

BIOME GUIDE PROTOCOL

Pregnancy & Infant Microbiome Protocol

Evidence-based strategies for building your
baby's microbiome from conception through
the first year of life

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16 peer-reviewed citations



The Pregnancy Microbiome Revolution

Pregnancy is one of the most dramatic periods of microbial change in a woman's life. From the first trimester to the third, the maternal gut microbiome undergoes a striking transformation -- expanding in Proteobacteria and Actinobacteria while losing overall species richness. In a landmark 2012 Cell study, Koren et al. demonstrated that when third-trimester microbiota were transferred to germ-free mice, the animals developed greater adiposity and insulin resistance compared to those receiving first-trimester microbiota. These changes, which resemble metabolic syndrome in non-pregnant individuals, appear to be adaptive -- driving energy storage to support fetal growth (Koren et al. 2012).

What makes this finding revolutionary is the implication that the microbiome is not just a passive bystander during pregnancy. It actively shifts to redirect maternal metabolism toward the needs of the growing fetus. This means that the state of your gut before and during pregnancy has a direct influence on both your metabolic health and the microbial inheritance you pass to your child.

Why This Matters

Your baby's microbiome begins forming at birth, and the microbial community they receive is shaped by choices you make during pregnancy -- from diet and probiotic use to delivery mode and breastfeeding. This protocol walks you through the evidence for each factor and provides actionable steps at every stage.

How Your Baby Gets Their Microbiome

The neonatal gut begins as a largely sterile environment and undergoes rapid colonization in the first hours and days of life. The sources, timing, and diversity of this initial colonization have far-reaching consequences for immune development, metabolism, and long-term disease risk. Multiple factors converge to shape this founding microbial community (Vandenplas et al. 2020; Kim et al. 2019).

Key Seeding Factors

Factor	Impact on Infant Microbiome	Evidence Quality
Vaginal delivery	Exposes infant to maternal vaginal and fecal microbiota -- Lactobacillus, Prevotella, Bacteroides spp. dominate early gut	Strong (multiple cohorts)
Cesarean section	Infant colonized by skin and hospital bacteria -- Staphylococcus, Streptococcus, Clostridium spp.; reduced Bacteroides for months	Strong (Sandall et al. 2018)
Breastfeeding	Human milk delivers 200+ HMOs that selectively feed Bifidobacterium; also provides IgA, lactoferrin, and live bacteria	Strong (Dinleyici et al. 2023)

Factor	Impact on Infant Microbiome	Evidence Quality
Maternal diet	High-fiber, diverse diet increases short-chain fatty acid producers in maternal gut, influencing microbes passed to infant	Moderate
Antibiotics	Intrapartum antibiotics reduce maternal-to-infant strain transmission; alter infant Bifidobacterium and Bacteroides colonization	Strong (Dubois et al. 2024)
Skin-to-skin contact	Immediate contact facilitates microbial transfer; paternal skin contact also contributes strains -- father's contribution becomes comparable to mother's by 1 year	Moderate (Dubois et al. 2024)

Birth Mode and the Microbiome

Cesarean section rates have risen to approximately 21% globally, and in some countries exceed 50%. While C-sections are life-saving when medically indicated, the Lancet's 2018 landmark review by Sandall et al. documented significant short- and long-term health effects on both women and children. Among these effects, altered immune development and reduced gut microbiome diversity in C-section-born infants stand out as consistent findings across dozens of cohort studies (Sandall et al. 2018).

C-section-born infants show delayed colonization by Bacteroides and Bifidobacterium -- two genera critical for immune education and short-chain fatty acid production in the infant gut. This difference can persist for 6-12 months and has been associated with increased risk of asthma, allergies, type 1 diabetes, and obesity in later childhood (Kim et al. 2019; Sandall et al. 2018).

Vaginal Seeding: The Evidence So Far

Vaginal seeding -- swabbing a newborn with maternal vaginal fluids after C-section -- has attracted significant interest as a way to restore the microbial transfer lost during surgical delivery. Mueller et al. (2023) published the first randomized controlled trial of vaginal seeding in mBio. The results showed that the intervention significantly increased mother-to-neonate microbiota transmission and produced compositional changes in the infant's skin and stool microbiota. While promising, the study was small and the long-term clinical significance remains unclear. Screening for Group B Strep, HSV, and other pathogens is essential before attempting this approach (Mueller et al. 2023).

