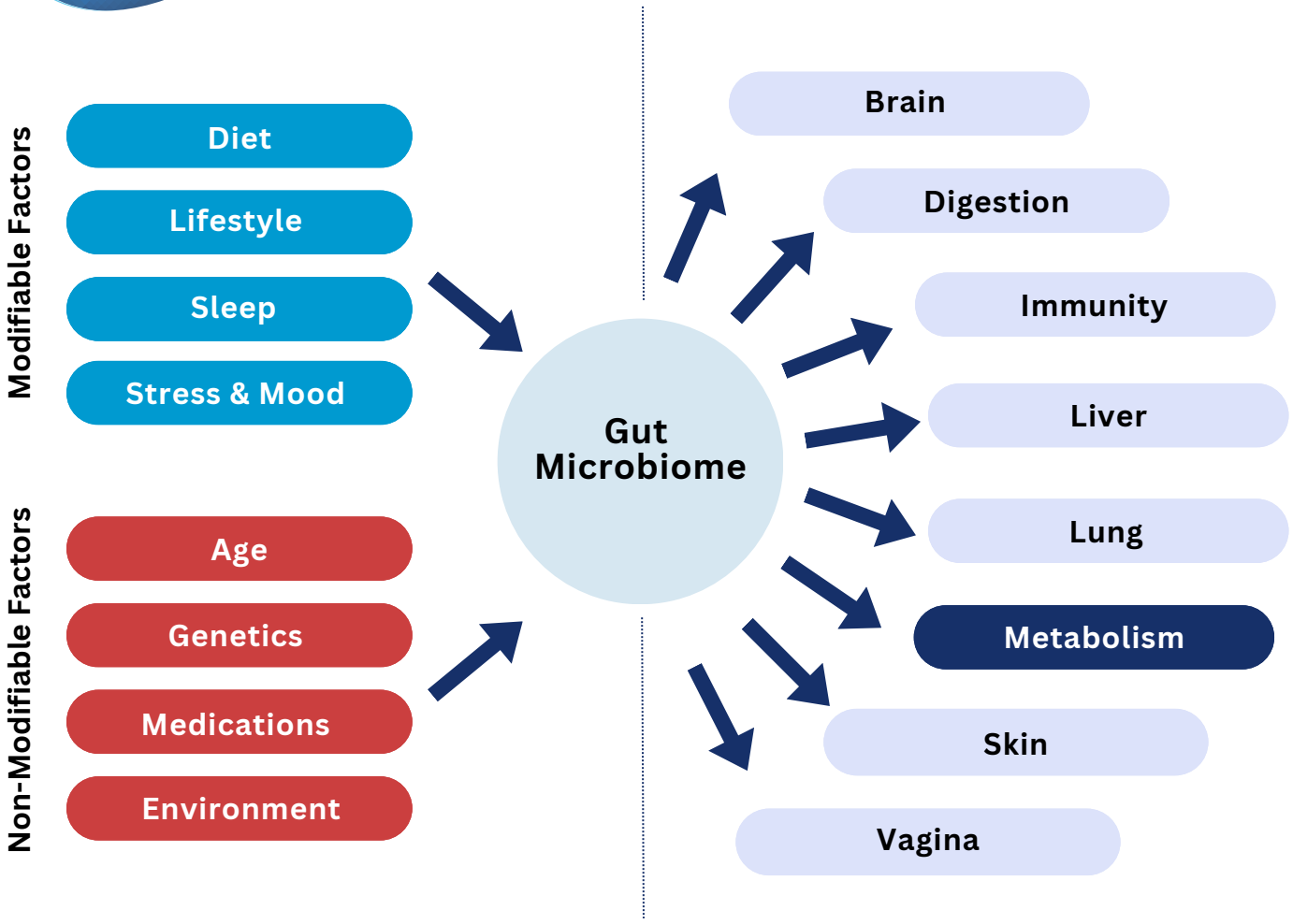
Yakult

Science for Health

GUT-METABOLISM AXIS HCP RESOURCE

The gut microbiome could be considered as "the conductor of health", orchestrating organs and systems in the human body, via different axes, to maintain homeostasis and optimal health.

The diagram below outlines some of the main axes whereby the gut communicates with other organs and systems in the body.



Gut Microbiome: The collection of microbes living in the digestive tract, their collective genetic material present and the metabolites that they produce.

This resource explains the relationship between the gut microbiome and body metabolism, known as the gut-metabolism axis. An overview of the gut microbiome and human metabolism is provided before explaining the role of microbial-mediated gut hormones on metabolic processes, and how to support metabolism with a particular focus on dietary approaches.

