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THE FUNCTIONAL NATUROPATH

CLINICAL WEBINAR

Rebuilding the Gut Microbiome: Clinical Strategies to Support Keystone Gut Bacteria

Presented by: Dan Sipple
Naturopath, Nutritionist
and Herbalist

Presenter | Dan Sipple

Dan Sipple BHSc (Naturopathy) is a fully qualified Naturopath, Nutritionist, and Herbalist known as “The Functional Naturopath.” Having journeyed from illness to recovery himself, Dan combines deep personal empathy with rigorous clinical expertise.

His practice is defined by fully customised treatment plans rather than generic protocols, frequently integrating Western diagnostics with functional testing to manage complex cases in autoimmunity, gut microbiome modulation, and hormone optimisation. Dan is an avid researcher committed to bridging the gap between allopathic and complementary medicine.

Dan resides in the Mollymook/Ulladulla region of South Coast NSW, where he practices solely online.

Host | Linda Dal Molin

Linda Dal Molin is the Director of Sales and Education for Designs for Health Australia.

Linda has a Masters in Human Nutrition, Bachelor of Health Science (Complementary Medicine), Advanced Diploma Naturopathy. She has been a practitioner for over 26 years and worked in the natural health space for 30 years having owned and managed health food stores and a large multi-modality allied health clinic. Linda practices on Saturday mornings in a busy clinic in Sydney's inner city with a focus on women's and gut health.

Linda has developed a strong relationship with the Designs for Health practitioner community. She will moderate the Q&A discussion with Dan in this webinar and engage our live Designs for Health practitioner community to bring insight and practical clinical pearls for all.

PART 1. Keystone and Beneficial Bacteria: The Architects of Gut Health

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Learning objectives:

1. Define keystone bacteria and their ecological roles in gut balance.
2. Identify keystone species such as *Faecalibacterium prausnitzii*, *Akkermansia muciniphila*, *Roseburia spp*. and others.
3. Understand their contribution to mucin regulation, immune tolerance, and production of key metabolites including butyrate and secondary bile acids.

Defining Keystone Bacteria

- Keystone bacteria are those whose presence disproportionately shapes the structure and function of the gut ecosystem — *much like keystone species in ecology.*
- They maintain microbial diversity, mucosal barrier integrity, and metabolic cross-feeding networks that sustain the broader microbiome.
- Loss of these keystone organisms is associated with dysbiosis, inflammation, and metabolic or autoimmune disorders.

Top Keystone Species & Their Ecological Roles

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1. *Faecalibacterium prausnitzii*

- One of the most abundant commensals in healthy adults.
- A major butyrate producer, fueling colonocytes and maintaining tight junction integrity.
- Exhibits anti-inflammatory effects via IL-10 induction and NF-κB inhibition.
- Depletion is consistently linked to IBD, IBS, depression, and metabolic syndrome.

2. *Akkermansia muciniphila*

- A mucin-degrading specialist residing in the mucus layer.
- Stimulates mucin turnover, indirectly strengthening the epithelial barrier.
- Enhances GLP-1 secretion, metabolic regulation, and immune tolerance.
- Its abundance is inversely correlated with obesity, insulin resistance, and inflammation.

Top Keystone Species & Their Ecological Roles cont...

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3. *Roseburia spp*.

- Another key butyrate-producing genus, synergistic with *F. prausnitzii*.
- Converts dietary fibers and polyphenols into short-chain fatty acids (SCFAs) that reduce intestinal pH and suppress pathogens.
- Supports gut motility and energy balance.

Other noteworthy beneficial species of the biome

- *Bifidobacterium adolescentis*,
- *Eubacterium rectale*
- These species contribute to cross-feeding — converting complex carbohydrates and polyphenol metabolites into substrates used by the aforementioned keystone species.

Keystone Species Role in Biome Homeostasis

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Function	Mechanism	Clinical impact
Mucin regulation	***Akkermansia*** stimulates goblet cell mucin synthesis while recycling mucin for microbial nutrition	Preserves barrier, prevents endotoxemia
Butyrate production	***Roseburia + F. prausnitzii*** ferment fibers to produce butyrate	Fuels colonocytes, tight junction integrity, anti-inflammatory signalling
Secondary bile acid metabolism	***Keystone species*** convert primary bile acids → secondary forms	Shapes microbial ecology, immune tone, lipid metabolism
Immune tolerance	SCFA-mediated Treg activation, GALT signalling	Reduces Th17 dominance, improves oral and systemic tolerance

Key Evidence

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Faecalibacterium prausnitzii Produces Butyrate to Maintain Th17/Treg Balance and to Ameliorate Colorectal Colitis by Inhibiting Histone Deacetylase 1
https://pubmed.ncbi.nlm.nih.gov/29796620/

Akkermansia muciniphila Adheres to Enterocytes and Strengthens the Integrity of the Epithelial Cell Layer
https://pubmed.ncbi.nlm.nih.gov/25795669/

Roseburia spp.: a marker of health?
https://pubmed.ncbi.nlm.nih.gov/28139139/

The Role of *Faecalibacterium, Roseburi*a, and Butyrate in Inflammatory Bowel Disease
https://karger.com/ddi/article-abstract/40/6/793/823101/The-Role-of-Faecalibacterium-Roseburia-and?

