MINI REVIEW

The development of human gut microbiota fermentation capacity during the first year of life

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Abstract
Fermentation capacity of microbial ecosystems intrinsically depends on substrate supply and the ability of a microbial community to deliver monomers for fermentation. In established microbial ecosystems, the microbial community is adapted to efficiently degrade and ferment available biopolymers which is often concurrently reflected in the richness of the microbial community and its functional potential. During the first year of life, the human gut microbial environment is a rather dynamic system that is characterized by a change in physiological conditions (e.g. from aerobic to anaerobic conditions, physical growth of the gastrointestinal tract, development of the intestinal immune system) but also by a change in nutrient supply from a compositionally limited liquid to a diverse solid diet, which demands major compositional and functional changes of the intestinal microbiota. How these transitions link to intestinal microbial fermentation capacity has gained comparatively little interest so far. This mini-review aims to collect evidence that already after birth, there is seeding of a hidden population of various fermentation organisms which remain present at low abundance until the cessation of breastfeeding removes nutritional restrictions of a liquid milk-based diet. The introduction of solid food containing plant and animal material is accompanied by an altering microbiota. The concurrent increases in the abundance of degraders and fermenters lead to higher intestinal fermentation capacity indicated by increased faecal levels of the final fermentation metabolites propionate and butyrate. Recent reports indicate that the development of fermentation capacity is an important step during gut microbiota development, as chronic disorders such as allergy and atopic dermatitis have been linked to lower degradation and fermentation capacity indicated by reduced levels of final fermentation metabolites at 1 year of age.
GASTROINTESTINAL FERMENTATION

Microbial fermentative systems occur in natural (e.g., soil, wetlands, gastrointestinal tract) and artificial (e.g., anaerobic digester, industrially produced fermented food) environments and are often coupled to methanogenesis. A complete degradation of carbohydrates through combined fermentation and methanogenesis theoretically mainly yields the gases CH₄ and CO₂ (Symons & Buswell, 1933; Tarvin & Buswell, 1934). Such optimal systems are dependent on factors such as carbohydrate supply and fermentation time and rarely occur in nature.

In the mammalian gastrointestinal tract, intestinal microbial degraders, which are predominantly bacteria accounting for 10¹⁰–10¹¹ cells g⁻¹ faeces and to a much lesser extent a fluctuating, low diversity population yeast and fungi (Nash et al., 2017; Raimondi et al., 2019), release mono-, di-, and oligosaccharides from undigested complex carbohydrates (e.g., plant derived and possibly technologically processed [resistant] starches, cellulose, hemicellulose, arabinoxylan, pectin) through the activity of carbohydrate active enzymes (CAZY) including glycosyl hydrolases (GH), glycosyl transferases (GT), polysaccharide lyases (PL) and carbohydrate esterases (CE) (Wardman et al., 2022).

Fermenting bacteria, yeast and fungi produce propionate or butyrate, or the fermentation intermediates lactate, acetate, succinate, formate and 1,2-propanediol which can be metabolized by fermentation specialists to propionate and butyrate (Figure 1) (Louis & Flint, 2017). Alternative metabolic pathways lead to the formation of propionate from lactate (acrylate pathway), succinate (succinate pathway) and 1,2-propanediol (propanediol pathway, Reichardt et al., 2014); some specialists are capable of the conversion of lactate and acetate to butyrate (Duncan et al., 2004). Methanogenic archaea use fermentation derived gases H₂ and the terminal electron acceptors CO₂, acetate or formate to produce CH₄ (Figure 1) (Gaci et al., 2014). In the human gastrointestinal tract, microbes not only compete for substrates but also rely on trophic interactions, that is, a nutrient-related relationship, to gain energy. Cross-feeding networks are thus an essential component of fermentative systems. Fermentation capacity is tightly linked not only to external factors, such as the supply of carbohydrates, but also to intrinsic factors including the metabolic cross-feeding potential of the microbial ecosystem.

The mammalian gastrointestinal tract is a dynamic system with intermittent and compositionally changing nutrient supplies (Pereira & Berry, 2017). Nutrient alterations are especially severe during the first year of life, when a physiologically developing gut system hosting a community of intestinal microbes is exposed to a dietary switch from a liquid breast milk or formula-based diet to solid food that gradually increases in compositional complexity. Yet, these transitions occur in most cases without obvious delay, and without major apparent adverse impact on the infant. The longitudinal development of the human gut microbiota during the first year(s) of life can thus act as a model system to study how changing of environmental conditions including alterations in dietary supply lead to shifts in microbial taxonomic community composition while concurrently altering the fermentation capacity.

