Statements and FAQ for media enquiries: GMfH panel

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1. Faecal microbiota transplantation (FMT)

Q. Covid transmission in FMT?

A. Early in the COVID19 pandemic it was reported that coronavirus DNA could be recovered from stool samples in patients with COVID19. However, live virus has never been grown from stool. Nevertheless, given the (albeit) theoretical risk of transmission of the virus from a donor with COVID, careful clinical screening and COVID testing is carried out on all FMT donors until now including nasal swab and faecal testing.

Supporting reference

Ianiro G et al (2020) Reorganisation of faecal microbiota transplant services during the COVID-19 pandemic. *Gut* 69(9):1555-1563.

Q. Obesity transfer in FMT

A. Early experiments in mice suggested that there was the possibility of transfer of obese trait from donors to recipients. A recent study looking at over 400 FMT procedures in patients from the Mayo Clinic found no evidence of a transfer of BMI from donors to recipients. It should be noted that obese donors are not accepted as donors for human FMT, being excluded at the stage of health screening.

Supporting reference

Sehgal K et al (2025) Body mass index changes after fecal microbiota transplantation for recurrent *Clostridioides difficile* infection. *Ther Adv. Gastroenterol* 18:1-12

Q. Is it safe to do your own FMT?

A. People should not be doing 'DIY' Faecal Microbiota Transplants (FMT) by themselves. This could cause serious harm. Even if you know the poo donor, you cannot test the safety of the faeces donated, and could risk infection which could be, and has been in some cases, life-threatening. Studies in animals have even shown that it might be possible to transfer mental health conditions like anxiety and depression in the poo.

FMT available on the NHS is only for those with recurrent or refractory *Clostridioides difficile (C. diff)* infection. It is always performed by an FMT specialist. FMT is so specialised that there are only two UK hospitals with a licensed stool bank. The poo donors undergo a rigorous screening process, with a full health questionnaire, blood and poo samples. The poo samples are strictly controlled. Much more research is needed in order to use FMT to treat other digestive condition but there are a range of trials being conducted in the UK and other countries.

Supporting material

- Guts UK/GMfH leaflet on FMT
- Ekezie et al (2021) Understanding the scope of do-it-yourself fecal microbiota transplant. *Am J Gastroenterol* 115: 603-607.

Q. Biobanking of stool samples

A. There are emerging clues that autologous FMT- i.e. using a person's own poo stored for later use-may be of benefit. For example, if a person undergoes immune system conditioning prior to bone marrow transplantation, this conditioning may damage the gut microbiome, and it is possible that re-introduction of poo stored before the treatment may improve clinical outcomes. This is far from proven, and, to date, there are no stool banks which store such samples for 'autologous' treatment.

Q. FMT trials

A. Current FMT trials in the UK include two focused on reducing the harm from the carriage of multi-drug-resistant organisms (MDRO). Both trials (FERARO and MAST) are being conducted in London. There are two trials in liver disease PROMISE (alcoholic liver disease) and FARGO which is recruiting patients with primary sclerosing cholangitis associated with inflammatory bowel disease.

Q. Routine NHS FMT

A. FMT was approved for the treatment of recurrent and refractory *Clostridiodes difficile* infection by NICE In 2022. The University of Birmingham Microbiome Treatment Centre (MTC) has held a GMP licence for FMT since in 2017 and has treated over 1100 patients with FMT in approximately 180 NHS Trusts to date.

FMT can be ordered from the MTC for CDI treatment (email bhs-tr.fmt@nhs.net). The specials licence also allows the use of FMT for other conditions to be discussed on a case-by-case basis.

2. Commercial gut microbiome (poo) testing

Q. Commercial gut microbiome testing

A. There is a lot of interest in commercial provision of microbiome testing, usually from stool. This can also sometimes be accompanied by recommendations based on the findings. Whilst this is an exciting and tempting proposition, this is not something that can be scientifically justified at present. Our group have previously produced a targeted leaflet on this topic with Guts UK (link below). This topic has also been the subject of an international consensus from 69 experts across 17 countries (link also below) which recognised the need to develop this sphere but also sounded caution on our readiness for such testing. In short, we do not know enough about what "normal" looks like in the microbiome to validate this in an individual; whilst variations in the scientific method applied to assessing this at each stage from collecting, storing and processing the sample, and undertaking the microbiome analysis can impact on the results. We therefore do not recommend commercial gut microbiome testing at present.

Supporting material

- Guts UK/GMfH leaflet on commercial gut microbiome (poo) testing
- Porcari et al (2025) International consensus statement on microbiome testing in clinical practice. Lancet Gastroenterol Hepatol 10:154-167.
- Servetas SL *et al.* (2026) Evaluating the analytical performance of direct-to-consumer gut microbiome testing services. *Commun Biol* **9**, 269.
- Video: Gut microbiome analysis – to test or not to test? (Blair Merrick)

3. Paediatrics

Q. Antibiotic effects on children's gut microbiome

A. The gut microbiome develops towards an adult pattern over the first three years of life and is probably fairly stable thereafter. There has therefore been a lot of interest in alterations to the microbiome in childhood, perhaps particularly through early antibiotic exposure which has been linked to a number of later conditions. Whilst it would be nice to be able to avoid antibiotic exposure during this critical window, this needs to be balanced against the very real need to use antibiotics to treat a number of childhood illnesses. Prescription of an antibiotic for a childhood infection is the responsibility of the prescribing physician, who will consider the benefits and risks of this within the clinical situation at play. Whilst it is not possible to offer guidance in a more general sense without specific clinical details, the majority of childhood infections are caused by viruses which do not respond to antibiotics, so it is helpful to avoid them when a viral cause is clear- fever from chicken pox is a good example. Discriminating between bacterial infections (that respond to antibiotics) and viral infections (that do not) can be a challenging undertaking and is not always accurately possible at the time. For this reason, many infections that are ultimately explained by viruses can be treated with antibiotics ahead of this information. This is an acceptable facet of paediatric practice and undertaken with the child's safety paramount. It may be that future tests will allow a more rapid and more accurate assessment of the cause of a child's fever to help reduce the need for antibiotics, but this is currently aspirational.

