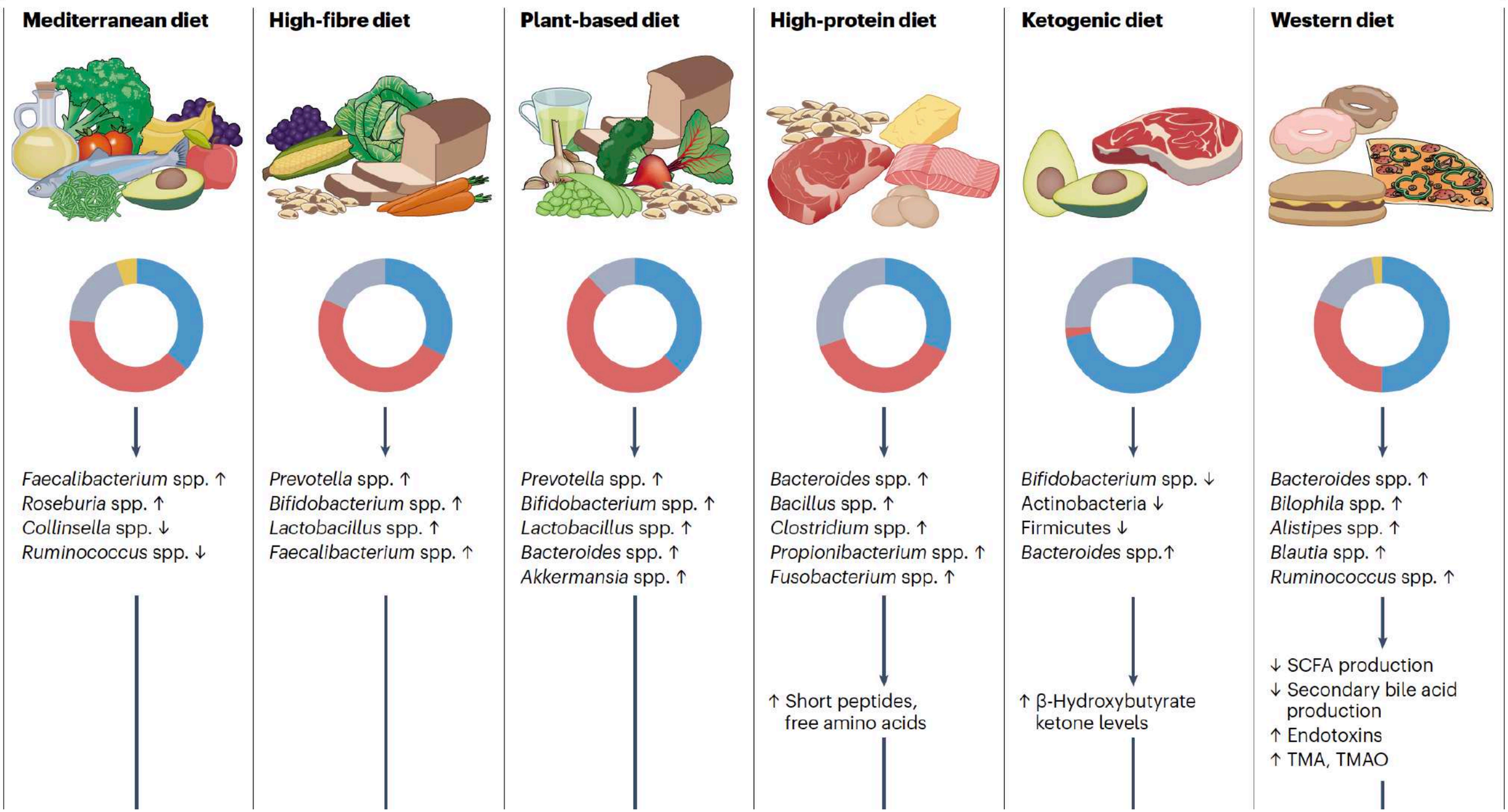
L07c

Focus on diet

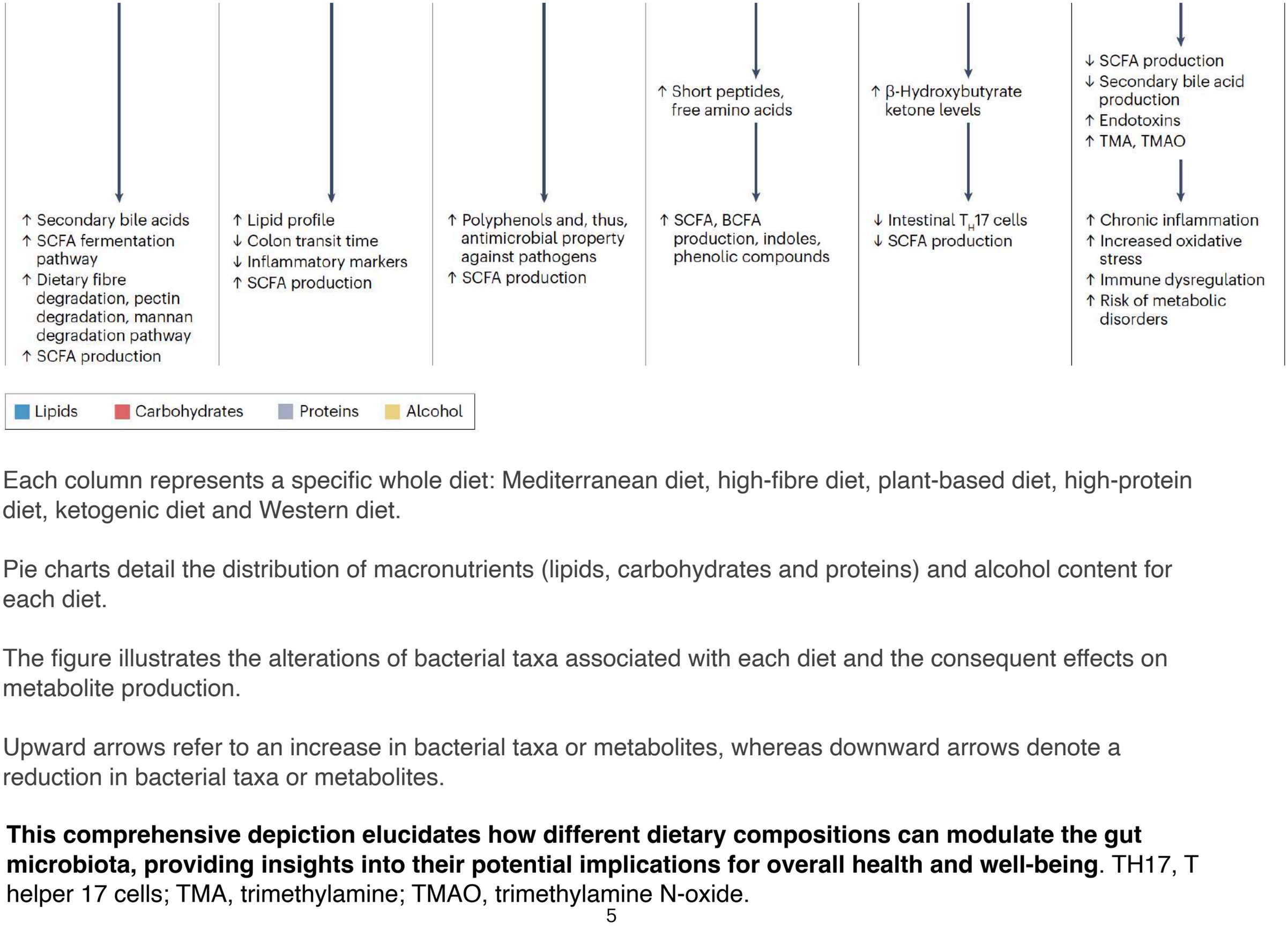
From the beginning to the end

Diet is generally recognized as a key determinant of gut microbiome variations

Interplay between diet and the gut microbiome



Ross et al., 2024



Each column represents a specific whole diet: Mediterranean diet, high-fibre diet, plant-based diet, high-protein diet, ketogenic diet and Western diet.

Pie charts detail the distribution of macronutrients (lipids, carbohydrates and proteins) and alcohol content for each diet.

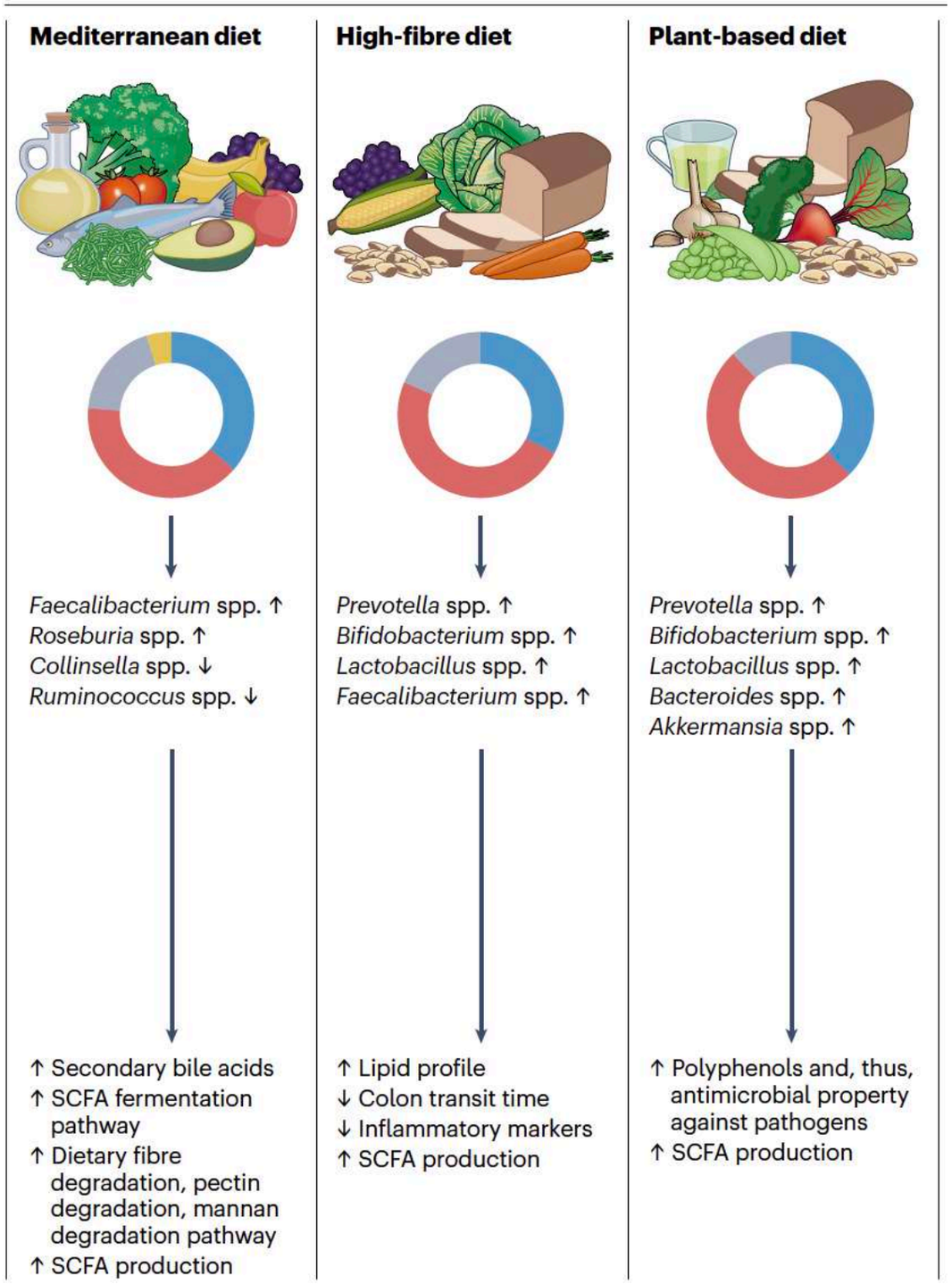
The figure illustrates the alterations of bacterial taxa associated with each diet and the consequent effects on metabolite production.

Upward arrows refer to an increase in bacterial taxa or metabolites, whereas downward arrows denote a reduction in bacterial taxa or metabolites.

This comprehensive depiction elucidates how different dietary compositions can modulate the gut microbiota, providing insights into their potential implications for overall health and well-being. TH17, T helper 17 cells; TMA, trimethylamine; TMAO, trimethylamine N-oxide.

Ross et al., 2024

We are what we eat, I



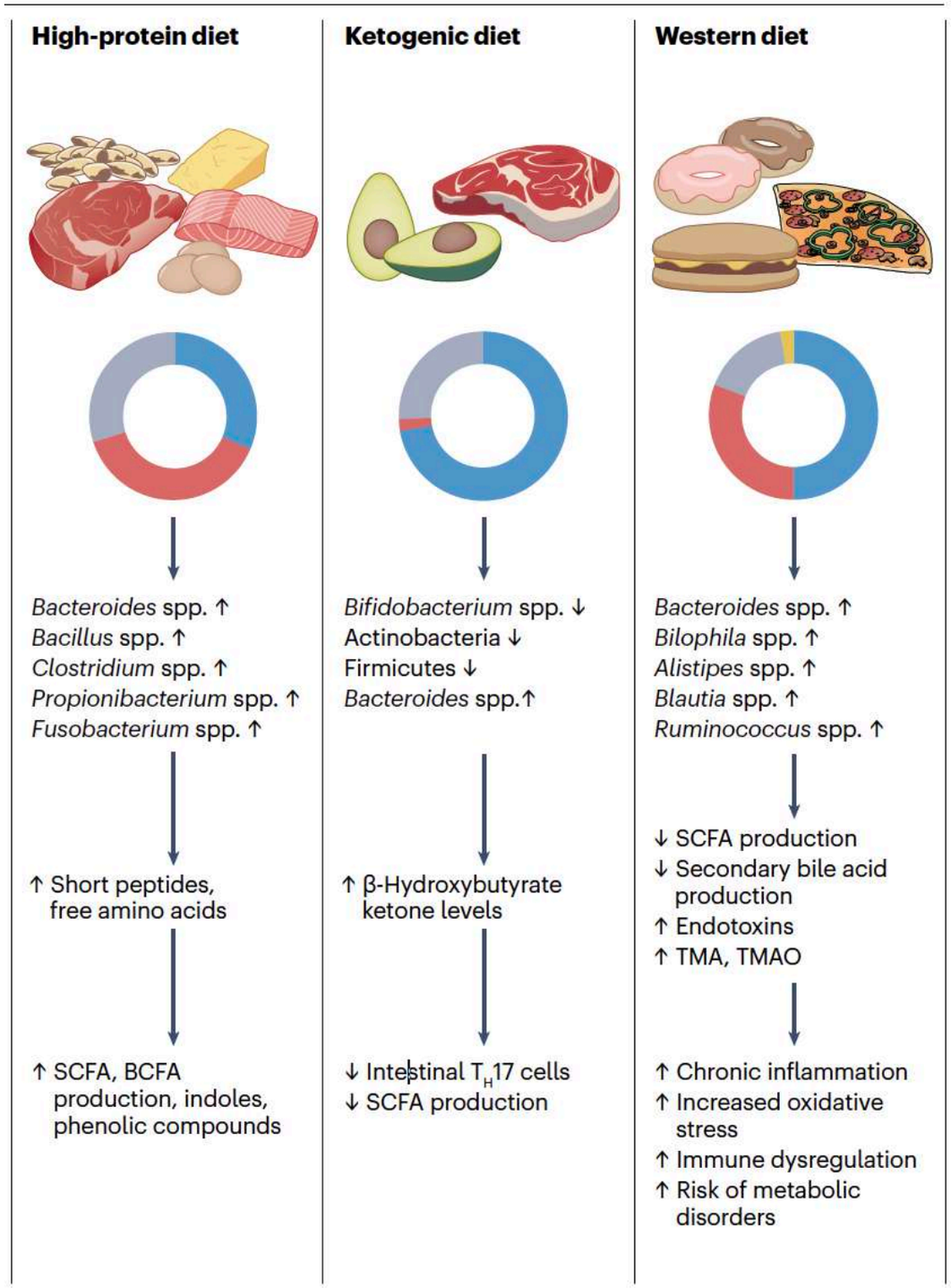
Ross et al., 2024

The Mediterranean diet is associated with increased *Faecalibacterium* spp. and is associated with **short-chain fatty acids (SCFAs)** and production of **anti-inflammatory molecules**

The high-fibre diet is associated with enriched *Prevotella* and *Faecalibacterium* species, which are associated with **enriched SCFA production** and also a **decrease in colon transit time**

A plant-based diet is associated with increased abundance of *Prevotella* and *Akkermansia* species, together with an **enrichment in polyphenols and SCFA production**