Dubois et al. (2024) provided additional insight by showing that maternal fecal microbiota transplantation (FMT) to C-section-born infants increased mother-infant strain sharing, raised average bacterial growth rates, and reduced pathogen colonization. They also demonstrated that paternal microbiota contributes strains independently of delivery mode, with the father's cumulative contribution becoming comparable to the mother's after one year (Dubois et al. 2024).

Important Safety Note

Vaginal seeding and fecal microbiota approaches are still experimental. They should only be performed under medical supervision after thorough screening for infectious pathogens. Professional medical societies have not yet endorsed routine vaginal seeding.

Human Milk Oligosaccharides (HMOs)

Human breast milk contains over 200 structurally distinct human milk oligosaccharides (HMOs) -- complex sugars that the infant cannot digest but that serve as the primary food source for beneficial gut bacteria. HMOs represent the third most abundant solid component of breast milk after lactose and lipids, yet they provide zero direct nutrition to the infant. Their entire purpose is to shape the developing microbiome (Dinleyici et al. 2023).

How HMOs Work

Function	Mechanism	Key Outcome
Prebiotic	Selectively feed <i>Bifidobacterium longum</i> subsp. <i>infantis</i> and other beneficial <i>Bifidobacterium</i> spp.	<i>Bifidobacterium</i> -dominant microbiome in breastfed infants
Anti-adhesion	Act as decoy receptors that mimic cell surface glycans, blocking pathogen attachment	Reduced incidence of diarrheal illness, necrotizing enterocolitis
Immune modulation	Signal directly to intestinal epithelial cells and immune cells via pattern recognition receptors	Balanced Th1/Th2 immune development, reduced inflammation
Barrier protection	Promote goblet cell mucin production and tight junction protein expression	Strengthened intestinal barrier, reduced intestinal permeability

The *B. infantis* Connection

Among all gut bacteria, *B. longum* subsp. *infantis* is uniquely equipped to consume the full spectrum of HMOs. It possesses a dedicated gene cluster for importing and metabolizing every major HMO structure. When *B. infantis* is present and HMOs are abundant (i.e., the infant is breastfed), it can dominate the infant gut at levels exceeding 80% of total bacteria. This dominance crowds out potential pathogens and produces lactate and acetate that lower gut pH to create an inhospitable environment for harmful organisms.

Prenatal and Perinatal Probiotics

Probiotic supplementation during pregnancy and early infancy is one of the most studied microbiome interventions. The evidence, however, is nuanced -- strain selection, timing (prenatal only vs. prenatal + postnatal), and the specific condition targeted all matter significantly. Below we review the strongest clinical trial evidence for key probiotic strains in the pregnancy and infant context.

Lactobacillus rhamnosus HN001: The Eczema-Prevention Standout

The New Zealand Probiotic Study by Wickens et al. demonstrated that *L. rhamnosus* HN001 given to mothers from 35 weeks' gestation through 6 months postpartum (if breastfeeding) AND directly to infants from birth to age 2 reduced eczema by 43% (HR 0.57) with protection persisting to at least age 4

(Wickens et al. 2012). Critically, a follow-up study showed that maternal-only supplementation (without direct infant dosing) was NOT effective for preventing infant eczema -- the infant must also receive the probiotic directly (Wickens et al. 2018).

LGG (*L. rhamnosus* GG): Prenatal-Only Insufficient

Boyle et al. (2011) conducted an important systematic review showing that prenatal-only LGG supplementation was not sufficient for eczema prevention. The evidence suggests that a postnatal component -- either through breastfeeding mothers who are supplemented or through direct infant supplementation -- is necessary for clinical benefit. This finding reinforced the need to select both the right strain and the right timing window (Boyle et al. 2011).

Evidence Summary: Prenatal/Perinatal Probiotic Strains

Strain	Dosing Window	Key Finding	Citation
<i>L. rhamnosus</i> HN001	Prenatal (35 wk) + infant (0-2 yr)	43% eczema reduction (HR 0.57), lasting to age 4+	Wickens et al. 2012
<i>L. rhamnosus</i> HN001	Maternal-only (no infant dose)	NOT effective for infant eczema prevention	Wickens et al. 2018
<i>L. rhamnosus</i> GG	Prenatal-only	Insufficient for eczema prevention alone; needs postnatal component	Boyle et al. 2011
<i>B. infantis</i> EVC001	Infant (Day 7-28, breastfed)	Safe, well-tolerated; 1000x increase in fecal <i>Bifidobacterium</i> ; fewer, better-formed stools	Smilowitz et al. 2017

B. infantis EVC001: Restoring the Ancestral Infant Gut

Historically, Bifidobacterium dominated the gut of breastfed infants in every culture studied. Yet in developed nations today, bifidobacteria are often scarce or absent from infant stool -- a phenomenon attributed to widespread antibiotic use, formula feeding, and reduced microbial transmission in hospital birth settings. B. infantis EVC001 was developed to address this gap.