Multiomics analysis reveals the biological effects of live *Roseburia intestinalis* as a high-butyrate-producing bacterium in human intestinal epithelial cells
https://pubmed.ncbi.nlm.nih.gov/37596881/

Part 2. Short-Chain Fatty Acids (SCFAs): Metabolic Messengers of the Microbiome

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SCFA Overview

Short-chain fatty acids (SCFAs) are microbial metabolites of dietary fibre produced primarily in the colon.

- Major SCFAs: acetate, propionate, butyrate
- Microbial sources of the three major SCFA's
  - **Butyrate:** *Faecalibacterium prausnitzii, Roseburia spp., Eubacterium rectale*
  - **Propionate:** *Bacteroidetes, Veillonella*, some *Firmicutes*
  - **Acetate:** most anaerobic gut bacteria, including *Bifidobacterium* and *Lactobacillus* species

Mechanisms & Metabolic Roles

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Acetate

- Serves as a systemic energy substrate for peripheral tissues.
- Influences appetite regulation via hypothalamic and gut–brain signalling.

Propionate

- Modulates hepatic gluconeogenesis and lipid metabolism.
- Stimulates satiety hormones (PYY, GLP-1) and affects energy balance.

Butyrate

- Primary fuel for colonocytes, supporting mucosal integrity.
- Acts as an epigenetic regulator via histone deacetylase (HDAC) inhibition. (Butyrate blocks these inhibition-enzymes so genes become easier to access / switch on. Imagine unrolling a tightly coiled carpet so the pattern becomes visible again)
- Exhibits anti-inflammatory activity, including promotion of colonic T-reg cells.

SCFAs and Host Signalling

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- SCFAs interact with G-protein coupled receptors (FFAR2/FFAR3) to influence immunity and metabolism. In simple terms, when these receptors are switched on, they help control inflammation, gut immunity, and regulate how your body uses energy.

- SCFA's create cross-talk with bile acid receptors (FXR and TGR5) which improves:
  - Hepatic lipid and glucose metabolism
  - Energy expenditure and systemic metabolic health
  - Intestinal barrier and mucosal homeostasis

Summary

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SCFA	Key Actions	Microbial Sources
Acetate	Energy substrate, appetite modulation	*Bifidobacterium, Lactobacillus*
Propionate	Hepatic gluconeogenesis, satiety, lipid metabolism	*Bacteroidetes, Veillonella*
Butyrate	Colonocyte fuel, HDAC inhibition, anti-inflammatory, T-reg activation	*Faecalibacterium, Roseburia, Eubacterium*

Key Evidence

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3. Furusawa Y, et al. Nature 2013; 504: 446–450 — butyrate promotes T-reg differentiation via HDAC inhibition.
4. Den Besten G, et al. J Lipid Res 2013; 54: 2325–2340 — SCFAs in host lipid and glucose metabolism.
5. Frost G, et al. Nat Commun 2014; 5: 3611 — acetate reduces appetite via central homeostatic mechanisms.
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8. Visekruna A, et al. Front Immunol 2021 — SCFA–bile acid receptor crosstalk.
9. Byrne CS, et al. Nutrients 2015; 7: 2839–2865 — SCFAs regulate appetite and energy homeostasis.
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Part 3. Bile Acids: The Overlooked Signalling Molecules in Gut-Liver Axis Health

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Learning objectives:

1. Describe the synthesis and transformation of bile acids (primary → secondary BA's).
2. Explain the microbiota's role in bile acid deconjugation and conversion (via *Bacteroides*, *Clostridium*, *Lactobacillus*).
3. Identify the impact of bile acid deficiency or stagnation on fat digestion, microbiome diversity, and mucosal health.
4. Understand the interaction between SCFAs and bile acid receptors (FXR, TGR5) in regulating metabolism and inflammation.
5. Discuss dietary and supplemental strategies to restore healthy bile acid metabolism.

1. “Physiology primers”: From BA Synthesis → Enterohepatic Cycle → Cell Signalling

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- Synthesis: Primary bile acids (*cholic acid, chenodeoxycholic acid*) are synthesised from cholesterol in the liver, conjugated with glycine or taurine, stored in the gallbladder and released into the duodenum after a meal to emulsify dietary fats.

- Enterohepatic circulation: ~95% of bile acids are reabsorbed in the terminal ileum and returned to the liver; a small fraction **reaches the colon and is transformed by the microbiota into secondary bile acids** *(eg. deoxycholic acid [DCA], lithocholic acid [LCA]).*

Where TUDCA fits in:
TUDCA (tauroursodeoxycholic acid) is a taurine-conjugated form of the secondary bile acid UDCA. It is naturally present in small amounts within the bile acid pool and forms when microbes convert primary bile acids, after which the liver reconjugates them with taurine. TUDCA supports bile flow, protects hepatocytes, and enhances endoplasmic reticulum and mitochondrial stability— which highlights its therapeutic potential.

Bile Acids continued…

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- **Signalling roles:** Bile acids are ligands for nuclear and membrane receptors — chiefly **FXR** (farnesoid X receptor) and **TGR5** (GPBAR1). Activation of these receptors regulates hepatic lipid and glucose metabolism, gut motility, GLP-1 release and intestinal barrier function.

Thus, bile acids when made cleanly and in appropriate amounts, can be thought of as therapeutic tools not only to optimise digestion, but also to prime metabolic signalling and be useful in weight loss strategies.

2. Microbial Transformations of BA's — Who Does What ?

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- **Deconjugation**: Many gut bacteria (eg. Lactobacillus, Bifidobacterium, Clostridium species) express bile salt hydrolases (BSH) that remove taurine/glycine from conjugated bile salts — the first step to further conversion.