We are what we eat, II



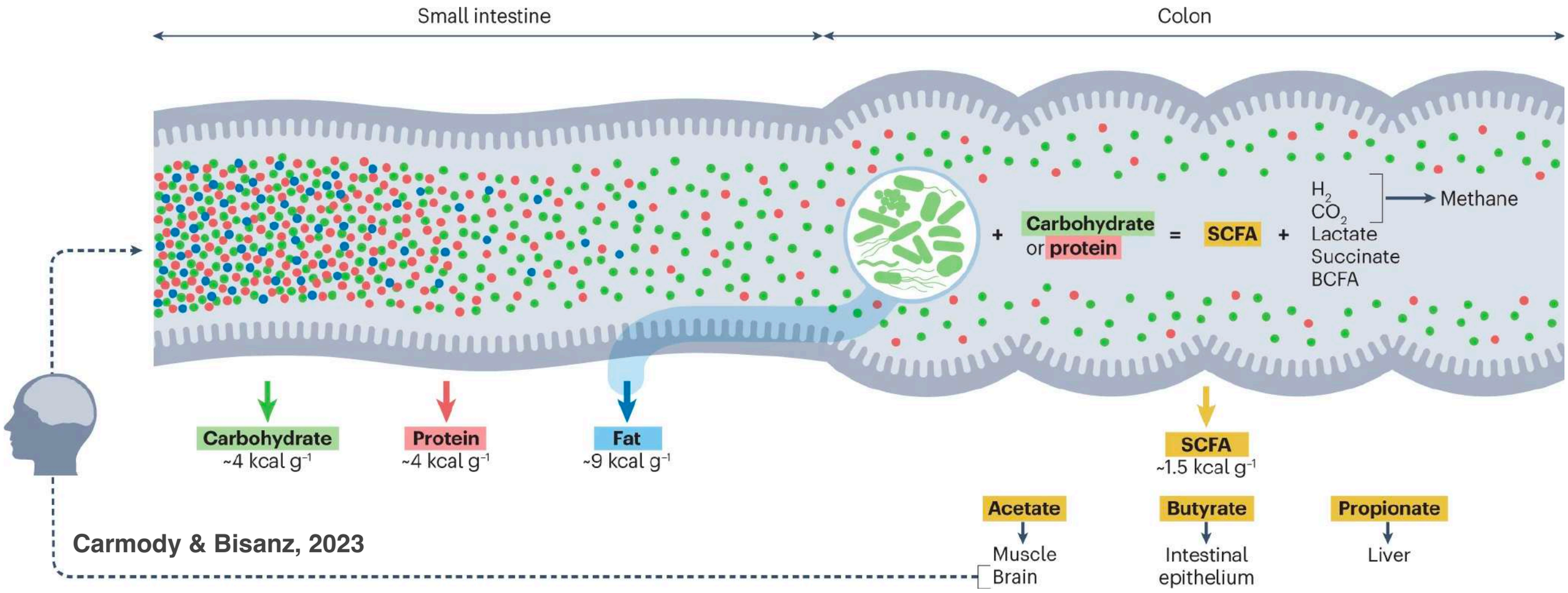
Ross et al., 2024

A high-protein diet is associated with enriched *Bacteroidetes* and *Fusobacterium* species, with **higher production of branched-chain fatty acids (BCFAs), indoles and short peptides**

The ketogenic diet is linked to decreased *Firmicutes* and *Actinobacteria* species and shows **high ketone levels**

A Western diet is associated with increased abundance of *Blautia* spp., *Bacteroides* spp. and *Ruminococcus* spp., which is in turn linked to **increased risk of metabolic disorders and chronic inflammation**

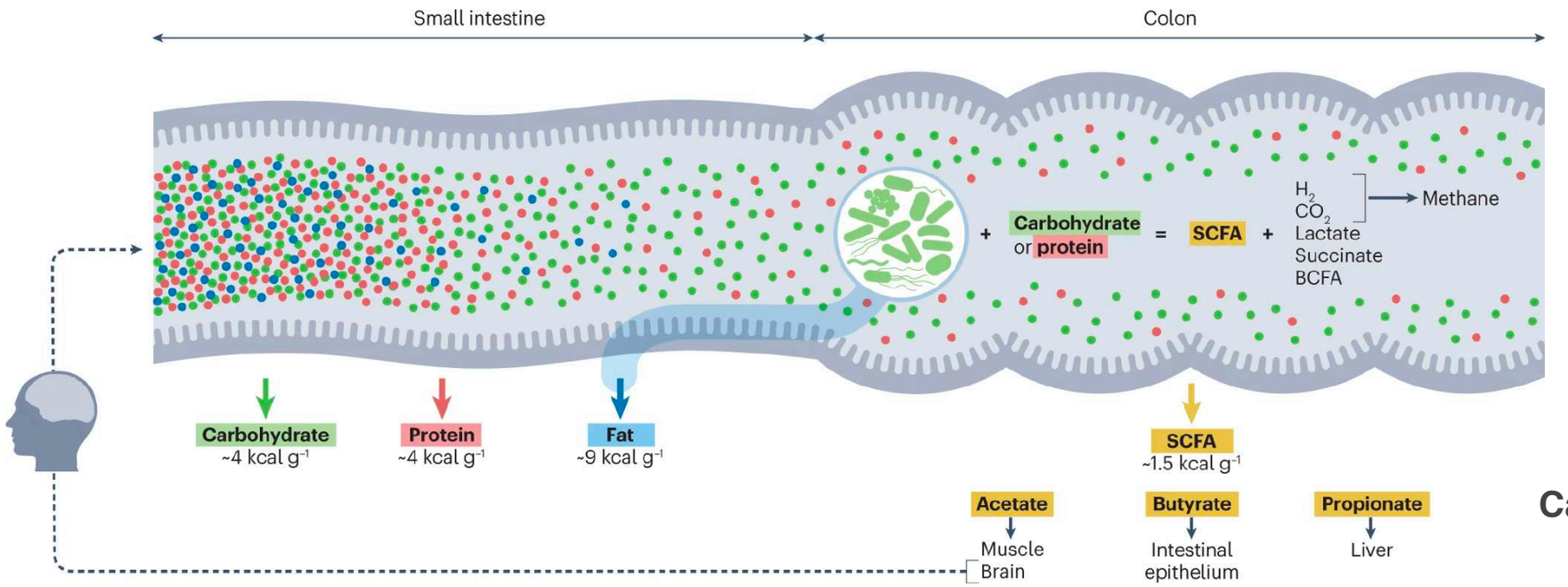
Gut microbiome enhances dietary energy harvest



Undigested carbohydrates are the principal fuel for microbial **fermentation**, from which the gut microbiome **generates the short-chain fatty acids (SCFAs) acetate, butyrate and propionate**

SCFAs enhance 1. gut barrier function by increasing mucus secretion and decreasing luminal pH, thereby protecting the intestinal lining from damage and preventing the entry of harmful pathogens into the bloodstream; 2. have antinflammatory and immunomodulatory effects, contributing to overall gut health and reducing the risk of gastrointestinal diseases

Gut microbiome enhances dietary energy harvest

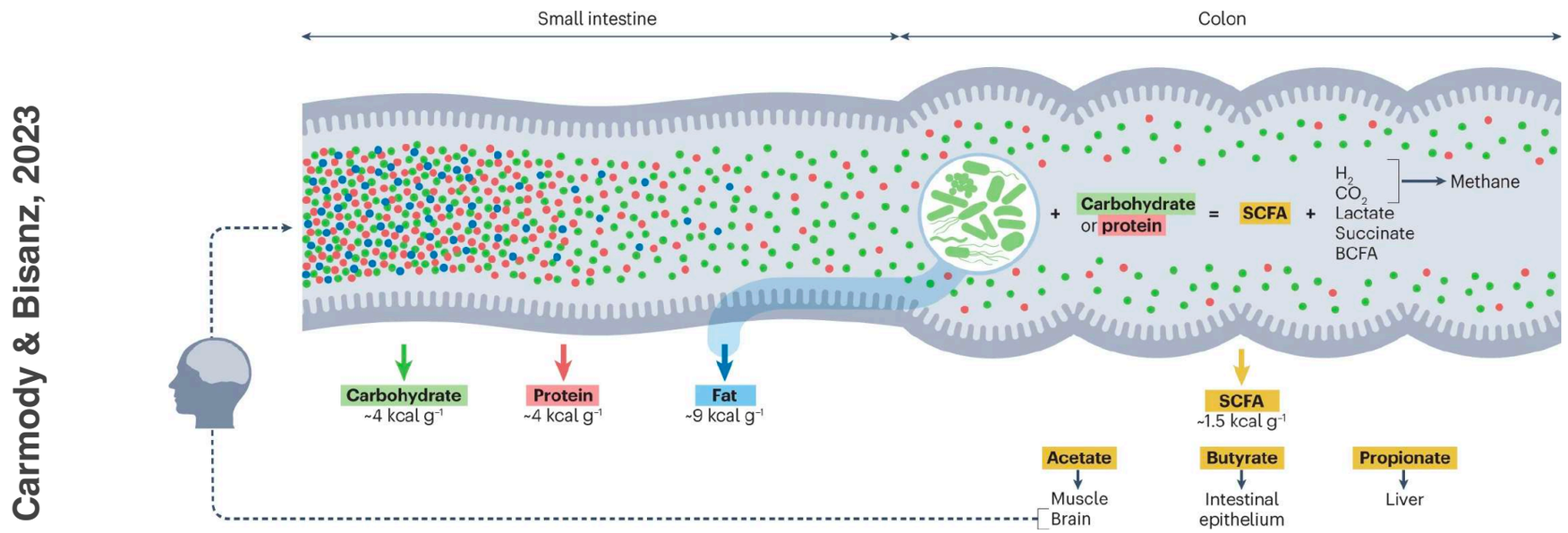


Carmody & Bisanz, 2023

Macronutrients available for breakdown by host enzymes are digested in the small intestine
Small intestinal macronutrient absorption supplies the host with energy predictable by biochemistry (carbohydrate, ~4 kcal g^{-1}; protein, ~4 kcal g^{-1}; fat, ~9 kcal g^{-1})
Dietary fat is readily absorbed in the proximal small intestine —> fat digestion canonically depends exclusively on host enzymes, but there are evidence of gut microbiome contributions to small intestinal lipid absorption

Microorganisms augment carbohydrate and protein digestion
The fractions of carbohydrate and protein digested in the small intestine vary with macronutrient structural form (for example, higher for sugar versus fibre), meal composition (for example, higher for fibre-poor versus fibre-rich meals), thermal processing (for example, higher for cooked foods) and physical processing (for example, higher for smaller particle sizes)
Nutrients that escape small intestinal digestion undergo fermentation by the colonic gut microbiota, producing an array of metabolites with energetic implications
The gut microbiome produces branched-chain fatty acids (BCFAs) from dietary valine, leucine and isoleucine, plus other organic acids such as lactate and succinate

Gut microbiome enhances dietary energy harvest



SCFAs are absorbed by the host and contribute to energy metabolism in diverse tissues, with acetate supporting muscle and brain, butyrate supplying up to 60–70% of the energetic needs of the colonic epithelium and propionate used in hepatic gluconeogenesis

Energy returns from SCFAs have been estimated at ~1.5 kcal g^{-1} < than half the rate for carbohydrates digested in the small intestine

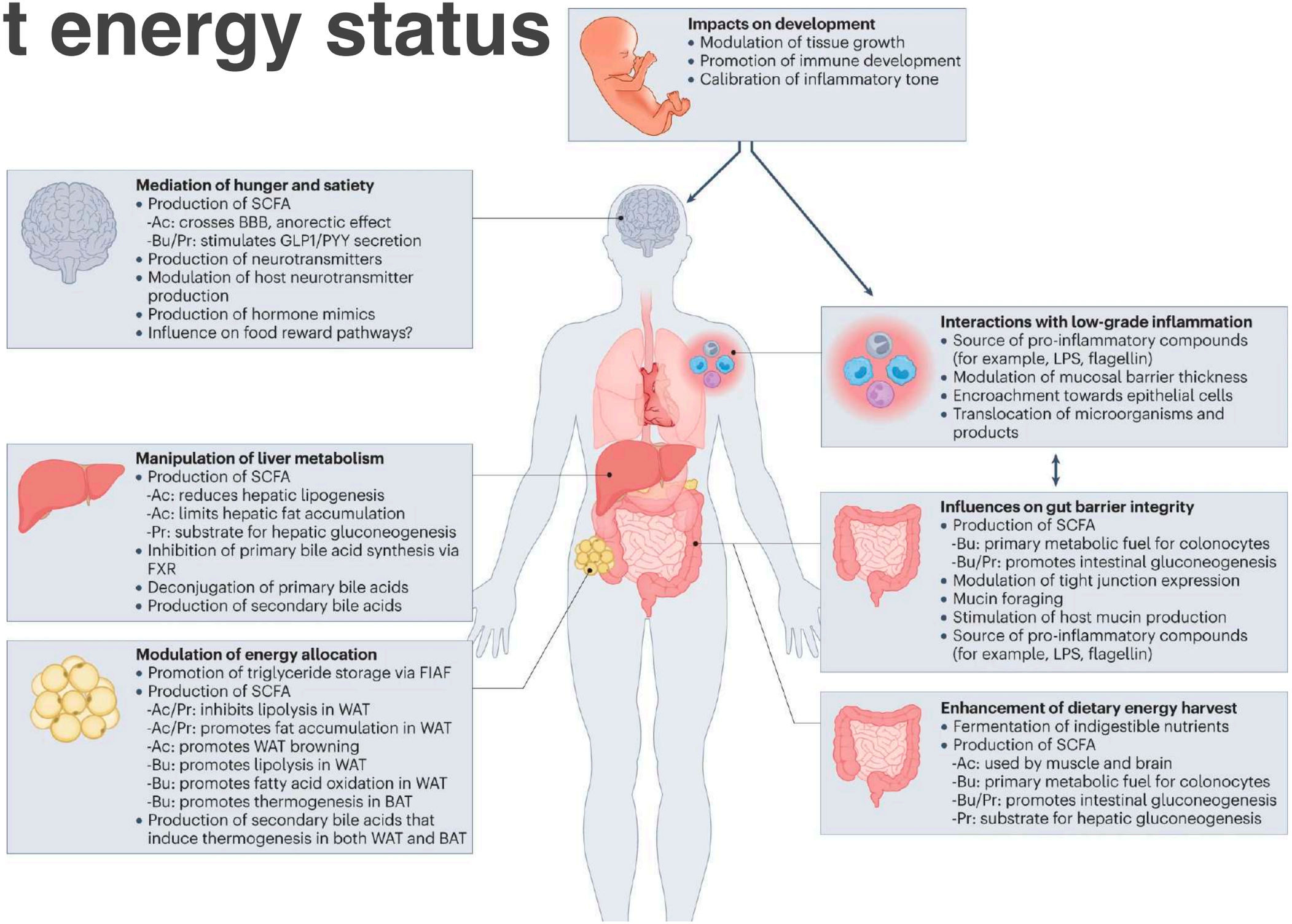
More energy is harvested by the host when nutrients are digested directly versus fermented

SCFAs account for ~5–10% of daily energy requirements in industrialized populations and almost certainly a >> fraction in populations with minimally processed and/or fibre-rich diets

SCFAs were long appreciated primarily as vehicles for energy salvage and have potent signalling functions that modulate energy intake, energy utilization and inflammation

Host metabolites, including bile acids and immune factors , also interact bidirectionally with the gut microbiome and influence its contributions to energy balance —> the metabolism of dietary fibre by gut microbiota leads to a cascade of beneficial effects on human health, including improved insulin sensitivity and fatty acid oxidation, and reduced inflammation

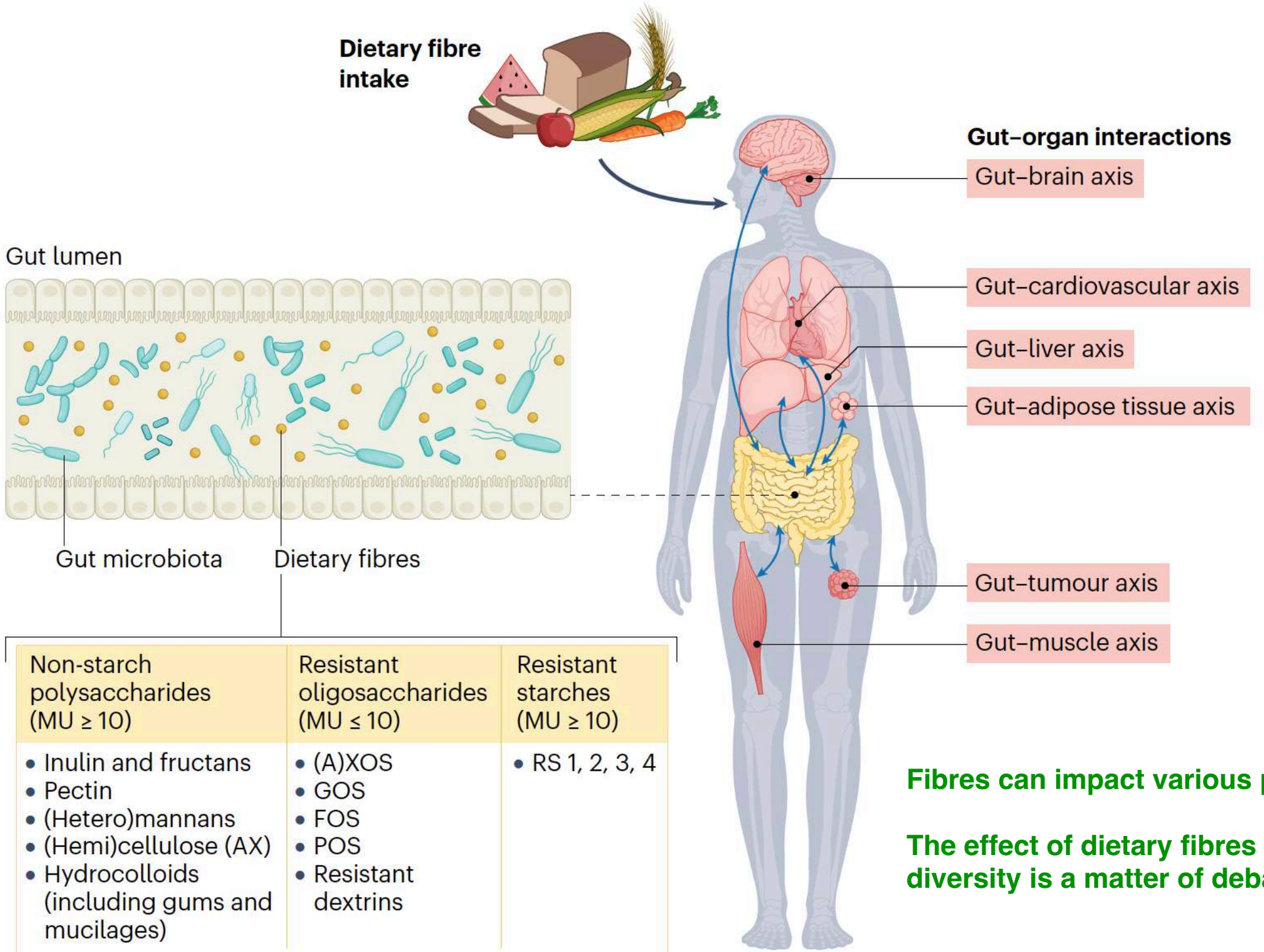
Mechanisms of gut microbial influence on host energy status