The Phase I IMPRINT trial (Smilowitz et al. 2017) enrolled 80 mother-infant dyads and randomized breastfed infants to receive either B. infantis EVC001 ($1.8\text{--}2.8 \times 10^{10}$ CFU daily in breast milk from Day 7 to Day 28) or lactation support alone. The supplemented group showed a mean Log10 increase of 6.6 in fecal Bifidobacterium (vs. 3.5 in controls, $p = 0.0002$). Supplemented infants also had fewer daily stools with improved consistency -- watery stools decreased and soft stools increased by 36% over baseline. No adverse events differed between groups (Smilowitz et al. 2017).

Clinical Takeaway

B. infantis EVC001 supplementation in breastfed infants safely restores a Bifidobacterium-dominant gut profile. The combination of breast milk (providing HMOs as fuel) and B. infantis (uniquely equipped to metabolize those HMOs) recapitulates the ancestral infant gut ecosystem. This is especially relevant for infants born by C-section or exposed to antibiotics.

Antibiotics and the Infant Microbiome

Intrapartum antibiotic prophylaxis (IAP) -- administered to roughly 30% of women in labor, primarily for Group B Streptococcus prevention -- has well-documented effects on the infant microbiome. IAP partially disrupts maternal-to-infant microbial seeding, reducing the transfer of beneficial Bacteroides and Bifidobacterium strains while selecting for antibiotic-resistant organisms (Kim et al. 2019; Dubois et al. 2024).

Dubois et al. (2024) quantified this disruption in the HELMi cohort, showing that cesarean birth and antibiotic exposure both independently reduced maternal strain transmission. However, their findings also offered hope: paternal strains partly compensated for reduced maternal seeding, and induced seeding approaches (maternal FMT) could restore transmission in C-section-born infants. Skin-to-skin contact with both parents was associated with increased strain sharing.

Practical Implication

If you received antibiotics during labor, your baby's microbiome may need active support. Strategies include extended breastfeeding (providing HMOs), skin-to-skin contact with both parents, probiotic supplementation (particularly *B. infantis* for breastfed infants and *L. rhamnosus* HN001 for eczema risk reduction), and avoiding unnecessary postnatal antibiotics for the infant.

Gestational Diabetes and the Microbiome

Gestational diabetes mellitus (GDM) affects 5-20% of pregnancies worldwide and is associated with adverse outcomes including macrosomia, preeclampsia, and increased long-term metabolic risk for both mother and child. Emerging evidence suggests that the gut microbiome plays a role in GDM development and may be a target for dietary and probiotic interventions.

Mokkala et al. (2021) conducted a randomized, double-blind, placebo-controlled trial analyzing the gut microbiota of 270 overweight/obese pregnant women using metagenomic sequencing. They found that while specific bacterial species did not predict GDM onset, GDM status appeared to reduce the gut microbiota's ability to respond to dietary interventions (fish oil + probiotics). Women without GDM showed significant changes in bacterial species abundance over pregnancy, particularly in the fish oil + probiotics group, while women with GDM did not -- suggesting that GDM impairs the metabolic flexibility of the gut microbiome (Mokkala et al. 2021).

The broader picture, reviewed by Godfrey et al. (2017) in *The Lancet Diabetes & Endocrinology*, highlights how maternal obesity and metabolic dysfunction reshape the microbial environment that the infant inherits. Maternal obesity is associated with altered gut microbiota composition, increased inflammatory markers in breast milk, and epigenetic changes in offspring -- creating an intergenerational cycle of metabolic risk (Godfrey et al. 2017).

GDM and the Microbiome

Managing blood sugar during pregnancy may also help preserve the metabolic flexibility of your gut microbiome. A diet rich in fiber, fermented foods, and omega-3 fatty acids -- combined with appropriate probiotic supplementation -- supports both glycemic control and microbial diversity.

