Ayurveda & Microbiome
Typing, Re-setting & Diet

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PLOS ONE

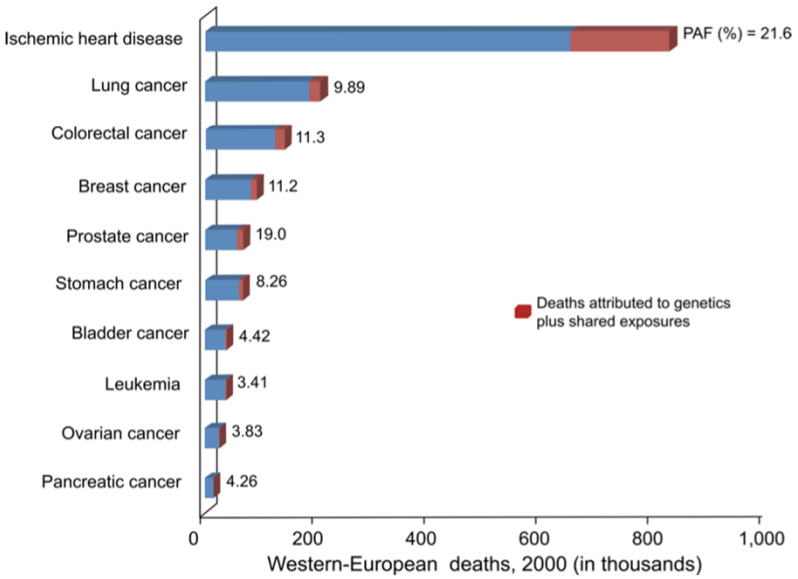

Fig 2. Numbers of Western-European deaths in 2000 estimated for ischemic heart disease and nine cancer types (1.53 million total deaths from these causes). The contributions attributed to genetics plus shared exposures are based on the population attributable fractions (PAFs) estimated from Western European monozygotic twins (Table 2).

RESEARCH ARTICLE

Genetic Factors Are Not the Major Causes of Chronic Diseases

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Commensal Host-Bacterial Relationships in the Gut

Lora V. Hooper and Jeffrey I. Gordon*

One potential outcome of the adaptive coevolution of humans and bacteria is the development of commensal relationships, where neither partner is harmed, or symbiotic relationships, where unique metabolic traits or other benefits are provided. Our gastrointestinal tract is colonized by a vast community of symbionts and commensals that have important effects on immune function, nutrient processing, and a broad range of other host activities. The current genomic revolution offers an unprecedented opportunity to identify the molecular foundations of these relationships so that we can understand how they contribute to our normal physiology and how they can be exploited to develop new therapeutic strategies.

The first draft of our complete DNA sequence represents a historic event in our quest for self-knowledge (*1*, *2*). Knowing our genotype highlights the need to understand how environmental factors interact with our genetic traits to influence health and predispose us to illness. In the midst of the current revolution in comparative and functional genomics, it is therefore appropriate to consider another form of self-knowledge: the contributions of our microbial partners to our biology. From birth to death, we are colonized by a vast, complex, and dynamic consortium of microorganisms that may outnumber our somatic and germ cells (*3*). The Nobel laureate Joshua Lederberg has suggested using the term "microbiome" to describe the collective genome of our indigenous microbes (microflora), the idea being that a comprehensive genetic view of *Homo sapiens* as a life-form should include the genes in our microbiome (*4*).

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Bacteria have inhabited Earth for at least 2.5 billion years (*5*). As a result, our predecessors have had to adapt to a biosphere dominated by microbes. However, we have minimal knowledge of how coevolution with indigenous microorganisms has shaped our genome and microbiome, as well as our physiology and postnatal development. For example, the human genome encodes 223 proteins with significant homology to bacterial but not eukaryotic proteins, suggesting that they were acquired through horizontal transfer of bacterial genes (*1*). Unfortunately, the components of our microbiome remain poorly defined. Like most complex ecosystems, enumerating membership in the various microbial societies that reside on our body surfaces has been hindered by the fact that most societal members cannot be cultured ex vivo. Moreover, most microbial genome-sequencing projects have focused on pathogens. Those that have embraced nonpathogens have turned to Archaea to understand the evolutionary diversification of protocytes and eukaryotes or to extremophiles to examine their adaptations to harsh environments and their potential for performing commercially applicable chemistry (*6*).