- **7α-dehydroxylation** → secondary BAs: A narrower set of anaerobes (usually Clostridium families) carry the enzymatic machinery for 7α-dehydroxylation, converting primary bile acids into secondary bile acids → DCA/LCA. These conversions profoundly change bile acid signalling properties.

- **Ecological effects:** Bile acids themselves are antimicrobials — they shape the microbiome by selecting bile tolerant bacterial species (eg. Bilophila, some Bacteroides) while suppressing others. In other words, some pathobionts like Bilophila wadsworthia may persist or even thrive in certain bile acid environments, especially when host diet or bile acid conjugation favors their metabolism. The antimicrobial effect of BA's is selective and depends heavily on bile acid type and the hosts diet i.e. high saturated fat intake can be detrimental to BA synthesis by promoting the wrong types.

3. Clinical Consequences of Bile Acid Deficiency or Stagnation

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When bile flow or transformation is insufficient (cholestasis, low bile production, post-cholecystectomy patients with poor bile release, or poor biliary motility), you may see:

- Impaired fat digestion & fat-soluble vitamin malabsorption → pale, greasy or floating stools, post-prandial bloating after fat.
- Lower microbiome diversity and dysbiosis — reduced secondary bile acid pool can reduce colonisation resistance and alter taxa (loss of Clostridium XIVa, Faecalibacterium, Akkermansia).
- Mucosal vulnerability: less detergent action and reduced signalling through FXR/TGR5 impairs barrier function, mucosal immunity and nutrient sensing. Low bile acid activity is linked with steatosis, cholestatic liver dysfunction and altered intestinal motility.

Clinical indicators: pale/greasy stools, steatorrhea, RUQ discomfort after fatty meals, bloating after fats, and lab clues (fat-soluble vitamin deficits, abnormal LFTs).

4. SCFAs ↔ Bile Acid Receptor Cross-talk (Why This Matters for Metabolism & Inflammation)

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- SCFAs regulate bile-responsive receptors: Butyrate and other SCFAs modulate expression/activity of TGR5 and downstream pathways, promoting GLP-1 release and anti-inflammatory signalling in the gut and liver. This amplifies insulin sensitivity and metabolic regulation. *(Emerging evidence shows butyrate can increase TGR5 signalling and promote M2 macrophage polarization via TGR5-linked pathways)*.

- Bile acids shape microbiome & SCFA producers: Secondary bile acids influence the abundance/activity of SCFA-producing taxa such as Faecalibacterium and Akkermansia; conversely, SCFA patterns influence bile acid receptor expression — *a reciprocal regulatory loop that links diet → microbes → host metabolism*.

5. Practical Strategies to Restore Healthy Bile Acid Metabolism — Strategy & Mechanism

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1. Bitter herbs (gentian, dandelion, artichoke, andrographis, ginger)

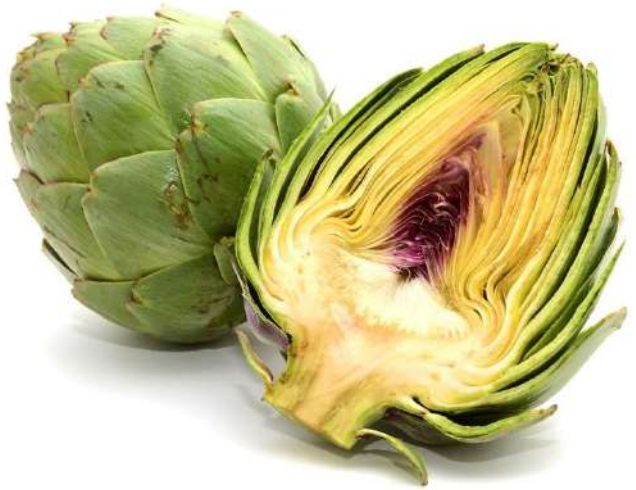
- Mechanism: Oral bitter receptors trigger a cephalic reflex increasing saliva, gastric secretions and gallbladder contractility; some bitters directly increase bile flow and improve fat emulsification. Useful pre-meal to improve choleresis and fat digestion.

2. Dietary fat timing & composition

- Mechanism: Small, regular moderate-fat meals stimulate physiologic gallbladder emptying; include monounsaturated and omega-3 fats (olive oil, oily fish) rather than large saturated-fat meals which can impair bile handling and inflammation.
- Meal timing (avoid large, late meals) supports coordinated bile release.

3. Phosphatidylcholine (PC) / lecithin

- Mechanism: PC is a major biliary phospholipid that protects epithelial cells from detergent injury and improves micelle formation; dietary/supplement PC may improve bile quality and protect mucosa, and animal data show PC modifies gallstone risk and bile composition



5. Practical Strategies to Restore Healthy Bile Acid Metabolism — Strategy & Mechanism Continued…

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4. Taurine & glycine precursors

Mechanism: Conjugation with taurine/glycine increases bile acid solubility and detergent efficiency; ensuring adequate protein and amino acid availability supports conjugation and effective bile action. Taurine supplementation is sometimes used to support bile acid conjugation.

5. Choline (and avoidance of severe choline deficiency)

Mechanism: Choline is required for hepatic VLDL export and phosphatidylcholine synthesis — both support normal bile composition and prevent cholestasis. Dietary choline (eg eggs, soy, lecithin) supports bile health.