Carmody & Bisanz, 2023

Ac, acetate; BAT, brown adipose tissue; BBB, blood–brain barrier; Bu, butyrate; FIAF, fasting-induced adipocyte factor; FXR, farnesoid X receptor; GLP1, glucagon-like peptide 1; LPS, lipopolysaccharide; Pr, propionate; PYY, peptide YY; SCFA, short-chain fatty acid; WAT, white adipose tissue

Fibres



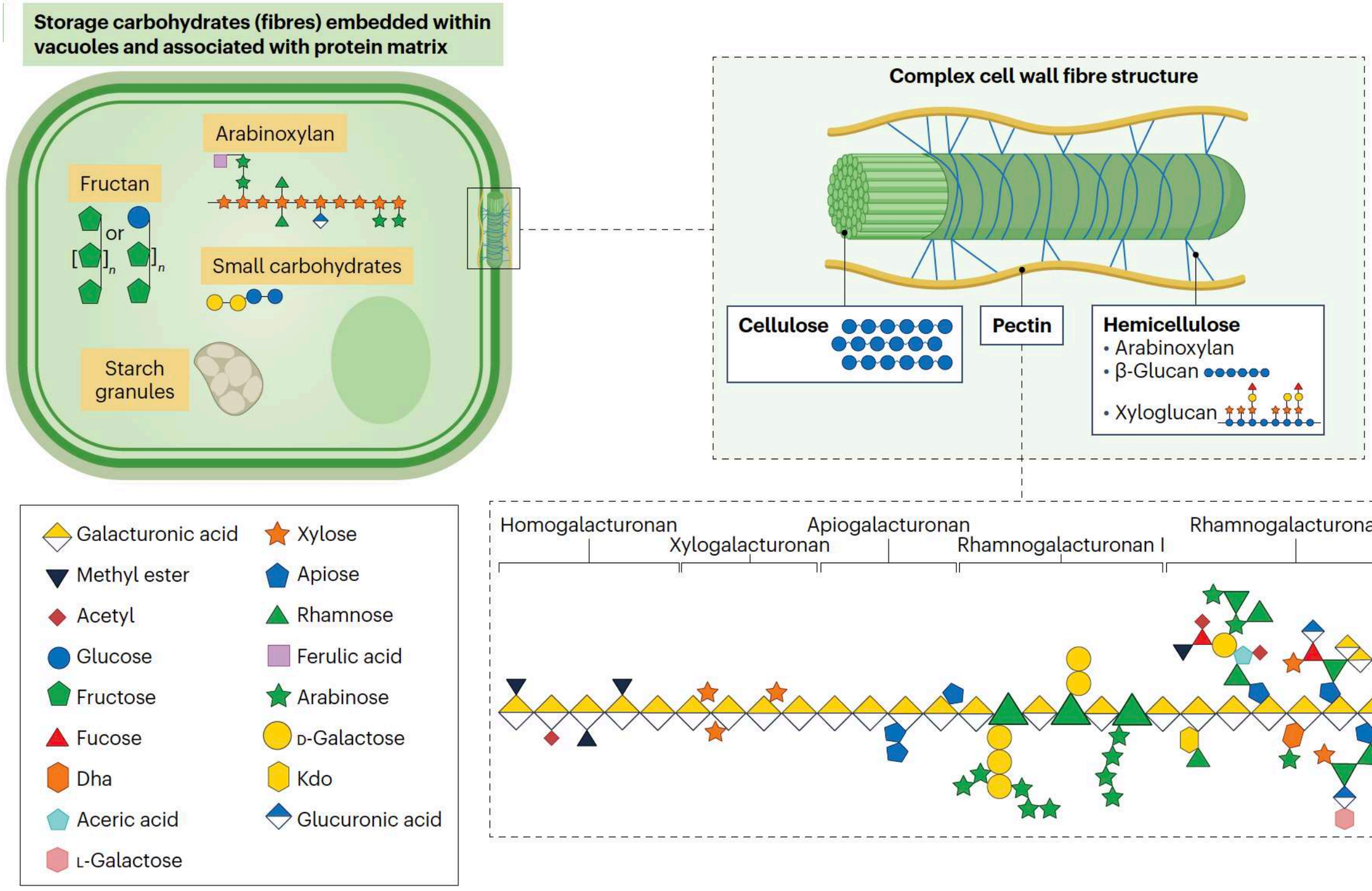
Non-starch polysaccharides (MU ≥ 10)	Resistant oligosaccharides (MU ≤ 10)	Resistant starches (MU ≥ 10)
• Inulin and fructans • Pectin • (Hetero)mannans • (Hemi)cellulose (AX) • Hydrocolloids (including gums and mucilages)	• (A)XOS • GOS • FOS • POS • Resistant dextrins	• RS 1, 2, 3, 4

Fibres can impact various physiological axes

The effect of dietary fibres on microbial diversity is a matter of debate

Delzenne et al., 2024

Fibres, II

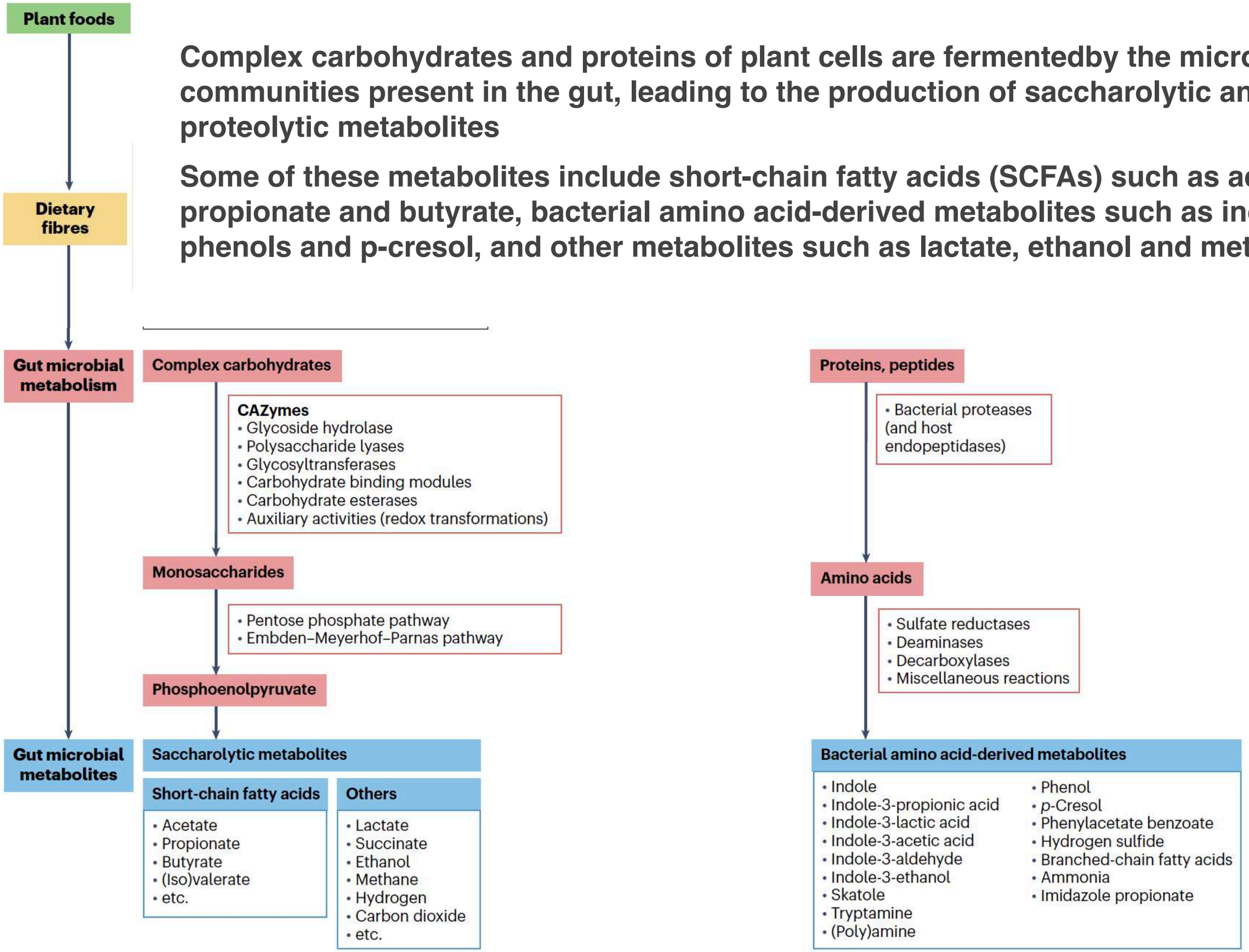


Delzenne et al., 2024

Dietary fibres either form a complex 3D structure that constitutes the backbone of plant cells, or are encapsulated as storage carbohydrates with various other nutrients such as lipids, proteins and polyphenols in the plant vacuole

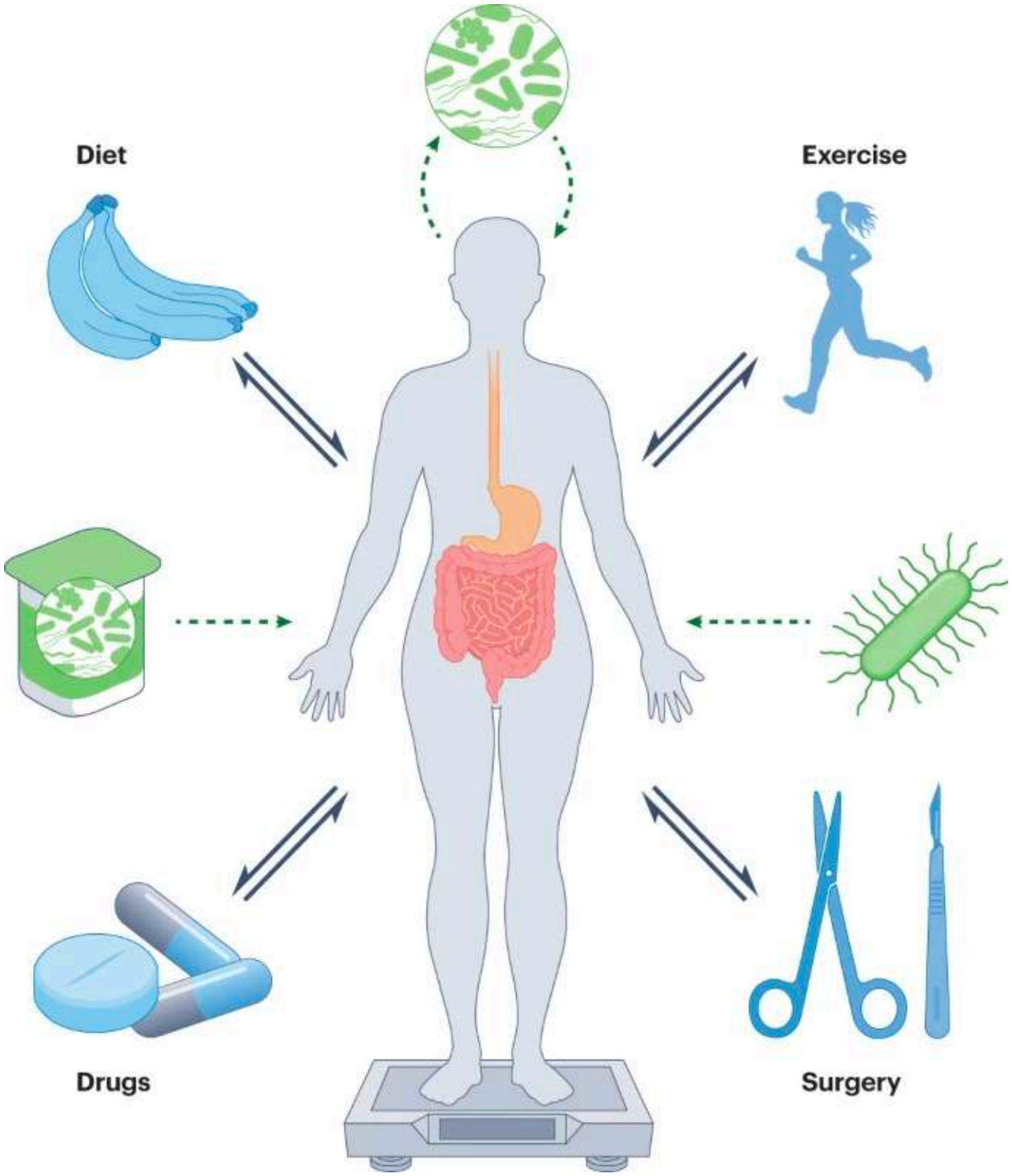
Complex carbohydrates and proteins of plant cells are fermentedby the microbial communities present in the gut, leading to the production of saccharolytic and proteolytic metabolites

Some of these metabolites include short-chain fatty acids (SCFAs) such as acetate, propionate and butyrate, bacterial amino acid-derived metabolites such as indoles, phenols and p-cresol, and other metabolites such as lactate, ethanol and methane



Delzenne et al., 2024

Reciprocal influences between the gut microbiome and key lifestyle and clinical approaches for weight management



Common weight-modulating interventions (blue) such as diet, exercise, drugs and surgery impact gut microbial structure and function, and these changes in the gut microbiome in turn alter intervention efficacy

Gut microbial contributions to weight management are targeted by emerging microbiome-directed therapies (green), including foods engineered to support the engraftment or growth of beneficial microorganisms, autologous faecal microbiota transplantation after weight loss and next-generation probiotics

Carmody & Bisanz, 2023

Microbial ALPHA and BETA- diversity changes with food

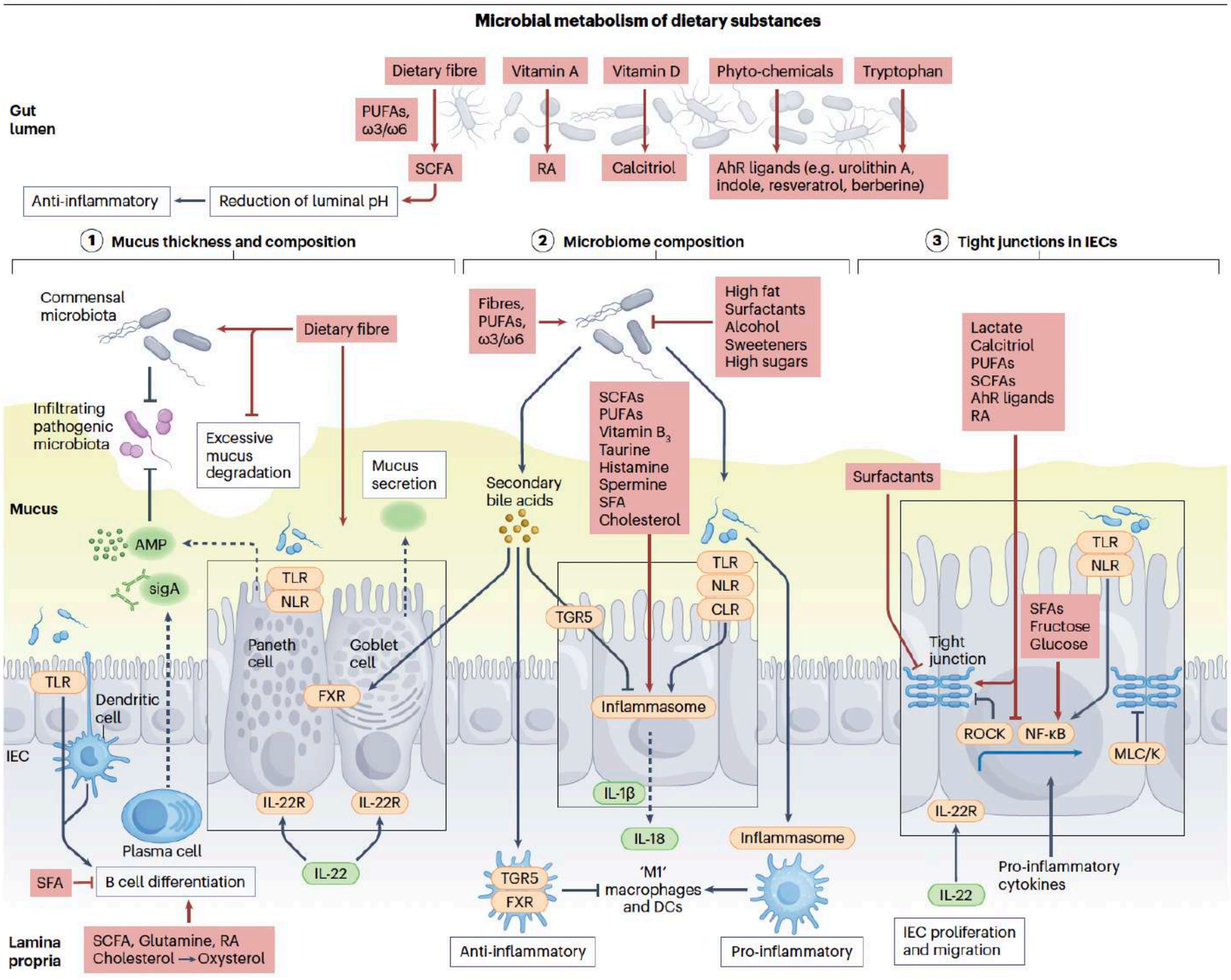
Table 2 | Associations between food groups and gut microbiota α-diversity and β-diversity

Diversity	Association	Food group
α-Diversity	**Increased diversity**	Fruit and berries[35,38–41,47,48], vegetables[35,38,48], fruit and vegetable intake biomarkers (carotenoids)[37] Fibre-rich bread[41] Dairy products (yogurt[46], buttermilk[35], low-fat cheese[41]) Fish[17,38,39], poultry[41] Coffee or caffeinated beverages[35,38], tea[35], alcoholic drinks (red wine)[35,38] Chocolate type[36], sweets[48]
	Decreased diversity	Sugary drinks[35,36,38,39,48], beer[35,36] (White) bread[35,48], potatoes/pasta/rice[38,48] Whole-fat milk/cream[35,40] Meat and processed meat[39,48] Desserts, ice cream, fatty-sweet products[38–40] Fried products[39], ready-cooked meals[39], snacks[35], sauces/spreads[35] Legumes and pulses[35], fruit and vegetable intake biomarkers (tocopherol and retinol)[37]
	Explained variance[a]	Grains, low-fibre rice and pasta, vegetables, olive oil, other oils, salad dressing, sugar-sweetened beverages, bread fillings[45]
β-Diversity	**Explained variance**[b]	Fruit and berries[17,35,36,39–41,47,48], juices[41], compote and jams[48], vegetables[17,35,40,41,48], fruit and vegetable intake biomarkers (tocopherol, carotenoids)[37], potatoes[17,48], legumes[17,40], soy products[36], nuts, seeds[17,40] (Whole) grains[17,40,48], (fibre-rich) bread[35,36,41,48] Dairy products[17,42] (cheese[39–41], milk, cream, ice-cream[40]) Meat and processed meat[17,36,41,48], fish[41,48], poultry[41] Sugary drinks[17,35,36,48], alcoholic drinks (beer, red wine)[17,35,36], coffee, tea[17,35,36,48] Sweets or desserts[17,48], chocolate type[36] Snacks[35], ready-cooked meals[39], fried products[39], dressings and oils[41]

[a]Food groups contributing to explaining the differences in α-diversity measures between individuals, with unspecified direction of associations. [b]Food groups contributing to explaining the dissimilarities in gut microbiota composition between individuals.