Yakult Science - making the science of the gut microbiota easy to digest

CONTENTS

KEY POINTS

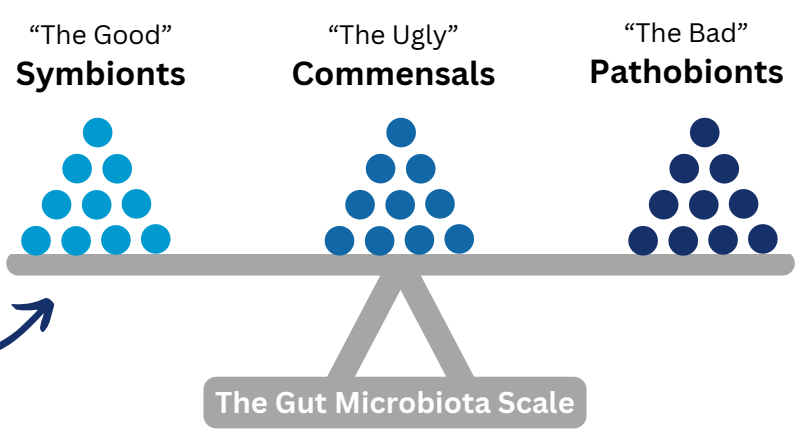
- A clear link exists between the gut microbiome, metabolism and metabolic health.
- A healthy gut microbiota is key for the body's metabolism, playing a role in **energy management, nutrient uptake, glucose tolerance, insulin sensitivity** and **appetite regulation**.
- The gut microbiome influences metabolism through the **regulation of gut hormone release**.
- The gut microbiome may play a role in **metabolic syndrome** (MetS) and subsequent conditions such as **cardiovascular disease** and **diabetes**.
- **Observational data** suggests a relationship between **gut diversity** and **MetS**. Many modifiable (e.g., diet, physical activity) and non-modifiable factors (e.g., age, genetics) influence gut microbial diversity.
- Diet impacts the gut microbiota composition; dietary interventions which target the gut microbiome could soon be used as a tool for the **treatment of metabolic syndrome.**

GUT MICROBIOTA

What is the gut microbiota?

The gut microbiota is the **collection of microbes** (e.g., bacteria, archaea, fungi and viruses) living throughout the **gastrointestinal tract**. Everyone has a **unique** gut microbiota.

There is no core "healthy" microbiota that is common to all individuals. Instead, a gut microbiota is considered to be optimal when balanced (a state called **symbiosis**). A balanced gut microbiota is important for supporting immunity.



METABOLISM

What is metabolism?

Metabolism plays a fundamental role in our overall health. It involves a range of complex biochemical reactions and processes that occur within all cells of the human body.

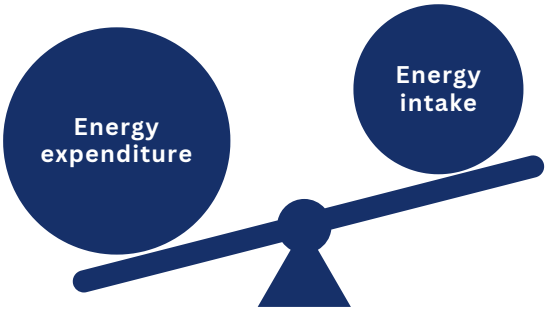
The three main functions of metabolism are:

- Conversion of food into energy
- Production of cell components
- Removal of waste products

ENERGY BALANCE

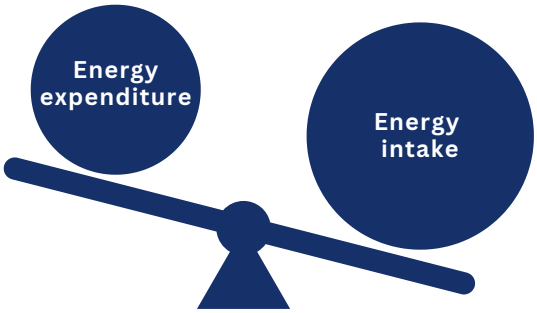
Energy balance refers to the relationship between calories taken into the body (i.e., food and drink) and calories being used in the body (e.g., physical activity) for our daily energy requirements. Body weight is stable when the body is in an energy balance.