Q. Is there any benefit from vaginal seeding for neonates born by C-section?

A Although there has been interest in this procedure, to try to mimic what would naturally occur during a vaginal delivery, there is insufficient clinical evidence of short term or long benefit, and safety concerns. There is a risk that pathogens could be transferred to the baby.

4. Diet

Probiotics

Q. *What are probiotics?*

A. Probiotics are “live microorganisms that, when administered in adequate amounts, confer a health benefit on the host” (1)

Supporting reference

Hill C et al (2024) Expert consensus document. The International Scientific Association for Probiotics and Prebiotics consensus statement on the scope and appropriate use of the term probiotic. *Nat Rev Gastroenterol Hepatol* 11:506-14.

Q. *What types of microorganisms are probiotics?*

A. The most common probiotics fall into the bacterial genera *Lactobacillus* (now known as *Lacticaseibacillus*) and *Bifidobacterium*, with a few in the genera *Bacillus, Enterococcus* and *Saccharomyces* (a yeast). Probiotics are identified by genus, species/subspecies and strain. Examples of some common commercial strains that have evidence for beneficial effects include:

- *Lactobacillus rhamnosus* strain GG
- *Lactobacillus casei* strain Shirota
- *Lactobacillus casei* strain DN-114001
- *Lactobacillus acidophilus* NCFM
- *Bifidobacterium animalis* subsp *lactis* DN-173010
- *Bifidobacterium longum* 35624
- *Saccharomyces boulardii* CNCM 1-745

It is important to note that not all food and supplements labelled as probiotics have proven health benefits. It should also be noted that in the UK and EU there are regulations governing health claims made on food and food supplements. At present, the term ‘probiotic’ is considered a health claim and, at present, is not allowed on label or commercial communications to the general public.

Q. *How are probiotics consumed?*

A. They can be incorporated into yoghurts e.g. ‘Activia’ (NB not all yogurts contain additional probiotics, check the label), yogurt drinks e.g. “Yakult’ ‘Actimel’, or sold in the form of tablets, capsules or liquids often containing mixtures of probiotics in a freeze-dried form to preserve their viability, e.g.’Bio-kult’, ‘Optibac’.

Q. *How do probiotics work?*

A. It is important to note that probiotics can exert effects by non-specific, species-specific and strain-specific mechanisms. Actions at the non-specific level include inhibition of pathogenic organisms in the gut and production of bioactive microbial metabolites such as short chain fatty acids. Vitamin synthesis and gut barrier enhancement are examples of species-specific effects, and at the strain level, effects on the immune system and cytokine production are seen. Some of these effects e.g. on pathogen inhibition, require the probiotics to reach the colon, but others, particularly the modulation of immune function are exerted in the small intestine.

Q. *What are the potential health benefits of probiotics?*

A. The potential health benefits of probiotics have been extensively studied, particularly in the gut, but it is clear that the effects can be species and strain specific and hence cannot be extrapolated from one probiotic to another. Also, many of the studies use relatively small numbers of subjects.

The areas where effects have been demonstrated for certain strains include:

1. Reduction in duration of acute infectious diarrhoea in infants (usually caused by rotavirus)
2. Reduction in risk and/or severity of necrotizing enterocolitis in premature babies
3. Possible reduction in severity or maintenance of remission of ulcerative colitis (but not the

other major inflammatory bowel disease - Crohn's disease), however there is little supporting evidence and no recommendations in clinical guidelines.
4. Reduction in severity of global symptoms and abdominal pain in irritable bowel syndrome (IBS). Meta-analyses mostly show the effects are modest
5. Reduction in incidence, severity and/or duration of upper respiratory tract infections (colds and flu) Several systematic reviews and meta-analyses of studies in infants, children and adults indicate beneficial effects, although it is clear that these are often probiotic strain specific.

There has also been research with certain probiotics into their prophylactic use with antibiotics to help reduce risk of antibiotic-associated diarrhoea and C. diff infection. Probiotics are not recommended by (NICE) but some Trusts do encourage probiotic use in this regard.

Q. *Are probiotics safe?*

A. Probiotic genera, especially lactobacilli and bifidobacteria, have a long history of safe use in foods and also are commonly present in the gut, and so are unlikely to cause harm in healthy people. However, there have been safety concerns around use of probiotics, especially *Saccharomyces boulardii*, in immunocompromised or severely ill patients, with instances of systemic bacterial or fungal infections.

Supporting material
BDA. Neutropenic Dietary Advice for Haematology Patients

Ultra processed food

Q. *What are Ultra processed foods (UPFs)?*

A. A new approach to food classification has been developed recently, which is based on the degree of processing rather than on the nutrient composition. The most common classification system used is NOVA. This assigns foods into one of four categories:

1. Minimally/unprocessed foods (e.g., milk, plain yoghurt)
2. Processed culinary ingredients (e.g., butter, oils, sugar)
3. Processed foods (e.g. canned vegetables, cured meats)
4. Ultra-processed foods defined as "ready-to-eat, industrially formulated foods" and includes chocolate, ice cream, biscuits, and fruit yoghurts. The foods in category 4 are often characterised as having high energy density, high glycaemic index, and low satiety.

Q. *Are UPFs harmful?*

A. A growing number of epidemiological studies have reported associations between the consumption of UPFs and the risk of obesity, metabolic syndrome, colon cancer, and mortality but the use of this system in nutritional epidemiology studies has been challenged. There are few if any intervention trials which would needed to establish causative relationships and concerns exist about the lack of plausible mechanisms by which processing might influence disease processes. Nevertheless, there is evidence from observational studies of an association between diets with high level in UPFs and gut disease and disorders, including inflammatory bowel disease, colorectal cancer and irritable bowel syndrome.