Sanz et al., 2025

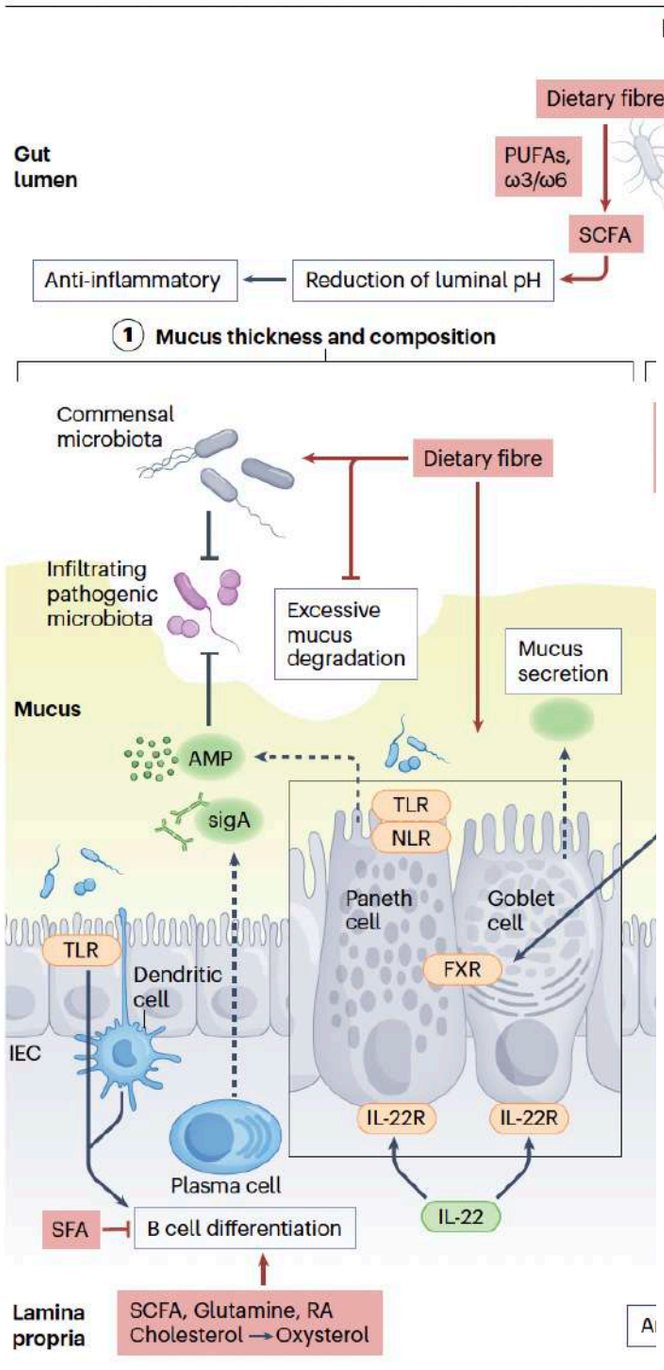
Dietary orchestration of gut barrier and immunity is linked to the microbiome



Dietary substances and their microbially produced metabolites (in red) modulate intestinal barrier integrity and immunity through various mechanisms involving the resident microbiome.

Sanz et al., 2025

Dietary orchestration of gut barrier and immunity is linked to the microbiome



1

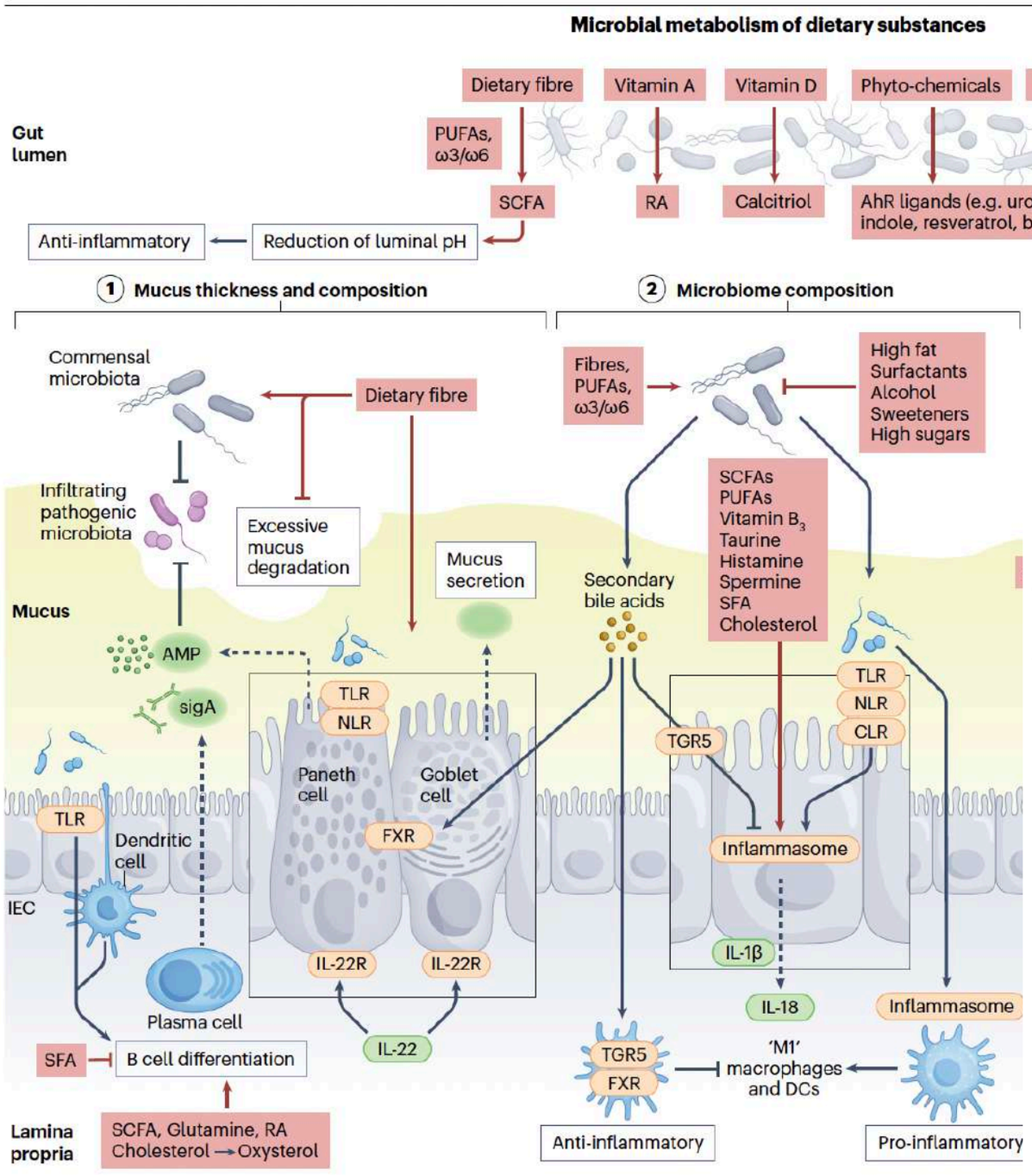
Dietary fibre mechanically stimulates mucus-producing goblet cells and prevents mucus digestion by nurturing commensal bacteria.

Microbiota sensing via Toll-like receptors (TLR) and NOD-like receptors (NLR) triggers plasma cells and Paneth cells to secrete secretory IgA (sIgA) and antimicrobial peptides (AMP).

Dietary metabolites such as retinoic acid (RA) and intestinal epithelial cell (IEC)-derived cytokines further facilitate plasma cell development.

Sanz et al., 2025

Dietary orchestration of gut barrier and immunity is linked to the microbiome

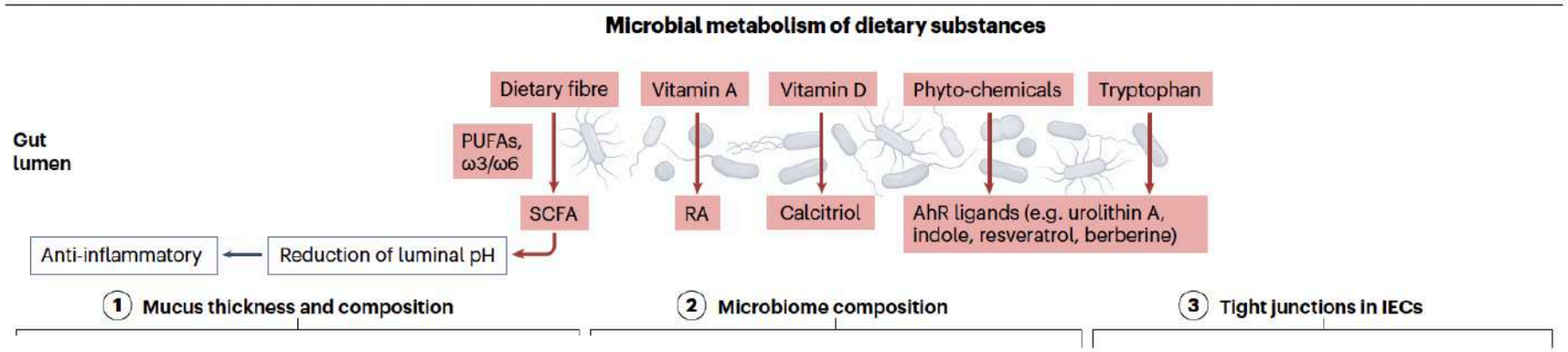


(2) Dietary substances shape microbiome composition, thereby indirectly influencing anti inflammatory
and mucus-promoting bile acid levels and triggering TLR–NLR–C-type lectin receptor (CLR)-mediated inflammasome activation in IECs and immune cells.

Inflammasome activity is also directly influenced by dietary metabolites.

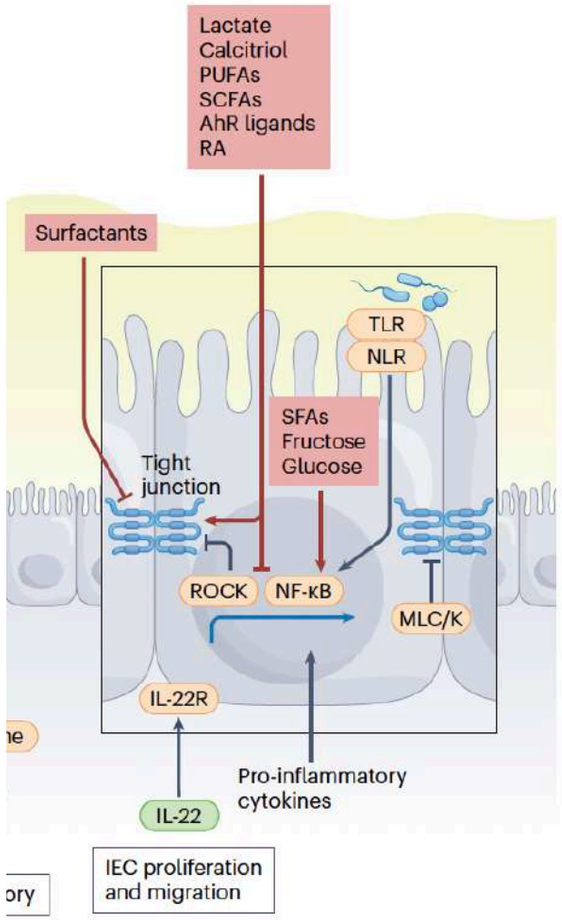
Sanz et al., 2025

Dietary orchestration of gut barrier and immunity is linked to the microbiome



(3) Tight junctions are disrupted by TLR signalling, pro-inflammatory cytokines and diet-induced reactive oxygen species or transcriptional reprogramming as it triggers nuclear factor -κB (NF-κB)-regulated and rho-associated coiled-coilcontaining
protein kinase (ROCK)-regulated myosin light chain and myosin
light chain kinase (MLC/K) signalling.

Dietary metabolites such as short-chain fatty acid (SCFA) or aryl hydrocarbon receptor (AhR) ligands counteract these
disruptions and enhance tight junction protein expression

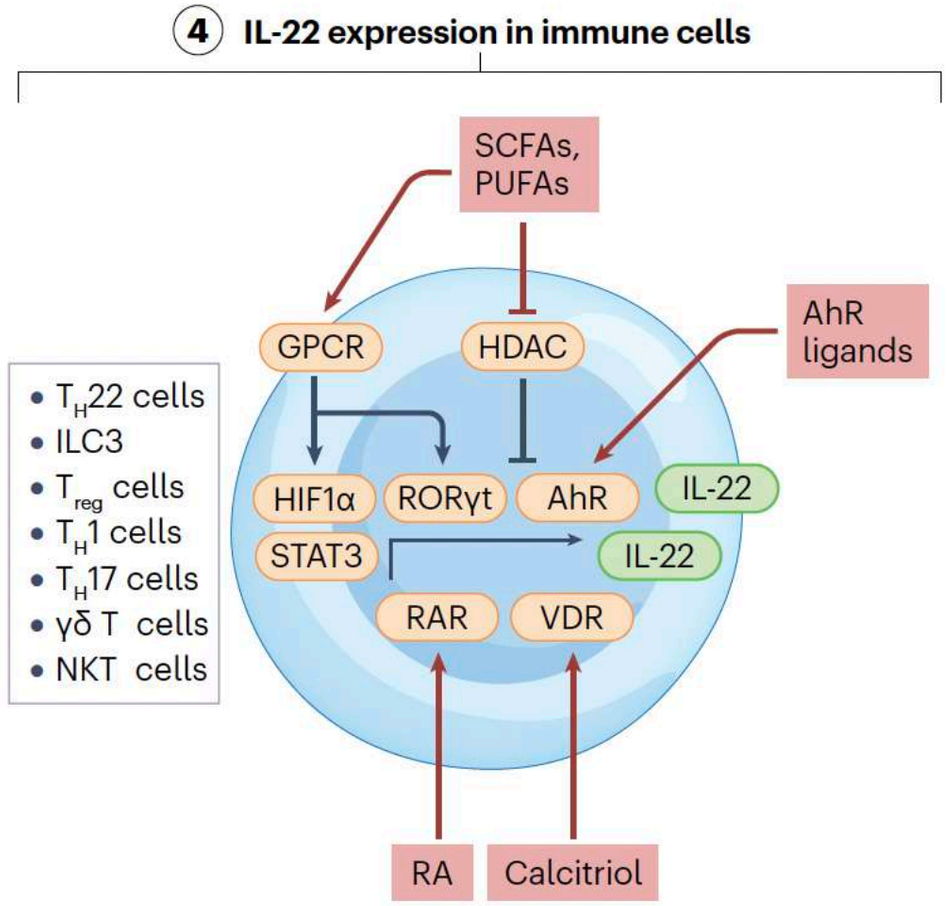


Sanz et al., 2025

Dietary orchestration of gut barrier and immunity is linked to the microbiome

(4) IL-22 signalling, primarily from type 3 innate lymphoid cells (ILC3) and T helper 22 (T_H22) cells, is crucial for mucus production and IEC maintenance, and is multifactorially supported by dietary substances.

SCFAs and polyunsaturated fatty acids (PUFAs) activate G protein-coupled receptors (GPCRs) and downstream transcription factors, and SCFAs inhibit histone deacetylases (HDAC), further promoting transcription. AhR ligands and vitamin metabolites directly activate different IL-22 transcription factors.