The Pregnancy Microbiome Protocol

This protocol is organized into three phases: preconception/early pregnancy, late pregnancy/birth, and the first year of life. Each phase targets the specific microbial processes most active during that window.

Phase 1: Preconception and First Trimester

Goal: Build a Diverse Maternal Microbiome

The microbes you carry into pregnancy form the foundation of what you will pass to your baby. A diverse, fiber-fueled gut microbiome before conception gives your baby the best starting point.

Action	Details	Rationale
High-fiber, diverse diet	30+ different plant foods per week; emphasis on prebiotic-rich foods (garlic, onions, leeks, asparagus, oats, bananas)	Feeds SCFA-producing bacteria; increases microbial diversity that will seed baby's gut
Fermented foods daily	Yogurt, kefir, sauerkraut, kimchi, miso -- aim for 2-3 servings/day	Introduces live beneficial bacteria; associated with reduced inflammatory markers
L. rhamnosus HN001	Begin at 35 weeks gestation (or earlier); continue through breastfeeding	43% eczema reduction in offspring when combined with infant dosing (Wickens et al. 2012)
Prenatal omega-3 (DHA/EPA)	At least 200 mg DHA daily from fish oil or algal source	Supports anti-inflammatory milieu; may enhance microbiome response to probiotics (Mokkala et al. 2021)
Avoid unnecessary antibiotics	Work with your provider to use antibiotics only when clinically indicated	Preserves maternal microbial diversity for transfer to infant at birth

Phase 2: Late Pregnancy and Birth

Goal: Maximize Microbial Transfer at Birth

The birth process is the single largest microbial seeding event in your baby's life. Every decision around delivery, antibiotics, and immediate postnatal care shapes the founding community.

Action	Details	Rationale
Vaginal birth when safe	Discuss birth preferences with your provider; vaginal delivery provides optimal microbial exposure	Vaginal birth seeds infant with Lactobacillus, Prevotella, Bacteroides (Sandall et al. 2018)
If C-section is necessary	Request immediate skin-to-skin; discuss vaginal seeding with provider (if screened); consider infant probiotics	Helps compensate for reduced microbial transfer (Mueller et al. 2023; Dubois et al. 2024)
Immediate skin-to-skin	Both parents -- not just mother. Minimum 1 hour uninterrupted after birth	Father also contributes strains; paternal contribution grows over first year (Dubois et al. 2024)
Initiate breastfeeding within 1 hour	Colostrum is rich in IgA, HMOs, and live bacteria; early initiation promotes Bifidobacterium colonization	HMOs selectively feed B. infantis and beneficial bifidobacteria (Dinleyici et al. 2023)
Minimize antibiotic exposure	If IAP is needed, request narrow-spectrum when possible; support infant gut recovery afterward	IAP reduces beneficial strain transmission (Kim et al. 2019; Dubois et al. 2024)

Phase 3: The First Year of Life

Goal: Nurture and Diversify the Infant Microbiome

The first 12 months represent a critical window for microbial ecosystem assembly. Breastfeeding, probiotic supplementation, and the introduction of solid foods are the three primary levers.

Action	Details	Rationale
Exclusive breastfeeding (0-6 months)	WHO recommends exclusive breastfeeding for 6 months; continue alongside solids to 12+ months	HMOs are the primary prebiotic driving Bifidobacterium dominance (Dinleyici et al. 2023)
B. infantis EVC001 supplementation	1.8×10^{10} CFU daily in breast milk, starting Day 7 for at least 21 days	Restores ancestral Bifidobacterium dominance; safe and well-tolerated (Smilowitz et al. 2017)
L. rhamnosus HN001 for infant	Begin at birth, continue to age 2; dose per manufacturer	Requires direct infant dosing for eczema protection (Wickens et al. 2012; Wickens et al. 2018)
Diverse solids introduction (6+ months)	Introduce a wide variety of vegetables, fruits, legumes, and whole grains; include small amounts of fermented foods	Drives microbial diversification; fiber from solids begins replacing HMOs as primary prebiotic
Avoid unnecessary infant antibiotics	Use only when clinically indicated; if antibiotics are given, follow with probiotics after course completion	Early antibiotic exposure associated with reduced diversity and increased allergy/obesity risk (Kim et al. 2019)
Pet and sibling exposure	Household pets (especially dogs) and older siblings increase microbial diversity in infants	Associated with reduced allergy and asthma risk; broader microbial exposure in first year (Kim et al. 2019)

Supplement Stack: Pregnancy Through Infancy

Below is a consolidated supplement reference for both mother and infant, organized by phase. Always discuss supplementation with your healthcare provider before starting.