FIGURE 1 Scheme indicating the succession of microbial degradation, fermentation and methanogenesis of dietary carbohydrates. Microbial communities in anaerobic ecosystems consisting of degraders, fermenters and methanogens interact (i) in releasing fermentable monosaccharides from complex carbohydrates, (ii) fermenting and cross-feeding on fermentation metabolites and (iii) delivering substrates for methanogens. Overall fermentation capacity, that is, the potential to form CO₂ and CH₄ from carbohydrates, is determined by microbial community composition and functional diversity and fermentation potential. In infants, fermentation capacity increases during weaning.
increasing intestinal fermentation capacity. While the focus of this review is set on the role of carbohydrate-based nutrients, short chain fatty acids (SCFAs) can also be produced during fermentation of amino acids, but that likely constitutes only a minor proportion of overall fermentation activity (Vital et al., 2017).

**BREASTFEEDING AND HUMAN MILK OLIGOSACCHARIDES DRIVE MICROBIAL COMMUNITY COMPOSITION BEFORE WEANING**

During recent years, several longitudinal cohort studies not only using 16S rRNA gene sequencing or metagenomics but also (targeted) cultivation have largely contributed to the investigation of the infant gut microbiota development (Bäckhed et al., 2015; Ferretti et al., 2018; Pham et al., 2016; Stewart et al., 2018). The infant gut microbiota evolves from an initially diverse and heterogeneous composition during the first days, to a simpler, and finally a complex adult like community at around 2 to 3 years of age (Ferretti et al., 2018; Tsukuda et al., 2021). This non-random and sequential transition is driven by nutrient availability and composition, the establishment of an anaerobic environment and microbial interactions (Bäckhed et al., 2015). First, colonizers are transmitted vertically during birth or breastfeeding through skin contact or milk, or are obtained from the hospital environment (Ferretti et al., 2018). Immediately after birth, facultative anaerobes, mainly *Enterococcus*, *Staphylococcus*, *Streptococcus* spp. and members of the family *Enterobacteriaceae* reach high densities (Bäckhed et al., 2015; Obermajer et al., 2017), which and mainly use mono- and disaccharides for growth. Initial colonization and concurrent oxygen depletion allow for the growth of strict anaerobic bacteria including *Bifidobacteriaceae* (Bäckhed et al., 2015; Obermajer et al., 2017).

Concurrently, breastfeeding provides a strong nutritive selective factor due to its content in human milk oligosaccharides (HMOs, 5–15 g L$^{-1}$), that is, complex glycans not digested by the host that can be degraded and metabolized by a group of highly adapted intestinal bacteria. HMOs are composed of the five monomeric building blocks, d-glucose (Glc), d-galactose (Gal), N-acetyl-d-glucosamine (Glc-NAc), L-fucose (Fuc) and sialic acid (Sia) (Figure 2) (Bode & Jantscher-Krenn, 2012). All HMOs contain lactose (Galβ1–4Glc) at the reducing end. Lactose can be elongated by the addition of the disaccharides lacto-N-biose (Galβ1–3GlcNAc) or N-acetyllactosamine (Galβ1–4GlcNAc). Di- and oligosaccharides can be modified with Fuc in α1–2-, α1–3-, or α1–4-linkages, or with Sia in α2–3- or α2–6-linkages (Bode & Jantscher-Krenn, 2012). HMO content and composition change during lactation phases (Plows et al., 2021). Two- and 3-fucosyllactose (2-FL and 3-FL) are usually among the most abundant HMOs (Plows et al., 2021). Milk of mothers that lack the fucosyltransferase 2 encoding gene (*fut2*) do not secrete...
α1–2 fucosylated HMO and might have overall lower HMO content (Azad et al., 2018). Around 95% of HMOs reach the colonic environment. Microbial degradation and fermentation of these structurally and compositionally diverse HMOs asks for a specialized repertoire of GH enzymes and for additional metabolic pathways to ferment the HMO monomers L-fucose (Bunesova et al., 2016; Tsukuda et al., 2021) and sialic acid (Egan et al., 2014). Specialization for HMO degradation and fermentation is widespread among infant-associated Bifidobacterium species such as Bifidobacterium bifidum, Bifidobacterium breve and Bifidobacterium longum subsp. infantis (Lawson et al., 2020; Tsukuda et al., 2021). In contrast to B. longum subsp. infantis, strains of B. bifidum possess extracellular GH and release HMO monomers to the intestinal microbial community for cross-feeding (Garrido et al., 2012). Intra-genus cross-feeding of Bifidobacterium species in the presence of sialyllactose and fucosyllactose has been reported (Egan et al., 2014; Lawson et al., 2020; Schwab et al., 2017, Tsukuda et al., 2021).