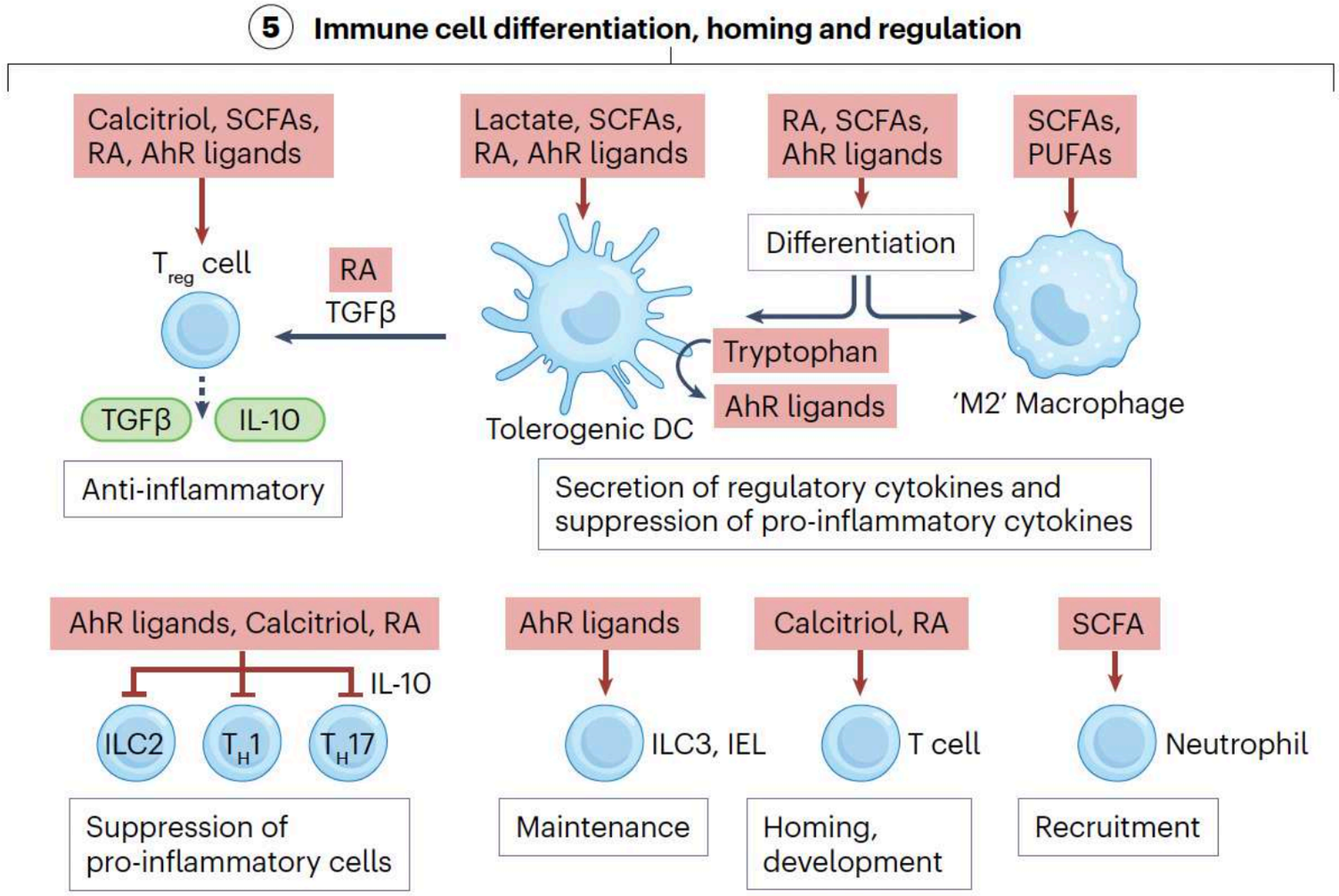
DC, dendritic cell; FXR, farnesoid X receptor; HIF1α, hypoxia-inducible factor 1α; IEL, intraepithelial lymphocyte; IL-22R, IL-22 receptor; NKT, natural killer T cell; RAR, retinoic acid receptor; RORγt, RAR-related orphan receptor-γ; SFA, saturated fatty acid; STAT3, signal transducer and activator of transcription 3; TGFβ, transforming growth factor-β; TGR5, Takeda G protein-coupled receptor 5; TH1, T helper 1 cell; TH17, T helper 17cell; VDR, vitamin D receptor.

Sanz et al., 2025

Dietary orchestration of gut barrier and immunity is linked to the microbiome

(5) Dietary metabolites influence the abundance and function of several intestinal immune cells.

Particularly, vitamin A-derived RA and AhR ligands integrate with cytokines to support regulatory T (Treg) cells and establish an anti-inflammatory milieu.



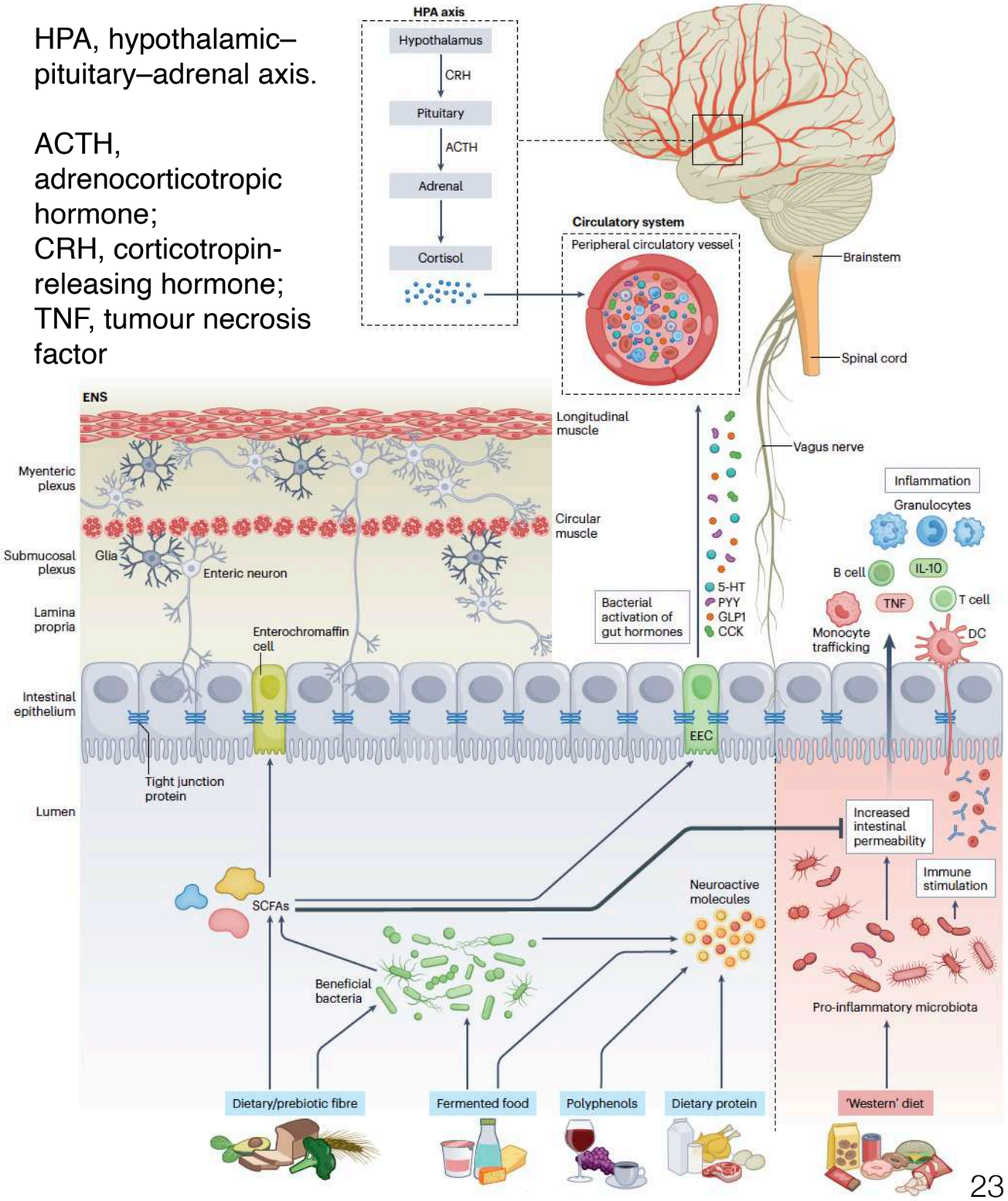
DC, dendritic cell; FXR, farnesoid X receptor; HIF1α, hypoxia-inducible factor 1α; IEL, intraepithelial lymphocyte; IL-22R, IL-22 receptor; NKT, natural killer T cell; RAR, retinoic acid receptor; RORγt, RAR-related orphan receptor-γ; SFA, saturated fatty acid; STAT3, signal transducer and activator of transcription 3; TGFβ, transforming growth factor-β; TGR5, Takeda G protein-coupled receptor 5; TH1, T helper 1 cell; TH17, T helper 17cell; VDR, vitamin D receptor.

Sanz et al., 2025

Diet and gut microbiome interactions orchestrate nervous system function

HPA, hypothalamic–pituitary–adrenal axis.

ACTH, adrenocorticotropic hormone;
CRH, corticotropin-releasing hormone;
TNF, tumour necrosis factor



Diets rich in fibre, fermented foods, and polyphenols, and with moderate levels of proteins sustain the growth of mutualistic microorganisms (beneficial bacteria) and contribute to the generation of **metabolites that favourably regulate nervous system function**.

Short-chain fatty acids (**SCFA**) **induce the secretion of the anorexigenic peptides glucagon-like peptide 1 (GLP1), peptide tyrosine-tyrosine (PYY) and cholecystokinin (CKK) by enteroendocrine cells (EEC),** which **act on** the **hypothalamus centres of food intake control.**
SCFAs strengthen the gut barrier integrity and induce protective immune responses, preventing chronic inflammation.

Gut bacteria participate in the provision and metabolism of amino acids that are **precursors of neurotransmitters such as tryptophan, which can be transformed to 5-hydroxytryptamine (5-HT) in enterochromaffin cells, or tyrosine which can be converted to catecholamines** (for example, noradrenaline and dopamine) **which can interact with the enteric nervous system (ENS) or stimulate vagal sensory neurons in the gut, leading to activation in the brain structures, controlling mood, behaviour and mental health**

DC, dendritic cell

Sanz et al., 2025

Diet and gut microbiome interactions orchestrate nervous system function

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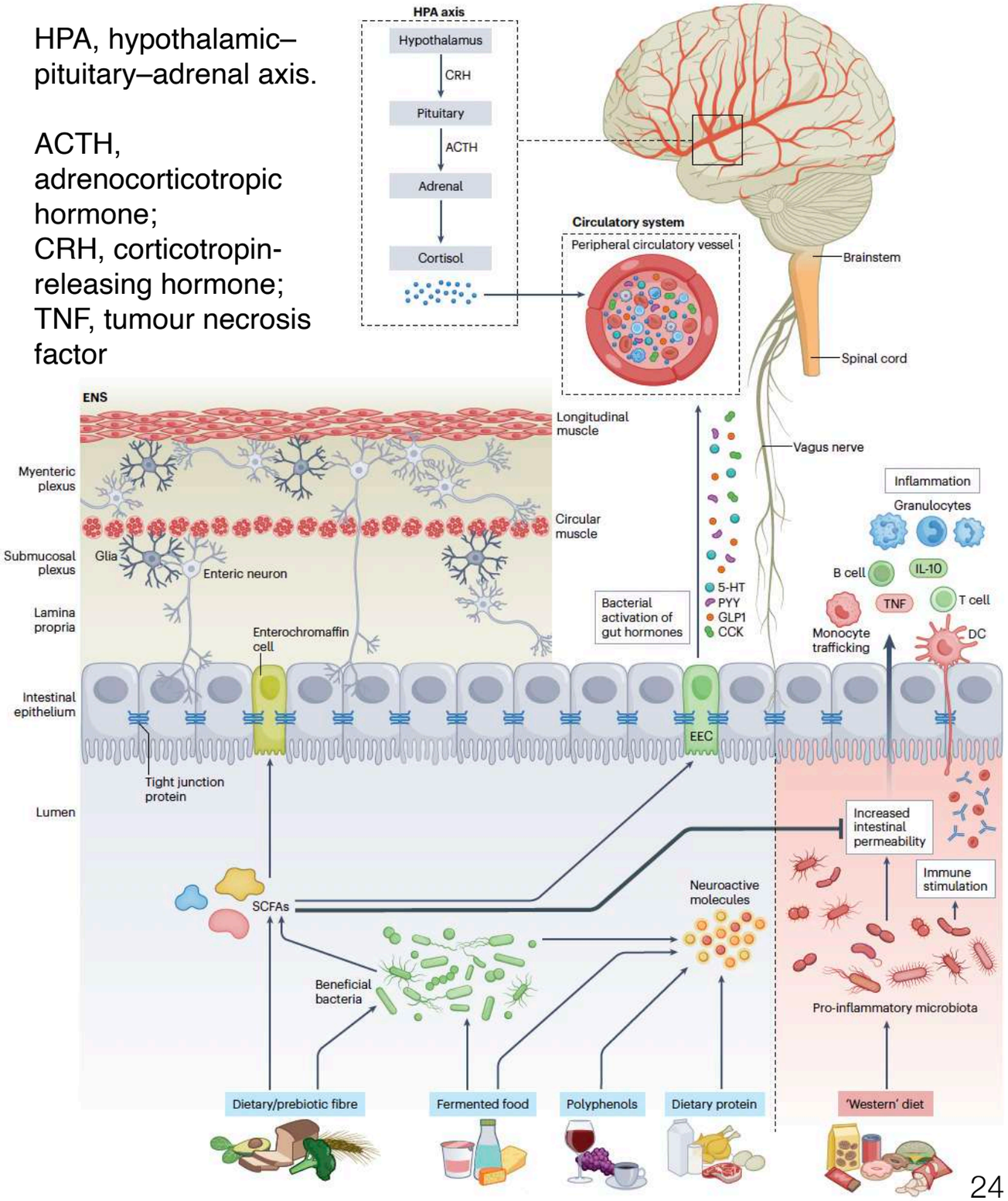


Unhealthy diets (that is, Western diets rich in energy, saturated fat and simple sugars) alter the composition and function of the gut microbiome, damage gut integrity and contribute to inflammation in the gut and systemically through the translocation of endotoxins from the gut lumen to the bloodstream and other inflammatory mediators that can induce systemic inflammation associated with behavioural and mental disorders.

DC, dendritic cell

Sanz et al., 2025

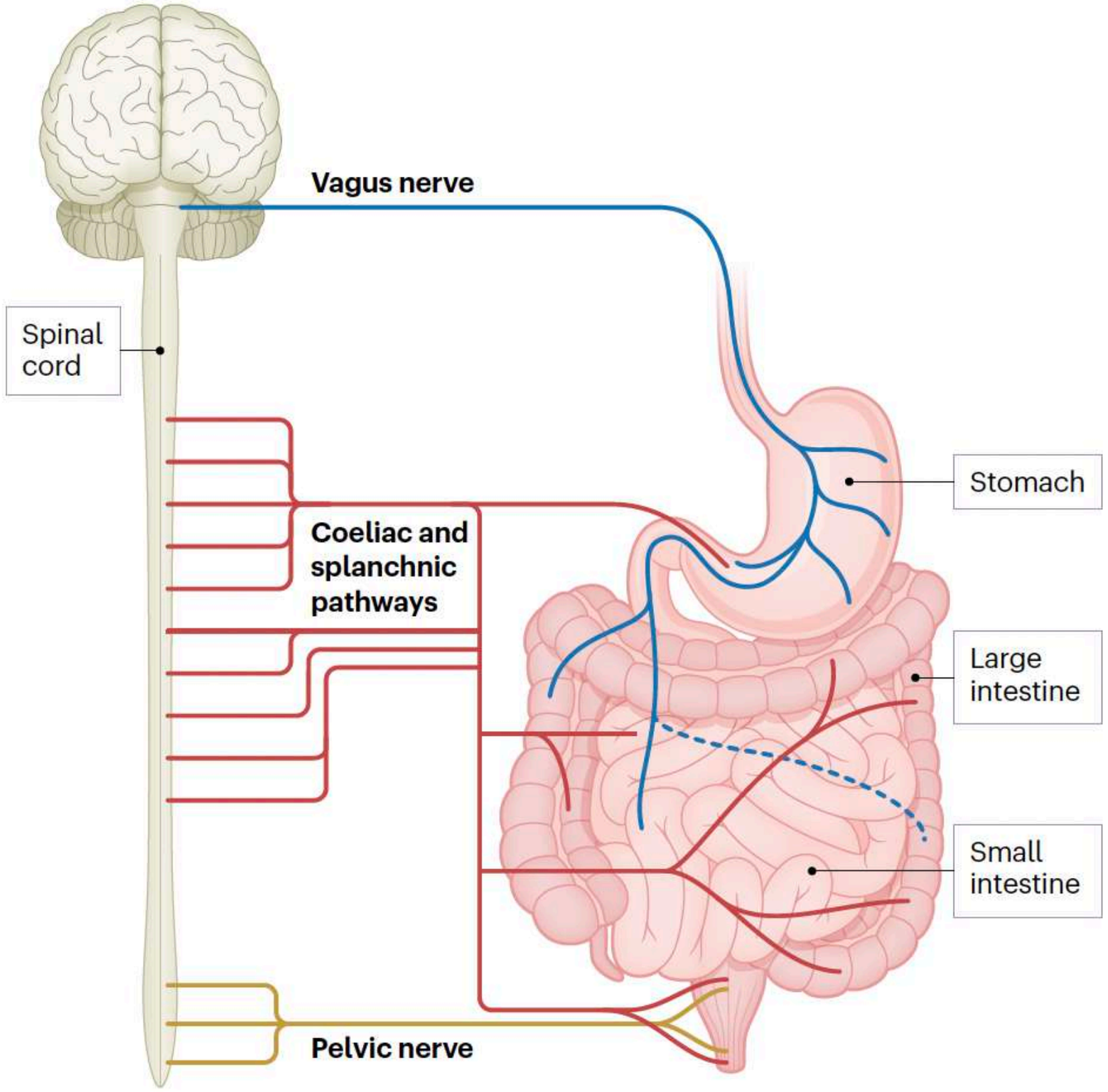


Fig. 1 | Major sensory pathways linking gut to brain. Schematic diagram showing the major sensory pathways linking gut to brain, including the vagal (blue), thoracolumbar spinal (red) and lumbosacral spinal (yellow) sensory pathways.