Q. *Do UPFs affect the gut microbiota*

A. A recent review (Brichacek et al 2024) identified four observational studies to investigate links between UPFs and gut microbiota. There were considerable differences in the way the NOVA classification was applied to the dietary assessments as well as differences in the methodology used for microbiota analysis. None of studies reported effects on beta diversity of the microbiota and only one study showed an effect on alpha diversity. There were no consistent effects of UPF on Firmicutes/Bacteroidetes ratio, and few consistent effects seen at the genus level. Clearly more studies, especially carefully controlled intervention studies are needed.

Fermented foods

Q. *What are fermented foods?*

A. The term fermented foods has been defined broadly as "foods made through desired microbial growth and enzymatic conversions of food components". Fermented foods have been consumed for many thousands of years and include a huge variety of food materials ranging from bread, cheese, coffee and chocolate, to sauerkraut, wine, and yogurt. For some of these products (e.g. bread, coffee wine, beer) the microbes used in the fermentation have been removed or killed. Current scientific interest focusses on those foods in which the microbes (bacteria, yeasts and moulds) remain alive.

Q. *What fermented foods contain live organisms?*

A.

1. Fermented milk products
   - Yogurt: all contain *Streptococcus thermophilus* and *Lactobacillus debrueckii* susp l*actis*, sometimes with additional probiotic strains
   - Kefir: a drink derived from cow's, goat or sheep's milk fermented with kefir grains. These contain a diverse, complex, symbiotic consortium of bacteria, yeasts and filamentous fungi, the composition of which still not fully established. It is important to note that the composition varies according to the source of the grains, milk type used, processing and storage technique so kefir is not a well-defined entity so its health benefits may vary according to the type used in a study
2. Fermented soy products (mainly from Korea, China; Japan)
   - Miso. Soybeans fermented with a mould Koji
   - Natto. Soybeans fermented with *Bacillus subtilis*
   - Tempeh. Soybeans fermented with the fungus *Aspergillus oryzae*

3. Fermented vegetables
   - Sauerkraut. Cabbage fermented with various lactic acid bacteria
   - Kimchi Various vegetables fermented with lactic acid bacteria

4. Kombucha – Sugared tea fermented with a variable symbiotic mixture of organisms usually including *Saccharomyces cerevisiae.*

Q. *Do fermented foods influence the gut microbiota?*

A. As might be expected given their microbial content, fermented foods with live microbes, in particular fermented dairy products, have been shown in some human studies to modulate the gut microbiota, although effects are inconsistent.

A comparative study of consumers and non-consumers of fermented plants found subtle but statistically significant differences in overall gut microbial community, with higher ratios of certain microbes such as *Bacteroides, Faecalibacterium* and *Prevotella* in consumers (Taylor et al 2020).

A review of kombucha studies (de Campos Costa et al 2023) reported some beneficial effects on gut microbiota dysbiosis and microbiota changes after kimchi consumption have also been reported. Increases in faecal lactobacilli have seen in patients with inflammatory bowel disease.

Q. *Do fermented foods have beneficial health effects in the gut?*

A. Consumption of live yogurt containing *Lactobacillus delbrueckii* subsp. *bulgaricus* and *Streptococcus thermophilus* has been extensively investigated as a method of improving lactose digestion in subjects who are lactose maldigestors. Virtually all human studies have demonstrated enhanced digestion of lactose and the evidence was sufficiently convincing for EFSA to issue a positive scientific opinion for a health claim.

For most other fermented foods (kombucha, tempeh kimchi, natto miso), there is only limited evidence from clinical studies for beneficial effects on gastrointestinal health, with most trials being of poor quality in terms of size, randomisation and controls. Kefir has been the subject of a few randomised controlled trials, which suggest that it may have benefits for lactose malabsorption and reduction of *Helicobacter pylori* carriage.

Other dietary topics

Q. What is precision nutritional therapy in gastrointestinal disease?

A. Precision nutritional therapy refers to disease management which is tailored to a person's characteristics and own biology, including the gut microbiome. Such treatment paradigms may lead to a more effective management of a non-communicable diseases, including diseases of the gastrointestinal tract. While for certain conditions, patient treatment can be stratified to specific disease characteristics, there is currently insufficient evidence to make recommendations for precision nutritional therapy for people with gastrointestinal diseases. Laboratories and commercial entities offering such services owe to provide robust evidence base to support their commercial interests.

Q. What is the internal shower drink, and does it work?

A. The internal shower drink is a blend of water, chia seeds and lemon juice. It has gained traction through celebrities, social media and nutritionists as a drink to help improve gut health and digestion. Searching scientific literature, we did not identify any article to have tested the effect of the blend above in improving the gut microbiome and gut health in general. There is currently no scientific evidence to support the benefit of internal shower in improving a person's gut health and digestion.

Q. Are food additives and artificial sweeteners harmful?

A. Food additives and artificial sweeteners have been implicated in the underlying pathogenesis of various conditions of the gastrointestinal tract, mainly inflammatory bowel disease. Seminal articles in animal models of disease and in the lab, showed that consumption of food additives and artificial sweeteners can initiate gut inflammation through mechanisms involving modification of the gut microbiome to a pro-inflammatory status. While such preclinical research is of utmost importance in understanding disease aetiology, and in the development of drugs, confirmation within human clinical trials is mandated before recommendations can be made. To date, studies by James Lewis in US and Emma Halmos in Australia failed to improve disease biomarkers in patients with active Crohn's disease who followed diets low in food additives, including food emulsifiers. While more research is undergoing in this area, we currently do not have robust evidence to suggest that elimination of food additives from the diet of a person with gut diseases will improve their conditions. Until such evidence becomes available, general public are recommended to follow their national dietary recommendation for healthy eating and patients with gastrointestinal disease to follow their healthcare professional's advice.