Interactions between bacteria and their hosts can be viewed in terms of a continuum between symbiosis, commensalism, and pathogenicity, with symbiosis and commensalism grouped under the general heading of mutualism (Fig. 1). "Symbiosis" refers to a relationship between two different species where at least one partner benefits without harming the other and is typically centered on metabolic capabilities that allow either or both partners to exploit an otherwise unavailable or poorly utilizable nutrient foundation (*7*, *8*). The term "commensal" comes from the medieval Latin "commensalis," meaning "at table together," and generally refers to partners that coexist without detriment but without obvious benefit. A pathogenic relationship results in damage to the host. Symbiosis and commensalism have been viewed as potential outcomes of a dynamic "arms race" (*9*) initiated when a pathogen encounters a vulnerable host. In this race, a change in one combatant is matched by an adaptive response in the other. In some settings, the arms race evolves toward attenuation of virulence and peaceful coexistence, with or without frank codependence (symbiosis). In other circumstances, the pathogenic relationship is sustained by the development of effective countermeasures that bypass the host's innate or adaptive defenses (Fig. 1). Ewald has coined the term "evolutionary epidemiology" to underscore how a comprehensive analysis of disease prevalence and spread must include the set of adaptive responses of host and pathogen to one another and their outside environment over time (*10*). He and others have emphasized that the concept of obligate evolution of parasites (pathogens) to benignness should be rejected on the

Microbiome Research
What has it revealed?

- the previously unappreciated bacterial diversity
- the ‘unanticipated’ variability between individuals
- numerous correlations between gut community composition and various host states

REVIEW

The gut microbiota — masters of ho
development and physiology

Felix Sommer[1,2] and Fredrik Bäckhed[1,2,3]

Abstract | Establishing and maintaining beneficial interactions between the host and
associated microbiota are key requirements for host health. Although the gut microb
previously been studied in the context of inflammatory diseases, it has recently beco
that this microbial community has a beneficial role during normal homeostasis, modu
the host's immune system as well as influencing host development and physiology, in
organ development and morphogenesis, and host metabolism. The underlying molec
mechanisms of host–microorganism interactions remain largely unknown, but recent
have begun to identify the key signalling pathways of the cross-species homeostatic
regulation between the gut microbiota and its host.

Microbiota
The sum of all microorganisms (including bacteria, archaea, eukaryotes and viruses) that reside in and/or on a host or a specified part of a host, such as the gastrointestinal tract.

All higher animals are associated with a diverse microbial community that is composed mainly of bacteria but also includes archea, viruses, fungi and protozoa. Microorganisms cover essentially all host mucosal surfaces, but most reside within the gastrointestinal tract. Studies had traditionally focused on examining the role of the microbiota during human disease, for example in inflammatory diseases such as colitis. However, in the past decade, the field of microbiota research has exploded, resulting in the publication of a plethora of reports that describe both the individual members of our intestinal microbiota and their wide-ranging impact on host physiology. Thus, the traditional anthropocentric view of the gut microbiota as pathogenic and solely an immunological threat has been substituted with an appreciation of its mainly beneficial influence on human health.

The ‘normal’ gut microbiota is dominated by anaerobic bacteria, which outnumber aerobic and facultative anaerobic bacteria by 100- to 1,000-fold[1]. In total, the intestinal microbiota consists of approximately 500–1,000 species that, interestingly, belong to only a few of the known bacterial phyla[2,3]. By far the most abundant phyla in the human gut are Firmicutes and Bacteriodetes, but other species present are members of the phyla Proteobacteria, Verrumicrobia, Actinobacteria, Fusobacteria and Cyanobacteria[2,4]. Two gradients of microbial distribution can be found in the gastrointestinal tract. First, microbial density increases both from the proximal to the distal gut (the stomach contains 10^3 microbial cells per gram of content, the duodenum 10^3 cells per gram, the jejunum 10^4 cells per gram, the ileum 10^7 cells per gram and the c
10^{11} cells per gram) and along the tissue-l
(with few bacteria adhering to the tissue or
a large number being present in the lumen)
bacterial diversity increases in the same axes a
as microbial density[4]. Many bacterial species
in the lumen, whereas fewer, but well-adapt
including several proteobacteria and *Ak*
mucinphila, adhere and reside within the m
close to the tissue[5,6]. Colonization of the host
ing birth, and the composition of the microbi
throughout host development (BOX 1).