Negative Energy Balance
Output > Input



Stored adipose tissue is used to meet energy demands

Positive Energy Balance
Output < Input



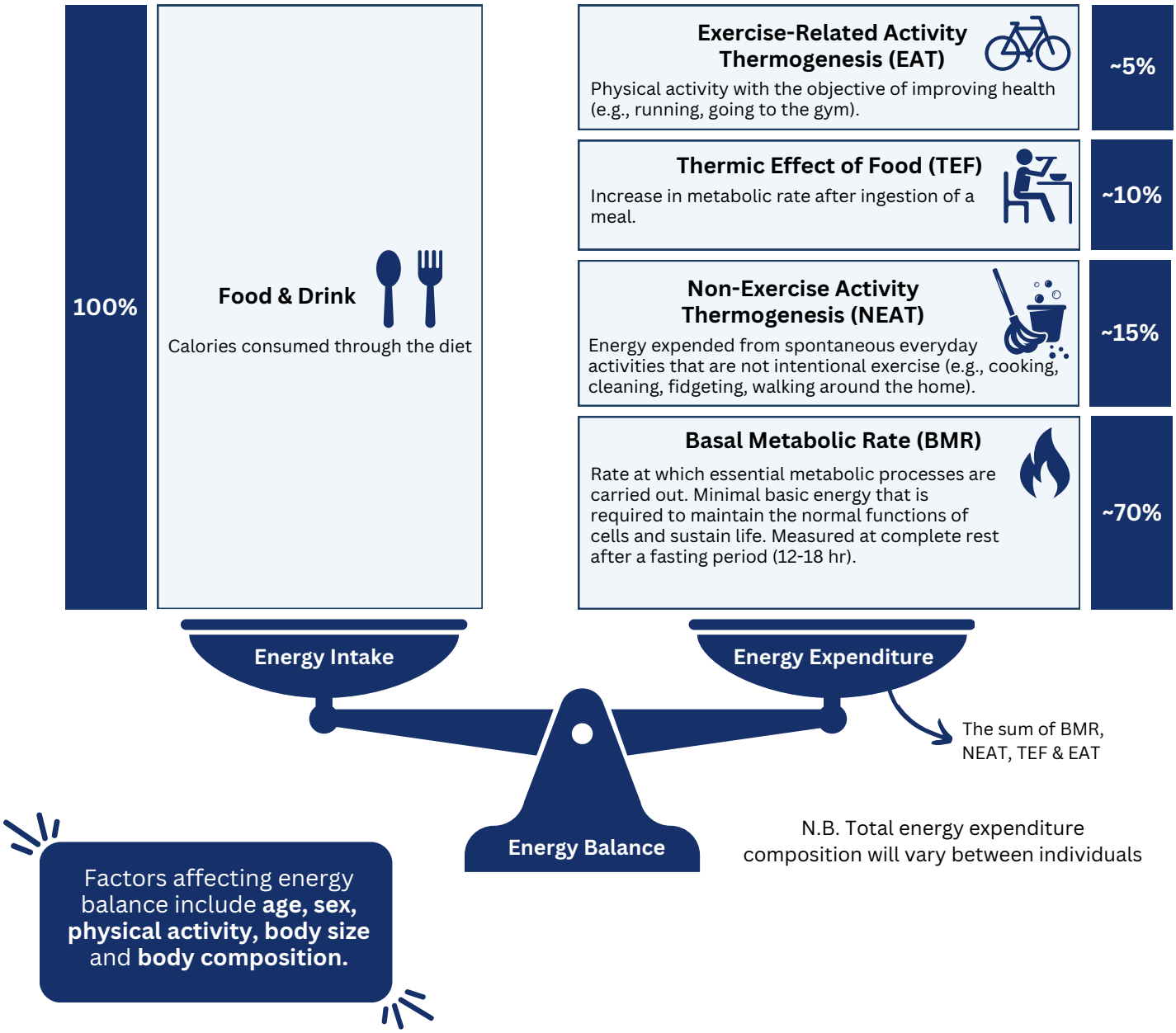
Excess energy stored in adipose tissue

FACT CHECKER

"Can I boost my metabolism?"

THE FACTS: The desire to boost or speed up metabolism is often associated with the desire for rapid weight loss and wanting to find a potential "silver bullet" for optimised health. Whilst it is possible to potentially influence metabolism by consuming certain food and drink, these effects are likely modest and short-term. Eating food can temporarily increase metabolism for a few hours and is referred to as the thermic effect of food (TEF). TEF increases after larger meals and consumption of carbohydrates and protein.[1] Exercise and body composition can have an impact on metabolism rate, either directly or indirectly.[2][3] Muscle is metabolically more active than fat and therefore individuals with greater muscle mass will burn more calories.

METABOLIC CONCEPTS



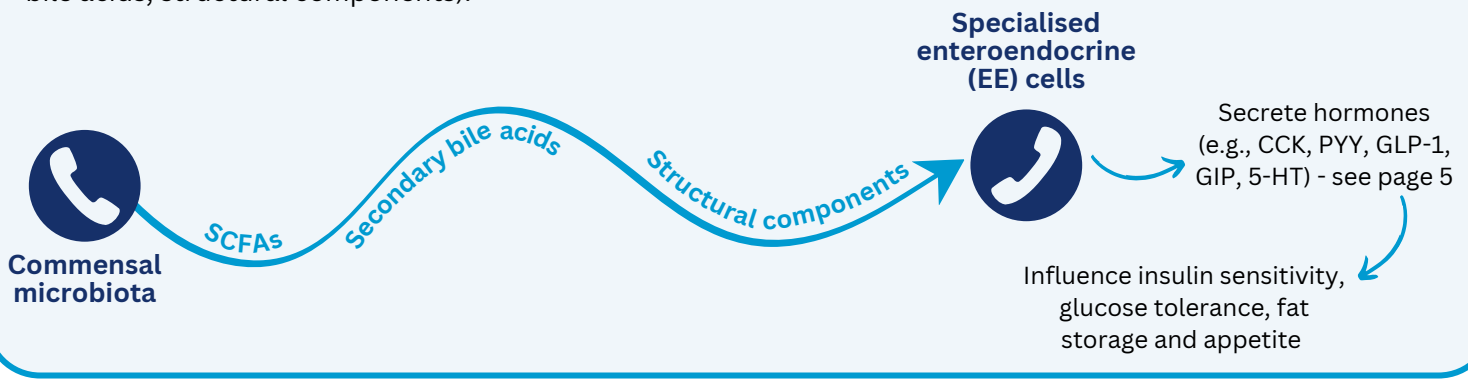
Does the gut microbiome influence energy balance and therefore body weight?

This question is complex as many factors impact body weight including **diet**, **lifestyle**, **genetics** and **socioeconomic status**, to name a few. While **animal studies** have shown some interesting results whereby transferring gut microbes from lean and obese donors to germ-free mice resulted in increased weight in those receiving the obese gut microbiota, this has not been seen in human clinical trials.[4–7] Nevertheless, the gut microbiota can impact metabolism, the secretion of hormones, neurotransmitters and inflammatory factors, all of which can impact weight gain or loss. Understanding the role of the gut microbiome in weight management may lead to future revolutionary strategies and interventions to help treat obesity.

ABOUT THE GUT-METABOLISM AXIS

The gut microbiota and its metabolites regulate metabolism via gut hormone release.