The dominance of Bifidobacterium spp. in the infant gut might explain the lower overall microbial richness of breastfed infant gut microbiota compared with that of formula-fed infants observed in some studies (Ferretti et al., 2018; Stewart et al., 2018). HMOs are likely supplied in surplus as spent HMOs and fucose were recovered in faeces with concentrations decreasing during the first months of breastfeeding (Borewicz et al., 2020; Sasaki et al., 2022). The faecal presence of HMOs and fucose suggests that the carbohydrate-dependent energy needs of the Bifidobacterium-dominated intestinal microbiota are fulfilled.

**HMO UTILIZATION BY BIFIDOBACTERIA DEFINES FAECAL FERMENTATION METABOLITE PROFILES**

Microbial composition and fermentation activity of major microbial groups during the breastfeeding phase is tightly linked to faecal fermentation profiles. Bifidobacterium spp. produce mainly acetate and lactate via the bifid shunt, and/or form formate and ethanol from pyruvate as alternative to lactate (de Vuyst et al., 2013). Accordingly, acetate is SCFA produced immediately after birth constituting up to 94% of the total major SCFAs (acetate, propionate and butyrate) in faeces until 3 months of age (Table 1).

The fermentation intermediates lactate and formate were regularly detected in faecal samples collected from infants during the first year of life (Pham et al., 2016; Tsukuda et al., 2021). Levels of lactate and formate were higher before than after weaning (Tsukuda et al., 2021). In addition, 1,2-propanediol, a metabolite of Bifidobacterium L-fucose metabolism (Bunesova et al., 2016), accumulated...
in faeces of breast milk-fed infants (He et al., 2019) suggesting that bifidobacteria were driving HMO degradation and fermentation to acetate, lactate, 1,2-propanediol and formate. The faecal accumulation of fermentation metabolites lactate, succinate and formate indicates the limited overall fermentation capacity of the infant gut microbiota during the breastfeeding period.

**CAN EARLY OCCURRING INTESTINAL PROPIONATE AND BUTYRATE PRODUCERS UTILIZE HMOS OR HMOS DEGRADATION PRODUCTS?**

Fermentation of breast milk oligosaccharides selects for a *Bifidobacterium*-dominated profile with mainly acetate, lactate, formate and 1,2-propanediol recovered as faecal fermentation metabolites. Utilization of acetate and lactate, or 1,2-propanediol would allow specialized cross-feeders to produce butyrate (Duncan et al., 2004) or propionate (Schwab et al., 2017), respectively, and indeed several longitudinal studies repeatedly observed faecal propionate and butyrate in meconium or as early as 1–2 weeks after birth (Appert et al., 2020, Gio-Batta et al., 2020, Nilsen et al., 2020; Norin et al., 2004; Pham et al., 2016; Tsukuda et al., 2021; Table 1). Before weaning, levels of propionate and butyrate were usually significantly lower than of acetate only representing a maximum of 10–18% of total SCFAs. Until 6 months of age, the concentration of propionate was generally higher than of butyrate (Table 1). While not all infants were identified as producers of propionate and butyrate based on faecal analysis (Table 1), the reliable and repeated recovery of low levels of propionate and butyrate from infant faeces gives strong indication that butyrate and propionate producers are a metabolically active part of the infant gut microbiota already days and weeks after birth.

