Mistakes in the management of carbohydrate intolerance and how to avoid them

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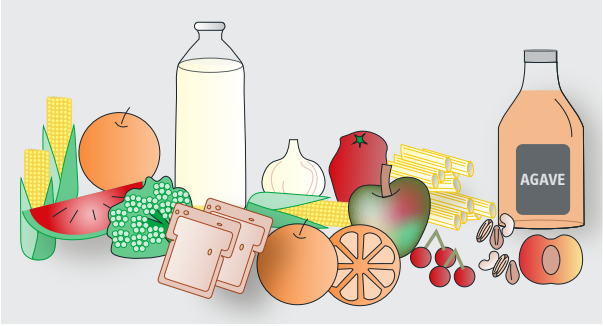

Carbohydrates not absorbed in the small intestine are fermented by colonic bacteria to organic acids and gases[1] (e.g. carbon dioxide, hydrogen and methane), part of which is absorbed in the colon, the other part remains in the lumen.[2,3] Large interindividual differences have been demonstrated for the production of such acids[4] and gas.[5,6] Fermentation products may cause symptoms of bloating, abdominal pain, diarrhoea and nausea;[6] however, although colonic fermentation occurs in everyone, most people do not report symptoms.[6-8]

In a recently published European Guideline,[9] the importance of correctly using the terms "malabsorption" and "intolerance" has been highlighted. Carbohydrate malabsorption is diagnosed by using a breath test, whereas intolerance is diagnosed when this process is related to the occurrence of abdominal symptoms. The European Guideline recommends that this should be done using validated methods such as the carbohydrate perception questionnaires (CPQ)[9] which are available for use in adult (aCPQ)[10] and pediatric (pCPQ)[11] patients and which are available in validated translations into several languages.[12] The clarification of the terms and the identification of "malabsorption" and "intolerance" with these validated tests now allows for a better discrimination of the role of carbohydrate intolerances in disorders of gut-brain interaction.[8] This is important as treatment targeted at the underlying causes of symptoms brings with it a higher likelihood of therapeutic success.[13-15]

Here, we discuss mistakes that are made when managing patients who have bloating, abdominal pain, diarrhoea and nausea, in whom carbohydrate malabsorption or intolerance has been diagnosed, or are thought to contribute to the condition. The discussion focuses on lactose malabsorption, because of its well-known pathophysiology and high prevalence; however, similar mechanisms apply for intolerances to other poorly-absorbable carbohydrates, like dietary fibres or FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides and polyols). As treatment focuses on symptom relief, evaluation of complaints thought to be related to carbohydrate ingestion has to place equal emphasis on the objective detection of carbohydrate malabsorption and symptom assessment.[16-19]

Mistake 1 Failing to distinguish food intolerance from food allergy

Many patients report having a reaction to food that is ascribed to an allergy; however, especially in adults, most food reactions are caused by intolerance. For practical purposes, patients have to be made aware of the difference between food allergy and food intolerance.

Classical food allergy is caused by an apparently dose-independent reaction of the immune system that has acute effects on many organs and systems, which can be life-threatening. By contrast, the symptoms and clinical consequences of food intolerance are dose dependent, generally less serious and are often limited to digestive problems.[20,21]

Symptom development and severity in those with a food intolerance depends on the amount of the food ingested, the digestion and assimilation of the food, and whether or not this process is tolerated. Different mechanisms that may be involved in food intolerance are shown in Figure 1. In the case of food allergy, the responsible allergen has to be completely avoided. By contrast, in the case of intolerance, the focus is on reducing the intake of the offending food. In addition, drugs that assist the digestion of certain foods, like pre- and probiotics,[22] lactase supplements[23] or xylose isomerase[24], or treating underlying conditions can be administered as part of the treatment for those with a food intolerance.

Mistake 2 Not considering the mechanisms underlying the relationship between food ingestion and symptom development

Patients who notice abdominal symptoms after eating a particular food frequently rely on its avoidance to treat their symptoms. However, in clinical practice, the association between food intake and symptom development may have different causal relationships (Figure 2). These relationships must be considered so that diagnostic evaluation and treatment of any underlying disease is not delayed.

