo, and found antibiotic associated diarrhoea in 10.8 percent of the probiotic group and 10.4 percent of the placebo group, with Clostridioides difficile diarrhoea in 0.8 and 1.2 percent respectively [27]. PLACIDE does not refute the meta analytic finding so much as delimit it: a particular multistrain product, given to unselected older inpatients, many at modest baseline risk, did not reproduce the average benefit, which may be concentrated in higher risk patients and in particular strains.

The Cochrane review by Goldenberg and colleagues, focused specifically on Clostridioides difficile associated diarrhoea, pooled 31 trials with 8,672 patients and reached a similarly conditional conclusion [28]. Probiotics reduced the incidence of C. difficile associated diarrhoea, but the benefit was concentrated in trials where the baseline risk exceeded 5 percent; in that subgroup the incidence was 3.1 percent with probiotics against 11.6 percent with control, corresponding to a relative risk of 0.30 and a number needed to treat for an additional beneficial outcome of 12, whereas in low risk settings the overall number needed to treat rose to 42 [28]. Probiotics did not reduce the detection of C. difficile infection itself, indicating an effect on disease expression rather than eradication [28]. Read together, the diarrhoea literature supports targeted prophylaxis with studied strains in older patients at genuinely elevated risk, for example those receiving broad spectrum or multiple antibiotics or with previous C. difficile disease, and does not support indiscriminate co prescription with every antibiotic course.

Immune outcomes have been tested mainly through respiratory infection endpoints. The Cochrane review by Hao, Dong and Wu pooled trials of probiotics for acute upper respiratory tract infections and found that participants taking probiotics had lower odds of

experiencing at least one episode, with an odds ratio of 0.53, experienced episodes that were on average 1.89 days shorter, and had lower odds of receiving an antibiotic prescription, with an odds ratio of 0.65 [29]. The reviewers rated the quality of this evidence as low, the trials spanned children and adults, and older adult specific and vaccine response data remain comparatively thin [29]. For geriatric practice the finding is best treated as supportive but not decisive: a modest reduction in the frequency and duration of common respiratory infections is plausible for some strains, yet it does not displace vaccination, which remains the primary preventive measure in this age group.

Constipation, by contrast, is an outcome where the population, the mechanism and the evidence align well. Dimidi and colleagues meta analysed 14 randomised trials with 1,182 adults and found that probiotics reduced whole gut transit time by 12.4 hours and increased stool frequency by 1.3 bowel movements per week, with effects that were significant for Bifidobacterium lactis, which increased stool frequency by about 1.5 movements per week, but not significant for Lactobacillus casei Shirota [30]. Given the prevalence of constipation in older adults, its contribution to discomfort, delirium and laxative dependence, and the benign safety profile of the tested strains, this is among the most defensible routine uses, provided the strain used is one with demonstrated effect and the trial period is long enough, typically at least two to four weeks, for benefit to appear. Across all outcomes, the pattern is consistent: effects are real but modest, strain bound, and largest where baseline risk or symptom burden is highest.

7. Indonesian Context: Traditional Fermented Foods

Indonesia offers a distinctive setting for this discussion because fermented foods are not a niche health product but a dietary inheritance. Tempe, produced by fermenting soybeans with Rhizopus moulds, chiefly Rhizopus oligosporus, is a primary protein source for much of the population. Nout and Kiers, reviewing tempe science, note that fermented foods supply roughly one third of food consumption worldwide, and that tempe fermentation improves digestibility, generates bioactive compounds and yields a food of attractive flavour and texture whose starter ecology, although dominated by the mould, also includes lactic acid bacteria and yeasts [31]. Tape, made from cassava or glutinous rice fermented with mixed starter cultures of yeasts and lactic acid bacteria, and dadih, the buffalo milk fermented in bamboo tubes in West Sumatra, similarly deliver live lactic acid bacteria when eaten fresh, together with the nutrients of their substrates.

The scientific literature nevertheless requires a careful distinction between cultural and nutritional value and probiotic status. Under the ISAPP fermented foods consensus, a traditional fermented food qualifies as a probiotic only if its strains are characterised and a health benefit has been demonstrated, which is not the case for tempe, tape or dadih as ordinarily produced and sold [16]. A review of fermented foods and gastrointestinal health by Dimidi and colleagues found that the foods actually tested in randomised gastrointestinal trials were kefir, sauerkraut, natto and sourdough; there are no randomised trials of tempe for gastrointestinal health outcomes [32]. Practical considerations reinforce the caution. Tempe is almost always cooked, commonly fried, before eating, which destroys the live organisms that any probiotic effect would require, and its dominant fermenting organism is a mould rather than a characterised probiotic strain. The honest position is therefore twofold. Tempe, tape and dadih should be valued and retained in the diets of older Indonesians as affordable, protein rich and culturally accepted foods that plausibly contribute to a diverse dietary pattern of the kind associated with a healthier microbiota [19], [23]. But they should not be marketed to families, or

relied upon by clinicians, as substitutes for characterised probiotic products where a specific clinical indication exists, and research groups in Indonesia have a clear opportunity to characterise the strains in these foods and test them properly, which would convert a plausible tradition into evidence.