Supplement	Who	Dose	Timing	Key Evidence
L. rhamnosus HN001	Mother	6×10^9 CFU/day	From 35 weeks through breastfeeding	Wickens et al. 2012
L. rhamnosus HN001	Infant	6×10^9 CFU/day	Birth to age 2	Wickens et al. 2012, 2018
B. infantis EVC001	Infant (breastfed)	1.8×10^{10} CFU/day in breast milk	Day 7 to Day 28+	Smilowitz et al. 2017

Supplement	Who	Dose	Timing	Key Evidence
Prenatal DHA/EPA	Mother	200+ mg DHA/day	Preconception through breastfeeding	Mokkala et al. 2021
Prenatal vitamin with folate	Mother	Per provider	Preconception through pregnancy	Standard of care
Vitamin D	Mother + Infant	Mother: 2000-4000 IU/day; Infant: 400 IU/day	Throughout	AAP recommendation for infant

Lifestyle Factors That Shape the Infant Microbiome

Maternal Obesity

Maternal pre-pregnancy BMI is one of the strongest predictors of infant microbiome composition. Godfrey et al. (2017) reviewed the evidence showing that maternal obesity alters the gut microbiota passed to offspring, increases inflammatory markers in breast milk, and drives epigenetic modifications that predispose the child to metabolic disease. Maintaining a healthy weight before pregnancy, or working to improve metabolic markers during pregnancy through diet and exercise, can help break this intergenerational cycle (Godfrey et al. 2017).

Formula Feeding Considerations

When breastfeeding is not possible, infant formula supplemented with HMOs (such as 2'-fucosyllactose and lacto-N-neotetraose) is the next best option. These synthetic HMOs do not replicate the full diversity of human milk's 200+ oligosaccharides, but they do provide some prebiotic support for Bifidobacterium colonization. Combining HMO-supplemented formula with B. infantis EVC001 may offer a synbiotic approach for formula-fed infants, though more research is needed (Dinleyici et al. 2023; Vandenplas et al. 2020).

Environmental Exposures

The hygiene hypothesis has evolved into the "old friends" hypothesis -- the idea that reduced exposure to diverse environmental microbes in modern life contributes to immune dysregulation. Practical implications for infant microbiome development include allowing outdoor play and soil contact, having household pets, avoiding excessive use of antibacterial cleaning products, and encouraging interaction with other children. Kim et al. (2019) documented that pet exposure and having older siblings were both associated with increased microbial diversity and reduced atopy risk in infants (Kim et al. 2019).

Quick Reference Timeline

Window	Priority Actions	Key Supplements
Preconception	Diversify diet (30+ plants/week); add fermented foods; optimize weight; begin prenatal vitamin	Prenatal vitamin with folate, DHA/EPA, Vitamin D
First trimester	Continue diverse diet; minimize medication use; manage nausea with ginger/small meals	Prenatal vitamin, DHA/EPA, Vitamin D
35 weeks gestation	Begin L. rhamnosus HN001; finalize birth plan; discuss skin-to-skin and breastfeeding with provider	L. rhamnosus HN001 + existing stack

Window	Priority Actions	Key Supplements
Birth	Vaginal delivery when safe; immediate skin-to-skin (both parents); breastfeed within 1 hour	Continue maternal HN001
Day 7-28 postnatal	Begin <i>B. infantis</i> EVC001 in breast milk; continue exclusive breastfeeding; begin infant HN001	<i>B. infantis</i> EVC001, <i>L. rhamnosus</i> HN001 (infant dose)
1-6 months	Exclusive breastfeeding; skin-to-skin contact; minimize antibiotics; pet/sibling exposure	Continue <i>B. infantis</i> + HN001 for infant; maternal HN001 + DHA
6-12 months	Introduce diverse solids; continue breastfeeding alongside; include fermented foods	Continue infant HN001; transition from <i>B. infantis</i> as solids increase