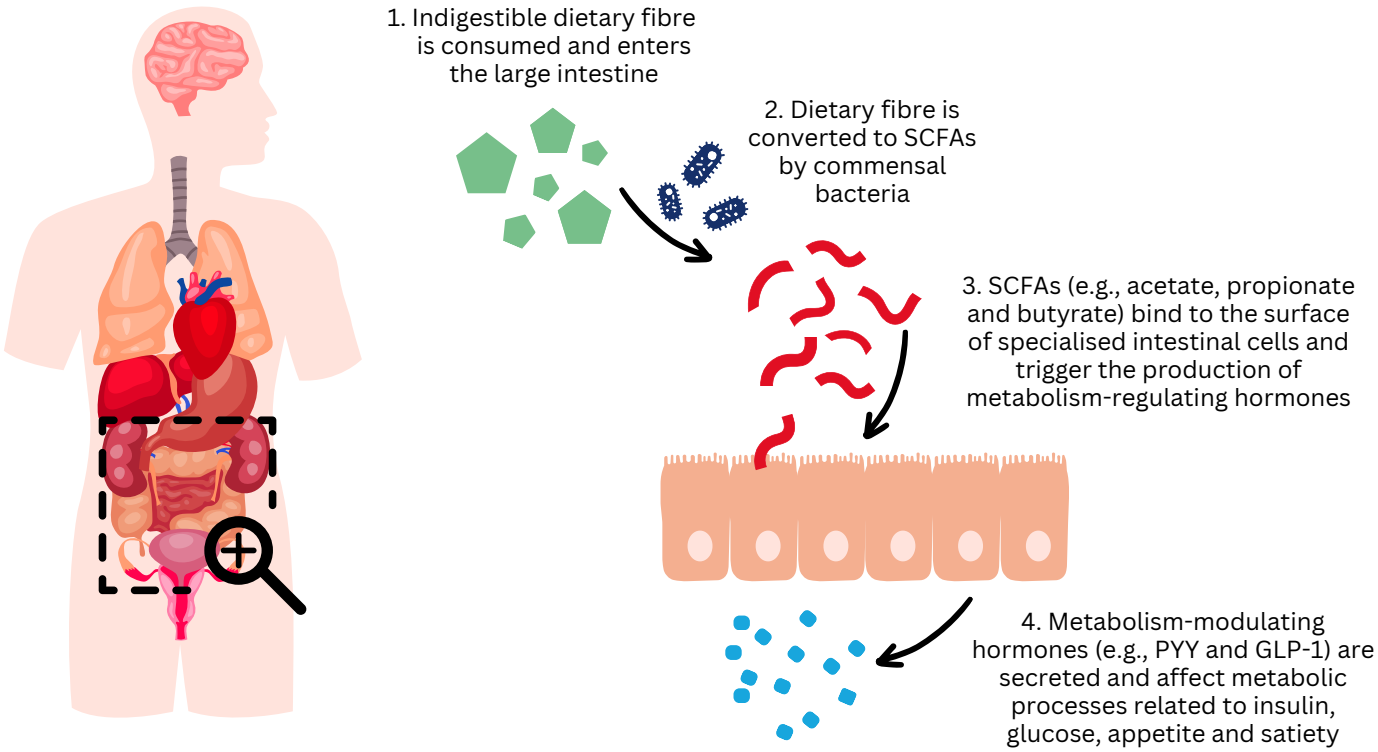
The gut microbiota, specifically the commensal microbiota, communicate to specialised enteroendocrine (EE) cells in the mucosal lining of the gut via microbial metabolites (e.g., short-chain fatty acids (SCFAs), secondary bile acids, structural components).[8]



ROLE OF SCFAs IN THE GUT-METABOLISM AXIS

Short-chain fatty acids (SCFAs) such as acetate, butyrate, propionate and lactate are **metabolites from gut microbiota fermentation of non-digestible dietary fibre**. The amount of SCFAs available is largely dependent on the abundances of certain bacterial species known to have SCFA-producing capability such as *Bifidobacterium, Lactobacillus, Faecalibacterium, Eubacterium* and *Roseburia*.[9]

SCFAs play a key role in **gut-mediated metabolism** by inducing the expression of gut hormones such as PYY and GLP-1,[10,11] leading to **appetite suppression**[12] and improvement of **glucose tolerance** and **insulin sensitivity**.[13,14]



KEY HORMONES IN GUT-METABOLISM AXIS

Hormones released by EE cells include **cholecystokinin** (CCK), **peptide YY** (PYY), **glucagon-like peptide 1** (GLP-1), **glucose-dependent insulinotropic peptide** (GIP) and **serotonin** (5-hydroxytryptamine, 5-HT). Their secretion from EE cells is influenced and regulated by the gut microbiota and its metabolites.[8] These microbial-mediated gut hormones have varying effects on metabolism, including:

Glucose levels & insulin sensitivity

Satiety & appetite

Fat storage

Energy levels

CCK

- Regulates the ingestion, digestion, and absorption of nutrients, and is released in response to dietary fat and protein intake.
- Its key roles include stimulation of pancreatic secretion and gallbladder contraction (i.e., release of bile acids), regulation of gastric emptying and appetite regulation via induced satiety.

PYY

- Regulates food intake and satiety.
- In a fasted state, PYY levels are low and subsequently increase rapidly in response to food intake.