Among the early detected succinate/propionate producers were members of the *Bacteroidota* (Bäckhed et al., 2015). This phylum includes taxa that are specialized in degrading plant complex polysaccharides or the endogenous mucin (Salyers et al., 1977), a heavily glycosylated protein that is secreted by the goblet cells of the intestinal epithelium (Pelaseyed et al., 2014). In addition, the *Bacteroides* species *B. fragilis* and *B. vulgatus* were capable of metabolizing HMOs, and were able to degrade a broad range of HMOs in comparison to *Bifidobacterium*, which preferred short chain HMOs (Marcobal et al., 2010). Succinate, a metabolite of *Bacteroidota* fermentation, was observed in faeces of breastfed and formula-fed infants and thus both in the presence and absence of HMOs (Bridgman et al., 2017; He et al., 2019) suggesting that the occurrence of *Bacteroidota* does not rely on HMO. Nonetheless, in the presence of HMOs, *Bifidobacterium* spp. might be more competitive in vivo (Marcobal et al., 2011). Human milk oligosaccharides degradation has also been reported for the mucin degrading propionate producer *Akkermansia muciniphila* and *B. bifidum*. Similar to *Bacteroides*, *A. muciniphila* and *B. bifidum* employ the same enzymes for mucus and for HMOs degradation (Kostopoulos et al., 2020; Marcobal et al., 2011; Turroni et al., 2010). Occurrence and abundance of *A. muciniphila* was nevertheless reported as low at 3 and 6 months (Sasaki et al., 2022).

Gut intestinal butyrate producers encompass a taxonomically and functionally diverse group mainly within the phylum *Bacillota* and the families *Lachnospiraceae*, *Ruminococcaceae*, *Clostridiaceae* and *Peptostreptococcaceae*. There are two major pathways that use either butyryl-CoA: acetate CoA-transferase (ButCoAT) or butyrate kinase (Buk) in the final step of the butyrate formation pathway (Louis & Flint, 2017). Endospore-forming members of the *Clostridiaceae* and *Peptostreptococcaceae*, which rely on Buk, are likely the butyrate forming pioneers within the infant gut microbiota (Appert et al., 2020; Rada et al., 2008; Tsukuda et al., 2021; Vital et al., 2017). Members of both families are anaerobes but can tolerate oxygen to various extent (Browne et al., 2016), and are often saccharolytic and proteolytic with access to a wide variety of substrates (Wiegel et al., 2006). Strains of *Clostridiaceae* (e.g. *Clostridium perfringens*) and *Peptostreptococcaceae* (e.g. *Clostridoides difficile*) showed an overall reduced efficiency to produce butyrate from glucose when compared with ButCoAT-dependent *Faecalibacterium prausnitzii* (*Ruminococcaceae*), *Eubacterium rectale* /Roseburia spp. and *Anaerobutyricum hallii* (*Lachnospiraceae*) during growth in yeast extract-casitone-fatty acid medium with glucose as the only carbohydrate source (Appert et al., 2020). Nevertheless, *Clostridiaceae* were more abundant in faeces of infants before weaning and might account for the rather low faecal butyrate levels that were observed (Appert et al., 2020). In addition, the occurrence of *Clostridiaceae* and *Peptostreptococcaceae* might be inversely related to breastfeeding and the presence of *Bifidobacterium* (Bäckhed et al., 2015; Rada et al., 2008). Strains of *C. perfringens* and *C. difficile* did not, or only poorly grew in the presence of 2-fucosyllactose (Salli et al., 2021) indicating that HMOs are an unlikely carbohydrate source. Some strains of *C. perfringens* grew in the presence of fucose indicating the potential for cross-feeding (Salli et al., 2021).

Human milk oligosaccharides degradation and utilization has been reported for butyrate producing *Roseburia hominis* or *Roseburia inulivorans* (Pichler et al., 2020), yet such species were only present at low abundance before weaning constituting around 3–4
log cells g⁻¹ faeces (Appert et al., 2020). Similarly, the butyrate-producing *F. prausnitzii* was only detectable at low abundance (Appert et al., 2020), and was not able to utilize HMOs or to cross-feed on bifidobacteria HMOs degradation metabolites in vitro (Cheng et al., 2020). The cross-feeding specialist *A. hallii*, which formed butyrate and propionate in co-cultures with bifidobacteria and 2-FL (Schwab et al., 2017) in vitro, was only detected at low abundance and not in all infants at 3 and 6 months (Appert et al., 2020) but could contribute to SCFA formation.