Spencer et al., 2025

Parental diet and offspring health: a role for the gut microbiome via epigenetics

Yang et al., 2025

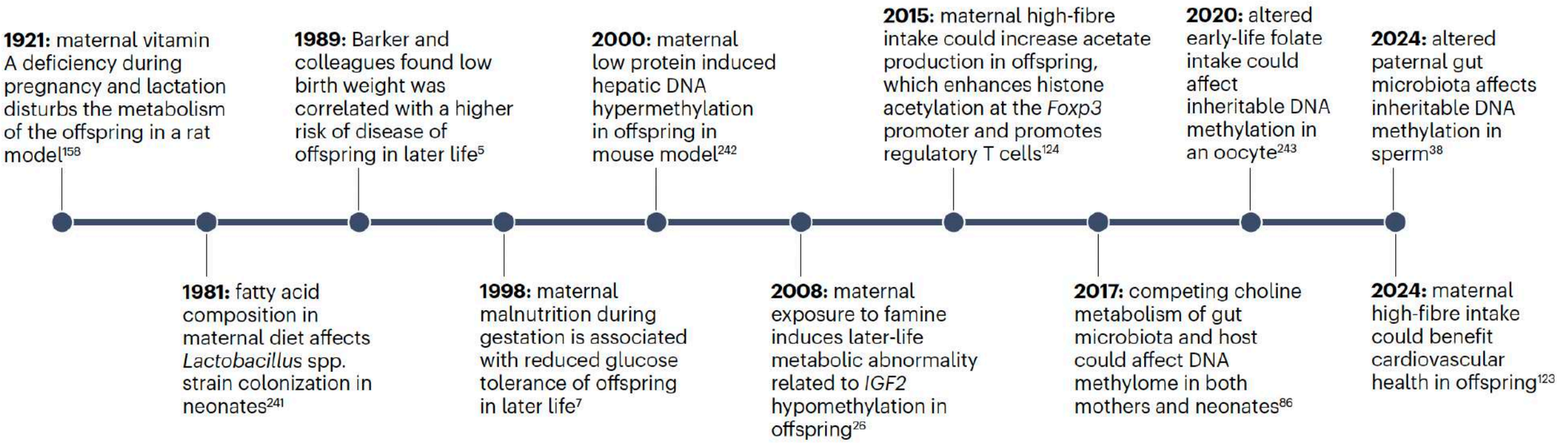


Fig. 1 | Key historical findings on the effect of maternal diet on the offspring. Key findings[5,7,26,38,86,123,124,158,241–243] include: 1921: discovery that maternal vitamin A deficiency disrupts offspring metabolism in rats, the earliest evidence that maternal diet affects offspring health; 1981–2000: gut microbiota and the epigenome identified as key mediators of the effects of maternal diet on offspring health; 2015–2024: recent findings show the roles of crosstalk between epigenome and gut microbiota in shaping offspring health in response to maternal diet.

Association versus causation

An important consideration in gut microbiome studies is the **distinction between association and causation**. These can usually be distinguished using **forward and reverse microbiome approaches**.

Causality is more complex to determine in pregnancy studies.

For example, maternal dietary patterns affect the composition of gut microbiota populations in the adult offspring of primates and mice.

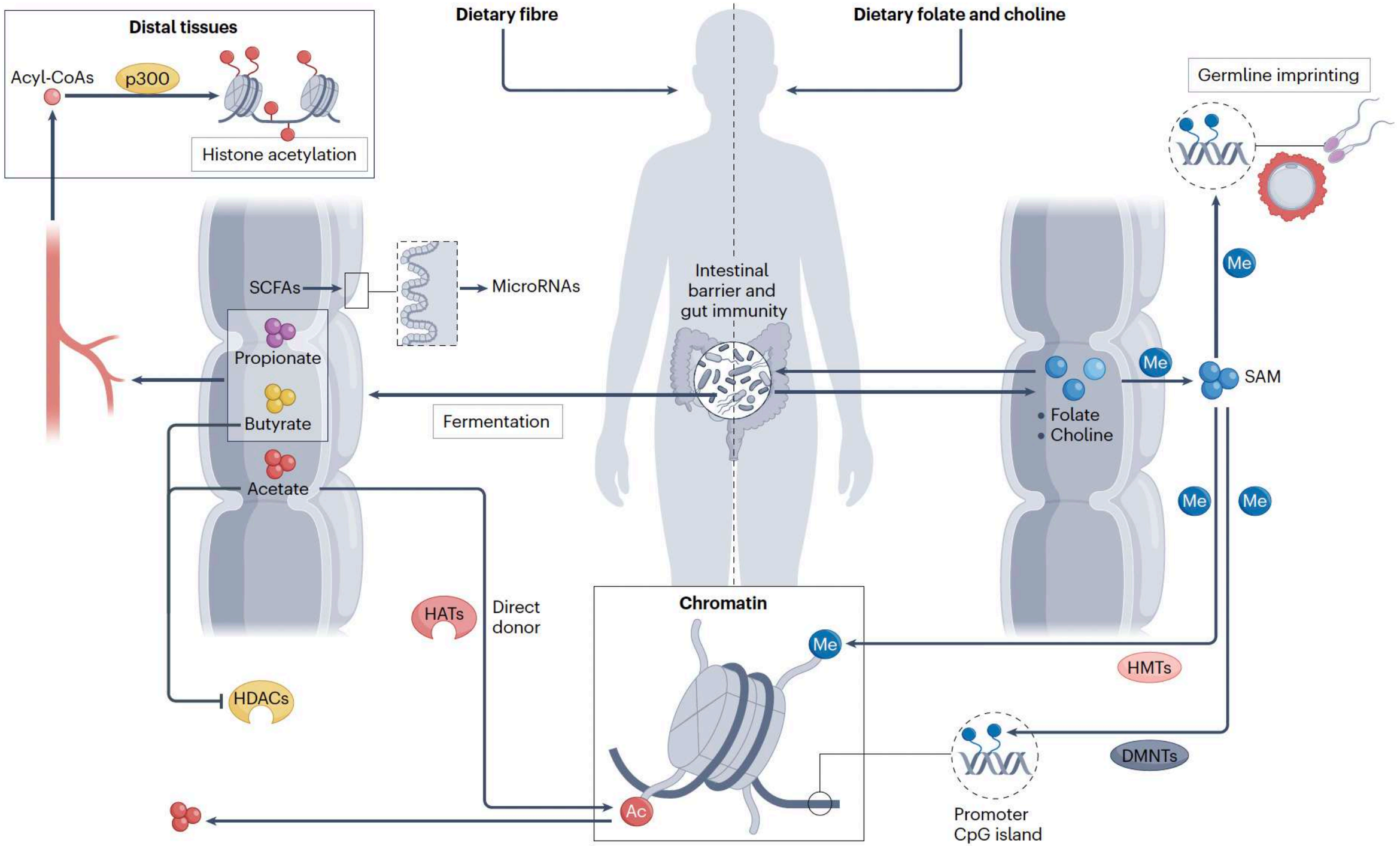
It is less clear whether health-related phenotypes observed in the offspring are due to in utero epigenetic modifications or to the passage of the microbiota from the mother to the offspring at birth.

To distinguish between these possibilities, studies usually leverage caesarean sections with cross-fostering, germ-free animals, or antibiotics during pregnancy (albeit many antibiotics are not safe for the fetus) and can evaluate the difference between interventions during pregnancy and interventions during breastfeeding.

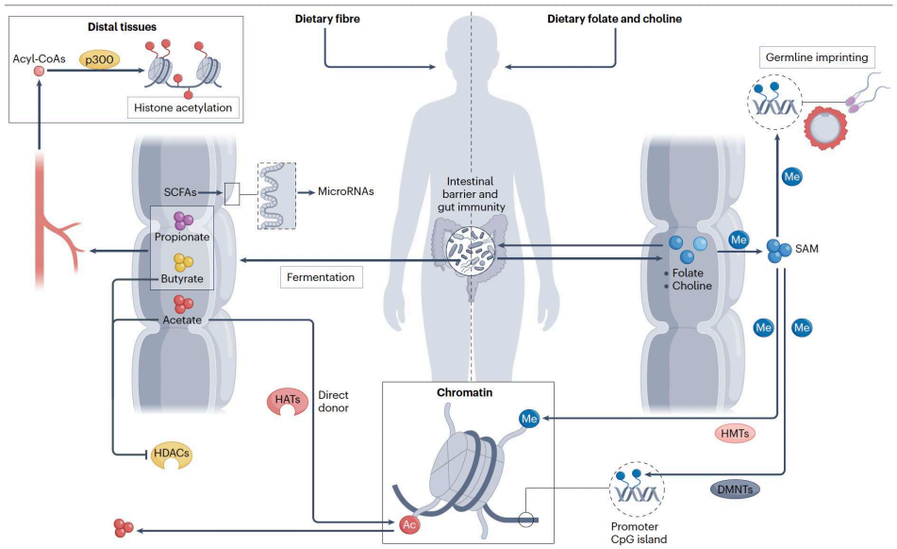
Studies need to distinguish the effects of diet in preconception, prenatal and postnatal end points and how these shift and interact with the parental gut microbiome.

Yang et al., 2025

Effects of the gut microbiome on epigenetic modifications



Yang et al., 2025



Effects of the gut microbiome on epigenetic modifications

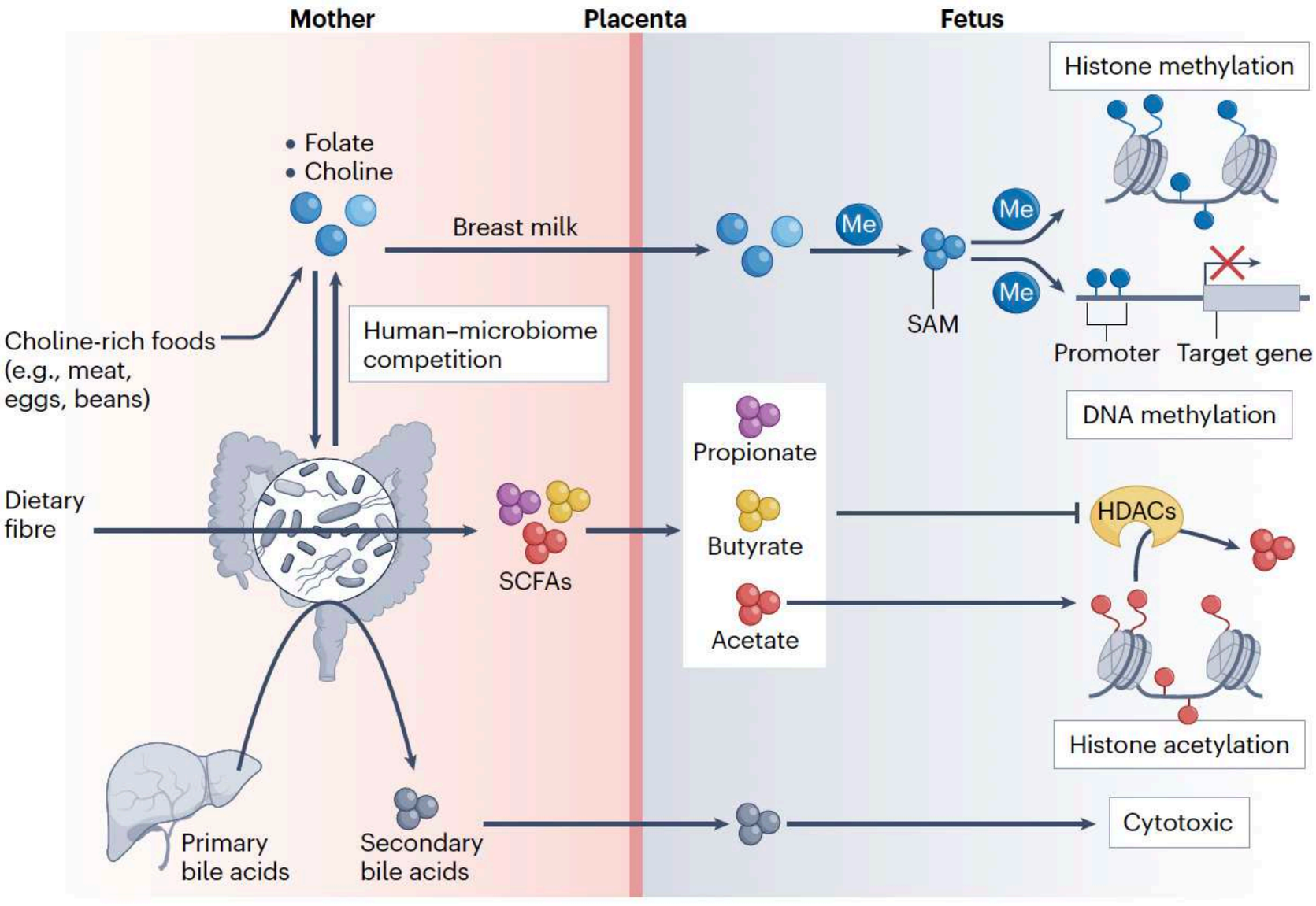
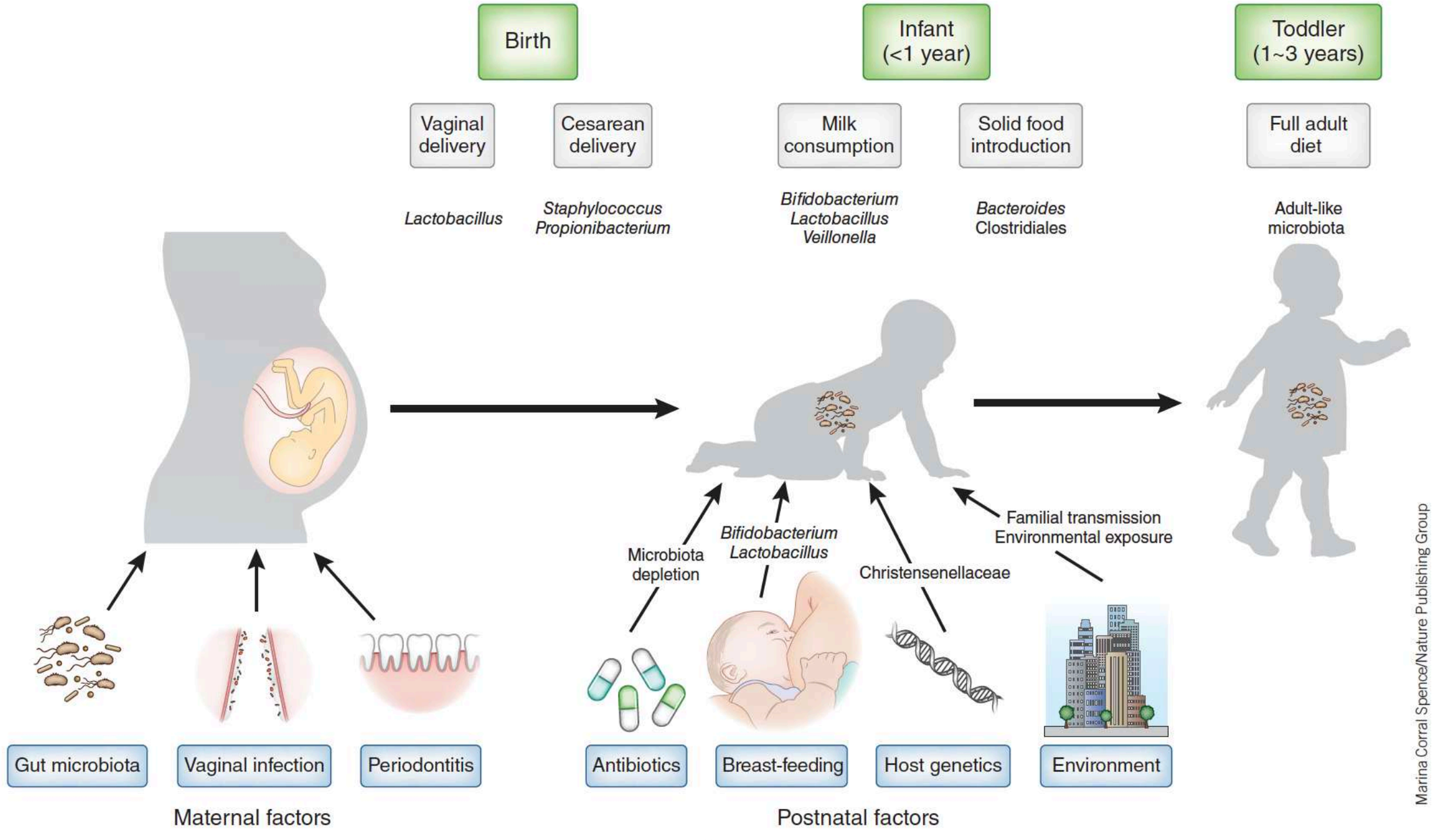


Fig. 3 | Epigenetic effects of maternal dietary intake. Choline and folate, obtained from dietary sources such as meat, eggs and beans, or breast milk, could be metabolized by both the host and the gut microbiota, with competition and dynamics between the two. Choline and folate can penetrate the placental barrier and affect the synthesis of *S*-adenosylmethionine (SAM) via one-carbon metabolism in the fetus. Affected SAM production could alter the fetus DNA and histone methylation processes. Dietary fibres are fermented by the gut microbiota to produce short-chain fatty acids (SCFAs), including acetate, propionate and butyrate. These SCFAs could also penetrate the placental barrier and affect epigenetic regulation by inhibiting histone deacetylases (HDACs), promoting histone acetylation. Furthermore, primary bile acids, produced by the liver, are transformed into secondary bile acids by the gut microbiota. Secondary bile acids penetrating the placental barrier can affect fetal development because secondary bile acids in high concentrations are cytotoxic. Me, methyl.