Q. What is the low FODMAP diet, and does it work?

A. The low FODMAP diet is an established dietary treatment for people suffering for irritable bowel syndrome (IBS). It is a structured diet which aims to reduce the intake of certain classes of carbohydrates and polyols which are poorly digested or absorbed in the small intestine, subsequently causing bloating and abdominal discomfort. Eliminating FODMAPS from a person's diet is likely to reduce the amount and alter the type of fibre a person consumes. In turn, this will have significant effects on the gut microbiome with evidence showing a reduction of fibre-fermenting organisms and producers of beneficial metabolites in the gut originating from fibre fermentation. The long-term implications of such microbial changes on gut health remain unknown, but they are unlikely to cause harm when short-term restriction of food high in FODMAPs is required. Patients should discuss with their dietitians to learn about alternative plant-based foods that are good sources of fibre but low in FODMAP contents, like carrots, lettuce, grapes and kiwis. This way irritable bowel syndrome symptoms will controlled following a low FODMAP diet, with minimal negative effects on the composition and function of the gut microbiome.

Note: FODMAP stands for Fermentable Oligosaccharides, Disaccharides, Monosaccharides, and Polyols.

Q. What is the Mediterranean diet and does it work?

A. There is a vast amount of data from nutritional surveys and dietary trials showing the benefits of the Mediterranean diet on cardiometabolic health and prevention of cardiovascular disease. The Mediterranean diet is a dietary pattern characterised by high consumption of vegetables, whole grains, legumes and nuts, and the main oil used is olive oil. It limits the intake of red meat, sweets and confectionaries whereas fish, poultry and eggs intake are consumed in moderation. How the Mediterranean diet protects the development of cardiovascular disease is mostly unclear but evidence the past decade strongly points toward mechanisms involving the gut microbiome. People who consume a Mediterranean diet have higher levels of beneficial microbes which have the genetic machinery to ferment fibre and produce beneficial metabolites for host health, and fewer microbes which are labelled as opportunistic pathogens and can produce presumably harmful metabolites for gut health.

Q. What is the Candida diet and does it work?

A. The Candida diet restricts the intake of certain foods, such as sugar, gluten, alcohol and some dairy products, that are believed to promote the growth of Candida (a yeast) in the body. There is limited and inconclusive research on its effectiveness.

https://www.thecandidadiet.com
https://www.medicalnewstoday.com/articles/326795#what-is-candida
https://pubmed.ncbi.nlm.nih.gov/31215785/

5. Cancer

Q. Is there any link between cancer and the gut microbiome?

A. Modern oncological management of most solid cancers often involves a combination of approaches such as surgery, chemotherapy, and immunotherapy, that are used over the course of a long and complex care journey. The gut microbiome, sometimes called the "oncomicrobiome" in cancer settings, is unique to each person and constantly changing.

There is growing evidence that the gut microbiome plays a key role not only in how cancer is treated, but also in how it develops in the first place. The balance of microbes in the gut can influence inflammation, the immune system, and even DNA damage—factors known to contribute to the onset of cancer. Some bacteria, such as *Fusobacterium nucleatum* and certain strains of *Escherichia coli*, have been linked to the development of colorectal cancer. These bacteria can produce toxins or create a pro-inflammatory environment that promotes tumour formation. Conversely, beneficial microbes may help to maintain a healthy gut lining and suppress harmful processes that can lead to cancer (Brennan and Garrett, 2019; Sears and Garrett, 2014).

Q. Are any gut bacteria linked to cancer?

A. The balance of microbes in the gut can influence inflammation, the immune system, and even DNA damage—factors known to contribute to the onset of cancer. Some bacteria, such as *Fusobacterium nucleatum* and certain strains of *Escherichia coli*, have been linked to the development of colorectal cancer. These bacteria can produce toxins or create a pro-inflammatory environment that promotes tumour formation. Conversely, beneficial microbes may help to maintain a healthy gut lining and suppress harmful processes that can lead to cancer (Brennan and Garrett, 2019; Sears and Garrett, 2014).

This understanding has led scientists to investigate whether the microbiome could be used as a tool for cancer prevention, early detection, or even as a marker for prognosis. For example, researchers are exploring whether microbial signatures in stool samples could act as non-invasive early warning signs for colorectal cancer. There's also interest in whether modifying the microbiome—through diet, probiotics, or targeted antibiotics—could help lower cancer risk or slow its progression. Harnessing the gut microbiome may one day help identify high-risk individuals, guide screening strategies, or even offer new ways to prevent cancer altogether (Zackular et al., 2014; Wong and Yu, 2019).

Q. Does the gut microbiome influence how well cancer therapy may work?

A. Recent research has also shown that the gut microbiome can have a powerful impact on how well cancer treatments work, particularly chemotherapy and immunotherapy (Chrysostomou et al, 2023). For example, *Fusobacterium nucleatum*, a bacterium often found in the gut of patients with bowel cancer, has been shown to reduce the effectiveness of a common chemotherapy drug, 5-fluorouracil, by interfering with the way cancer cells die (Yu et al., 2017). Other gut microbes produce substances like short-chain fatty acids (SCFAs) which help control inflammation and protect the gut lining, both of which can influence how a patient responds to treatment and whether they experience side effects (Alexander et al., 2017; Coutzac et al., 2020).

The microbiome also plays a key role in newer cancer treatments that stimulate the immune system, such as immune checkpoint inhibitors. These drugs work by helping the immune system recognise and attack cancer cells. Studies have found that patients with higher levels of certain gut bacteria—such as *Akkermansia muciniphila*, *Bifidobacterium longum*, and *Faecalibacterium prausnitzii*—tend to respond better to these therapies (Gopalakrishnan et al., 2018; Routy et al., 2018). These bacteria appear to boost the body's immune response, making treatment more effective. On the other hand, using antibiotics before or during

immunotherapy may disrupt the microbiome and reduce the effectiveness of these drugs. This has led scientists to explore ways to support or restore a healthy microbiome—through diet, probiotics, or even faecal microbiota transplantation (FMT)—to improve cancer outcomes. This area of research is still developing, but it shows promise for making cancer treatment more personalised and effective in the future (Derosa et al., 2022).