For example, in patients who are lactose intolerant, it may be unclear whether acquired primary lactase deficiency or secondary lactase deficiency, due to another small intestinal disorder (e.g. infection, coeliac disease or inflammatory bowel disease [IBD]), is responsible for lactose malabsorption or lactose intolerance. Therefore, it may be necessary to exclude other malabsorptive disorders, especially if the patient's ethnic background is associated with a low prevalence of acquired primary lactase deficiency.

For practical purposes, food intolerances may have different functional or organic backgrounds, the clinical consequences of which range from

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Mechanism	Example
Maldigestion, malabsorption	Absence of an enzyme needed for digestion (e.g. lactase deficiency)
Physiologically incomplete absorption	FODMAPs, magnesium
Dysregulated handling of bowel contents	IBS
Reaction to the products of digestion	Histamine, gas, short-chain fatty acids
Sensitivity to food additives or contents	Sorbitol, fructose, xylitol
Concurrent medical conditions	Previous surgery, concurrent diseases
Concurrent psychological conditions	Stress, psychological factors

Figure 1 | Mechanisms involved in food intolerance.

being harmless nuisances to diseases requiring medical evaluation and treatment.[20,21]

Mistake 3 Assuming that the mechanisms underlying intolerance are completely understood

The typical symptoms of lactose intolerance (i.e. abdominal pain, bloating, flatulence, nausea and diarrhoea) are generally attributed to bacterial fermentation of lactose in the large intestine. Fermentation products increase the osmotic gradient, causing water to shift into the lumen to restore an isotonic milieu[25] that may contribute to abdominal pain and diarrhoea.[4] The gases released by colonic fermentation contribute to the sensation of bloating and to flatulence,[5] especially if there are conditions which are associated with increased perception of luminal distension, like irritable bowel syndrome (IBS) or IBD.[26,27]

Although colonic events have a major role in symptom generation, some symptoms develop rapidly, before intestinal contents have reached the colon. This may be a consequence of functional dyspepsia, an overactive gastro-colic reflex or of distension of the small intestine.[28] The latter mechanism is marked in the presence of small bowel bacterial overgrowth, in which fermentation with luminal distention due to gas production and fluid flux occurs already in the mid-gut.[29] Notwithstanding the above, the perception of bloating is not determined only by mechanical factors. Increased visceral sensitivity to the presence of gas is a very frequent finding in patients who have functional gastrointestinal disorders, especially in those with anxiety disorders.[30]

Practically speaking, it is important to remember that different factors are responsible for the development of symptoms in patients with carbohydrate malabsorption. The complex interplay between products of bacterial carbohydrate metabolism and the structures and functions of the gastrointestinal tract results in marked interindividual differences in the sensitivity to incompletely absorbed carbohydrates and symptom development.

Mistake 4 Ignoring the possibility that comorbidities influence symptoms in patients with carbohydrate malabsorption

Abdominal pain, bloating and a variable bowel habit are nonspecific symptoms that can occur with various functional or organic diseases, with or without carbohydrate malabsorption. In particular, intolerance of numerous foods is a hallmark of IBS.[31] Up to 82% of patients with IBS report food intolerances, with dairy products (49%), beans/lentils (36%), apple (28%) and wheat flour (24%) being the most frequently reported food items.[32] Symptoms were also frequently reported after intake of fried and fatty foods (52%). Although self-reported lactose intolerance is often not confirmed on breath testing, anxiety related to food intake has a negative impact on diet and quality of life.[33,34]

There is a large overlap between the occurrence of carbohydrate intolerance and IBS, both of which are common conditions worldwide. Altering dietary intake of fermentable carbohydrates, including lactose in patients with documented lactase intolerance, is known to improve symptoms in IBS.[35,36] In controlled studies, the risk of developing symptoms after lactose ingestion is related not only to the dose of lactose ingested but also to patient factors.[20] These factors include a history of abdominal surgery or recent gastrointestinal disease,[20] evidence of an activated mucosal immune system (e.g. increased mast cells in biopsy samples from the small intestine and colon)[30] and colonic dysbiosis.[15] Psychosocial factors, such as the presence of psychological disease and/or high levels of "life event stress", are also very important.[30,37] Many of these factors, especially inflammation and anxiety, are associated with visceral hypersensitivity in patients with IBS.[30] The shared aetiology of these conditions suggests that carbohydrate intolerance is a clinical manifestation of functional bowel disease.[38]