Taken together, these observations show that propionate and butyrate producers with the potential for HMO utilization and cross-feeding are present during the first months. The ability to use HMOs as alternative or secondary substrate might support the colonization or seeding of mucin utilizing propionate producers such as the genera *Bacteroides* and *Akkermansia* or of primary degraders and butyrate producers including *R. hominis*, *R. inulinovans* and *F. prausnitzii* (Lopez-Siles et al., 2012; Scott et al., 2014) during infant age. Such an early colonization might prime the intestinal microbial community for the development of degradation and fermentation capacity during and after weaning. The presence of such hidden functional populations seems not to be limited to degrading and fermenting microorganisms but might also include methanogens. Hudson and Roberts (1993) identified 20% of 1-week old infants as carriers of methanogens. With the introduction of solid food, the diversity and complexity of dietary carbohydrates increases (Figures 2 and 3). In addition, the omission of the selective pressure of a HMO containing breast milk diet slowly reduces the competitive advantage of HMOs utilizers. Studies reported either a decreasing or a stable abundance of *Bifidobacterium* during or after weaning, possibly due to continuation of supplementary breastfeeding. Concurrently, the abundance of degraders of complex carbohydrates increased in the months after the switch to solid feeding (Appert et al., 2020; Nilsen et al., 2020; Roger & McCartney, 2010). Plant-derived carbohydrates introduce novel dietary hexoses (e.g. D-mannose, D-fructose) and pentoses (e.g. L-arabinose, D-xylene), and sugar acids (D-glucuronic and D-galacturonic acid) that are connected through a variety of α- and β-type linkages (Figure 2). Additionally, plant carbohydrates contain a variety of non-carbohydrate components such as acetyl and methyl groups, hydroxyycinnamic acid or borate (Figure 2). The efficient degradation of these polymers asks for a more complex

**FIGURE 3** Scheme depicting the changes of microbial communities and faecal fermentation profiles that occur with the introduction of solid food. The intestinal microbiota changes from a *Bifidobacterium* dominated microbiota that produces mostly acetate and lactate to compositionally and functionally more diverse microbial communities with the introduction of solid food. This transformation leads to increasing faecal levels of propionate and butyrate. Figure was prepared using biorender. Ace, acetate; but, butyrate; lac, lactate; pro, propionate.
CAZY machinery including GH, PL and CE that are hosted by specialist microbial degraders, and is connected to a change in faecal fermentation profiles.

The transition process that leads to increased fermentation capacity after weaning takes several months. Infant age at the beginning of complementary feeding of any solids or non-water liquids might affect microbial fermentative response. In a previous study, an early start of complementary feeding (≤3 months) was associated with higher faecal butyrate and propionate levels at 12 months compared with introduction of complementary feeding at 3 months of age (Differding et al., 2020). There was no detectable difference of SCFA levels at 3 months shortly after the start of complementary feeding (Differding et al., 2020) indicating the time-consuming response of the infant gut microbiota.

The faecal SCFA with most dramatic higher levels after the introduction of solid food is butyrate together with major butyrate producers such as *E. rectale*/*Roseburia* spp., *F. prausnitzii*, and *A. hallii* becoming more abundant (Appert et al., 2020; Nilsen et al., 2020; Vitali et al., 2017). Strains of *E. rectale* and *Roseburia* are able to hydrolyze and ferment structurally and compositionally different carbohydrates while *F. prausnitzii* can use pectin (Lopez-Siles et al., 2012; Scott et al., 2014). The ability of *A. hallii* to utilize complex dietary carbohydrates is limited, but members of this species can cross-feed on fermentation intermediates (Schwab et al., 2017).

The change of diet during weaning is tightly linked to alterations of the faecal microbial community and of fermentation profiles. With the introduction of solid food, the α-diversity of faecal microbiota increases (Appert et al., 2020; Bäckhed et al., 2015). In infants from Western countries, a lower β-diversity of weaned infants indicated the development of a more homogenous microbiota (Bäckhed et al., 2015).