6. Prokinetic & pro-digestive supports (ginger, motility exercises, bitters)

Mechanism: Improve gastric emptying and coordinated vagal drive to the gallbladder; reduces stasis and enhances enterohepatic circulation.

7. Probiotics / targeted microbial modulation (Lactobacillus, Bifidobacterium, selected Clostridia)

Mechanism: BSH-expressing strains can modulate deconjugation; other probiotics can support SCFA production and restore taxa that influence secondary bile acid pools. Use targeted strains rather than indiscriminate high-dose blends.

5. Practical Strategies to Restore Healthy Bile Acid Metabolism — Strategy & Mechanism Continued…

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8. Bile acid replacement / stimulants (pharmaceutical)

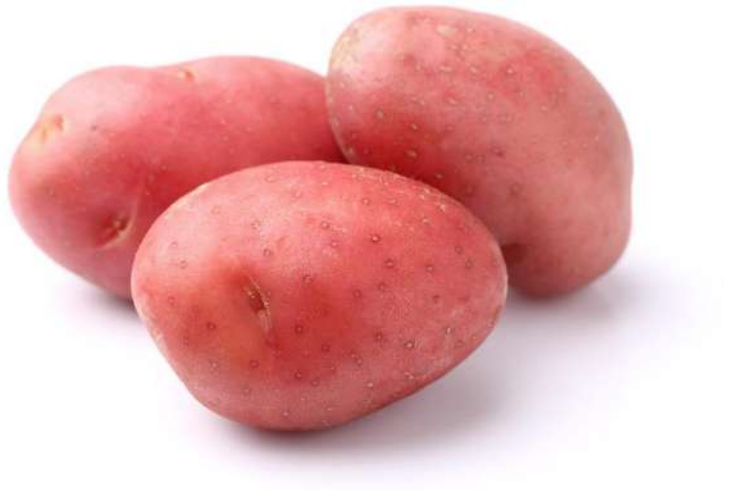
Mechanism: In confirmed bile acid deficiency or cholestasis, ursodeoxycholic acid (UDCA) or controlled bile salt therapy under specialist guidance may be indicated; conversely, bile sequestrants are used in bile acid diarrhoea. These are prescription tools, not first-line nutraceuticals.

9. Support liver detox & phase II pathways (glutathione, taurine, glycine, B-vitamins)

Mechanism: Improve hepatic handling of bile acids, conjugation capacity and prevent toxin-mediated cholestasis.

10. Dietary prebiotics & fibre diversity

Mechanism: Feed SCFA producers (eg. resistant starch, inulin) to encourage butyrate production and downstream TGR5/FXR benefits — but introduce gradually to avoid gas if SIBO present.



Cautions and When to Hold Off

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- Acute gallbladder inflammation (cholecystitis), biliary obstruction or suspected stones — do not use pro-choleretic herbs or stimulants until obstruction is ruled out. In obstruction, increasing flow can cause pain, cholangitis or pancreatitis.
- Active bile acid diarrhoea / BAM or severe diarrhoea — strategies that increase free bile acids in the colon may worsen diarrhoea.
- Suspected sphincter of Oddi dysfunction or recent biliary surgery — specialist review required before choleretic use.
- Drug interactions & mineral effects: bile sequestrants reduce absorption of fat-soluble meds and vitamins; phosphatidylcholine may alter lipid panels in some contexts. Monitor labs if you're supplementing.

Quick Clinical Summary

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- Primary bile acids (cholic, chenodeoxycholic) are made in the liver; secondary bile acids (DCA, LCA) are formed by gut microbes via deconjugation and 7α-dehydroxylation.
- Healthy bile flow shapes the microbiome by suppressing overgrowth and supporting beneficial species like Akkermansia and Faecalibacterium.
- When bile acids are low or stagnant → reduced diversity, SIBO tendencies, mucosal irritation, and fewer secondary bile acids.
- Bile acids activate FXR/TGR5, regulating motility, inflammation, and epithelial turnover.
- SCFAs (especially butyrate) enhance TGR5 signalling and epithelial repair, creating a feedback loop that supports bile acid function.
- Together, bile acids + SCFAs maintain rhythmic gut motility and healthy mucosal renewal.

Key Evidence

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Part 4. Polyphenols : Clinical Impact on SCFAs and Bile Acid Balance

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Learning objectives:

1. Review human trial evidence for Pomella® (pomegranate extract) increasing SCFA production and microbial diversity.
2. Discuss mechanisms linking polyphenols to bile acid metabolism
3. Explore antioxidant and anti-inflammatory synergy between punicalagins, urolithin A, and microbial metabolites.

Human Trial Evidence for Pomella® — SCFA Production & Microbial Diversity

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Clinical trial summary

Design: Randomized, double-blind, placebo-controlled trial, 4 weeks, healthy adults (age 25–55), Pomella® 250 mg/day vs placebo; whole-genome shotgun sequencing of stool + plasma SCFA & urolithins measured.

Results;

- Circulating SCFAs: Increases in propionate (large % rise reported) and acetate with Pomella supplementation vs placebo; SCFA changes correlated with microbial functional shifts.
- Microbial taxa / diversity: No large change in overall alpha diversity reported in small cohort, but select increases in beneficial SCFA-producing taxa were observed (e.g., *Roseburia, Faecalibacterium, Ruminococcus spp*. reported in press summaries and trial text). The study was small (n≈18) so changes were selective rather than global.
- Urolithins: Higher circulating urolithin levels observed in the Pomella group (consistent with enhanced gut microbial conversion of ellagitannins → urolithins).

