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Traditional tribal diets and gut health: A review

P. Rajakumari*

Department of History, Holy Cross College (Autonomous) Tiruchirappalli, Affiliated to Bharathidasan University, Tiruchirappalli, Tamil Nadu, India; *Corresponding author

Affiliation URL:
https://www.hcctrichy.ac.in/

Author contact:
P. Rajakumari -E-mail: srisabellascc@gmail.com; isabellarajakumari@hcctrichy.ac.in

Abstract:
The human gut microbiome evolved with diverse, fiber-rich ancestral diets, whereas modern western diets promote microbial dysbiosis and metabolic diseases. Therefore, it is of interest to examine the effect traditional tribal diets on gut microbial composition and function. Diets rich in microbiota-accessible carbohydrates, wild plants, fermented foods and ancient grains enhance microbial

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diversity, short-chain fatty acid production and gut barrier integrity. Thus, data shows ancestral diets as a promising strategy for restoring gut health and preventing non-communicable diseases.

Keywords: Gut microbiome, metagenomic sequencing, traditional foodways, evolutionary discordance, short-chain fatty acids, metabolic endotoxemia

Background:

The human gastrointestinal tract hosts a complex ecosystem of trillions of microorganisms that has co-evolved over millions of years with diet. The relationship is mutualistic in that the human host provides an anaerobic nutrient-rich environment while the microbial consortia decode, ferment and bio transform complex environmental substrates that the human genome does not have the enzymatic machinery to process **[1]**. But the pace of industrialisation, urbanisation and agricultural monoculture has accelerated rapidly over the last two centuries, outpacing human genomic adaptation. This phenomenon is the basis of the evolutionary discordance hypothesis, which argues that there is a striking mismatch between our ancestral metabolic blueprint and the modern nutritional environment. The modern Western diet (WD) is rich in a cellular, fast-absorbing macronutrients, refined sugars, unhealthy fats and synthetic xenobiotics (*e.g.* emulsifiers, artificial sweeteners and chemical preservatives). This diet depletes the distal gut ecosystem by absorbing nutrients early in the upper gastrointestinal tract **[2]**. The industrial gut microbiota undergoes a significant collapse in its constitution in the absence of complex, structurally intact plant matter. This collapse is characterised by a sharp drop in alpha-diversity (the richness and evenness of species within a single ecosystem) and the extinction of important ancestral bacterial taxa with highly specialised metabolic capabilities. Disruption of this symbiotic ecosystem results in direct causality of dysbiosis. In the absence of energy the microbiota switches metabolic activity from fermentation of dietary fibres to degradation of the protective colonic mucus layer of the host. Breakdown of the mucus barrier results in direct exposure of the underlying intestinal epithelial monolayer to microbial antigens. Tight junction proteins (claudins, occludin and zonula occludens-1) undergo systematic degradation to seal the paracellular spaces **[3]**. This increased intestinal permeability allows the paracellular translocation of immunogenic bacterial moieties, mainly lipopolysaccharides (LPS), outer membrane components of Gram-negative bacteria, into the portal vein and systemic circulation. This systemic influx then leads to metabolic endotoxemia, which is defined as the chronic low-grade activation of toll-like receptor 4 (TLR4) complexes on circulating and tissue resident macrophages. This causes a systemic inflammatory cascade that promotes insulin resistance, ectopic lipid deposition and tissue fibrosis. This pathway is the common pathological background of current non-communicable diseases (NCDs) such as obesity, type 2 diabetes mellitus, non-alcoholic fatty liver disease (NAFLD), cardiovascular diseases and autoimmune diseases. To comprehend the origin of this metabolic catastrophe, we must return to the historic baseline of human nutrition: the traditional foodways of indigenous and tribal peoples **[4].** Therefore, it is of interest to examine the evidence linking traditional tribal diets with gut microbiome diversity, microbial functional metabolism and protection against diet-associated non-communicable diseases.

Traditional foodways:

The historical baseline:

Dietary pillars across global indigenous populations:

While traditional tribal foodways vary greatly, they are well adapted to local ecosystems, ranging from the arid savannas of East Africa to the dense rainforests of the Amazon and the remote highlands of Asia **[5].** Despite this geographic diversity, these foodways share some basic ecological and biochemical features that differ from modern industrial food production.