In the adult intestine, a total of about
rial cells are present, which is ten times the
human cells in the body[7]. Their combinat
(known as the microbiome) contain more t
lion genes, thus outnumbering the host's ge
tial by two orders of magnitude[3,8]. This large
gene products provides a diverse range of bi
and metabolic activities to complement ho
ogy. In fact, the metabolic capacity of the gut
equals that of the liver, and the intestinal mic
therefore be considered as an additional org
bacteria are essential for several aspects of h
For example, they facilitate the metabolism o
indigestible polysaccharides and produce ess
mins; they are required for the development
entiation of the host's intestinal epithelium a
system; they confer protection against in
opportunistic pathogens[9]; and they have a
maintaining tissue homeostasis. Recent st
also revealed that the human microbiota

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Microbiome Research
3 Questions it Raises

- How can one categorize in a consistent way, the immense variation in the microbiome of a population?
- How can one restore and reset a disturbed microflora?
- What food sources are available to contribute to healthy microbiome?

An Alternate Viewpoint

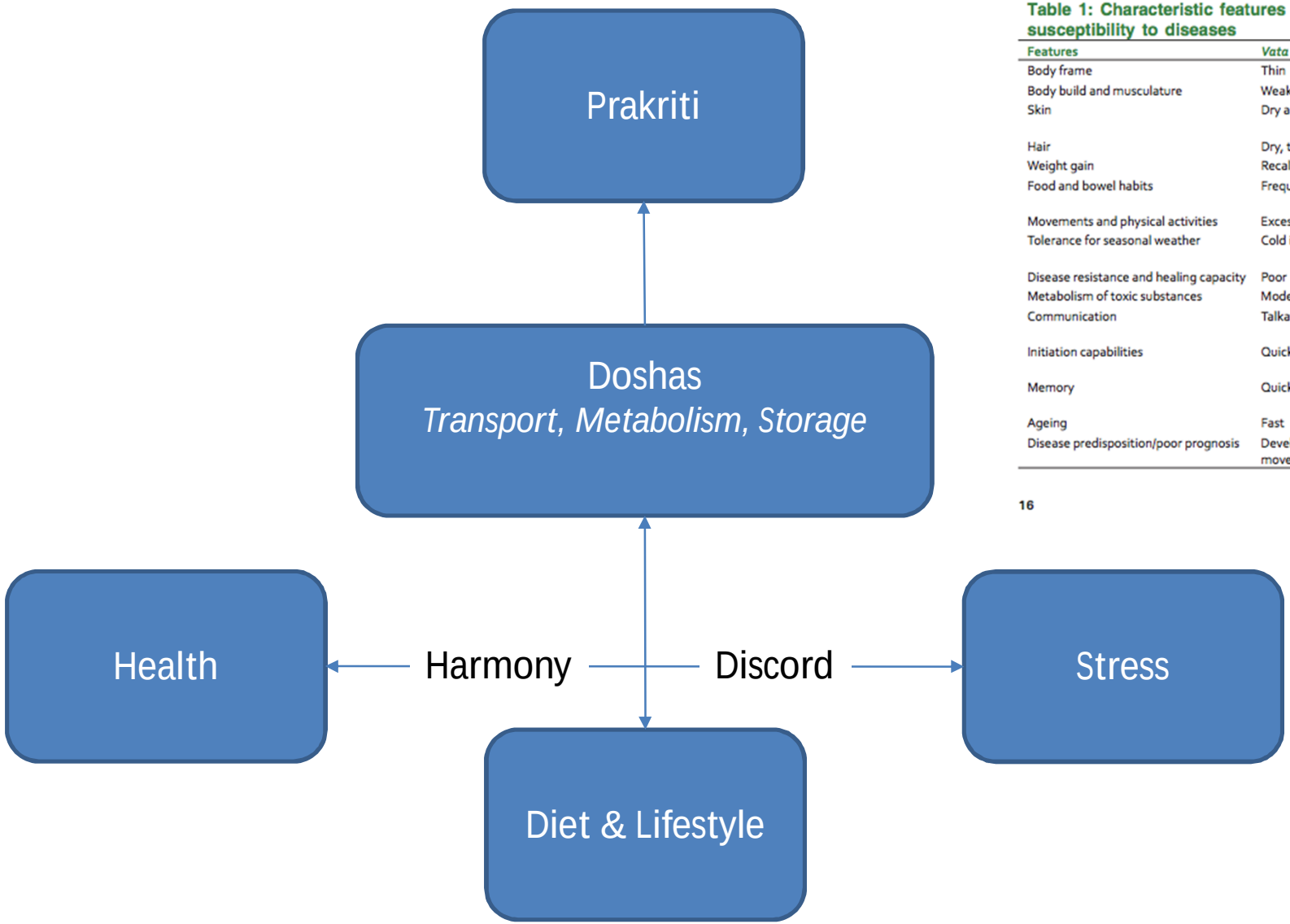
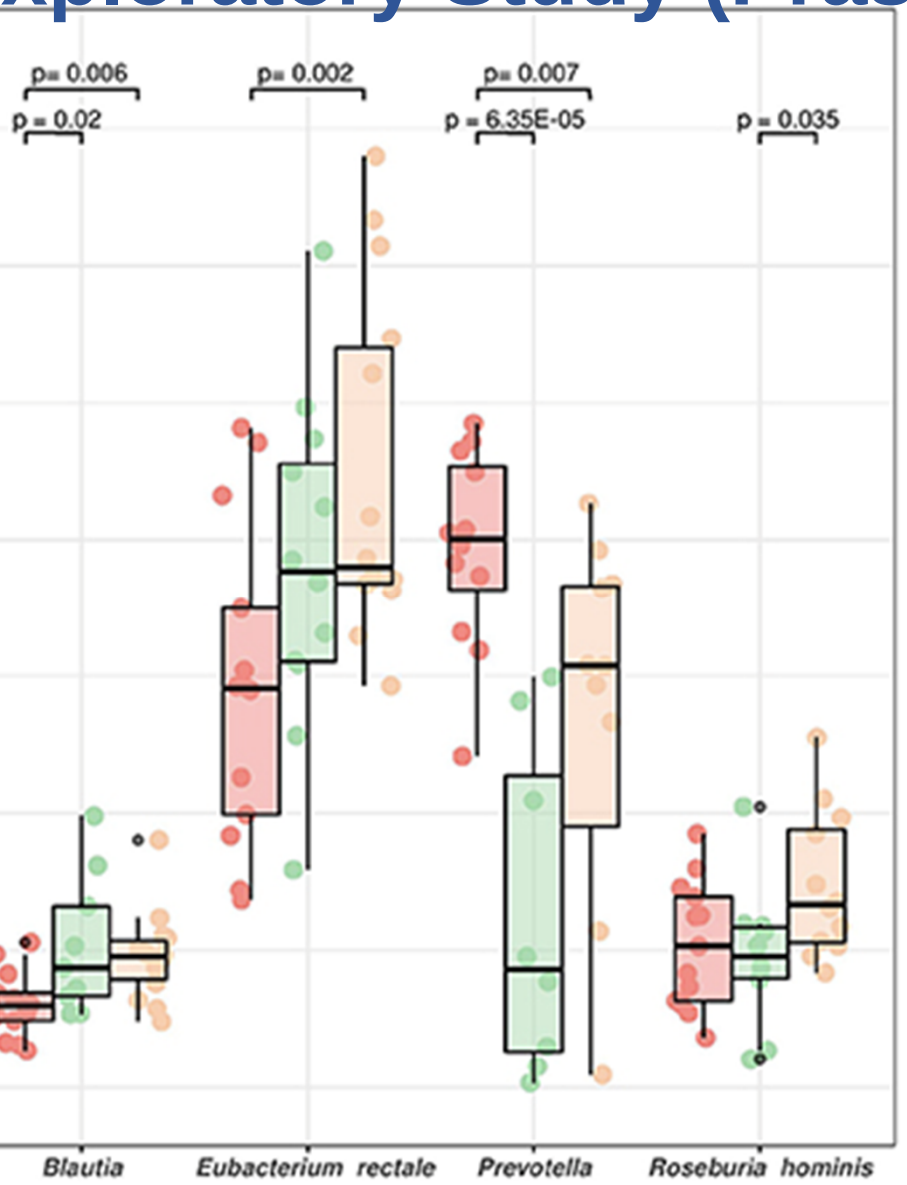