Mechanisms Linking Polyphenols → Microbiome → Bile Acid Metabolism

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Overview (conceptual flow)

1. **Ingestion**: Pomegranate ellagitannins (punicalagins, punicalin, ellagic acid glycosides) reach the colon largely intact. Microbial enzymes hydrolyse punicalagins → ellagic acid → further microbial conversion to urolithins (Uro-A, Uro-B, etc.). (namely - *Gordonibacter* species!)

2. **Microbial community shifts:** Ellagitannin metabolism selects for taxa that can degrade these polyphenols and/or benefit from their breakdown products; concurrently, polyphenols have antimicrobial/modulatory effects (suppressing some species, favoring SCFA producers). This alters microbial functional potential relevant to bile acid transformations.

3. **Bile acid pathway modulation:** Changes in microbiome composition and function alter bile salt hydrolase (BSH) activity and 7α-dehydroxylation capacity *(the key microbial steps that convert primary → secondary bile acids).* Polyphenol-driven shifts can therefore reduce or reshape secondary bile acid formation. Clinical/experimental data show PE (pomegranate extract) intake reduced fecal secondary bile acids (and coprostanol) in a small dyslipidaemic cohort, an effect correlated with Uro-A production.

Explore Antioxidant and Anti-inflammatory Synergy Between Punicalagins, Urolithin A, and Microbial Metabolites

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Antioxidant & anti-inflammatory synergy: punicalagins, urolithin A, microbial metabolites (SCFAs)

How the pieces fit:

- Punicalagins (parent compounds): Strong direct antioxidant activity in vitro; inhibit inflammatory enzyme pathways (e.g., NF-κB, COX2) and reduce oxidative stress markers in animal and cell models. As large tannins they have low systemic bioavailability but act in the gut and as substrates for microbial metabolism.
- Urolithin A (microbial postbiotic): Better bioavailability than parent tannins; shown in human trials and preclinical studies to stimulate mitophagy, improve mitochondrial function, reduce systemic inflammation markers (e.g., CRP), and have antioxidant/anti-inflammatory actions via modulation of NF-κB, mitochondrial quality control, and other pathways. Clinical UA trials report decreased inflammatory biomarkers and improved mitochondrial biomarkers.
- SCFAs (microbial metabolites): Propionate and butyrate have recognized anti-inflammatory roles (regulate Tregs, epithelial barrier, and host metabolism) and act locally and systemically. Increased SCFA production (observed with Pomella) provides a further anti-inflammatory/metabolic axis.

Part 5. Fibre & Resistant Starch (Solnul®) and Prebiotics (Livaux®)

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Learning objectives:

1. Identify the fibre types that best feed keystone bacteria and SCFA production.
2. Understand when to use and when to pause high-fibre or resistant starch interventions (e.g., SIBO, methane dominance, diarrhoea).
3. Integrate Solnul® (RS2 potato starch) and **Livaux®** (gold kiwifruit) evidence for restoring *Faecalibacterium* and *Akkermansia*.

Human Trial Evidence — What the Trials Show

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Solnul® (Resistant Potato Starch — RPS)

- Design / dose: Randomized, double-blind, placebo-controlled trials and secondary analyses tested Solnul™ RPS at 3.5 g/day (and higher arms) for 4 weeks.
- Microbiome effects: Significant increases in Bifidobacterium and Akkermansia relative abundance in the low-dose (3.5 g/day) arm vs placebo. Improvements correlated with better stool form (reduced diarrhea/constipation associated bowel movements).
- Tolerability: Low dose was well tolerated with low incidence of gas/bloating — suggesting Solnul is suitable as an introductory/maintenance fermentable fibre.

Mechanisms — how these fibres shift keystone taxa and SCFAs

- Slow fermentation in the colon: RS2 resists small-bowel digestion and is fermented by saccharolytic bacteria in the proximal colon, producing acetate and propionate and, through cross-feeding, butyrate.
- Bifidogenic and Akkermansia stimulation: Low-dose RPS selectively promotes Bifidobacterium (primary degraders) and Akkermansia muciniphila creating an environment that supports butyrate producers indirectly.

Human Trial Evidence — What the Trials Show

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Livaux® (Gold kiwifruit powder)

- Design / dose: Randomised crossover trials of Livaux® (gold kiwifruit powder) capsules in healthy and functionally constipated groups (4-week interventions).
- Microbiome effects: In functionally constipated participants, Livaux® significantly increased Faecalibacterium prausnitzii (e.g., from ~3.4% → ~7.0% in one dataset) and improved bowel function/subjective symptoms.
- Effects were consistent across nearly all constipated participants in the study.

Mechanisms — how these fibres shift keystone taxa and SCFAs

- Prebiotic fibre + polyphenols: Gold kiwifruit supplies soluble fibre, oligosaccharides and polyphenols that preferentially feed Faecalibacterium prausnitzii and other butyrate-producers, improving butyrate output and mucosal anti-inflammatory signaling.
- Osmotic + motility effects: Kiwifruit also contains actinidin and other components that improve stool frequency and transit — creating favourable ecological conditions for F. prausnitzii to recover.