Mistake 5 Not considering the role of all poorly absorbed, fermentable carbohydrates in patients with suspected carbohydrate intolerance

In addition to the commonly considered carbohydrates lactose or fructose, many other incompletely absorbed carbohydrates may reach the colon and be fermented by bacteria.[39,40] Indeed, the mechanisms by which bacteria digest different carbohydrates (monosaccharides, disaccharides, polyols, oligosaccharides and digestible dietary fibres like pectin) follow the same principles[41] and therefore intolerance of lactose or fructose may be shared by many other types of carbohydrate intolerances, including

Causal relationship	Example	Clinical consequence
Food content is the cause of a disease	Food allergy, coeliac disease, alcoholic pancreatitis	Remove the offending food
Symptoms after food ingestion are a clinical manifestation of an underlying gastrointestinal, biliopancreatic or hepatic disease or abnormality	Biliary disease, irritable bowel syndrome (IBS), functional dyspepsia, small bowel obstruction, lactase deficiency	Detect and treat the underlying disease, reduce the offending food
Food contents stimulate or alter normal functions, possibly with the prerequisite of perturbed gastrointestinal function	Caffeine, fat, capsaicin (chilli), glutamate, histamine	Symptoms unrelated to a disease, reduce the offending food
Excessive ingestion of certain foods overwhelm normal physiologic absorptive capacities	FODMAPs, magnesium	Symptoms unrelated to a disease, reduce the offending food component

Figure 2 | Causal relationships between food intake and the GI tract in the pathogenesis of food-associated symptoms.

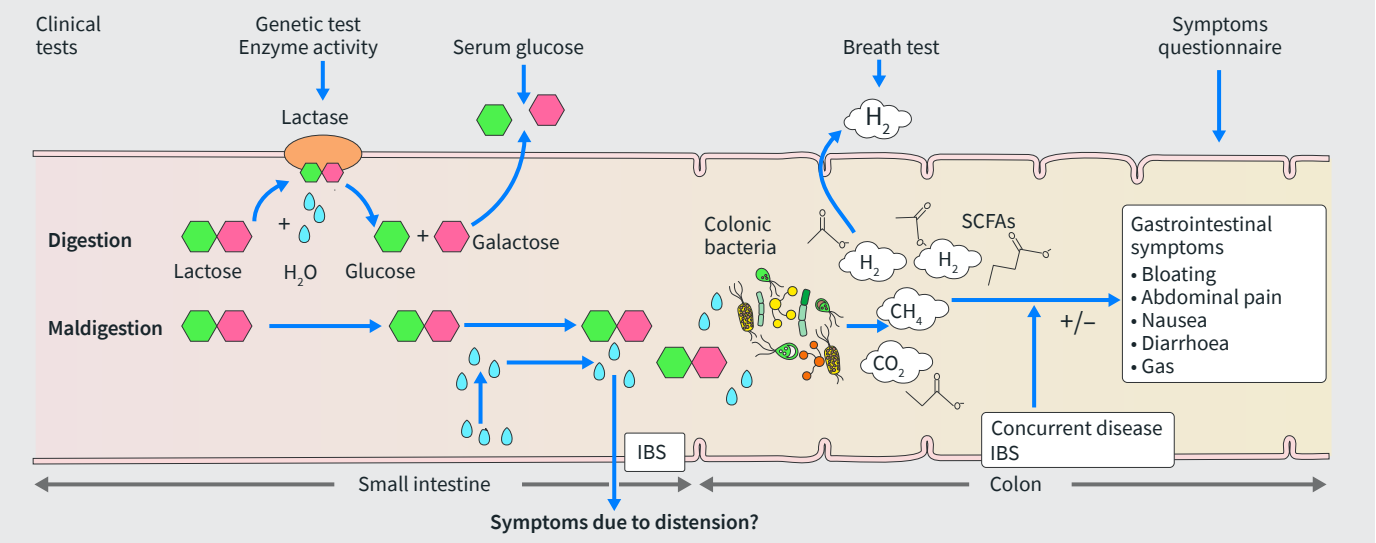

Figure 3 | Processes involved in lactose digestion, lactose malabsorption and lactose intolerance. In individuals with lactase persistence, lactose is digested by lactase to glucose and galactose, which are absorbed from the small intestine. Lactase activity can be measured in biopsy samples, and genetic testing can detect mutations associated with lactase persistence. Glucose absorption can be demonstrated by a rise in serum glucose concentration. In lactase deficiency, lactose enters the lower parts of the small and the large intestine along with water. Colonic bacteria ferment lactose, which generates gas and short-chain fatty acids. Absorbed hydrogen can be measured in breath. The interplay with concurrent diseases, such as IBS, leads to the development of gastrointestinal symptoms.