Table 1: Characteristic features of the three extreme *prakriti* types: *Vata, pitta,* and *kapha* and t susceptibility to diseases

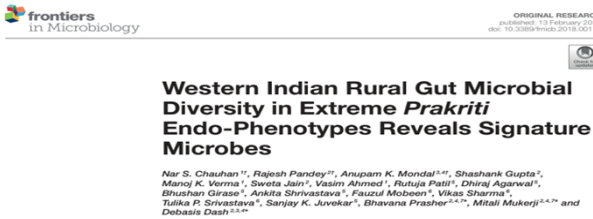
Features	*Vata*	*Pitta*	*Kapha*
Body frame	Thin	Medium	Broad
Body build and musculature	Weakly developed	Moderate	Well-developed
Skin	Dry and cracked	Soft, thin, with tendency for moles, acne and freckles	Smooth and firm, c complexion
Hair	Dry, thin, prone to breaks	Thin, oily, early greying	Thick, smooth, and
Weight gain	Recalcitrant	Fluctuating	Tendency to obesit
Food and bowel habits	Frequent, variable, and irregular	Higher capacity for food and water consumption	Low digestive capa and stable food hal
Movements and physical activities	Excessive and brisk	Moderate	Less mobile and slo
Tolerance for seasonal weather	Cold intolerant	Heat intolerant	Tolerant to both he and cold
Disease resistance and healing capacity	Poor	Good	Excellent
Metabolism of toxic substances	Moderate	Quick	Poor
Communication	Talkative	Sharp, incisive communication with analytical abilities	Less vocal with goo communication ski
Initiation capabilities	Quick, responsive, and enthusiastic	Moderate, upon conviction and understanding	Slow to initiate new things
Memory	Quick at grasping but poor retention	Moderate grasping and retention	Slow grasping but retention
Ageing	Fast	Moderate	Slow
Disease predisposition/poor prognosis	Developmental, neurological, dementia, movement and speech disorders, arrhythmias	Ulcer, bleeding disorders, skin diseases	Obesity, diabetes, atherosclerotic cor

Prakriti & Microbiome

xploratory Study (Prasher, Mukerji, Dash eta al, IGIB)



Prakriti	Microbiome Features	Phenotype Feature
Vata	B. Vulgaris Oscillibacter valericegenes Eubacterium rectale R. hominis	Irregular, unpredictable digestion lower immune response
Pitta	Enrichment of butyrate producing microbes	In general good digestion & stron metabolism. But prone to inflammation (Juyal et al. 2012),
Kapha	Prevotella and P. copri	Obesity, susceptibility to type 2 diabetes, artherosclerosis (Prash al. 2008, Govindraj et al. 2015, Doddoli et al. 2016)


frontiers in Microbiology

ORIGINAL RESEARCH

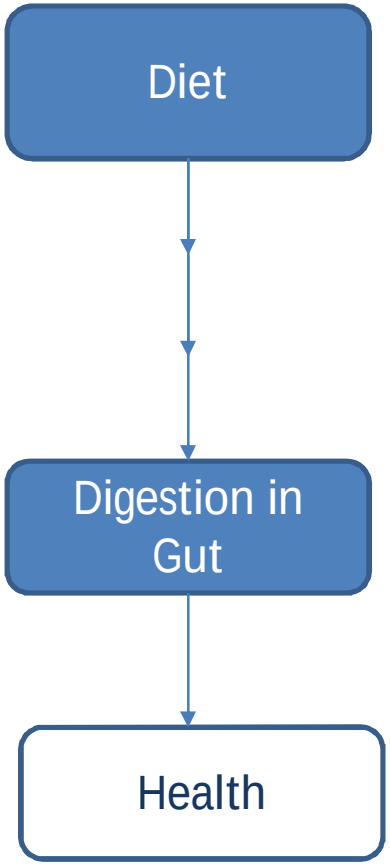
Western Indian Rural Gut Microbial Diversity in Extreme *Prakriti* Endo-Phenotypes Reveals Signature Microbes