Yang et al., 2025

Factors shaping the neonatal microbiome

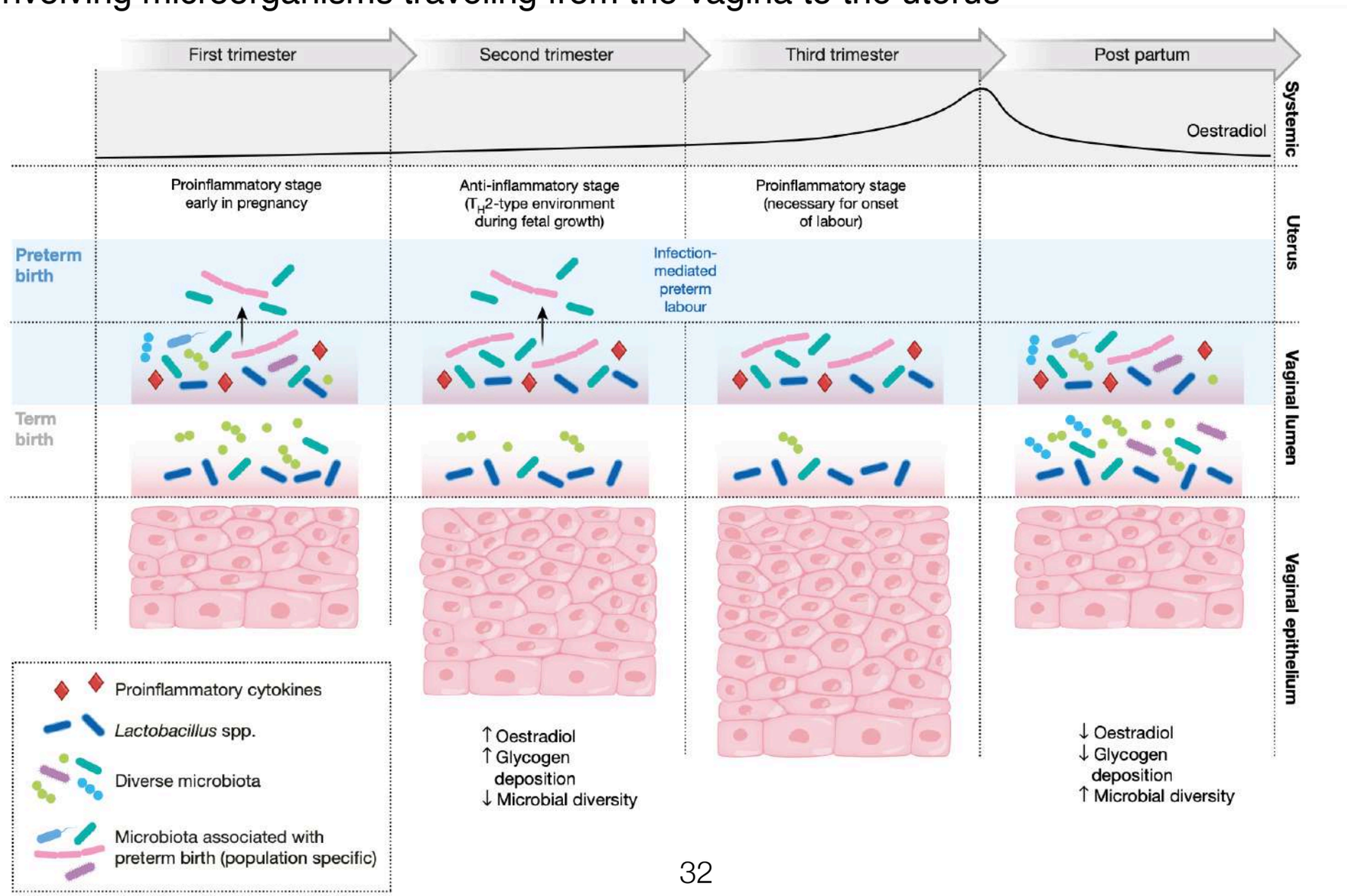


Marina Corral Spence/Nature Publishing Group

Tamburini et al, 2016

Dysibiosis and pregnancy in preterm birth

- As **pregnancy** progresses, with **predictable changes in systemic oestradiol levels**, the uterine and **vaginal environments** undergo various **changes**
- The **uterus switches** from an **early pro-inflammatory** condition **to** an **anti-inflammatory** condition in the second trimester, and then back **to a pro-inflammatory** condition before the onset of **labour**
- Specific changes in the microbiome of the vaginal lumen can be associated with preterm birth, possibly through mechanisms involving microorganisms traveling from the vagina to the uterus

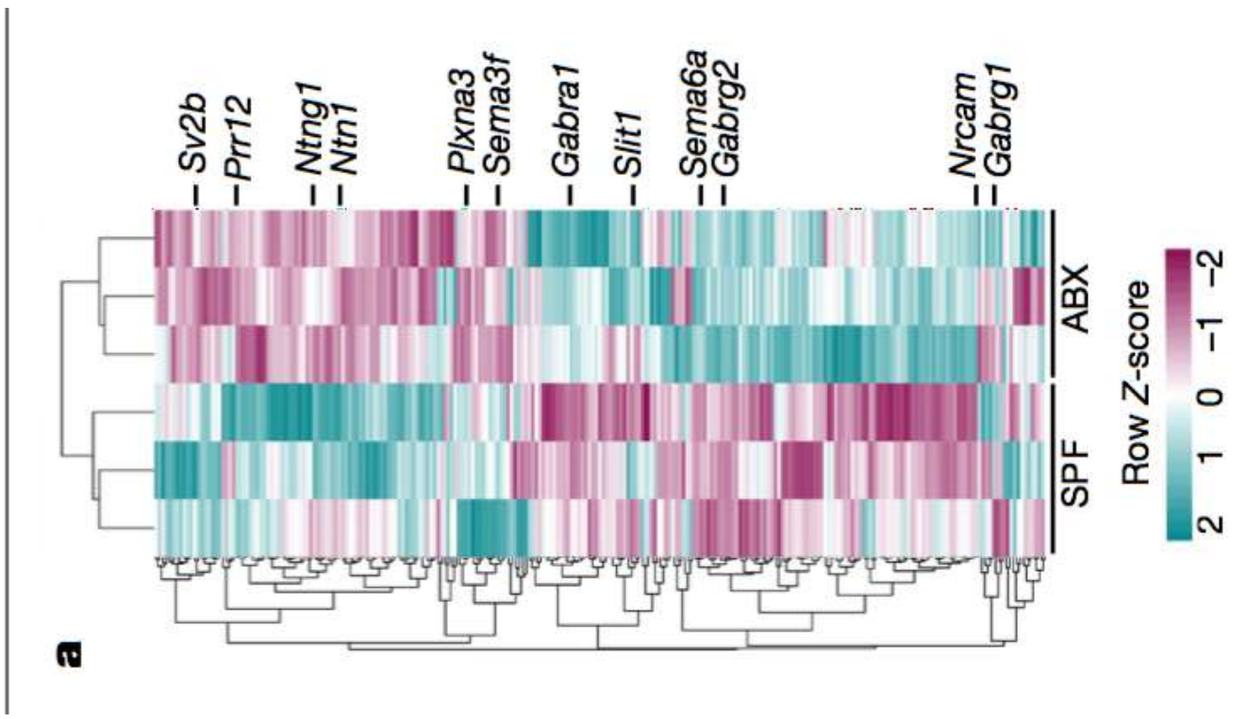


iHMP, 2019

The maternal microbiome modulates fetal neurodevelopment in mice

‘**Dysbiosis’ of the maternal gut microbiome**, in response to challenges such as infection, **altered diet and stress during pregnancy**, has been increasingly associated with **abnormalities in brain function and behaviour of the offspring**

Manipulation of the maternal microbiome and microbial metabolites during pregnancy yielded adult o spring with **altered** tactile sensitivity in two aversive somatosensory behavioural tasks



SPF: specific-pathogen-free

Genes that are differentially expressed in E14.5 brains from offspring of SPF and ABX dams

Vuong et al., 2020

Pregnancy and maternal gut microbiome

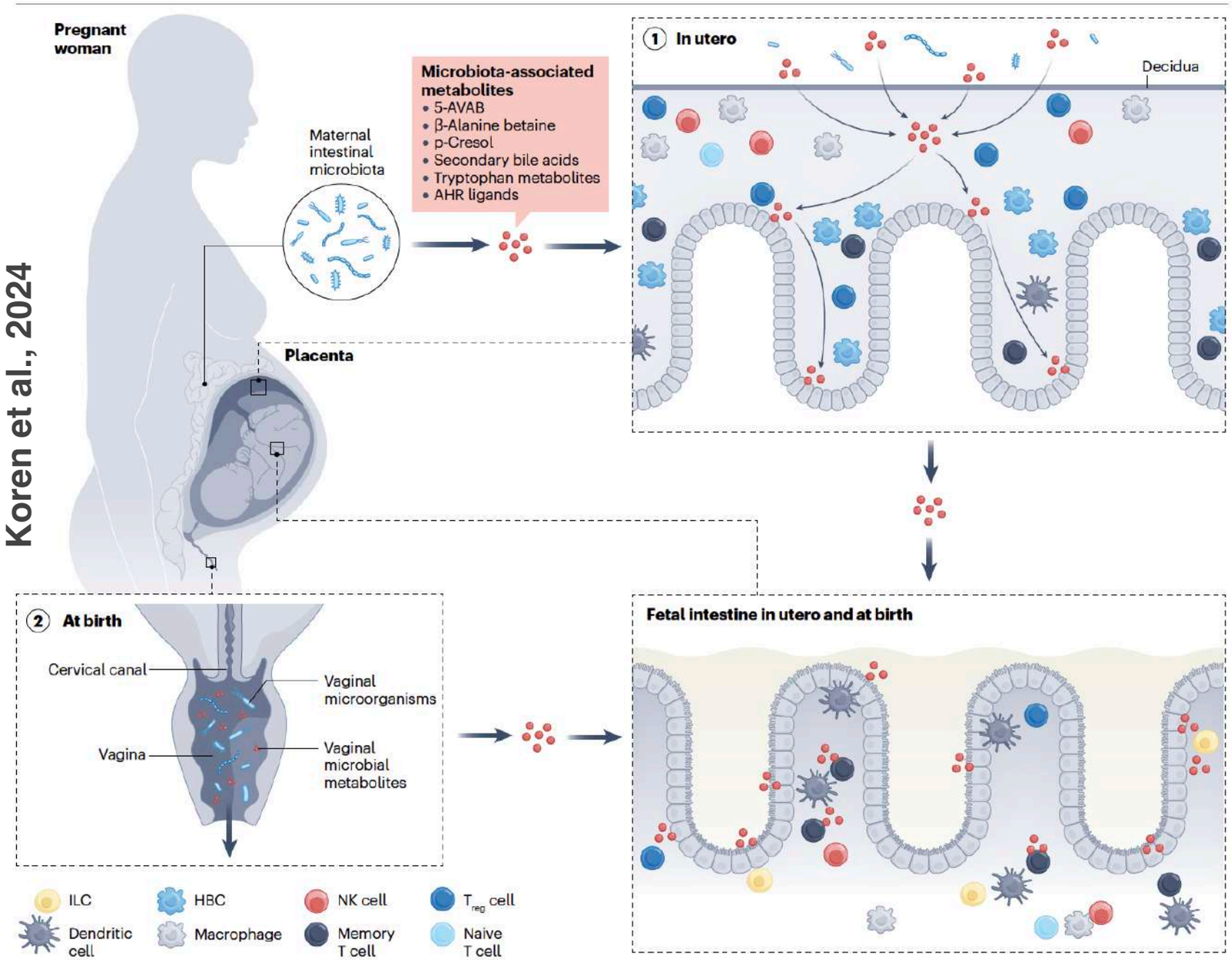
- The maternal gut microbiome changes during pregnancy, with the most drastic changes occurring towards the end of pregnancy

- Increase in Proteobacteria and Actinobacteria in late pregnancy as well as a decrease in short-chain fatty acid (SCFA) producers as gestation progresses

- The maternal gut microbiome has been shown to be involved in multiple phenotypes: including weight gain, low-grade inflammation and insulin resistance

- **Maternal immunity and microbial metabolites during pregnancy, microbial transfer during birth, and transfer of immune factors, microorganisms and metabolites via breastfeeding provide critical sources of early-life microbial and immune training, with important consequences for human health**

Microbial metabolites from the maternal microbiome contribute to fetal and neonatal immune development



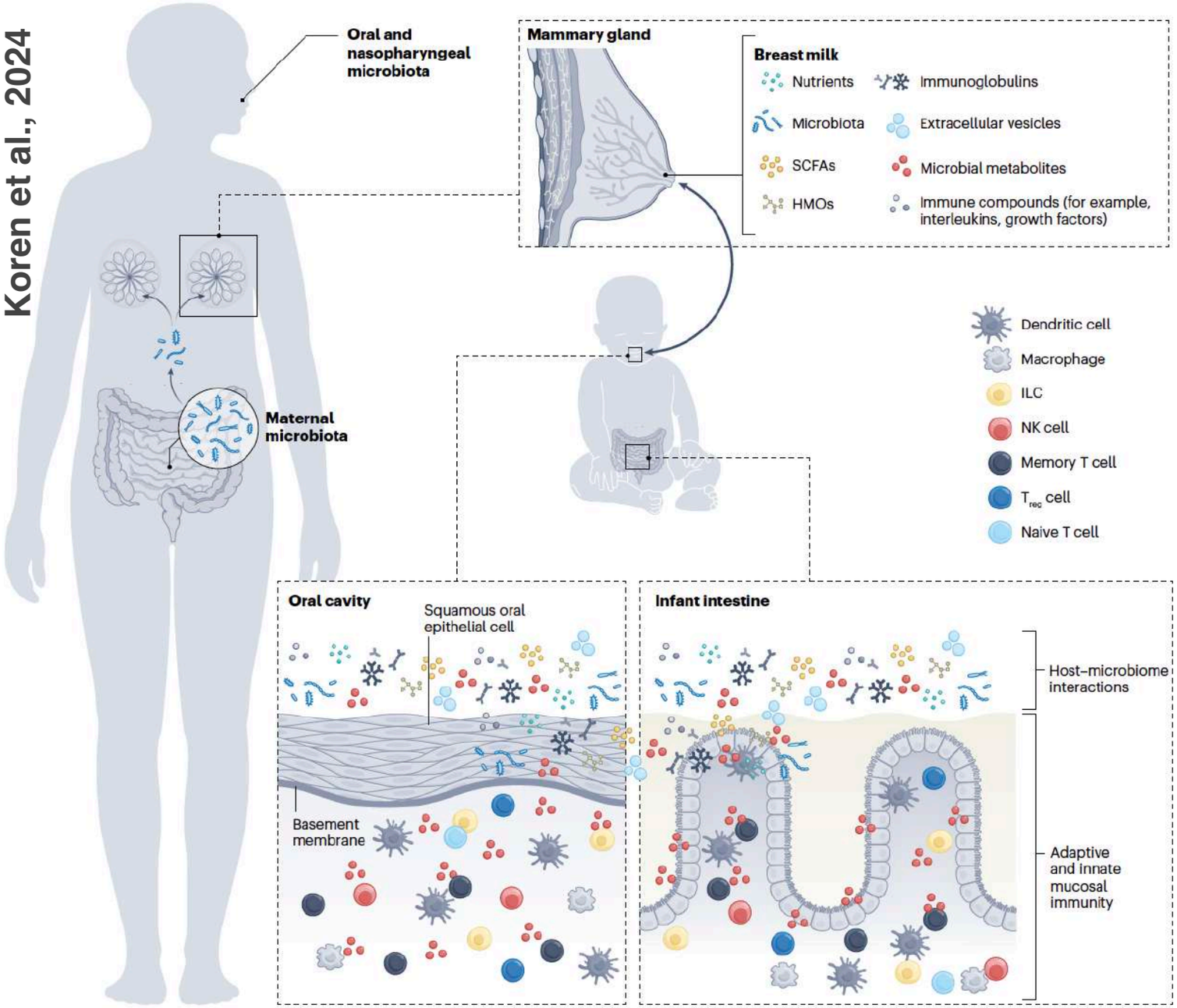
Koren et al., 2024

1) Maternal intestinal microbiota derived metabolites influence immune development in the placenta and fetal intestine in utero

2) The vaginal microbiome and microbial metabolites contribute to intestinal immune development at birth

5-AVAB, 5-aminovaleric acid betaine; AHR, aryl hydrocarbon receptor; HBC, Hoffbauer cell; ILC, innate lymphoid cell; NK, natural killer; Treg cell, regulatory

Human milk microorganisms and their metabolites support the gut microbiome and immune system in the offspring