intolerances to starch [42] and nonstarch polysaccharides, [28] dietary fibres, [43,44] fructans and other FODMAPs. [35,45] Further, it has been confirmed in placebo-controlled trials that fructans, rather than gluten, induce abdominal symptoms, even in patients with self-reported non-coeliac gluten sensitivity (NCGS). [46,47]

Studies originating from diverse geographical backgrounds, like Australia [47,48], France, [49] Spain, [50] Saudi Arabia, [51] New Zealand [52] and China [33] which have reviewed dietary components of the FODMAP group have shown that lactose and/or wheat products (including fructans) are quantitatively the most important components of the FODMAP group, together contributing more than 60% to the total amount of FODMAP. Therefore, taking the common example of lactose intolerance, the following two questions have to be raised:

- Why should IBS patients who tolerate lactose be submitted to a low-FODMAP diet in which a major component is restriction of lactose, and thereby an unnecessary impact on quality of life, costs of living and nutrient supply due to restriction of milk products?
- Why should IBS patients with lactose intolerance be submitted to a low-FODMAP diet, which includes dietary restriction of other poorly absorbable carbohydrates and thereby make the diet much more complicated to follow, rather than restricting lactose intake?

Permanent reduction of dietary FODMAPs is not recommended as a treatment for IBS, which is a chronic condition. [53] Furthermore, the long-term effects of low FODMAP diets are unclear. Possible negative effects include alterations of the microbiome [54] and deficiency of nutrients. [51]

Future studies will show whether demonstration of specific carbohydrate intolerances can help to focus on specific dietary components which should be avoided, thereby reducing the complexity of a low-FODMAP diet with its potentially restrictive effect on costs, quality of life, long-term safety, nutrition and faecal microbiota. [55] To minimise the negative impact of dietary restrictions, the use of a step-up approach in which specific foods (e.g. lactose, wheat products, fructose) are sequentially removed from the diet may be preferable to the early application of highly restrictive low-FODMAP diets, with subsequent step-down reintroduction of foods. [56]

Mistake 6 Putting too much trust in breath testing

Hydrogen-breath tests (HBT) are the most commonly used tests for evaluating lactose malabsorption. However, a positive result of an HBT is neither sufficient to explain the cause of symptoms which led to the referral of the patient, nor is it enough to warrant the start of treatment. [9] HBT should be performed as part of a comprehensive clinical assessment. [9] Diagnostic evaluation with the HBT and symptom assessment by a validated questionnaire, for example, with the aCPQ [10] or the pCPQ, [11] can be performed with different carbohydrates, which makes it possible to also test for incomplete absorption of carbohydrates other than lactose.

Interpreting the findings of combined breath and intolerance studies is challenging in patients who report abdominal symptoms after carbohydrate ingestion without evidence of malabsorption (i.e. no increase in breath hydrogen). A study [57] of fructose and fructose oligomers showed that short-chain and long-chain carbohydrates had different effects in the small intestine and colon, raising the possibility that symptoms after carbohydrate ingestion may occur without carbohydrates having to reach the colon (that is, without malabsorption).

In assessing the relation between dietary history and symptoms, it is helpful to consider the pretest probability of lactase deficiency (according to ethnic background). If the pretest probability of lactase deficiency is high, then the occurrence of typical symptoms 30–180 min after ingestion of milk products may be sufficient to establish the diagnosis of lactose intolerance, and formal breath testing and intolerance testing may not need to be performed. Conversely, if the pretest probability of lactase deficiency is low, then it is useful to identify intolerance as a cause of symptoms and to identify patients who benefit from treatment. [13]

It should also be noted that patients who report symptoms within a few minutes (<15 min) after ingestion of a test carbohydrate without an increase in breath hydrogen are likely to have functional dyspepsia triggered by gastric distention rather than a specific food intolerance. Patients who do have an early increase in symptoms with an increase in breath hydrogen (even only 10-20 ppm) may have small intestinal bacterial overgrowth. Both of these diagnoses require specific management.