GLP-1

- Secreted in response to dietary intake of certain nutrients (e.g., glucose, fatty acids, protein).
- Influences satiety,[15–17] food intake[15–18] and the release of insulin,[15,17] therefore contributing to postprandial glycaemic response.
- Notably, SCFA can induce GLP-1 secretion.

GIP

- Involved in postprandial insulin secretion, energy storage and bone formation.[19]

Serotonin 5-HT

- Plays an important signalling role in various physiological pathways and processes, including gastrointestinal (GI) motility, glucose homeostasis,[20] lipid metabolism, bone density and metabolic conditions such as obesity[20–22] and type 2 diabetes.[23]

Approximately 95% of serotonin is located and used locally within the gut to aid gut motility.[24]

THE LINK BETWEEN GUT DIVERSITY AND METS: THE EVIDENCE

Metabolism is linked to many conditions and health outcomes. Observational research suggests an association between low diversity of the gut microbiota and MetS. Specifically, a deficiency in microbial metabolites such as SCFAs and bile acids can contribute to the development of MetS.[25]

Metabolic Syndrome (MetS)

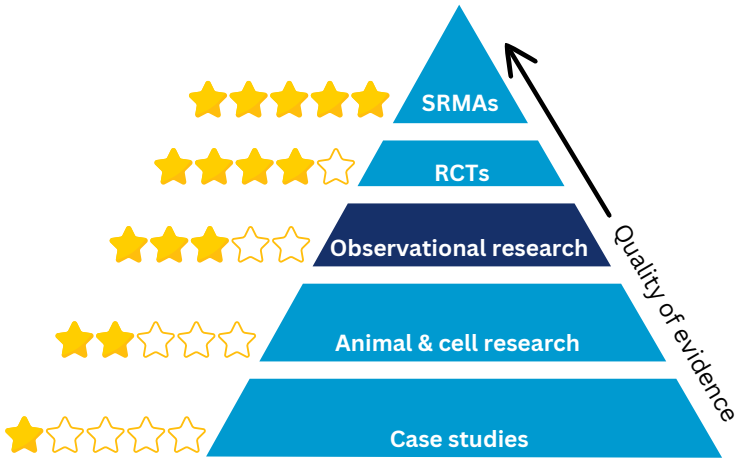
Collection of health conditions (e.g., hypertension, high blood sugar, obesity) that occur together, increasing the risk of cardiovascular disease and type 2 diabetes.

WHAT'S THE EVIDENCE?

Research is now starting to explore if modulation of the gut microbiota could have implications on disease incidences.

Much of the evidence currently comes from **observational studies** which cannot demonstrate **causality**. Therefore, it remains unknown whether an imbalanced gut microbiota (dysbiosis) precedes the condition or vice versa.

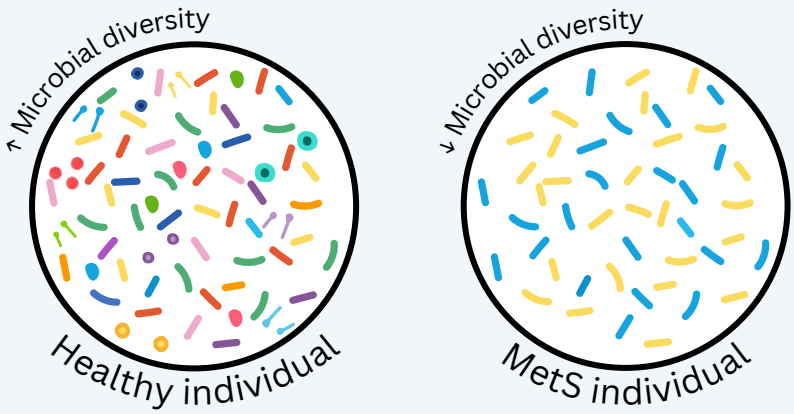
More research is needed to better understand whether gut dysbiosis may cause, or contribute to the development of certain conditions or whether the condition itself reduces microbial diversity.



RCTs = Randomised controlled trials
SRMAs = Meta-analyses & systematic reviews

The diversity of dietary intake correlates with the diversity of gut commensal bacteria, which is associated with greater abundance of SCFAs.[26–28]

Evidence from observational studies suggests that **dysbiosis** and **reduced gut microbiota diversity**, generally seen in individuals living with various health conditions such as obesity and/or type 2 diabetes, can result in more pronounced metabolic dysfunction, low-grade inflammation, impaired blood sugar control and dyslipidaemia.[29–32]



GUT-METABOLISM AXIS THROUGHOUT LIFE

This flowchart describes age-related metabolic changes and compositional changes in the gut microbiota throughout life.

Up to **~3 years** is the **critical window of opportunity** to modulate the GM composition with greatest intra- and inter-variability. After this age, the microbiota is more **stable** and **mature** (40%-60% similarity with the adult microbiota).[33–35]

Pregnancy

Maternal microbiota is the major influence on neonatal GM. During pregnancy, metabolism adapts to meet the energy demands of pregnancy.[36–38]

Birth Delivery Mode

Impacts neonatal GM. Metabolic adaption can vary between preterm infants and those born at full-term;[39] the former has been linked to increased risk of metabolic disorders e.g., cardiovascular disease.[40–42] C-section delivery may be associated with metabolic risk factors in later-life such as obesity.[43,44] However, more research is needed to confirm clinical significance.