**THE IMPACT OF BIRTH MODE AND BREASTFEEDING ON FERMENTATION CAPACITY**

It is likely that any disturbances at the beginning of life that impact the seeding of microbes, or dietary alternatives to breastfeeding affect the development of fermentation capacity. Seeding of the infant gut microbiota is affected by birth mode, which also impacts microbiota development during the first year of life (Bäckhed et al., 2015; Reyman et al., 2019). However, breastfeeding is a confounding factor, as frequently a lower proportion of caesarean born infants is breastfed, and for a reduced duration (Reyman et al., 2019). A study that compared faecal SCFA profiles in exclusively (30%), partly (42%) and non-breastfed (28%) infants (*n* = 158) at 4 months found significant differences depending on breastfeeding status but no association with birth mode (Bridgman et al., 2017). Infants that were still exclusively breastfed at 4 months had lower levels of propionate and butyrate, and higher levels of lactate compared with non-breastfed infants. In contrast, faecal levels of butyrate were higher in caesarean-born compared with vaginally delivered infants in a study with 70 participants and infants that were breastfed for a median of around 1 month only (Mueller et al., 2021).

Then again, differences in faecal microbiota composition and fermentation metabolites between non, partly and exclusively breastfed infants indicate that, as soon as the nutritional pressure of breastfeeding and HMOs consumption is reduced or stopped, fermentation capacity increased indicated by higher propionate and butyrate, and lower lactate levels, and that this might already happen before weaning. Concurrently, in exclusively formula fed infants, faecal microbial α-diversity was higher compared with breastfed infants (Bäckhed et al., 2015; Bridgman et al., 2017; Ho et al., 2018), preceding observations made during and after weaning.

**ALTERATIONS OR DISTURBANCES IN FERMENTATION CAPACITY DEVELOPMENT MIGHT BE LINKED TO HEALTH OUTCOMES LATER IN LIFE**

While the comparatively limited fermentation capacity of the infant microbiota during the breastfeeding period might not be detrimental to health outcomes, the potential for butyrate formation after weaning seems beneficial for health in later life and has been negatively linked to the occurrence of chronic diseases such as allergy and atopic dermatitis (AD) (Cait et al., 2019; Roduit et al., 2019). Sandin et al. (2009) reported that lower levels of butyrate at 1 year were associated with food allergy at 4 years. While the cause has not been completely clarified, the risk of developing AD was lower in infants with higher faecal butyrate at 1 year of age (Roduit et al., 2019).

Recent studies observed that lower butyrate formation capacity of infants with AD might be linked to primary degradation potential, as there was lower abundance in CAZY related to HMO degradation in infants with AD at 3 months and of CAZY associated with plant-cell wall, animal and fungal carbohydrates at 1 year (Cait et al., 2019). These differences in CAZY profiles might be indicative of a disrupted carbohydrate degradation and cross-feeding scheme which might impact the development of fermentation capacity. In another study, the abundance of the starch-degrader *Ruminococcus bromii* and of the mucin-utilizing *A. muciniphila* was lower at 1 year (Sasaki et al., 2022).
in infants AD compared with infants without AD, which again links health status to alterations in carbohydrate degradation capacity.

**CONCLUSION**

This mini-review aims to provide a perspective on how the gastrointestinal fermentation system develops from a comparatively limited fermentation state that is characterized by the accumulation of fermentation intermediates to a more complex fermentation system. The surplus supplies of HMOs during breastfeeding not only provide competitive advantage for HMO degrading specialists but also limit fermentation pathways. Seeding of a low abundant population of more diverse fermentation specialists even before the introduction of solid food prepares the microbial community for the diet change that occurs during weaning. The origin of these strict anaerobes has not been completely clarified. Recent metagenome studies reliably identified strains of *Bifidobacterium*, *Bacteroides* and *Enterobacteriaceae* including *E. coli* (Asnicar et al., 2017; Ferretti et al., 2018; Li et al., 2021) as being vertically transmitted during the first week after birth but did rarely report the transmission of any butyrate producers. This gap might be due to low faecal abundance of taxa such as *Faecalibacterium*, *Roseburia* and *A. hallii* in infant faeces, which interferes with their detectability in shotgun metagenome sequencing based analysis. Novel targeted cultivation approaches might enable to isolate and characterize such low abundant community members (Watterson et al., 2021). A second possibility is the transfer of endospore formers. Browne et al. (2016) estimated that up to 50% of taxa present in adult humans are able to form endospores. As the occurrence of chronic diseases has been linked to alterations in fermentation capacity compared with healthy control infants, future efforts can be directed towards a targeted seeding of degraders and fermenters, for example as probiotics, to ensure the development of a functional intestinal microbial fermentation system.

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clarissa schwab: Conceptualization (lead); writing – original draft (lead); writing – review and editing (lead).

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**CONFLICT OF INTEREST**

There is no conflict of interest to declare.

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