However, the roles of different microbiomes in cancer development and treatment are constantly being researched and revised; one particular recent development relates to the finding that the gut microbiome might influence how likely people with blood cancer are to respond to treatments that they are receiving. For example, in a study called MAST, the scientists and clinicians are investigating the impact of giving a stool transplant to patients with blood cancer, before they have their bone marrow transplant. This study is being run from Imperial College London (https://tinyurl.com/3v6dfa9p) and involves 6 other hospitals in London (The Royal Marsden, King's College and UCLH), Birmingham, Leeds and Manchester.

And finally…

While our understanding of the role played by the gut microbiome in cancer development and treatment is ever increasing, it is important to note that at present there is insufficient evidence to support microbiome testing or microbiome manipulation to predict, prevent or treat cancer in routine practice.

Supporting material

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- Yu, T et al. (2017) *Fusobacterium nucleatum* promotes chemoresistance to colorectal cancer by modulating autophagy. *Cell*, 170(3), pp.548–563.e16.
- Zackular, J.P. et al (2014) The human gut microbiome as a screening tool for colorectal cancer. *Cancer Prevention Research*, 7(11), pp.1112–1121.

6. Antimicrobial resistance (AMR)

Q. What is AMR and why does it matter?

A. Antimicrobial resistance (AMR) occurs when bacteria and other microbes evolve to withstand the drugs, such as antibiotics, designed to kill them. As a result, infections that were once easily treatable become more difficult, and in some cases, impossible to cure.

AMR is widely recognised as **one of the biggest threats to global public health**, food security, and development today. It affects people of all ages and increases the risk of complications during routine medical procedures, including surgeries, cancer chemotherapy, organ transplantation, and care of premature infants.

Key facts and figures:

- **An estimated 1.27 million deaths** were directly caused by AMR in 2019 worldwide, with nearly **5 million associated deaths** in total (The Lancet, 2022).
- In the UK alone, **AMR is linked to over 7,000 deaths annually**, a figure expected to rise if no action is taken (UKHSA, 2024).

- Without urgent intervention, global AMR deaths could **exceed 10 million per year by 2050**—surpassing deaths from cancer (O'Neill review on AMR, 2016).
- AMR costs the global economy **tens of billions of pounds** annually in healthcare expenses and productivity losses (WHO, 2023)

Overuse and misuse of antibiotics in **human health**, **veterinary medicine**, and **agriculture** accelerate the development of resistance. Resistant bacteria can spread between people, animals, and the environment, making AMR a **One Health issue** requiring a coordinated, multi-sector response.

Combating AMR is essential to preserving the effectiveness of modern medicine. Without effective antibiotics, even minor infections or injuries could once again become life-threatening.

Q. What does it mean to carry resistant bacteria in your gut?
A. Many people unknowingly carry **antibiotic-resistant bacteria** in their gastrointestinal tract – **asymptomatic colonisation**. These bacteria live harmlessly in the gut without causing illness, but their presence is significant for individual and public health.

This "silent carriage" doesn't typically cause symptoms, but it presents two key risks:

1. **Infection risk** – If a person becomes immunocompromised, has surgery, or sustains trauma, these bacteria can translocate from the gut to other parts of the body (such as the bloodstream or urinary tract), potentially causing **hard-to-treat infections**.
2. **Transmission risk** – Carriers can unknowingly spread resistant bacteria to others, particularly in hospitals, care homes, or within households. The bacteria can survive on hands, surfaces, or medical equipment, especially in environments with weakened infection control. Washing hands and good infection control are key to reducing the risk.

Key facts and figures:

- Up to **5–10% of healthy people** in the UK population may be colonised with **extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriales**. (unpublished local data) These bacteria are resistant to a wide range of antibiotics including penicillins and cephalosporins.
- After international travel, particularly to regions with high antibiotic use or poor sanitation, up to **70% of people** may return temporarily colonised with resistant gut bacteria. (Ostholm-Balkhed et al, 2013)
- **Carbapenem-resistant organisms (CRO)**, considered among the most dangerous AMR threats, can persist in the gut for **months or even years** after exposure. (Haverkate et al, 2016)

Screening for gut colonisation is usually reserved for high-risk settings – such as hospitals – where **early identification can prevent outbreaks**. However, the public plays a key role in prevention through hygiene, responsible antibiotic use, and food safety practices.

Gut colonisation acts as a **reservoir for resistance genes**, meaning resistant traits can potentially be transferred to other, more harmful bacteria in the microbiome – a process that accelerates the overall spread of AMR.

Q. How do resistant bacteria get into the gut?
A. Resistant bacteria can enter the gut through a variety of everyday exposures. While most people think of hospitals or illness, many carriers acquire resistant microbes in the community—often without realising it.

Main routes of entry include:

- *Contaminated food or water*
  Resistant bacteria are commonly found in raw or undercooked meat, unwashed fruits and vegetables, and improperly handled food. They can also be present in drinking water, particularly in areas with inadequate sanitation or water treatment.
- *Person-to-person transmission*
  Close contact—especially in settings like households, care homes, or hospitals—can result in the spread of resistant organisms. Poor hand hygiene is a major contributor.

- *Antibiotic use*
  When antibiotics are taken—particularly broad-spectrum types—they not only kill harmful bacteria but also disrupt the normal gut microbiome. This imbalance can allow resistant strains to multiply and establish themselves.
- *Healthcare exposure*
  Hospital stays, especially involving invasive procedures or antibiotic treatments, significantly increase the risk of acquiring resistant bacteria in the gut.
- *International travel*
  Travelling to regions with high levels of AMR (e.g. South Asia, parts of Africa and Latin America) increases the chance of acquiring resistant gut bacteria—often through food, water, or medical care.