When To Use And When to Pause High-fibre / RS Interventions

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Use (good candidates)

- Low-grade constipation or intermittent constipation with low Faecalibacterium/Akkermansia
- Microbiome recovery after antibiotics (low dose maintenance)
- Wanting to promote mucosal health and butyrate production without aggressive fermentable prebiotics

Pause or be cautious (risk scenarios)

- Active SIBO (small intestinal bacterial overgrowth) — especially if proximal fermentation symptoms (bloating, rapid gas, early satiety) occur; RS can feed small intestinal microbes. Test/clear SIBO or start very low and titrate.
- Methane-dominant constipation/IMO — methane producers often flourish with carbohydrates that increase hydrogen substrate; cautious use and monitoring recommended.
- Active diarrhoea from rapid fermentation — lower dose or stop until stool form stabilises.
- Histamine intolerance / severe MCAS — some fibres/polyphenols transiently shift microbiome and can trigger symptoms in sensitive patients.

Practical rule: When in doubt, start low (e.g., Solnul 3.5 g/day), track Bristol stool, bloating and gas, and consider breath testing if symptoms worsen.

References

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Solnul / Resistant Potato Starch

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Livaux® / Gold kiwifruit

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Part 6. Lifestyle Impacts on Bile Acids and Beneficial Microbes

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Learning objectives:

1. Recognise how stress, circadian disruption, low dietary diversity, alcohol, and high PUFA/fried fat diets impair bile flow and SCFA synthesis.
2. Identify evidence-based lifestyle strategies:
   - Circadian-aligned meal timing
   - Regular movement and hydration to promote bile flow
   - Diverse plant-based diet for fibre/polyphenols
   - Stress reduction (vagal tone directly influences biliary secretion)

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Circadian disruption / irregular meal timing

- Bile acids and bile acid–related gene expression follow a daily rhythm; circadian misalignment (shift work, erratic eating) disrupts bile acid homeostasis and is linked to metabolic dysfunction. Meal timing interventions (early/large breakfast, time-restricted feeding) re-align bile acid rhythms and the microbiome.

Psychological stress & low vagal tone

- Stress alters gut microbiota composition and reduces vagal efferent activity; vagal efferents and CCK-vagal loops are key drivers of gallbladder contraction and bile release. Chronic stress therefore reduces biliary secretion and alters microbial metabolites including SCFAs.

THE FUNCTIONAL NATUROPATH

Low dietary diversity, low fibre, low polyphenols

- Diverse plant fibre and polyphenol intake consistently increase SCFA producers and SCFA concentrations; low diversity collapses keystone taxa and reduces butyrate output.

Alcohol and high-PUFA / fried fat diets

- Excess alcohol and repeatedly oxidised dietary lipids impair hepatocellular function and bile acid synthesis pathways, increase oxidative stress, and promote dysbiosis — all of which can blunt bile production and downstream microbial SCFA synthesis.

Movement & hydration (protective)

- Regular physical activity improves gallbladder motility, lowers gallstone risk, reduces hepatic steatosis, and correlates with healthier bile dynamics and microbiome profiles supportive of SCFA production. Hydration supports bile viscosity and transit.

Summary

THE FUNCTIONAL NATUROPATH

- Chronic stress and circadian misalignment blunt bile acid rhythm and secretion and dampen SCFA production by disrupting microbial rhythms and lowering keystone SCFA producers.
- Low dietary diversity, heavy alcohol use, and diets high in oxidised/polyunsaturated fats or fried foods further impair hepatic bile synthesis, gallbladder motility and the microbial networks needed to make butyrate.
- Simple, evidence-based countermeasures — timing meals to the biological day, regular low-moderate movement and good hydration, diverse plant fibre and polyphenols, and practices that boost vagal tone (breathing/meditation/sleep hygiene) — restore bile flow and SCFA output and are practical first-line interventions.

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Case Study Integration:

Transforming the Microbiome With Polyphenols & Targeted Prebiotics (Using Pomella® + Livaux® as in GI Biome Nourish)

Patient Overview

THE FUNCTIONAL NATUROPATH

Name: Jacinta, 39-year-old female

Occupation: High-stress finance job

History: Mild IBS-M, persistent bloating, irregular stools, post-meal cramping, fatigue, and brain fog. Heaviness in the RUQ, foul-smelling gas, 4-6 bowel motions daily (loose & inconsistent) < w/fatty meals.

Background:

- 2 rounds of antibiotics in past year (URTI)
- Low dietary plant diversity (≈8 plant foods/week)
- High PUFA/fried foods (convenience at her work place)
- Frequent meal skipping and late-night eating due to meetings and agenda shifts
- Sleep average 5.5 hrs/night

THE FUNCTIONAL NATUROPATH

Keystone Bacteria (Low)

- Faecalibacterium prausnitzii: Very low
- Akkermansia muciniphila: Low
- Bifidobacterium adolescentis: Borderline

SCFA Production

- Total SCFAs: Below range
- Butyrate: Low
- Propionate: Normal
- Acetate: Low-normal

Bile Acid Levels

- Secondary bile acids: Low-medium (dysbiosis pattern)
- Primary bile acids: Low (fat intol/RUQ pain)
- Indicating: sluggish bile flow + poor microbial conversion

Dysbiosis Features

- ↑ Alistipes
- ↑ Bilophila wadsworthia (bile-sulfide producer; linked to bloating and pain)
- Mild yeast overgrowth (non-pathogenic species)

Inflammatory Markers

- Calprotectin: Mildly elevated
- Zonulin: Borderline high
- Secretory IgA: Low

Intervention Plan (8 weeks)

THE FUNCTIONAL NATUROPATH

Clinical Impression & pattern:

- Low-diversity microbiome + impaired bile flow + low butyrate → high secondary bile acids
- → mucosal irritation → bloating and dysmotility.