Resetting the Microbiome

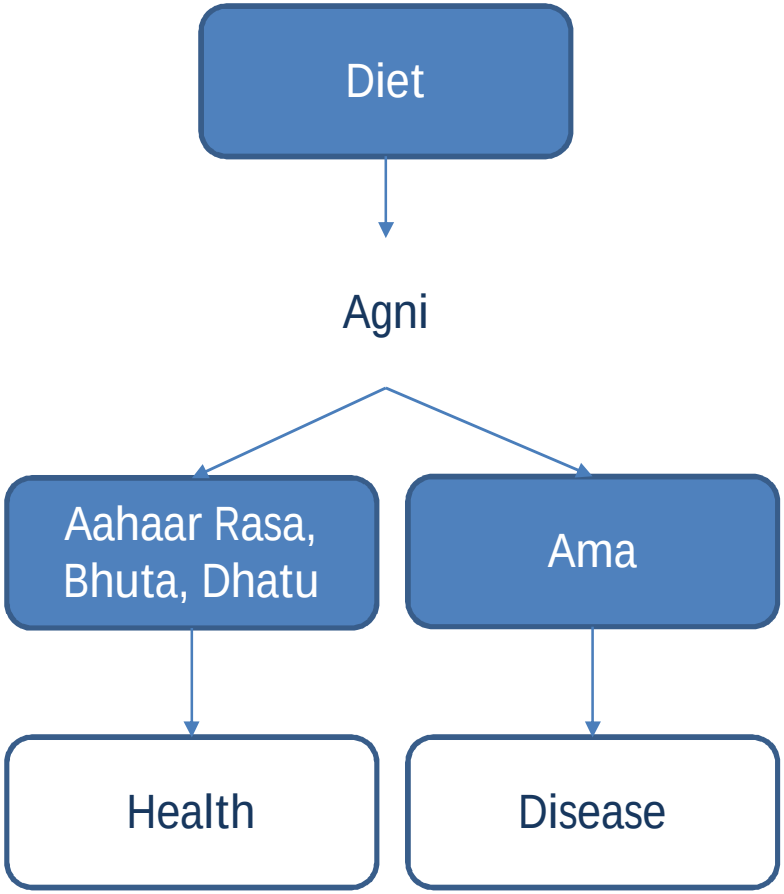
The Holistic Approach

Prebiotics

Probiotics



Fecal Transplant



Agni vardhak

Fermented mil
products
Takra arishta
Jeerakady asav

Panchakarma

Dairy-based Products

Role in Treating Diarrhoeal Diseases

INDICATION	PREPARATION
Diarrhea and Dysentery	• Light food consisting of buttermilk is given when there i hunger after diarrhea • In diarrhea with *kapha* dominance (passing stools with mucous and with continuous pain), drugs like *Woodfor fruticosa*, *Symplocos racemosa*, *Zingiber officinalis* adde with buttermilk is given. • Buttermilk with *Plumbago zeylanica* is given during abdominal discomfort . • Pomegranate juice with *Holarrhena antidysenterica* alo with buttermilk cures diarrhea.
Chronic diarrhea: Lactose or gluten intolerances/ Crohn's disease	• Buttermilk is taken as post meal drink • Buttermilk is wholesome in chronic diarrhoea and othe abdominal disorders • Buttermilk is appetizing • After taking meal prepared from horse gram, buttermilk given • Buttermilk with *Plumbago zeylanica* in chronic indigesti
Others	• Astringency of butter milk for blood mixed stools • For electrolyte loss • Ensures improved metabolism for those suffering from overall debility

But What is Buttermilk

An Innovators Dream!