Key facts and figures:

- A 2015 study found that **up to 90% of chicken meat samples** in some UK supermarkets contained bacteria resistant to at least one antibiotic (FSA, 2015).
- After short-term international travel, **30–70% of travellers** become colonised with resistant Enterobacterales—especially ESBL-producing strains. (Ostholm-Balkhed et al 2013)
- Use of antibiotics increases the risk of resistant gut colonisation **up to 3-fold**, particularly with repeated or inappropriate use (Wuerz et al, 2020).

Reducing the risk involves a combination of **safe food handling**, **good hygiene**, **responsible antibiotic use**, and being informed about travel-related health risks. Public awareness and behaviour change are critical in preventing the spread of AMR into and through the gut.

Q. Who is most at risk?

A. While anyone can carry antibiotic-resistant bacteria in their gut, certain individuals and groups are at **higher risk** due to factors that increase their exposure or disrupt their natural gut microbiome.

High-risk groups include:

- *Hospitalised patients*
  Especially those in intensive care, undergoing surgery, or receiving invasive treatments like catheters or ventilators. Hospital environments can harbour resistant bacteria, and antibiotic use is common.

- *Residents of care homes or long-term care facilities*
  Frequent antibiotic use, shared living spaces, and underlying health conditions make this group particularly vulnerable to colonisation and transmission.

- *People who have recently taken antibiotics*
  Antibiotics disturb the natural balance of gut bacteria, giving resistant strains an opportunity to grow and persist.

- *International travellers*
  Visiting regions with high AMR prevalence—particularly South and Southeast Asia, Africa, and parts of South America—significantly increases the risk. Resistant strains can be acquired from food, water, and healthcare exposure. Travellers can protect themselves by practicing good hygiene, avoid risky food and water, and don't take antibiotics unless necessary.

- *People with weakened immune systems*
  Individuals with chronic diseases (like diabetes or cancer), transplant recipients, or those undergoing immunosuppressive therapy are more susceptible to colonisation and infection.

- *Healthcare workers and carers*
  Frequent contact with patients and clinical environments raises the risk of acquiring and transmitting resistant organisms.

- *People with frequent contact with animals*
  Farmers, veterinarians, and abattoir workers can be exposed to resistant bacteria through livestock, particularly when animals have been given antibiotics.

Key facts and figures:

- In UK hospitals, up to **20% of patients in high-dependency units** may carry resistant Gram-negative bacteria such as ESBL-producing *E. coli* or *Klebsiella*.
- Residents in long-term care facilities may carry AMR organisms at rates **2–5 times higher** than those in the general population. (OECD review, 2022)

Understanding who is most at risk is key to shaping screening, prevention, and targeted public health strategies to limit the spread of AMR through the gut.

Q. Can antibiotics cause resistant bacteria in the gut?

A. Yes, antibiotics - especially broad-spectrum ones - can **significantly increase the risk** of resistant bacteria establishing themselves in the gut. While antibiotics are essential for treating infections, their overuse and misuse are major drivers of AMR.

How it happens:

- **Antibiotics disrupt the gut microbiome**, killing not only the harmful bacteria causing infection but also many of the beneficial or neutral bacteria that help maintain gut health.
- This disruption creates a vacuum that allows resistant bacteria—often already present in small numbers—to thrive, multiply, and dominate.
- The gut then becomes a **reservoir of resistance**, potentially harbouring bacteria that are resistant to multiple antibiotics.

Broad-spectrum antibiotics:

- These are drugs that target a wide range of bacteria, used when the specific cause of an infection is unknown.
- While useful in emergencies, broad-spectrum antibiotics are more likely to **wipe out diverse populations of healthy gut bacteria**, creating more space for resistant organisms.
- They are particularly associated with colonisation by **ESBL-producing bacteria** and ***Clostridioides difficile***.

Key facts and figures:

- Patients who receive antibiotics are up to **3 times more likely** to become colonised with resistant bacteria (Wuerz et al, 2020).
- Inappropriate or unnecessary prescribing accounts for **up to 20% of antibiotic use** in UK primary care, according to NHS data.

The takeaway:

Antibiotics should be used **only when necessary**, and the choice of antibiotic should be as **narrow spectrum as clinically appropriate**. This reduces the risk of AMR developing in the gut and helps preserve the effectiveness of these potentially life-saving medicines for the future.

Q. How is gut carriage detected?

A. Detection of gut carriage of antibiotic-resistant bacteria is typically done through **screening tests**, which are most often used in hospitals, particularly in high-risk patients or during outbreaks.

The most common methods include:

- Culture-based testing

  A swab of the rectal area or a small sample of stool is collected and sent to a laboratory. These are the standard specimens used for detecting gut colonisation with resistant bacteria such as:
  - *Carbapenemase-producing Enterobacterales (CPE)*
  - *Extended-spectrum beta-lactamase (ESBL)*-producing organisms
  - *Vancomycin-resistant Enterococci (VRE)*

  In the lab, samples are cultured on special media designed to encourage the growth of resistant organisms while inhibiting others. The bacteria are then identified, and resistance patterns are confirmed.

- Molecular methods (PCR tests)
  Increasingly, some NHS laboratories use rapid molecular techniques to detect the presence of specific resistance genes (such as *blaKPC*, *blaNDM*, or *vanA*) directly from patient samples.

When is testing done?

- **Upon hospital admission** for patients with known risk factors (e.g. recent overseas hospitalisation, previous carriage, or exposure during an outbreak).
- **During contact tracing** when a known carrier has been identified in a healthcare setting.
- **Before high-risk procedures**, such as organ transplantation or intensive care admission, in some hospitals.