Sources

1. Koren O, Goodrich JK, Cullender TC, et al. Host remodeling of the gut microbiome and metabolic changes during pregnancy. *Cell*. 2012;150(3):470-480. doi:10.1016/j.cell.2012.07.008
2. Sandall J, Tribe RM, Avery L, et al. Short-term and long-term effects of caesarean section on the health of women and children. *Lancet*. 2018;392(10155):1349-1357. doi:10.1016/S0140-6736(18)31930-5
3. Vandenplas Y, Carnielli VP, Ksiazek J, et al. Factors affecting early-life intestinal microbiota development. *Nutrition*. 2020;78:110812. doi:10.1016/j.nut.2020.110812
4. Kim H, Sitarik AR, Woodcroft K, Johnson CC, Zoratti E. Birth mode, breastfeeding, pet exposure, and antibiotic use: associations with the gut microbiome and sensitization in children. *Curr Allergy Asthma Rep*. 2019;19(4):22. doi:10.1007/s11882-019-0851-9
5. Dinleyici M, Barbier J, Dinleyici EC, Vandenplas Y. Functional effects of human milk oligosaccharides (HMOs). *Gut Microbes*. 2023;15(1):2186115. doi:10.1080/19490976.2023.2186115
6. Wickens K, Black P, Stanley TV, et al. A differential effect of 2 probiotics in the prevention of eczema and atopy: a double-blind, randomized, placebo-controlled trial. *J Allergy Clin Immunol*. 2008;122(4):788-794; Wickens K, Stanley TV, Mitchell EA, et al. Early supplementation with *Lactobacillus rhamnosus* HN001 reduces eczema prevalence to 6 years: does it also reduce atopic sensitization? *Clin Exp Allergy*. 2013;43(9):1048-1057. doi:10.1111/j.1365-2222.2012.03975.x
7. Wickens K, Barthow C, Mitchell EA, et al. Effects of *Lactobacillus rhamnosus* HN001 in early life on the cumulative prevalence of allergic disease to 11 years. *Pediatr Allergy Immunol*. 2018;29(8):808-814. doi:10.1111/pai.12874
8. Boyle RJ, Ismail IH, Kivivuori S, et al. *Lactobacillus* GG treatment during pregnancy for the prevention of eczema: a randomized controlled trial. *Allergy*. 2011;66(4):509-516. doi:10.1111/j.1398-9995.2010.02507.x
9. Smilowitz JT, Moya J, Breck MA, et al. Safety and tolerability of *Bifidobacterium longum* subspecies *infantis* EVC001 supplementation in healthy term breastfed infants: a phase I clinical trial. *BMC Pediatr*. 2017;17(1):133. doi:10.1186/s12887-017-0886-9
10. Mueller NT, Differding MK, Engstrom A, et al. Vaginal seeding after caesarean section: a randomised clinical trial. *mBio*. 2023;14(2):e00491-23. doi:10.1128/mbio.00491-23
11. Dubois L, Valles-Colomer M, Ponsero A, et al. Paternal and induced gut microbiota seeding complement mother-to-infant transmission. *Cell Host Microbe*. 2024;32(6):1011-1024. doi:10.1016/j.chom.2024.05.004
12. Morkkela K, Paulin N, Houttu N, et al. Metagenomics analysis of gut microbiota in response to diet intervention and gestational diabetes in overweight and obese women: a randomised, double-blind, placebo-controlled clinical trial. *Gut*. 2021;70(2):309-318. doi:10.1136/gutjnl-2020-321643
13. Godfrey KM, Reynolds RM, Prescott SL, et al. Influence of maternal obesity on the long-term health of offspring. *Lancet Diabetes Endocrinol*. 2017;5(1):53-64. doi:10.1016/S2213-8587(16)30107-3
14. Slykerman RF, Kang J, Van Zyl N, et al. Effect of early probiotic supplementation on childhood cognition, behaviour and mood: a randomised, placebo-controlled trial. *Acta Paediatr*. 2018;107(12):2172-2178. doi:10.1111/apa.14590
15. Volery M, Ngyuen TTP, Engel D, et al. Study protocol for ABERRANT: antibiotic exposure effects on gut microbiota in mothers and neonates. *BMJ Open*. 2020;10(9):e036275. doi:10.1136/bmjopen-2019-036275
16. Li HY, Zhou DD, Gan RY, et al. Effects and mechanisms of probiotics, prebiotics, synbiotics, and postbiotics on metabolic diseases. *Nutrients*. 2021;13(9):3211. doi:10.3390/nu13093211

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