Breast milk composition is complex and unique

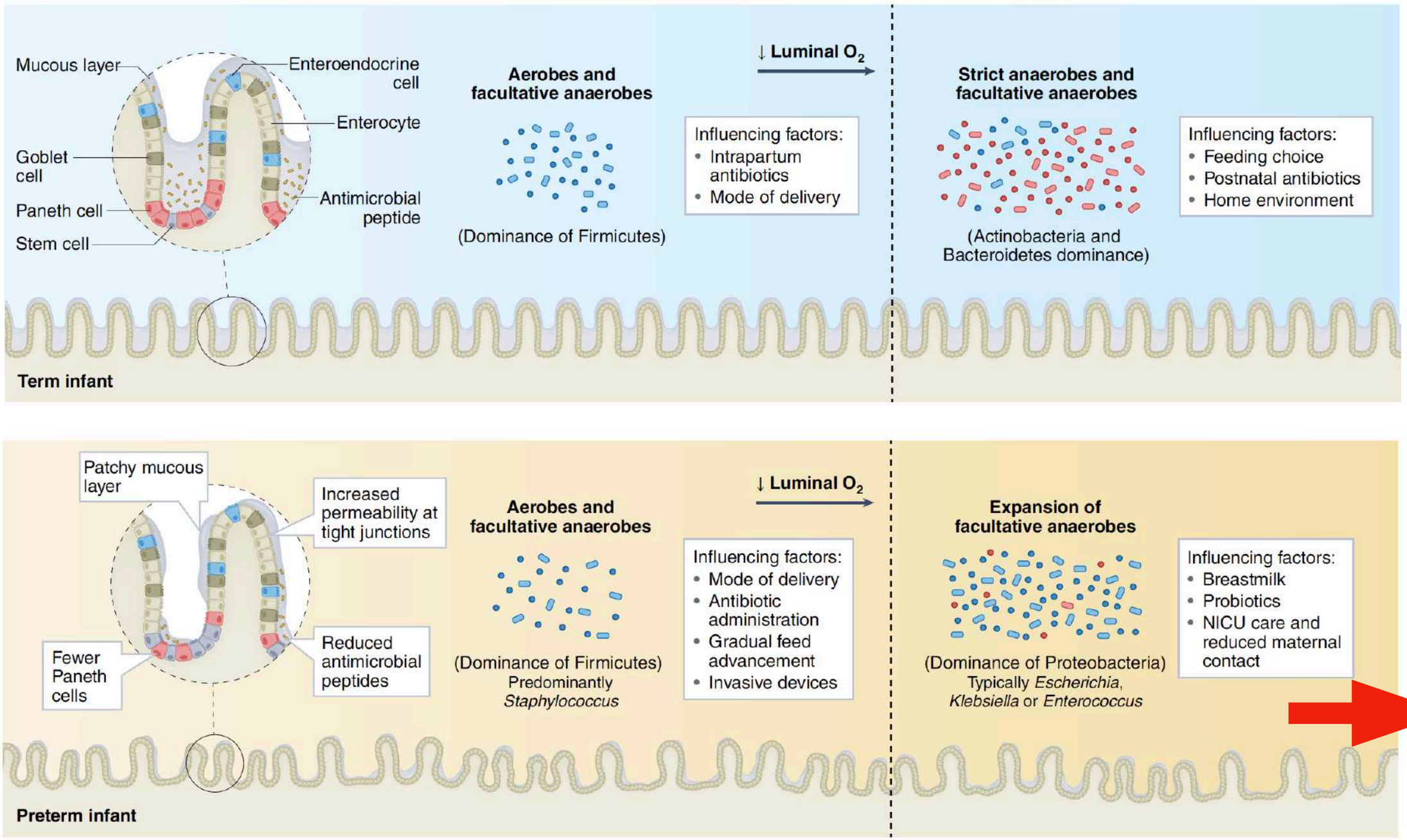
Milk contains nutrients and specific bioactive compounds:

- microbiota & metabolites (including SCFAs)
- microorganism-derived products (cell walls, membrane, DNA, specific secreted proteins, and other fragments or structures)
- human milk oligosaccharides (HMOs)
- immune-related compounds (secretory IgA, immunoglobulins, lactoferrin and lysozyme)
- CD14 T cells, cytokines, growth factors, defensins
- extracellular vesicles, which can cargo microRNAs, long non-coding RNAs, proteins and lipids, as well as maternal cells, including leukocytes and stem cells

This complexity is key to adaptive and innate mucosal immunity in the neonate and to support neonatal microbial assembly by interacting closely with intestinal epithelial cells and intestinal receptors signalling to the immune system (modulating the adaptive immune response via a T helper cell response and stimulating regulatory T (Treg) cells and regulatory B cells) and generating immune tolerance

ILC, innate lymphoid cell; NK, natural killer

Term and Preterm infant



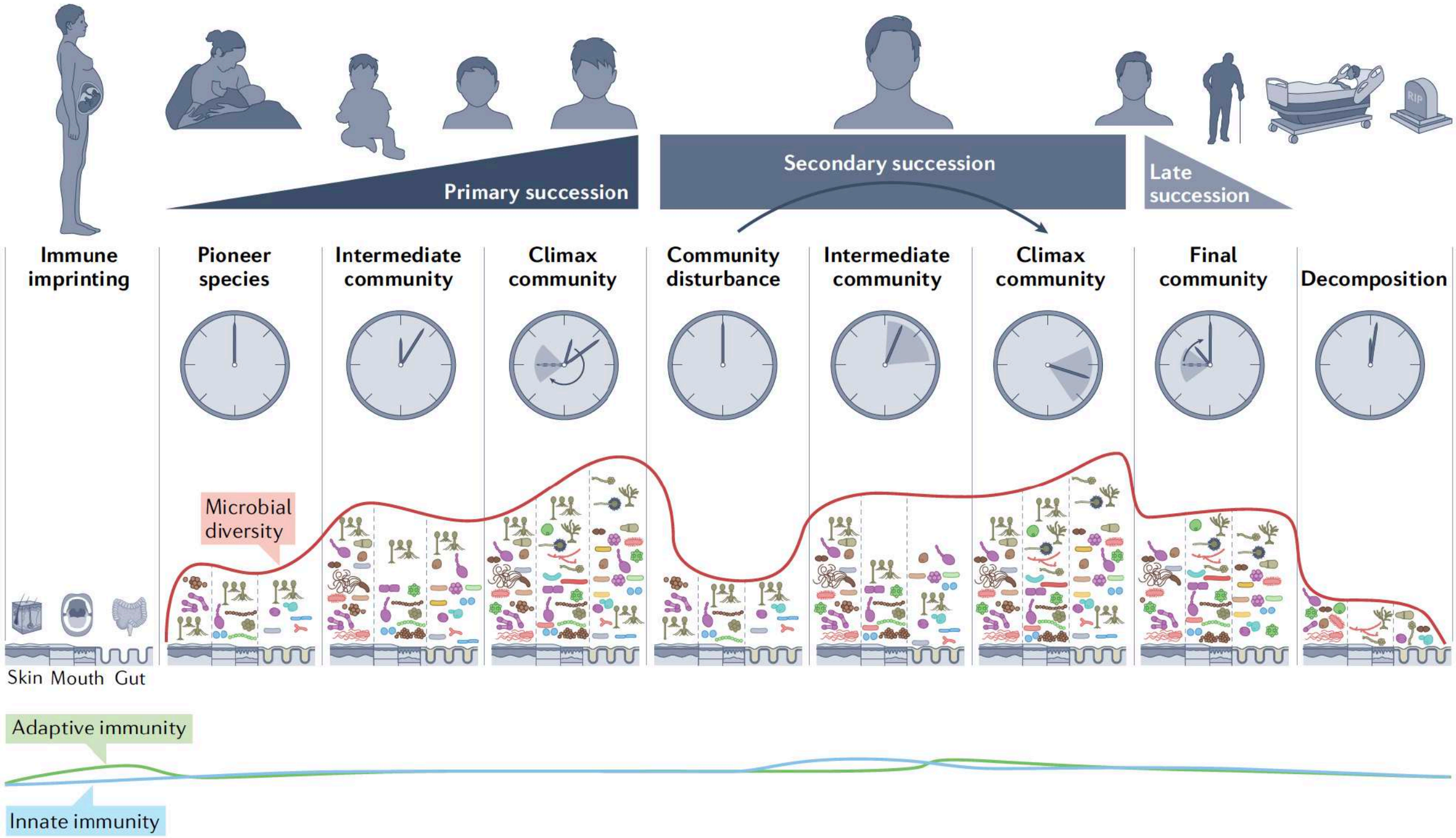
Healy et al., 2022

Anatomical differences, successional development of bacterial communities, and factors influencing microbiome establishment in term and preterm infants

The intestinal anatomy of the preterm infant is immature, with poor differentiation of epithelial cells leading to weakened gut-barrier defences

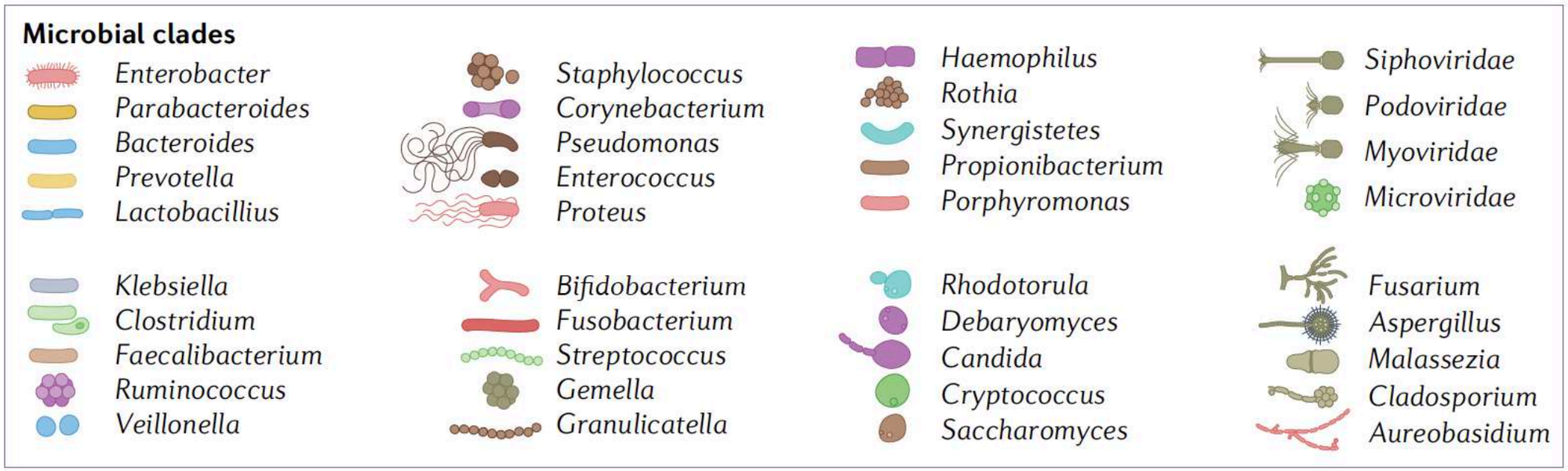
The initial colonizers of the gut are similar for term and preterm infants but, over time (hours to days) (represented by the vertical dashed line), various influencing factors and alterations in community dynamics lead to the establishment of considerably different populations of microbiota

The succession of the human microbiota from conception to death

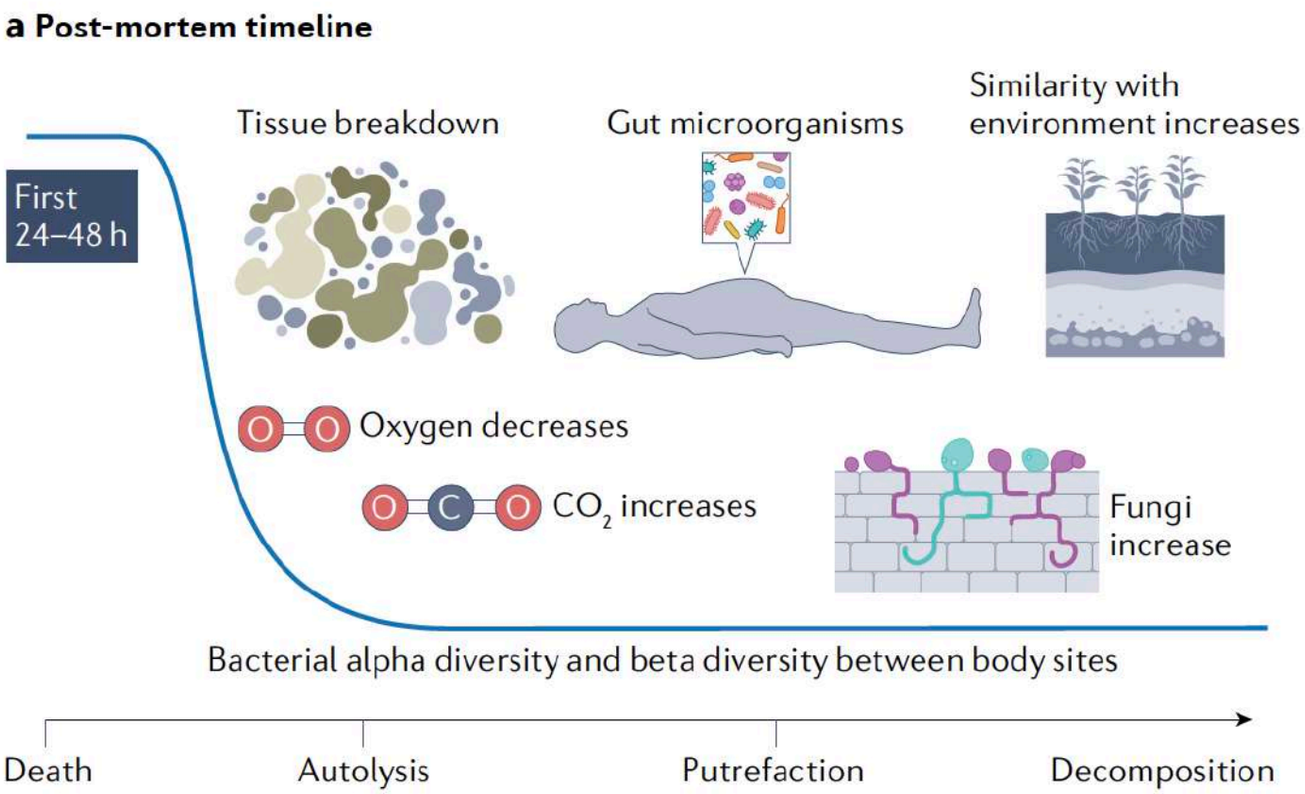


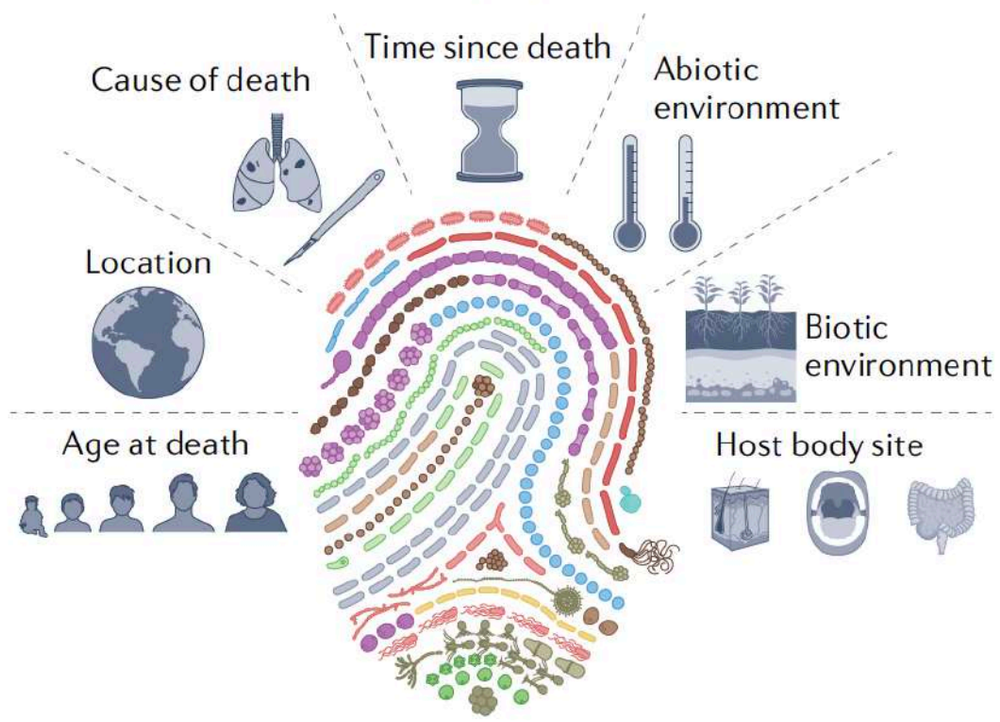
Martino et al., 2022

- **Immune imprinting begins before birth through the mother's microbiota and its metabolites**
- **Initial colonization of pioneer species begins at birth, and body site- specific microbial communities emerge**
- **These communities increase in complexity until they reach a relatively stable community structure**


Microbial clades
Enterobacter
Parabacteroides
Bacteroides
Prevotella
Lactobacillius
Staphylococcus
Corynebacterium
Pseudomonas
Enterococcus
Proteus
Haemophilus
Rothia
Synergistetes
Propionibacterium
Porphyromonas
Siphoviridae
Podoviridae
Myoviridae
Microviridae
Klebsiella
Clostridium
Faecalibacterium
Ruminococcus
Veillonella
Bifidobacterium
Fusobacterium
Streptococcus
Gemella
Granulicatella
Rhodotorula
Debaryomyces
Candida
Cryptococcus
Saccharomyces
Fusarium
Aspergillus
Malassezia
Cladosporium
Aureobasidium

The microbiota after death





Martino et al., 2022

- After death the microbiota is relatively stable in the first 24–48 h
- The tissue then begins to break down during autolysis, leading to bloom in the gastrointestinal microbiota and a decrease in alpha diversity and a decrease in beta diversity between body sites
- During putrefaction, the role of fungi increases, and the microbiota of the body becomes more similar to the microbiota of the surrounding environment
- The post- mortem microbiota is unique to each body and is distinct between bodies on the basis of the time since death, cause of death, environment, location and age at death, at the beginning, between body sites

In sum the roles of the microbes in the human ecosystem are:

1. Digestion and Metabolism

- Helps break down complex carbohydrates, fiber, and proteins that the human body cannot digest alone
- Produces essential **short-chain fatty acids (SCFAs)** like butyrate, acetate, and propionate, which provide energy to gut cells and regulate metabolism
- Aids in the synthesis of **vitamins** (*e.g.*, B vitamins, vitamin K)

2. Immune System Regulation

- Plays a crucial role in **training and modulating the immune system**, helping to distinguish between harmful and harmless microbes
- Prevents infections by **competing with pathogens** for nutrients and space (colonization resistance)
- Produces **anti-inflammatory** and immune-modulating compounds

3. Protection Against Pathogens (Defense Mechanism)

- Maintains gut barrier integrity by strengthening **tight junctions** between intestinal cells
- Produces antimicrobial substances to prevent pathogen overgrowth

4. Influence on Brain and Behavior (Gut-Brain Axis)

- Produces **neurotransmitters** like serotonin, dopamine, and GABA, which influence mood and cognitive functions
- Regulates the **gut-brain axis**, impacting mental health conditions like anxiety and depression

5. Role in Disease Prevention and Development

- **Dysbiosis (microbial imbalance)** is linked to diseases like **inflammatory bowel disease (IBD), obesity, type 2 diabetes, allergies, and even neurodegenerative disorders**
- **A healthy gut microbiome reduces inflammation and supports overall metabolic and immune balance**