Key facts:

- In the UK, national guidance recommends **routine screening for CPE** in high-risk patients using rectal swabs.
- Screening allows hospitals to implement **early isolation and tailored infection control measures**, preventing the spread of resistant bacteria.

Detecting gut carriage is not about diagnosing illness—it's about identifying **hidden reservoirs of resistance** and stopping transmission before infections occur.

Q. Are carriers isolated in hospital?

A. Yes—**in many cases, people who are known carriers of antibiotic-resistant bacteria in their gut may be isolated or placed under special infection control precautions** while in hospital. The decision depends on the **type of resistant bacteria**, the patient's condition, and the hospital's infection prevention policies

Q. Why does isolation matter?

A. Carriers can **unknowingly shed resistant bacteria**, which may contaminate surfaces, equipment, or the hands of healthcare workers. In vulnerable hospital populations – such as those in intensive care or undergoing surgery – even low levels of contamination can cause **serious infections or outbreaks**.

Infection control strategies may include:

- *Single-room isolation*
  Patients may be cared for in a single room, especially if colonised with high-risk organisms like *carbapenemase-producing Enterobacterales (CPE)*
- *Barrier nursing (contact precautions)*
  Healthcare staff wear gloves and aprons (or gowns) when entering the room and follow strict hand hygiene protocols when leaving.
- *Dedicated equipment*
  Medical equipment such as blood pressure monitors or thermometers may be assigned to the patient to prevent cross-contamination.
- *Enhanced environmental cleaning*
  More frequent and thorough disinfection of rooms and shared facilities is used, particularly for organisms that persist in the environment.

Q. When is isolation used?

- **Proactively**, when screening identifies resistant gut bacteria in high-risk patients.
- **Reactively**, during outbreaks or when a carrier develops an infection.
- **Contextually**, depending on the level of risk to others and the resources available (e.g. room availability).

Key facts:

- In the UK, NHS hospitals follow national guidelines such as those from the UK Health Security Agency (UKHSA) and NICE on managing patients colonised with AMR organisms.
- CPE carriage requires **prompt isolation** due to its high potential for spread and resistance to nearly all available antibiotics.
- Isolation aims to protect **both the patient and others**, and it is not a form of stigma.

Q. What role does the gut microbiome play?

A. The **gut microbiome**—the vast community of trillions of bacteria, viruses, fungi and other microbes living in the digestive tract—plays a **central role in both protecting against and facilitating the spread of antimicrobial resistance (AMR)**.

Protective role: colonisation resistance. A healthy and diverse gut microbiome acts as a **natural defence system**, a concept known as *colonisation resistance*. This means:

- Beneficial microbes **compete with harmful bacteria** for space and nutrients.
- They produce substances (e.g. bacteriocins, short-chain fatty acids) that can inhibit or kill invading pathogens.
- They stimulate the **immune system**, helping it to identify and eliminate harmful bacteria.

When the microbiome is intact, it helps **prevent colonisation by antibiotic-resistant bacteria**, reducing the chance of them taking hold and multiplying.

Disruption and risk. However, factors such as:

- **Antibiotic use** (especially broad-spectrum agents),
- **Poor diet**
- **Illness or hospitalisation**

can disrupt this balance—reducing microbial diversity and allowing **resistant bacteria to thrive**. Once established, these bacteria can persist for months and may pass resistance genes to other microbes through a process called **horizontal gene transfer**.

AMR reservoir. The gut microbiome is increasingly recognised as a **major reservoir of resistance genes**. These genes can be:

- Harboured by harmless bacteria,
- Shared with potentially pathogenic species (e.g. *E. coli*, *Klebsiella*, *Salmonella*),
- Spread to other people via faecal-oral routes (e.g. poor hygiene, contaminated food).

Key facts:

- The gut contains **more bacterial cells than the rest of the human body combined**, making it a critical environment for resistance development.
- Studies have shown that after a single course of antibiotics, **resistant strains can increase significantly and persist for up to a year**.
- Microbiome disruption is associated not only with AMR but also with ***C. difficile* infection**, inflammatory bowel disease, and metabolic conditions.

Implications for prevention and treatment:

- Strategies to preserve or restore the gut microbiome, such as **microbiome-friendly antibiotic prescribing**, **dietary interventions**, and **probiotics or faecal microbiota transplant (FMT)** – are being explored as potential strategies to combat AMR.
- Maintaining a healthy gut microbiome could be an important tool in the future of AMR prevention.

Q. What are bacteriophages?

A. Bacteriophages (or simply “phages”) are **viruses that infect only bacteria**. They outnumber every other biological entity on Earth (≈10^{31} particles) and are found wherever bacteria live – soil, water, food, and the human gut. Each phage consists of a nucleic-acid genome encased in a protein capsid and usually a tail-like structure that recognises a single bacterial species or even a single strain. When a phage attaches to its target it can follow:

> **Lytic cycle** – the phage replicates inside the bacterium and bursts the cell, killing it.
> **Lysogenic cycle** – the phage genome integrates or persists episomally inside the bacterium until triggered to enter the lytic phase.

This exquisite **host-specificity is the property exploited for therapy**, allowing phages to remove a pathogen without disturbing neighbouring “good” microbes.

Q. How does bacteriophage therapy work in practice?

1. **Selection / engineering of active phages:**
   - Clinicians send the patient's bacterial isolate to a reference laboratory or "phage bank". Phages with proven lytic activity (or cocktails that broaden host range) are matched to the strain; some centres also use genome-edited phages to enhance potency or evade bacterial defences.

2. **Administration routes:**
   - Topical (wounds, burns, fistulae)
   - Oral or nasogastric (gut infections or decolonisation)
   - Intravenous or intraperitoneal for deep-seated or disseminated infection

3. **Mode of action:**
   - Direct lysis of the bacterium during the lytic cycle.
   - Biofilm penetration – many phages carry depolymerase enzymes that digest the extracellular matrix, exposing bacteria to immune attack or adjuvant antibiotics.
   - Synergy with antibiotics – sub-minimum inhibitory concentrations (MIC) i.e. levels insufficient to kill the bacteria of antibiotics can boost phage adsorption, while phage-mediated damage increases antibiotic penetration. Multiple *in vitro* and animal studies have confirmed this additive effect.