Foraging, hunting and hyper-seasonality:

Native groups depend on extensive networks of wild foraging and hunting that are timed with the rhythms of the seasons. Instead of eating a fixed set of hyper-domesticated crops all year round, like the Hadza hunter-gatherers of Tanzania, they rely on over 100 different plant and animal species every year. The seasonal availability of dietary inputs varies greatly. Wild honey and berries are plentiful in the wet, while dry season depends on deeply buried fibrous tubers and wild game **[6]**. This hyper-seasonality leads to a dynamic succession of chemical compounds and dietary substrates entering the gastrointestinal tract. This variation prevents any single microbial taxon in the niche from ever permanently dominating the system and guarantees high ecosystem plasticity and functional redundancy.

Wild and spontaneous fermentation:

Before the advent of industrial refrigeration, traditional communities preserved food, detoxified and enhanced flavour using natural fermentation. Commercial fermentation relies on isolated monocultures of particular yeasts or bacteria, whereas tribal fermentation is based on wild, spontaneous inoculations from the local environment, plant surfaces and vessels. Examples are spontaneous fermentation of wild tubers, ancient grains and native dairy products (*e.g.* fermented mare's milk or sorghum beers). These foods are good sources of live non-dairy lactic acid bacteria (LAB) comprising a wide variety of strains of *Lactobacillus, Leuconostoc* and *Pediococcus* **[7].** These wild strains are resistant to gastric acidity and bile salts and act as transient environmental regulators in the host gut.

Underutilized ancient grains and wild tubers:

Tribal farming and pastoral communities grow hardy, climate-resilient ancient grain varieties (finger millet, pearl millet, proso millet, amaranth, sorghum and wild rice varieties) which have not been subjected to modern genetic hybridisation. These grains are eaten in their natural, unrefined state, with the bran layers

and cellular structure of the grain still intact. Caloric anchors also include wild-foraged tubers (*e.g.* Eminia, Vigna and wild yam species). These storage organs contain carbohydrates in highly cross-linked polymers and starches within tough lignin-cellulose walls that require long mastication and survive transit through the upper digestive tract **[8].**

Table 1: Ancestral dietary components, their structural characteristics and gastrointestinal targets

Dietary Component	Structural/Biochemical Characteristics	Primary Gastrointestinal Target
Microbiota-Accessible Carbohydrates (MACs)	Heterogeneous polymers: Inulin, pectin, mucilages, resistant starch (Type 2 and 3), hemicellulose.	Distal colon; fermented by specialized anaerobic taxa (*Prevotella*, *Treponema*).
Wild Polyphenolic Complexes	Condensed tannins, high-molecular-weight flavonoids, phenolic acids bound to cell-wall matrices.	Reaches the large intestine intact (90%); biotransformed into bioavailable anti-inflammatory metabolites.
Cellular Nutrient Matrices	Intact plant cell walls enclosing lipids, proteins and simple carbohydrates.	Retards upper GI digestion; ensures slow, sustained delivery of nutrients to the distal ileum and colon.
Absence of Ultra-Processed Xenobiotics	Complete absence of synthetic emulsifiers, chemical stabilizers, microcrystalline cellulose and artificial sweeteners.	Preserves the structural biophysical integrity of the colonic mucus bilayer.

Structural and biochemical components of traditional diets:
Macromolecular architecture and carbohydrate diversity:
The main difference between ancestral and industrial diets is the diversity of Microbiota-Accessible Carbohydrates (MACs) **(Table 1)**. The Western diet offers highly refined cellular carbohydrates that are rapidly assimilated in the duodenum, while traditional foodways provide a complex matrix of cellular carbohydrates **[9]**. These include structural polysaccharides like xylans, arabinogalactans, pectins, beta-glucans and resistant starches (Type 2 and Type 3). There are less than twenty enzymes in the human genome that can cleave these complex carbohydrate bonds. These molecules arrive in the large intestine intact and act as an energy source for the colonic microbiome.

Wild polyphenol profiles:
Wild plants that are not sprayed with synthetic pesticides produce high levels of secondary metabolites, including polyphenols, alkaloids and terpenoids, as natural defences against UV radiation and herbivores. Commercial varieties have much lower concentrations of polyphenols than foraged fruits, wild leaves and unrefined grains. More than 90% of these dietary polyphenols are not absorbed in the upper gastrointestinal tract due to their high molecular weight and association with plant cell-wall fibres. Specialised microbial enzymes bios transform them into smaller phenolic acids (*e.g.* urolithins and phenylacetic acids) that are highly bioavailable when they reach the colon. Absorption of these metabolites results in powerful systemic anti-inflammatory, antioxidant and mitochondrial-modulating effects **[10].**

Cellular versus A cellular food:
Traditional foods are inherently cellular, with nutrients locked into intact cell walls. This delays carbohydrate digestion kinetics preventing postprandial glucose spikes and allowing a large part of the energy content of the meal to reach the distal ileum and colon **[11]**. Industrial milling and ultra-processing destroy these cellular walls however and make a cellular nutrients. These nutrients favour rapid proximal absorption, resulting in functional starvation of the distal colonic microbiota and a tendency to shift toward host mucin degradation.