Mistake 7 Misinterpreting lactase deficiency or lactose malabsorption as lactose intolerance

Various methods are available to assess the different parts of the process that lead from lactose maldigestion to the generation of symptoms (Figure 3). These methods include

genetic testing for lactase deficiency, determining lactase activity in biopsy samples taken from the small intestine, and the HBT combined with a validated symptom assessment, such as the aCPQ or pCPQ.[9]

A major limitation of the lactose HBT without symptom assessment is that an increase in breath hydrogen after carbohydrate ingestion establishes malabsorption but not intolerance. The same is true for the other blood and biopsy tests listed above. These tests, therefore, establish lactose malabsorption, lactase deficiency or the genetic predisposition to lactase deficiency[58], but they do not establish lactose intolerance, which is the main focus of clinical evaluation and the decision on starting treatment of symptomatic patients referred for testing. Furthermore, in clinical practice, the lactose intolerance testing should not be done with high doses of lactose (e.g. 40-50 g) that are not representative of normal intake of milk products.[59] For clinically meaningful assessment of lactose intolerance, it is more appropriate to use lower doses (e.g. 20 g lactose) that more closely reflect normal dietary behaviour.

Given that genetic tests, enzyme activity testing of biopsy samples and breath tests only demonstrate enzyme deficiency, maldigestion, or malabsorption, validated symptom assessment is required for assessment of clinically relevant intolerance. Suggestions for adhering to restrictive diets[13] or using enzyme supplements (e.g. containing lactase[23] or xylose isomerase[24]) should be limited to cases of documented intolerance, that is the documented relationship between ingestion of a carbohydrate and development of symptoms.[9]

Mistake 8 Relying on unvalidated symptom assessment

Documentation of intolerance to the ingested carbohydrate is the main indication for dietary or drug treatment and has to be performed using standardised tests to avoid bias.[9, 10, 11, 17, 19, 60] These tests (aCPQ, pCPQ) have been translated into several languages[12] and are suggested to be used by European Consensus papers and Guidelines.[9, 17, 19, 60] In German language countries, the aCPQ is available as an App[61] which is registered as a CE-certified medical device. Unvalidated symptom questionnaires should be avoided as it is not known if these methods really measure what is intended and if the data are obtained in a consistent, uniform manner that can be compared to other centres.

Mistake 9 Overlooking the dose dependency of symptom development

Patients sometimes assume that small amounts of lactose, for example, those present as additives in drugs, cause symptoms of intolerance. Studies show that any occurrence of symptoms to the dose of lactose in tablets (<1 g) is not physiological but instead related to a nocebo response.[62] Some pharmaceutical companies have recognised this as a potential market and advertise their drugs as being lactose-free. As such, it is clinically relevant to understand the dose of lactose required to induce notable symptoms (i.e. intolerance).

Increasing the dose of lactose during a lactose challenge increases the number of individuals who report abdominal symptoms.[63] In one double blind study, ingestion of less than 10 g lactose rarely induced abdominal symptoms in healthy controls, but 73% reported symptoms after ingestion of 40 g lactose, which approximates the dose most often applied in clinical studies (35–50 g). It should also be noted that the daily tolerable dose of lactose appears to be greater if lactose intake is distributed throughout the day and taken with meals.[64]

Of the symptoms related to carbohydrate malabsorption, the pathophysiology of carbohydrate-induced diarrhoea is probably the most rigorously studied. Diarrhoeal response to a disaccharide load depends on the amount of malabsorbed carbohydrate.[4] The colon has a large capacity to absorb fermentation products and thus to avoid faecal excretion of osmotic loads.[25] This colonic salvage becomes saturated as the quantity of carbohydrates reaching the colon increases. For instance, in healthy individuals, ingestion of 45 g of nonabsorbable disaccharide lactulose increased faecal water excretion only minimally. Only when greater than 80 g of lactulose was ingested did significant diarrhoea develop.[25] The equivalent amount of lactose (45 g, which approximates the lactose content in 1 L of milk) can be expected to be partially digested and absorbed in the small intestine even in lactose malabsorbers,[65] making it unlikely that this amount alone is responsible for severe diarrhoea.