8-sources of milk

↓

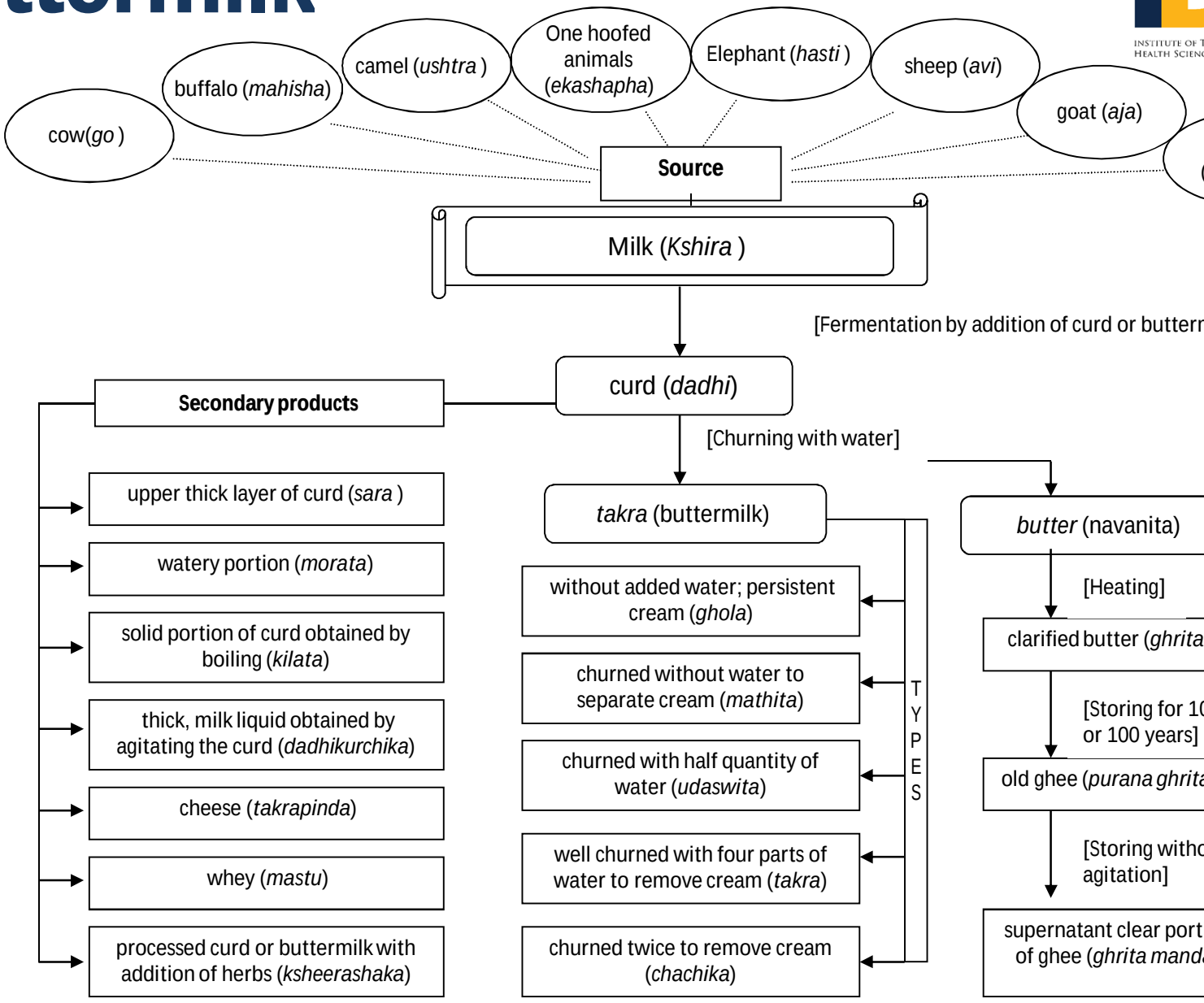
5 degrees of fermentation

↓

5 process variations

↓

200 product combinations



Milk and fermented milk products mentioned in Ayurveda (Kukkupuni et al., 2015)

Enhancing Agni

Dipaniya, Agni Vardhaka (Enhancers of digestion and metabolism)
Haritaki (*Terminalia chebula*)- fruits
Pippali (*Piper longum*) - fruits
Vibhitaki (*Terminalia bellerica*)- fruits
Kakamachi (*Solanum nigrum*)- fruits and leaves
Jiraka (*Cuminum cyminum*)- fruits
Dadima (*Punica granatum*)- fruits
Agnimantha (*Clerodendrum phlomides* L.F. or *Premna integrifolia* L.)- root/ stem bark