The microbial bridge:
Emerging scientific evidence:

Comparative metagenomics: industrial versus indigenous ecosystems:
The recent development of high throughput metagenomic sequencing has enabled researchers to comparatively analyse the biogeography of the human gut microbiome at different levels of industrialisation. Profiling studies of non-Westernized indigenous groups, like the Hadza hunter-gatherers of East Africa, the isolated Yanomami Amerindians of the Amazon basin, the Matses of Peru and rural tribal communities in Asia, have revealed structural patterns that do not fit Western-centric definitions of a "healthy" microbiome **[12].** Alpha diversity declines sharply as populations shift from nomadic foraging to rural agriculture and finally to urbanised, industrial lifestyles. This loss is characterised by gradual extinction of entire bacterial lineages over generations **(Figure 1 and 2)**. This process is exacerbated by widespread use of clinical antibiotics, hygienic sanitisation and MAC-depleted diets **[13].**

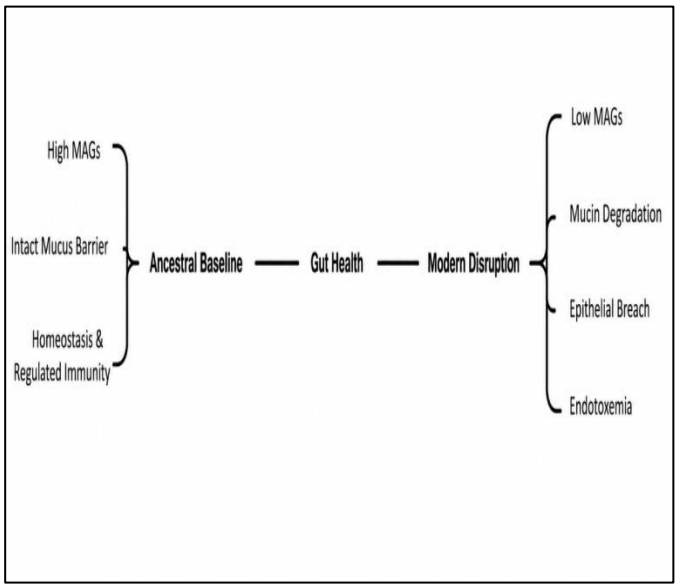

Figure 1: Evolutionary model of gut health showing the transition from ancestral high-MAC diets that maintain mucus integrity and immune homeostasis to modern low-MAC diets that promote mucin degradation, epithelial barrier disruption and endotoxemia**.**

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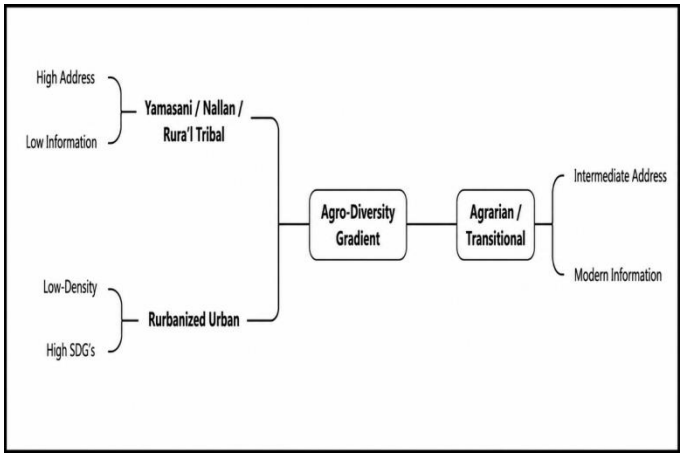

Figure 2: Alpha diversity gradient and health outcomes. The most remarkable result is the sharp gradient of the α-diversity. Indigenous populations display the highest microbial diversity, gene richness and taxonomic evenness ever documented in humans

Deep taxonomic and functional divergence:

The *prevotella-treponema* axis:

Taxonomically, the Western gut is dominated by members of the genus *Bacteroides* and a number of *Firmicutes* that are adapted to a diet high in simple sugars, animal proteins and saturated fats. In contrast, the indigenous and tribal gut microbiome is consistently dominated by the *Prevotella-Treponema* axis. The genus *Prevotella* (*P. copri* in particular) and several non-pathogenic, symbiotic species of *Treponema* (*phylum Spirochaetota*) are core dominant taxa in diverse, geographically isolated indigenous populations. These bacteria possess large repertoires of Carbohydrate-Active Enzymes (CAZymes) including specialised glycoside hydrolases, polysaccharide lyases and carbohydrate esterases organised within coordinated polysaccharide utilisation loci (PULs). These genetic toolkits allow them to break down the very cross-linked, insoluble cellulose and xylan structures found in wild tubers and ancient unrefined grains **[14].**

Functional redundancy and seasonal plasticity:

Metagenomic functional profiling suggests that the native gut microbiome encodes a more diverse set of metabolic pathways than the industrialised ones. This is mirrored in high functional redundancy, where different bacterial species perform the same metabolic tasks. This architecture creates a stable ecosystem: if environmental pressures cut into one species, other species will fill in to keep the metabolic output going. Moreover, longitudinal tracking of the Hadza has showed remarkable seasonal plasticity. During the wet season when foraging shifts to honey and berries, the abundance of fiber-degrading *Prevotella* declines and simple-sugar fermenters increase. Importantly, however, as the dry season comes again and fibrous tubers return as the dietary anchor, the *Prevotella* population bounces back fully. This cyclic plasticity is mostly absent in Western populations, where microbiomes remain relatively static due to the year-round consumption of ultra-processed foods **[15].**

Biochemical outputs and functional epigenetics:

The metabolic interplay between traditional dietary substrates and the indigenous microbiome produces a unique biochemical signature that influences host physiology. Dietary inulin, pectin, and celluloses (microbiota-accessible carbohydrates) are fermented by gut microbes through the activity of carbohydrate-active enzymes (CAZymes). The fermentation process leads to the formation of short-chain fatty acids (SCFAs), which are primarily acetate, propionate and butyrate, and play a significant role in maintaining host metabolic and immune homeostasis. Acetate and propionate are absorbed and transported to the liver where they regulate hepatic gluconeogenesis and lipogenesis and contribute to glucose and lipid metabolism. Butyrate, on the other hand, is the primary energy source for colonocytes and helps to maintain the integrity of the intestinal epithelium. Moreover, butyrate functions as a histone deacetylase (HDAC) inhibitor and thereby causes epigenetic regulation to induce differentiation and expansion of $FoxP3^+$ regulatory T (T_{reg}) cells, leading to immune tolerance and lessening of inflammation of the intestine.

Short-Chain Fatty Acid (SCFA) dynamics:

The primary products of anaerobic microbial fermentation of MACs are short-chain fatty acids, specifically acetate, propionate and butytrate. Metagenomic analyses show that the absolute and relative concentrations of these metabolites are significantly higher in indigenous populations than in people consuming a Western diet **[1].** Butyrate is the main energy substrate for colonic epithelial cells (colonocytes). Oxidation of it maintains local hypoxia within the colonic lumen (<1% oxygen tension). This anaerobic state is essential to maintain the obligate anaerobic structure of a healthy gut and prevent the overgrowth of facultative anaerobic pathogens such as *Enterobacteriaceae* **[16, 2].** Propionate is carried to the liver via the portal vein where it is used as a substrate for gluconeogenesis and interacts with specific receptors to down-regulate hepatic de novo lipogenesis and cholesterol synthesis.

Epigenetic signatures and immune homeostasis:

Apart from energetic substrates SCFAs are also signalling molecules that directly modulate the host immune system. Intestinal epithelial cells, dendritic cells and regulatory T-cells (T_{reg}) highly express the G-protein coupled receptors GPR 41 or FFA3, GPR 43 or FFA2 and GPR 109A. Moreover, both butyrate and propionate are powerful endogenous inhibitors of histone deacetylase (HDAC). These molecules inhibit HDACs in the promoter region of FoxP3 gene in naive CD4+ T-cells and induce hyperacetylation of core histones. This epigenetic modification upregulates FoxP3 expression, which leads to the differentiation of naive T-cells into immunosuppressive, anti-inflammatory $FoxP3^+$regulatory T-cells (T_{reg}). These cells secrete high levels of interleukin-10 (IL-10) and transforming growth factor-beta (TGF-β) that inhibit systemic inflammatory pathways, down-regulate

auto-reactive immune responses and maintain immune homeostasis **[17].**

Health implications and modern applications:

The "disappearing microbiota" hypothesis and chronic disease etiology:

The 'disappearing microbiota' hypothesis concerns the progressive loss of ancestral microbial diversity in industrialised countries. This model suggests that the rapid decline of our symbiotic microbial partners has deprived the modern human immune system of the regulatory signals it needs for appropriate development. If a diet is deficient in MACs, the gut microbiota must adapt to survive **[18]**. In this energy-starved state, specialised mucin-degrading bacteria (*e.g.*, *Akkermansia muciniphila*, *Bacteroides caccae*) switch their metabolism to consume the host colonic mucus layer, using the O-linked glycans of the mucus matrix as a surrogate carbon source. When the substrate deprivation is chronic, this degradation exceeds the host mucus production resulting in physical thinning and structural failure of the protective inner mucus layer. MAC Starvation → Mucin Glycan Consumption → Mucus Layer Thinning → Epithelial Contact → Tight Junction Breakdown → LPS Translocation → TLR4 Activation → Metabolic Endotoxemia. This structural breakdown permits luminal bacteria and their metabolic products to come into direct contact with the underlying colonic epithelial monolayer. This interaction leads to the disassembly of tight junction proteins, which creates paracellular pathways for systemic translocation of Gram-negative bacterial lipopolysaccharide (LPS)**.** Upon release into circulation, LPS is bound by soluble lipopolysaccharide-binding protein (LBP) and presented to the TLR4/MD-2 complex on the surface of macrophages, monocytes and dendritic cells. This binding induces an intracellular signalling cascade via MyD88, leading to the translocation of Nuclear Factor Kappa B (NF-$_{\kappa}$B) to the nucleus. This pathway leads to transcription and systemic release of pro-inflammatory cytokines including Tumour Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6) and Interleukin-1 beta (IL-1β). This chronic, low-grade inflammatory state, termed metabolic endotoxemia, perturbs insulin receptor signalling via activation of c-Jun N-terminal kinase (JNK) and contributes to the pathogenesis of insulin resistance, metabolic syndrome, atherosclerosis and autoimmune disorders **[19].**

Translating ancestral insights into modern clinical interventions:

The biochemical and structural principles of traditional tribal foodways serve as a blueprint for the development of clinical and dietary interventions to address modern dysbiosis.

Precision macroglycan architecture and prebiotic engineering:

Current clinical approaches often use single-substrate prebiotic supplements in isolation, such as synthetic inulin or fructooligosaccharides (FOS). These simple structures are however rapidly fermented in the proximal colon, resulting in gastrointestinal discomfort (gas and bloating) and not reaching the distal colon where dysbiosis and inflammation are often the most pronounced. Engineering precision macroglycan matrices is a form of modern functional food design that may mimic traditional foodways. These are complex, heterogenous carbohydrate matrices from underutilised ancient grains (*e.g.* finger millet) and structural plant fibres. In these matrices, cross-linking of different fibres slows fermentation kinetics to ensure SCFA are released over the entire length of the colon, thus favouring a broader diversity of microbial species **[20].**

Ecologically complex functional food design:

Instead of depending on industrial dairy-derived probiotics, functional foods can include wild, non-dairy fermentative consortia isolated from traditional tribal foodways. Such wild-type ferments are usually found in spontaneously fermented grains and tubers, with robust strains of lactic acid bacteria generating stable postbiotics such as bacteriocins, hydrogen peroxide and organic acids. These matrices, in combination with wild polyphenol extracts, act as synbiotics, protecting the living microorganisms during digestion and enhancing their therapeutic effect when reaching the colon **[21]**.

Next-generation probiotics (NGPs):

Comparisons in metagenomics have sped up the discovery of Next-Generation Probiotics (NGPs), specialised commensal organisms that are very abundant in healthy, non-Western populations and largely absent from the industrial gut. Clinical trial candidates include *Prevotella copri* clades, selected for their ability to restore plant-polysaccharide degradation and to increase propionate production; *Faecalibacterium prausnitzii* strains, known for their ability to produce high amounts of butyrate and to activate directly anti-inflammatory pathways; and *Christensenella minuta*, associated with lean phenotypes in epidemiological and animal model systems, potentially useful in metabolic regulation. Delivery of these live biotherapeutic products in protective, structurally complex plant matrices allows modern clinical science to attempt to re-establish ancestral metabolic pathways within the industrialised human gut **[22]**.

Conclusion:

Traditional food practices, supported by metagenomic and immunological evidence, show that traditional diets are fundamental to human–microbiome co-evolution. Indigenous populations exhibit greater microbial diversity, metabolic flexibility and short-chain fatty acid production, whereas industrialized diets disrupt gut barrier integrity through reduced microbiota-accessible carbohydrates. This evolutionary mismatch promotes metabolic endotoxemia, chronic inflammation and non-communicable diseases.

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