Mistake 10 Omitting professional dietary counselling and follow-up

Patients for whom there is a clear association between symptoms and lactose ingestion should be educated about appropriate dietary restrictions[13] and the appropriate use of enzyme supplements.[23] However, as many carbohydrates other than lactose are incompletely absorbed by the normal small intestine[39,35] and because dietary fibre is also metabolised by colonic bacteria,[41] symptom persistence while on a lactose-reduced diet is not uncommon. Extending the diet to include a global reduction of other poorly fermentable carbohydrates may be helpful for such patients[66,67] Because of the additive effect of different poorly absorbable carbohydrates, patients with IBS and lactose intolerance may require advice on a FODMAP-reduced diet rather than only a lactose-reduced diet; however, as discussed in “mistake 5”, a step-up dietary restriction plan may be more appropriate and better tolerated than the use of highly restrictive diets. Depending on local care provisions, this may be best served by well-trained dietitians, who can provide dietary counselling and follow-up. Ideally, decisions regarding dietary treatment should be supported by carbohydrate intolerance documented by the results of a structured and validated assessment of symptoms after ingestion of the suspected carbohydrate.[9-11]

Amongst the practical information provided, patients should be informed that the doses of lactose (up to a cup of milk), fructose and other carbohydrates usually consumed in the diet do not normally cause symptoms when ingested with a meal.[68] Regular or daily consumption of lactose-containing food may be better tolerated than intermittent consumption.[63] Yoghurt may be tolerated by such patients and provide a good source of calcium. Alternatively, supplementation of dairy products with lactase of microbiological origin can be suggested.[23] The results of older controlled studies on the use of lactose-reduced products or lactase capsules are, however, inconsistent[63] because of the failure to distinguish between lactose malabsorption and lactose intolerance in the past. With the current awareness of the different implications of lactose malabsorption and lactose intolerance, as defined in the European Guideline,[9] treatment studies have to be repeated with inclusion of patients with lactose intolerance documented by validated symptom questionnaires.[9-11, 13]

If symptoms persist after ingestion of small amounts of dairy products, then the possibility of an allergy, rather than intolerance, should be considered. Intolerance to fat is also prevalent in patients with reflux disease, functional dyspepsia and other gastrointestinal disorders and can be another reason why symptoms persist despite appropriate dietary restriction.[69, 70]

The rapid increase in the prevalence of obesity and guidelines that suggest limiting the consumption of simple sugars has increased interest in alternative sweeteners.[71] Some of these are poorly absorbed carbohydrates, such as sorbitol or xylitol, which may result in similar intolerance symptoms as fructose or lactose.

At the same time, dietary counselling must consider the supply of other nutrients, which may be affected by long-term adherence to a specific diet. For example, lactase deficiency may be a risk factor for the development of osteoporosis and bone fractures, either owing to the avoidance of dairy products[59] or interference with calcium absorption.[72] However, proof of lactase deficiency rarely leads to a lactose-reduced diet if lactose ingestion is not associated with symptoms of intolerance. Patients for whom a lactose-reduced diet is recommended should be advised to add calcium from other dietary sources. Patients in whom a strict FODMAP-reduced diet is recommended should be made aware that there are limited data on the long-term safety of this

diet, with respect to nutritional adequacy, effects on faecal microbiota and diseases for which a high-fibre diet is suggested as a prevention or treatment (e.g. colonic adenocarcinoma). Professional dietary counselling can help patients to adapt their diet to the severity of their symptoms and assist them in meeting their long-term dietary needs and nutritional requirements.

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Your carbohydrate intolerance briefing

Mistakes in …

- Dietary management of IBS and how to avoid them [https://gutflix.eu/r/Td29BVfELFS0]
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UEG Week

- What is the approach to malabsorption? Presentation at UEG Week 2024 [https://gutflix.eu/r/AD6yzmDYkqC6]
- FODMAP diet and DOMINO fodmap app. Presentation at UEG Week 2023 [https://gutflix.eu/r/uGPgKHk9luKF]