Human Milk Oligosaccharides –
Background and Metabolism

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Introduction
In recent years, there has been a tremendous increase in our knowledge regarding specific effects of human milk oligosaccharides (HMOs) which are not or only in trace amounts present in bovine milk (Table 1). Concomitantly with these studies, progress in biotechnology nowadays allows to produce at least some HMOs to potentially be added to infant formulas. To decide which compounds would be most suitable for supplementation, in which concentrations or combinations, and how long it should be given, studies are needed regarding their metabolic fate as well as their local and systemic effects.

History
Important observations with regard to infants’ health have already been made around 1900. The discovery of lactobacilli and bifidobacteria and their relevance for health and disease was an important milestone. At the same time, pediatricians realized that the chemical composition of breast-fed and bottle-fed infants differed. Observations indicated that this difference is particularly linked to the milk carbohydrate fraction. This was the starting point of research on human milk carbohydrates. In the following years, the first HMOs were identified [1]. Studies conducted after 1950 focused on the identification of various HMOs as the ‘bifidus factor’ in human milk [2]. Since then, about 150 single HMOs have been characterized. It is important to note that the Lewis blood group and the secretor/non-secretor status lead to very specific HMO patterns in milk which are discussed of having an influence on certain diseases [3].

Key Messages
Infants receive large amounts of human milk oligosaccharides (HMOs) with a high potential for local effects within the gastrointestinal tract and for systemic functions. In feces and urine, native HMOs and degradation products are present which partly reflect the mothers’ specific milk oligosaccharide pattern.

Physiological Observations as Background to HMO Research
Large amounts of HMOs, i.e. several grams per day, rinse the gastrointestinal tract of a human milk-fed infant, thereby potentially preventing pathogen adhesion to the intestinal mucosa or influencing gut maturation processes [5, 6]. HMOs are considered not to be degraded by human digestive enzymes and transported into the lower end of the intestine where they may be metabolized by the microflora or get excreted with feces [7–10]. As about 1–2% of HMOs are excreted via the infants’ urine (Fig. 2), several hundred milligrams per day may circulate in the infants’ blood, which is enough to suppose systemic functions such as anti-inflammatory or anti-inflammatory effects of HMOs.

Fig. 1. Composition of HMOs and potential modifications.

Table 1. Composition of major components in human and bovine milk (g/L)

<table>
<thead>
<tr>
<th>Component</th>
<th>Human milk</th>
<th>Bovine milk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proteins</td>
<td>10</td>
<td>33</td>
</tr>
<tr>
<td>Lactose</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>Lacto-N-Biose</td>
<td>65</td>
<td>47</td>
</tr>
<tr>
<td>Oligosaccharides</td>
<td>5–15</td>
<td>Not present or only in traces</td>
</tr>
</tbody>
</table>

Modified by
- Chain Elongation and Branching
- Fucose Attachment
- Sialic Acid Attachment

Fig. 2. Intake, metabolism, and potential functions of HMOs.

References