Q. Is bacteriophage therapy new?

A. No, it has been in clinical use for over 100 years – a timeline is given below.

- **1910s** Discovery by Frederick Twort and Félix d'Hérelle; first therapeutic use in 1919.
- **1920s–30s** Widely used in Europe and the former USSR to treat dysentery, cholera and staphylococcal infections.
- **1940s–60s** Interest waned in the West after the antibiotic era began; phage work continued in Georgia and Poland.
- **1990s–2010s** Resurgence driven by global AMR; modern sequencing and GMP production solved earlier issues of purity and reproducibility.
- **2020s** –Dedicated phage centres established in the USA, Australia, Belgium and now the UK; dozens of interventional trials registered worldwide.

Q. Are phage therapies currently available in the UK?

Licensed products: None yet. No phage medicine has a UK marketing authorisation.

Compassionate / "specials" use: Over a dozen UK patients with life-threatening, drug-resistant infections have received bespoke phage preparations since 2023, sourced from international GMP libraries under clinician responsibility.

Regulatory framework: In June 2025 the Medicines and Healthcare products Regulatory Agency (MHRA) issued the UK's first guidance for developing phage medicines (https://www.gov.uk/government/news/helping-bring-phage-medicines-to-uk-patients-guidance-for-industry). It sets out quality, safety, and clinical-trial expectations for both personalised and off-the-shelf phage cocktails and aligns with the UK's 2024-2029 AMR National Action Plan.

Pipeline: Multiple academic–industry consortia are building an onshore GMP phage manufacturing facility and a national phage library to streamline NHS access. Early-phase trials for *Pseudomonas* lung infection in cystic fibrosis and *E. coli* urinary tract infection are slated to start recruitment in late 2025.

Q. Are bacteriophages safe?

Clinical evidence: A systematic review in the Lancet of 52 clinical studies (Uyttebroek et al, 2022) reported phage therapy was well tolerated in most cases. Adverse events were reported more frequently in controls than those receiving phage therapy. Generally, any adverse events reported were mild and resolved after the phage therapy ended. Many patients receiving phage therapy died, but this was because of the severity of their disease rather than the receipt of phage therapy.

Immunogenicity: Phages are immunogenic proteins, but neutralising antibodies typically arise only after prolonged (>2 weeks) systemic exposure. Short courses for acute infections rarely trigger clinically relevant inactivation.

Endotoxin concerns: Modern GMP purification achieves <5 EU/kg endotoxin, comparable to injectable biologics.

Horizontal gene transfer: Therapeutic phages are obligatorily lytic (do not integrate into bacterial DNA) and undergo full-genome sequencing to exclude toxin or resistance genes before release.

Regulatory safeguards: UK phage specials are manufactured under MHRA-audited GMP or imported under strict "named-patient" controls; batch certificates accompany every dose.

Collectively, current data and post-treatment surveillance suggest that well-characterised phages have a safety profile on par with other biological therapeutics, though large, randomised trials are still required.

7. **Gut microbiome influence**

Q Is there a link between the gut microbiome and mood?
A. Emerging evidence suggests the gut microbiome can influence mood and mental wellbeing; a concept often termed the "gut-brain axis". Studies indicate that alterations in gut microbial communities can influence the production of neurotransmitters and inflammatory mediators, potentially affecting mood, anxiety, and depressive symptoms. Preclinical studies using animal models provide strong support for a causal role of microbiome changes in mood regulation; however, translating these findings to humans remains challenging. Most human research to date has been observational, and robust human intervention studies are essential to confirm causation, clarify mechanisms, and guide potential microbiome-based therapies for mood disorders.

Q Is there a link between the gut microbiome and allergies?
A. Early life gut microbiome development appears to play an important role in shaping the immune system, potentially influencing susceptibility to allergic conditions such as food allergies, eczema, and asthma. Research indicates that infants with lower microbiome diversity and reduced beneficial microbes, including certain species of *Bifidobacterium*, may have an increased risk of developing allergies. Modulating the microbiome using probiotics, prebiotics, or dietary interventions offers promising avenues. Currently, some clinical guidelines support probiotic use during pregnancy and early infancy, particularly for reducing the risk of eczema (as recommended by the World Allergy Organization - WAO), although broader and definitive clinical guidance for other allergic conditions still requires further rigorous research.

Q. Can the gut microbiome influence the efficacy of drug therapy?
A. The gut microbiome significantly influences how individuals respond to medications, either enhancing or reducing drug effectiveness and altering side-effect profiles. Gut microbes can metabolise drugs, changing their absorption, activity, or excretion, and conversely, many drug therapies - including antibiotics and chemotherapy - can profoundly alter microbiome composition. An exciting emerging area is microbiome-targeted therapies that enhance immune responses and improve patient outcomes, such as boosting the effectiveness of immune checkpoint inhibitors used in cancer treatment. A better understanding of these complex microbiome-drug interactions could lead to more personalised, effective, and safer therapeutic strategies, although clinical translation is still in its early stages.

Q. Is there a link between the gut microbiome and autism?
A. The link between autism and the gut microbiome remains uncertain and complex. While children with autism often show differences in gut microbiome composition compared to neurotypical peers, recent robust evidence suggests these differences may be driven more by restricted diets, altered eating patterns, or gastrointestinal symptoms commonly experienced by autistic individuals rather than autism itself. Although early studies indicate potential benefits from microbiome-targeted therapies (e.g., probiotics or faecal transplants) in improving gastrointestinal symptoms, there is currently no strong evidence these interventions improve core autism symptoms. Further carefully controlled studies are needed to clarify these complex relationships.