Intervention aim:

- Rebuild keystone species, increase butyrate, improve bile composition, reduce GI inflammation markers → improve IBS-M/GI symptoms + quality of life.

Core Formula: GI Biome Nourish

(+ vagus nerve exercises, nature exposure, timed meals, <caffeine)

Contains: Pomella® pomegranate extract + Livaux® gold kiwifruit extract + RS2 resistant starch.

Dose:

- 1 scoop/day with breakfast
- Duration: 8 weeks (matches Pomella® 4–12wk trials & Livaux® 4wk trials)

THE FUNCTIONAL NATUROPATH

Pomella® (Pomengranate extract / punicalagins)

Clinically shown to:

- ↑ total SCFAs, especially acetate → butyrate cross-feeding
- ↑ microbial diversity
- ↓ inflammatory markers
- Support healthier bile acid metabolism via microbial conversion
- Promote urolithin A–producing species (anti-inflammatory, mitochondrial support)

Livaux® (Gold kiwifruit extract)

Clinically proven to:

- ↑ Faecalibacterium prausnitzii (butyrate powerhouse)
- Improve stool form in IBS
- Support mucosal immunology
- Reduce constipation/irregularity patterns

RS2 Resistant Starch (Solnul®-like effect)

Evidence shows:

- ↑ bifidobacteria and Akkermansia
- ↓ bloating scores
- Improved stool consistency
- ↑ butyrate-producing pathways

Progress at 4 Weeks (Symptom Scores)

THE FUNCTIONAL NATUROPATH

- Bloating improved from 6/10 → 2/10
- Stools now 1–2/day, more formed
- Gas dramatically reduced
- Less cramping
- Brain fog down by 50%
- Energy more stable
- “Stomach feels lighter”

CASE STUDY

8 Week Follow Up Re-test (Shotgun Metagenomics)

THE FUNCTIONAL NATUROPATH

Keystone Bacteria

- Faecalibacterium prausnitzii: ↑ 170%
- Akkermansia muciniphila: ↑ 95%
- Bifidobacteria: ↑ 65%

SCFA Production

- Butyrate: up 80%
- Total SCFAs: up 60%

Bile Acid Balance

- Secondary bile acids: improved (within range)
- Primary bile acids: improved (within range)
- Symptoms consistent with improved bile flow

Dysbiosis markers

- Bilophila wadsworthia: ↓ 55%
- Alistipes: normalised

Inflammation

- Calprotectin: normal
- Zonulin: improved by 22%
- sIgA: improved, no longer low

Outcomes and Take Home Messages

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Jactinta now reports:

- "I barely think about my gut anymore."
- Normal daily stools
- Rarely gets bloating
- Better tolerance of meals that previously caused distress
- Mental clarity improved
- Increased exercise tolerance
- PMS reduced

Clinically, this reflects:

Healthy keystone repopulation + improved bile flow + better SCFA production → reduced inflammation → normalised motility.

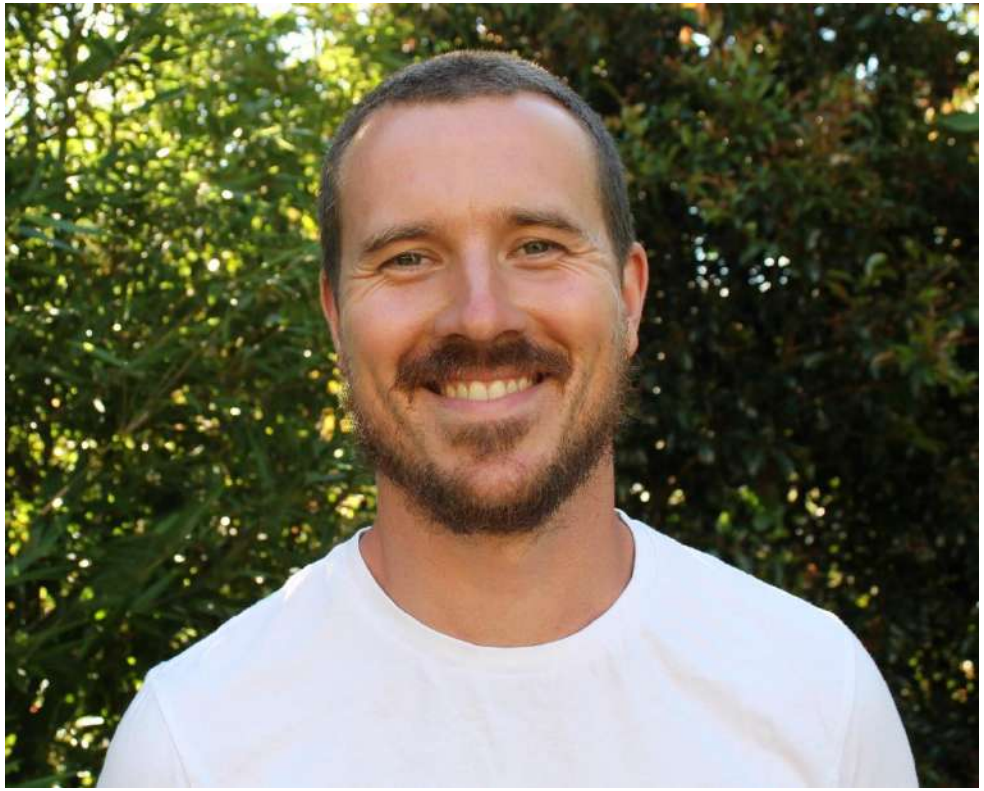
Clinical pearls:

The Pomella® + Livaux® + RS2 combination represents a high-evidence, low-irritation strategy for:

- Low microbial diversity
- Low butyrate
- Bloating/gas with inconsistent stools
- Bile acid dysregulation
- Post-antibiotic dysbiosis
- IBS-M, IBS-D and mixed symptom clusters

Its provides a gentle & tolerable way to rebuild the microbiome without provoking fermentation distress.