8. Safety in Older and Immunocompromised Adults

Probiotics have a long record of safe use in healthy people, and the trials summarised above reported few serious adverse events attributable to treatment [26], [28]. Older adults, however, are precisely the population in which the exceptions cluster. Doron and Snydman reviewed the safety literature and catalogued the theoretical and observed risks: systemic infection, including bacteraemia and, with Saccharomyces boulardii, fungaemia; deleterious metabolic effects; excessive immune stimulation in susceptible hosts; and transfer of antibiotic resistance genes [33]. Case reports of invasive infection overwhelmingly involve hosts with predisposing conditions, including immune compromise, central venous catheters, critical illness, severe underlying disease, short bowel, and damaged cardiac valves, and the reviewers advise avoiding probiotics in such patients or using them only with explicit justification and monitoring [33]. The cautionary trial signal comes from severe acute pancreatitis, in which a probiotic preparation given to critically ill patients was associated with increased mortality; although that setting is far removed from community use, it demonstrates that live microbial products are not inert in compromised hosts [33]. Frail older patients frequently carry several of the listed risk factors at once, including central lines during hospitalisation, immunosuppressive therapy, and chemotherapy, so a safety screen is a routine part of prescribing rather than an afterthought. Products should come from manufacturers that identify strains on the label and guarantee viable counts to the end of shelf life, in line with the FAO and WHO labelling recommendations [10], because misidentification and low viability have both been documented in commercial products and each converts a studied intervention into an untested one.

9. Practical Guidance for Clinicians and Caregivers

The literature supports a disciplined, indication first approach. The clinician should begin from the problem, whether antibiotic prophylaxis in a high risk patient, constipation, or recurrent respiratory infections, and then select a product whose strain or strain combination has human trial evidence for that problem, at the dose and duration tested [12], [28], [30]. Where baseline risk is low, as the PLACIDE trial and the Cochrane C. difficile analysis jointly show, expected benefit is small and may be zero, so restraint is evidence based rather than merely conservative [27], [28]. Probiotics should be framed to patients and families as adjuncts: they do not replace vaccination, antibiotic stewardship, adequate fibre and fluid intake, mobility, or review of constipating and microbiota disrupting medicines such as unnecessary acid suppression [19], [21]. Diet deserves explicit attention at the same consultation, because the NU AGE results show that a Mediterranean style pattern can shift the older gut microbiota favourably over a year, and because fibre provides the substrate on which both resident microbes and any prebiotic or synbiotic strategy depend [13], [23]. Safety screening should exclude or flag patients who are immunocompromised, critically ill, or carrying central venous catheters, and any older patient who develops fever or signs of sepsis while taking a probiotic, particularly a yeast based product, should have the product stopped and blood cultures taken with the laboratory informed of the exposure [33]. Caregivers, who usually administer these

products at home, should be told what improvement to expect and over what period, to store products as labelled, to avoid giving probiotics simultaneously with an antibiotic dose where separation is advised for bacterial products, and to report persistent diarrhoea, bloating or fever rather than simply increasing the dose. Finally, families in Indonesia should be helped to keep the categories distinct: tempe, tape and dadih are wholesome traditional foods to be enjoyed, while a therapeutic probiotic is a characterised product chosen for a defined purpose [16], [31], [32]. Documenting the strain, dose, indication and review date in the medical record, and stopping products that show no benefit at review, protects patients from indefinite, purposeless supplementation and keeps the intervention aligned with the evidence.

CONCLUSION

The published literature supports a real but bounded relationship between probiotic consumption and gut microbiota health in older adults: the ageing gut loses diversity and core health associated taxa in patterns linked to inflammaging and frailty, and selected, well characterised probiotic strains, given at tested doses, can modestly improve specific outcomes, most credibly the prevention of antibiotic associated and Clostridioides difficile associated diarrhoea in higher risk patients, the shortening of upper respiratory infections, and the relief of constipation, while large pragmatic evidence in unselected older inpatients, person specific colonisation resistance, delayed post antibiotic recovery in experimental studies, and genuine safety concerns in immunocompromised and critically ill patients all argue against indiscriminate use. In Indonesia, where older adults already form 12.00 percent of the population and are projected to reach about one fifth by 2045, the rational strategy is to protect the microbiota first through diet quality, fibre and judicious antibiotic and acid suppression use, to value traditional fermented foods such as tempe, tape and dadih as nutritious components of that diet without overstating their clinical evidence, and to deploy probiotics as targeted, strain specific, time limited adjuncts chosen by indication, screened for safety, and reviewed for benefit by clinicians and caregivers working from the published evidence.

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