Resetting the Microbiome

The 6 Rasa Diet

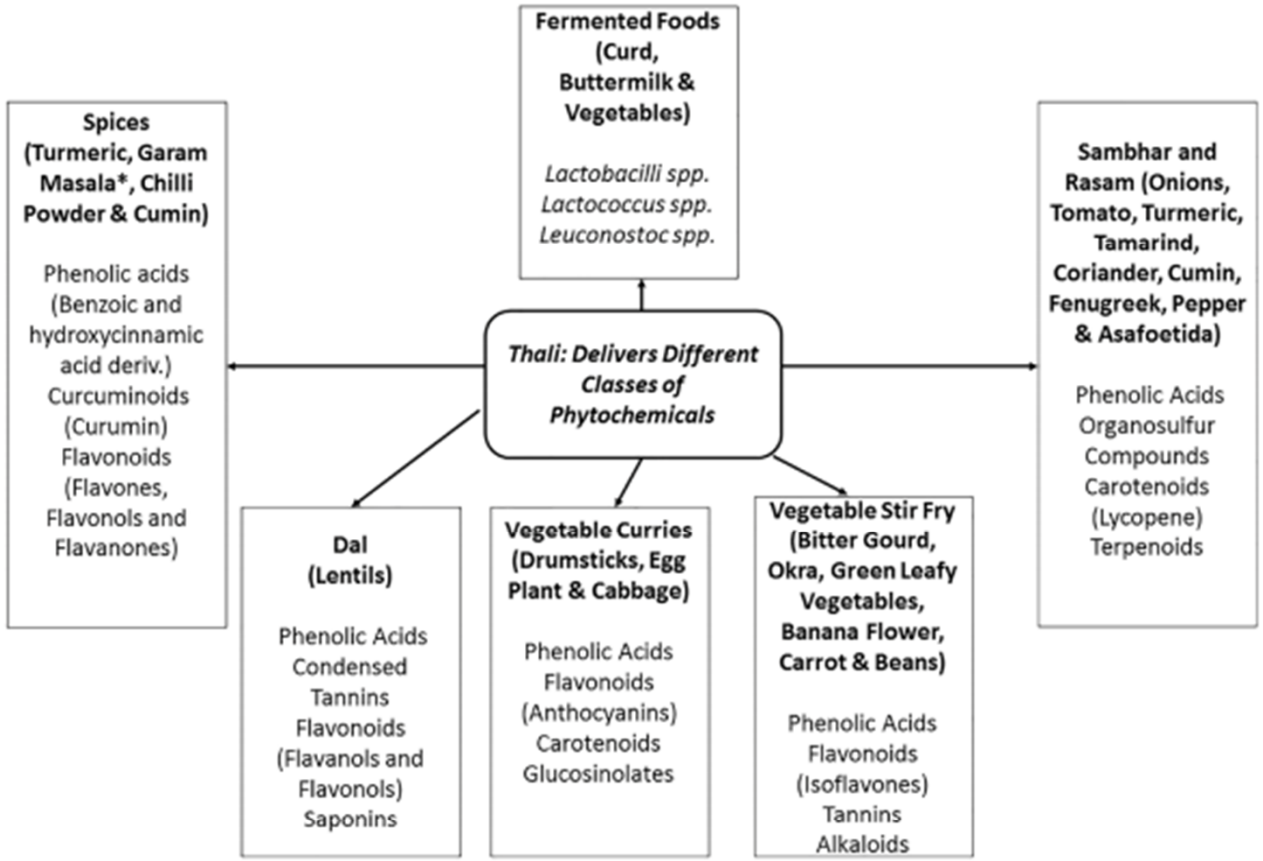


Figure 1. The *thali* diet promotes gut bacterial diversity by delivering probiotics, prebiotics, and different classes of phytochemicals from fermented foods, *dal* and vegetables and spices, respectively. Indeed, *sambar*, a component of the *thali* diet suppressed chemically-induced colon carcinogenesis *in vivo* [67]. *Black and white peppercorns, cloves, cinnamon, mace (part of nutmeg), black and green cardamom pods, bay leaf, cumin, and coriander.

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PERSPECTIVES

Ancient *Thali* Diet: Gut Microbiota, Imn and Health

Kaitlyn Shondelmyer[a], Rob Knight[b,c], Anusha Sanivarapu[d], Shuji Ogino[e,f,g], and Jai Vanamala[a,h,i,*]

We Are What We Eat & Drink

दीपो भक्षयते ध्वान्तं कज्जलं च प्रसूयते |
यदन्नं भक्षयेन्नित्यं जायते तादृशी प्रजा ||

dIpo bhakShayate dhvAntam kajjalam cha prasUyate |
yadannam bhakShayennityam jAyate tAdRishI prajA ||

Lamp eats darkness and produces [black] soot!
What food (quality) [one] eats daily, so will [one] produce.

All Diseases Begin in the Gut

Thank You