Childhood

Increased hygiene measures and broad-spectrum antibiotic usage reduces GM.[52,53] Total energy expenditure increase rapidly in first year of life and throughout childhood.[54] Differences in gut microbiota composition in children may predict predisposition to obesity.[55–57]

Milk Source

Influences infant GM.[45,46] Greater amounts of *Bifidobacterium* and *Bacteroides*, and lower amounts of *Streptococcus* and *Enterococcus* are seen in breastfed babies compared to those consuming formula milk.[47] Maternal energy requirements increase in response to lactation.[48] Breastfeeding is associated with a protective effect against early obesity in children.[49–51]

Adolescence/ Adulthood

GM is dependent on diet and lifestyle factors.[58] Puberty is a time of rapid growth and changing energy requirements owing to changes in fat-free mass, growth- and puberty-related hormones.[59] Metabolic rates are most stable during adulthood with little change to overall metabolism.[54]

Perimenopause/ Menopause

GM becomes less diverse and reflects the male gut microbiota.[60] Menopausal transition is associated with changes in body fat and lipid profile as well as increased risk of metabolic diseases.[61–63]

Older Adulthood

GM diversity and BMR decline with age.[64,65] Metabolic functions and energy expenditure declines with age, in addition to hormonal and neurological changes.[54,66]

Environment: GM is influenced by those you live with (including pet ownership) and those in the same household will have a more similar microbiota.[67,68] Exposures such as pollution and pathogens will also affect GM.[69–71]

GM = Gut Microbiota

Find out more at yakult.co.uk/HCP
Contact us: science@yakult.co.uk
@yakultscience_ukie
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FACTORS INFLUENCING THE GUT-METABOLISM AXIS

Many **modifiable and non-modifiable factors** can influence both the gut microbiota and metabolism. Arguably, the two modifiable factors which have the greatest influence on the gut-metabolism axis are **diet and exercise**. See **page 9** for more information on how to support your patients' gut-metabolism axis through diet.

Diet

- A **diverse diet** rich in fruits, vegetables, grains and legumes are recommended to promote **microbial diversity**.[72,73]
- Diet high in **fibre**, which is subsequently metabolised to SCFAs by the gut microbiota, has been shown to modulate the gut microbiota, decrease inflammation and increase insulin sensitivity.[74–77]

Exercise

- Increasing habitual physical activity can lead to **increased microbial diversity** and associated health benefits.[78,79]
- Can regulate the gut microbiota, increase **production of SCFAs** and enhance **metabolic capacity**.[80–82]
- Can play a protective role against MetS.[83–85]

Sleep

- Sleep deprivation has been linked to **dysregulation of hormones** responsible for **appetite control**, which can increase food consumption.[86]

Genetics

- Influence the **composition of the gut microbiome** and therefore, in turn, can impact metabolism.[87]
- Can play a role in **predisposition of obesity**.[88–90]

Stress

- Effect on metabolism varies.
- Can be associated with **reduced dietary intake** and lower body weight.[91]
- Can lead to **increased appetite and weight**, in addition to greater satisfaction when eating highly processed foods.[91–93]

Body Composition

- Fat free mass (FFM) has a significant influence on metabolism whereby having a **greater percentage of FFM** results in **increased overall metabolic rate** and energy expenditure.[94,95]
- Metabolic processes remain similar between males and females but differences in fat storage, muscle mass and hormonal **changes during puberty** impact metabolic rate.
- Females tend to have a lower metabolic rate compared to males.[96]

FOODS IN FOCUS

For practical dietary tips, read our **Diet Diversity Guide**

Diet can significantly alter gut microbial diversity. Therefore, there is potential for dietary interventions to act as an adjunct therapy in the future. General tips to increase gut diversity are explained below.

FIBRE

Dietary fibres are fermented by the gut microbiota, producing SCFAs. SCFAs play an important role in the gut-metabolism axis. Diets high in fibre are associated with greater microbial richness and diversity.[97] Sources of fibre include fruits, vegetables, wholegrain carbohydrates, beans and lentils, nuts and seeds, herbs and spices.

DIVERSITY

Restrictive diets can starve the gut microbiota, whereas a diet focused on adding more – more plants, more variety, and more fibre – can increase gut microbial diversity. Research has shown that those who ate more than 30 different plant-based foods per week, had a far more diverse gut microbiome compared to those who ate less than 10.[98]

MEDITERRANEAN DIET

The Mediterranean diet is characterised by an abundance of high-fibre plant foods and contains sources of omega-3 fatty acids, vitamin D and polyphenols, all of which have been associated with increased gut microbiota diversity.[99–105] Following a Mediterranean diet has been shown to help in the prevention and treatment of MetS.[106–109]

FERMENTED FOODS

Fermented foods (e.g., fermented milk drinks, kimchi, sauerkraut, kombucha) can be a source of live dietary microbes and are thought to support gut health. Some human studies have shown that fermented foods can play a part in modulation of the gut microbiota.[110–113]

Movement & Microbes

Exercise independently increases the diversity and growth of beneficial bacteria in the gut. It can reduce metabolic dysfunction and reduce the risks for metabolic disease states.[83,114]

Social Support: Encourage patients to surround themselves with like-minded and supportive individuals who can encourage them, especially when motivation is low!

Small Changes: Offer simple ways your patients can incorporate more movement into their day such as parking further away from the shops, taking the stairs rather than the escalator, organising walking meetings or moving during TV ad breaks.

