



# Exploring How Human Milk Oligosaccharides Affect the Gut Microbiome of Infants

Philippe Michiels\*

Department of Virology, University of Porto, Praca de Gomes Teixeira, Porto, Portugal

## ABOUT THE STUDY

Human Milk Oligosaccharides (HMOs) are complex carbohydrates abundantly present in human breast milk. These bioactive compounds have gained significant attention due to their potential role in shaping the infant gut microbiome, which in turn plays a crucial role in immune system development, nutrient absorption, and overall health. This commentary explores the intricate relationship between HMOs and the infant gut microbiome, highlighting the emerging research in this field and its implications for infant health and development [1-3].

HMOs are a diverse group of complex sugars unique to human breast milk. They are structurally complex, comprising various monosaccharide units and exhibiting vast structural diversity. HMOs are not digestible by the infant, but rather serve as prebiotics, selectively promoting the growth of beneficial bacteria in the gut [4]. Research has shown that the composition and abundance of HMOs in breast milk vary among individuals and across populations, emphasizing the personalized nature of this component of nutrition.

The gut microbiome, consisting of trillions of microorganisms residing in the gastrointestinal tract, plays a crucial role in infant health. During infancy, the gut microbiome undergoes dynamic development, influenced by various factors including diet, mode of delivery, and exposure to environmental factors [5]. The establishment of a diverse and balanced gut microbiome during this critical period is associated with optimal health outcomes, including reduced risk of allergies, asthma, obesity, and other chronic diseases.

Recent studies have highlighted the interplay between HMOs and the infant gut microbiome, revealing their mutual influence on each other [6]. HMOs act as a food source for specific beneficial bacteria, such as *Bifidobacterium* and *Bacteroides*, promoting their growth and colonization in the infant gut. These bacteria, in turn, contribute to the fermentation of HMOs, producing Short-Chain Fatty Acids (SCFAs) that provide energy to the intestinal cells and support their development.

Furthermore, HMOs possess antimicrobial properties, inhibiting the growth of harmful bacteria such as pathogenic strains of *Escherichia coli* and *Salmonella*. This selective growth and inhibition of bacteria by HMOs help shape the composition of the infant gut microbiome, favouring the establishment of a healthy microbial community [7,8].

The interaction between HMOs and the infant gut microbiome has significant implications for infant health and development. Studies have linked higher HMO concentrations in breast milk with a lower risk of infectious diseases, allergies, and autoimmune disorders in infants. The modulation of the gut microbiome through HMOs has been associated with enhanced immune system development, improved gut barrier function, and reduced inflammation. These factors contribute to overall health and may have long-term effects on the child's well-being.

Although significant progress has been made in understanding the relationship between HMOs and the infant gut microbiome, several challenges and avenues for future research remain. The structural complexity and diversity of HMOs make it challenging to fully elucidate their specific functions and mechanisms of action [9]. Further investigation is needed to determine the individual and combined effects of different HMO structures on the gut microbiome and their subsequent impact on infant health outcomes.

Additionally, while most research has focused on breastfeeding infants, it is important to consider the implications for formula-fed infants [10]. Attempts are being made to incorporate certain HMOs into infant formulas to mimic the benefits observed in breastfed infants. However, further research is needed to determine the optimal HMO composition and concentrations in formula to support healthy gut microbiome development.

## CONCLUSION

In conclusion, the interplay between HMOs and the infant gut microbiome is a crucial factor in infant health and development. Understanding this relationship sheds light on the importance

**Correspondence to:** Philippe Michiels, Department of Virology, University of Porto, Praca de Gomes Teixeira, Porto, Portugal, E-mail: michiels.philippe@upo.pt

**Received:** 02-Jun-2023, Manuscript No. CMCH-23-22141; **Editor assigned:** 05-Jun-2023, PreQC No. CMCH-23-22141 (PQ); **Reviewed:** 19-Jun-2023, QC No CMCH-23-22141; **Revised:** 26-Jun-2023, Manuscript No. CMCH-23-22141 (R); **Published:** 03-Jul-2023. DOI: 10.35248/2090-7214.23.20.465.

**Citation:** Michiels P (2023) Exploring How Human Milk Oligosaccharides Affect the Gut Microbiome of Infants. Clinics Mother Child Health. 20:465.

**Copyright:** © 2023 Michiels P. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

of breastfeeding and the unique benefits of human breast milk. Continued research in this field will enhance our understanding of the specific roles of HMOs, guide the development of targeted interventions, and potentially pave the way for the inclusion of beneficial HMOs in infant formula. By untangling the intricate relationship between HMOs and the infant gut microbiome, we can provide better nutritional support and foster optimal health outcomes for infants in their early stages of life.

## REFERENCES

1. Ipsa E, Cruzat VF, Kagize JN. Growth hormone and insulin-like growth factor action in reproductive tissues. *Front Endocrinol (Lausanne)*. 2019;10:777.
2. Qiao J, Wang ZB, Feng HL. The root of reduced fertility in aged women and possible therapeutic options: current status and future prospects. *Mol Aspects Med*. 2014;38:54-85.
3. Shapiro BS, Richter KS, Harris DC. Implantation and pregnancy rates are higher for oocyte donor cycles after blastocyst-stage embryo transfer. *Fertil Steril*. 2002;77(6):1296-1297.
4. Reh A, Fino E, Krey L. Optimizing embryo selection with day 5 transfer. *Fertil Steril*. 2010;93(2):609-615.
5. Roque M, Lattes K, Serra S. Fresh embryo transfer versus frozen embryo transfer in in vitro fertilization cycles: A systematic review and meta-analysis. *Fertil Steril*. 2013;99(1):156-162.
6. De Croo I, Colman R, De Sutter P. Blastocyst transfer for all higher cumulative live birth chance in a blastocyst-stage transfer policy compared to a cleavage-stage transfer policy. *Facts Views Vis Obgyn*. 2019;11(2):169-176.
7. Healy MW, Patounakis G, Connell MT. Does a frozen embryo transfer ameliorate the effect of evaluated progesterone seen in fresh transfer cycles? *Fertil Steril*. 2016;105(1):93-99.
8. Dyer S, Chambers GM, De Mouzon J. International committee for monitoring assisted reproductive technologies world report: Assisted reproductive technology 2008, 2009 and 2010. *Hum Reprod*. 2016;31(7):1588-1609.
9. Pereira N, Rosenwaks Z. A fresher perspective on frozen embryo transfers. *Fertil Steril*. 2016;106(2):257-258.
10. Franasiak JM, Forman EJ, Hong KH. The nature of aneuploidy with increasing age of the female partner: A review of 15,169 consecutive trophoctoderm biopsies evaluated with comprehensive chromosomal screening. *Fertil Steril*. 2014;101(3):656-663.