Thank you



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THE FUNCTIONAL NATUROPATH

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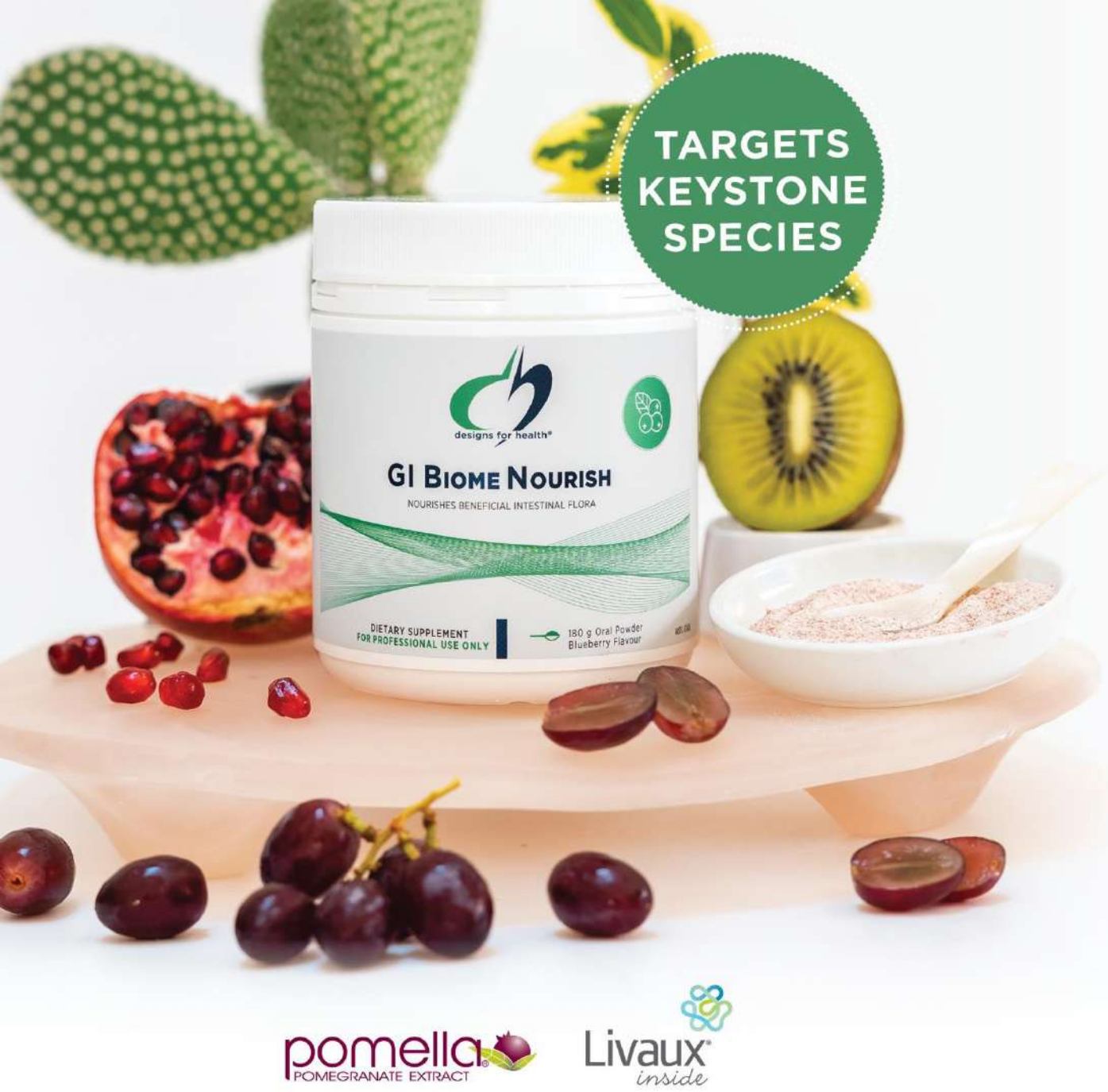

GI BIOME NOURISH

Precision prebiotics for potent digestive support.

Supercharge your gut health protocols with science-backed, polyphenol-powered nourishment. GI Biome Nourish is a premium prebiotic blend designed to fortify gut integrity, fuel beneficial bacteria, and foster colon health.

Featuring clinically researched Livaux® Gold Kiwifruit and Pomella® Pomegranate Extract, this powerhouse formula targets keystone gut species for optimal function.

Delicious, versatile, and free from common allergens – perfect for your GI protocols.

- Promotes Healthy Digestive System Flora
- Supports Healthy Bowel Function
- Nourishes Beneficial Intestinal Flora
- Supports GI Health

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