*"Whenever it comes to encouraging more fibre into your patients' diets, remind them to go **low and slow** - gradually increase the amount of fibre to avoid uncomfortable side effects and don't forget to **stay well hydrated**".*

DR HOLLY NEILL

Science Officer
Yakult UK & Ireland

REFERENCES

1. Calcagno et al. (2019) *J Am Coll Nutr* 38(6): 547–551.
2. MacKenzie-Shalders et al. (2020) *J Sports Sci* 38(14): 1635–1649.
3. LaForgia et al. (2006) *J Sports Sci* 24(12): 1247–1264.
4. Bell (2015) *Postgrad Med* 127(8): 863–868.
5. Ridaura et al. (2013) *Science* 341(6150): 1241214.
6. Turnbaugh et al. (2006) *Nature* 444(7122): 1027–1031.
7. Turnbaugh et al. (2009) *Nature* 457(7228): 480–484.
8. Martin et al. (2019) *Front Physiol* 10: 428.
9. Hamamah et al. (2023) *Nutrients* 15(15): 3365.
10. Larraufie et al. (2018) *Sci Rep* 8: 74.
11. Tolhurst et al. (2012) *Diabetes* 61(2): 364–371.
12. Horner and Lee (2015) *Appl Physiol Nutr Metab* 40(11): 1089–1099.
13. Cani et al. (2006) *Diabetes* 55(5): 1484–1490.
14. Zander et al. (2002) *Lancet* 359(9309): 824–830.
15. Holst (2007) *Physiol Rev* 87: 1409–1439.
16. Shah and Vella (2014) *Rev Endocr Metab Disord* 15(3): 181–187.
17. Müller et al. (2019) *Mol Metab* 30: 72–130.
18. Kinzig et al. (2002) *J Neurosci* 22(23): 10470–10476.
19. Baggio et al. (2007) *Gastroenterology* 132(6): 2131–2157.
20. Young et al. (2018) *Int J Obes* 42: 1880–1889.
21. Crane et al. (2015) *Nat Med* 21: 166–172.
22. Young et al. (2015) *Gastroenterology* 149: 253–255.
23. Martin et al. (2017) *Endocrinology* 158(5): 1049–1063.
24. Berding et al. (2021) *Adv Nutr* 12(4): 1239–1285.
25. dos Santos and Galiè (2024) *Nutrients* 16(3): 390.
26. Silva et al. (2020) *Front Endocrinol* 11:25.
27. Fusco et al. (2023) *Nutrients* 15(9): 2211.
28. Nogal et al. (2021) *Gut Microbes* 13(1): 1–24.
29. Lozupone et al. (2012) *Nature* 489(7415): 220–230.
30. Kriss et al. (2018) *Curr Opin Microbiol* 44: 34–40.
31. Le Chatelier et al. (2013) *Nature* 500(7464): 541–546.
32. Mosca et al. (2016) *Front Microbiol* 7: 455.
33. Bäckhed et al. (2015) *Cell Host Microbe* 17(5): 690–703.
34. Koenig et al. (2011) *Proc Natl Acad Sc USA* 108(Suppl 1): 4578–4585.
35. Yatsunenko et al. (2012) *Nature* 486(7402): 222–227.
36. Zeng et al. (2017) *Ann Nutr Metab* 70(1): 59–65.
37. Lain and Catalano (2007) *Clin Obstet Gynecol* 50(4): 938–948.
38. Wang et al. (2016) *BMC Med* 14(1): 205.
39. Platt and Deshpande (2005) *Semin Fetal Neonatal Med* 10(4): 341–350.
40. Casirati et al. (2022) *Front Nutr* 9: 978271.
41. Markopoulou et al. (2019) *J Pediatr* 210: 69–80.e5.
42. Parkinson et al. (2013) *Pediatr* 131(4): e1240–e1263.
43. Bernardi et al. (2015) *Am J Clin Nutr* 102(2): 295–301.
44. Mínguez-Alarcón et al. (2022) *Pediatr Res* 91: 672–680.
45. Carr et al. (2021) *Front Immunol* 12(604080).
46. Wiciński et al. (2020) *Nutrients* 12(1): 266.
47. Ma et al. (2020) *Sci Rep* 10(15792).
48. Butte and King (2005) *Public Health Nutr* 8(7A): 1010–1027.
49. Horta et al. (2023) *Acta Paediatr* 112(1): 34–41.
50. Qiao et al. (2020) *J Pediatr Nurs* 53: 57–66.
51. Arenz et al. (2004) *Int J Obes Relat Metab Disord* 28(10): 1247–1256.
52. Francino (2016) *Front Microbiol* 6(1543).
53. Patangia et al. (2022) *MicrobiologyOpen* 11(1): e1260.
54. Pontzer et al. (2021) *Science* 373(6556), 808–812.
55. Kalliomäki et al. (2008) *Am J Clin Nutr* 87(3): 534-538.
56. Vael et al. (2011) *Gut Pathog* 3(1): 8.
57. Cho (2023) *Clin Exp Pediatr* 66(4): 148–154.
58. Hasan and Yang (2019) *PeerJ* 7(e7502).
59. Cheng et al (2016) *Am J Clin Nutr* 104(4): 1061-1074.
60. Peters et al. (2022) *Int J Womens Health* 14: 1059-1072.
61. Jeong and Park (2022) *Metabolites* 12(10): 954.
62. Polotsky and Polotsky (2010) *Semin Reprod Med* 28(5): 426–434.
63. Stachowiak et al. (2015) *Menopause Rev* 14(1): 59–64.
64. Ragonnaud and Biragyn (2021) *Immun Ageing* 18(1): 2.
65. Roberts and Rosenberg (2006) *Physiol Rev* 86(2): 651–667.
66. Palmer and Jensen (2022) *J Clin Invest* 132(16): e158451.
67. Kates et al. (2020) *Front Cell Infect Microbiol* 10(73).
68. Song et al. (2013) *eLife* 2(e00458).
69. Bailey et al. (2020) *Gut Microbes* 11(5): 1188–1202.
70. Jin et al. (2017) *Environ Pollut* 222: 1–9.
71. Pickard et al. (2017) *Immunol Rev* 279(1): 70–89.
72. Garcia-Mantrana et al. (2018) *Front Microbiol* 9(890).
73. van der Merwe (2021) *Int J Food Sci Nutr* 72(5): 665–669.
74. Attaye et al. (2022) *Gastroenterol* 162(7): 1911–1932.
75. Thomas et al. (2022) *Metab Syndr Relat D* 20(8): 429–439.
76. Reynolds et al. (2020) *PLoS Medicine* 17(3): e1003053.
77. Ojo et al. (2020) *Nutrients* 12(11): 3239.
78. Monda et al. (2017) *Oxid Med Cell Longev* 3831972.
79. Hintikka et al (2023) *Sci Rep* 13, 11228 .
80. Zhang et al. (2023) *Nutrients* 15(6): 1539.
81. Mailing et al. (2019) *Exerc Sport Sci Rev* 47(2): 75–85.
82. Carey and Montag (2021) *BMJ Open Sport Exerc Med* 7(2): e000930.
83. Thyfault and Bergouignan (2020) *Diabetol* 63(8): 1464–1474.
84. Liang et al. (2021) *Rev Cardiovasc Med* 22(4): 1523–1533.
85. Myers et al. (2019) *Nutrients* 11(7): 1652.
86. Sharma and Kavuru (2010) *Int J Endocrinol* 270832.
87. Goodrich et al. (2014) *Cell* 159(4): 789–799
88. Golden and Kessler (2020) *J Am Assoc Nurse Pract* 32(7): 493–496.
89. Loos and Yeo (2022) *Nat Rev Genet* 23(2): 120–133.
90. Bouchard (2021) *Obesity* 29(5): 802–820.
91. Torres and Nowson (2007) *Nutrition* 23(11-12): 887–894.
92. Adam and Epel (2007) *Physiol Behav* 91(4): 449–458.
93. Rabasa and Dickson (2016) *Curr Opin Behav Sci* 9: 71-77.
94. Johnstone et al. (2005) *Am J Clin Nutr* 82(5): 941–948.
95. Stiegler and Cunliffe (2006) *Sports Med* 36(3): 239–262.
96. Yang et al. (2021) *Energy Build* 251: 111360.
97. De Filippo et al. (2010) *Proc Natl Acad Sc USA* 107(33): 14691-14696.
98. McDonald et al. (2018) *mSystems* 3(3): e00031-18.
99. Bellerba et al. (2021) *Nutrients* 13(10): 3378
100. Cheng et al. (2023) *Phytomedicine* 119(154979).
101. Costantini et al. (2017) *Int J Mol Sci* 18(12): 2645.
102. Fakhoury et al. (2020) *J Steroid Biochem Mol Biol* 200(105663).
103. Fleet (2022) *Adv Exp Med Biol* 1390: 155–167.
104. Fu et al. (2021) *Mediators Inflamm* 8879227.
105. Wan et al. (2021) *Crit Rev Food Sci* 61(4): 690–711.
106. Huo et al. (2015) *Eur J Clin Nutr* 69(11): 1200–1208.
107. Babio et al. (2009) *Public Health Nutr* 12(9A): 1607–1617.
108. Kastorini et al. (2011) *J Am Coll Cardiol* 57(11): 1299–1313.
109. Salas-Salvadó et al. (2015) *J Nutr* 146(4): 920S–927S.
110. Wastyk et al. (2021) *Cell* 184(16): 4137–4153.e14.
111. Stiemsma et al. (2020) *J Nutr* 150(7): 1680–1692
112. Leeuwendaal et al. (2022) *Nutrients* 14(7): 1527.
113. Illikoud et al. (2022) *Immunol Lett* 251-252: 91–102.
114. Egan & Zierath (2013) *Cell Metab* 17(2): 162–184.

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Scan here for our Microbiome Matters podcast

Season 3, Episode 4

'The effect of exercise on the gut microbiota'
with Dr Kevin Barrett, General Practitioner

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Science has always been at the heart of the Yakult company, since the microbiologist Dr Shirota selected and cultivated *Lacticaseibacillus paracasei* Shirota in Japan in 1935.

Yakult Science for Health is an educational hub that empowers healthcare professionals, researchers and students by offering a range of resources and services to support and expand their knowledge of the gut microbiome, probiotics and more.

Visit our Yakult Science Learning Hub for access to:

- Resources and e-learning
- Yakult Science events
- Microbiome Matters podcast with expert guests
- Tailored workplace talks
- Yakult Educational Grant
- Research updates

This resource is intended for healthcare professionals and is not to be distributed to patients.

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Find out more at yakult.co.uk/HCP
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