Vitamins & Minerals
ISSN: 2376-1318

Friel, et al., Vitamin Miner 2014, 3:2

Editorial Open Access

Maternal Risk Factors for Vitamin and Mineral Nutrient Deficiencies in Exclusively Breastfed Infants-The Potential Role of Membrane Transporters in the Mammary Epithelium

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Rec date: May 27, 2014, **Acc date:** May 29, 2014, **Pub date:** May 31, 2014

Editorial

Human milk is the acknowledged first food for all infants. Unlike other foods in the human diet it is assumed to be complete in and of itself. It is presumed that evolutionary forces optimized the nutrient content to fulfill the requirements of a growing breastfed infant.

The composition of human milk is the result of complex transport processes across different extracellular and intracellular membranes, which are in large part mediated by membrane, bound solute carrier proteins [1]. However, the identities of the proteins mediating the transfer of most essential trace elements and vitamins into human milk remain to be determined. Therefore there is a lack of knowledge about the molecular mechanisms regulating their concentrations [1].

This lack of knowledge hinders the progress on two largely unexplored research questions: A) is the set of nutrient transporters expressed in the human mammary alveolar epithelial cell sufficient to supply all essential elements to fully sustain an infant during prolonged exclusive breastfeeding? B) can variations in genes encoding membrane transporters decrease their capacity to transfer nutrients into human milk and therefore be defined as maternal risk factors for nutrients deficiencies?

To address question A), exclusive breast-fed infants who do not receive supplemental vitamin D and are not exposed to adequate sunlight are at risk of developing vitamin D deficiency [2]. Currently 10 µg vitamin D per day are recommended for healthy, full-term infants in Canada, which cannot be achieved through exclusive human feeding since the vitamin D content of human milk ranges from 0.5-1.5 µg/L [3]. Cross species comparisons show higher levels in rodents, e.g. rat milk contains 1.0-3.5 µg/L vitamin D [4], suggesting an additional set of transporters might be responsible for this discrepancy. Knowledge about the secretory pathways and the proteins involved will enable us, through interspecies comparison, to determine if humans might have lost or never attained the ability to fully sustain infants' wellbeing in relation to essential nutrients when exclusively breast fed.

In addition it recently became evident that human mutations can impact on the nutrient content of human milk. Although the transfer of zinc into human milk is not fully understood, two membrane transporters ZnT2 (SLC30A2) and ZnT4 (SLC30A4) are known to be involved in the process [5]. Variations in the *SLC30A2* gene have been associated with reduced zinc concentrations in human milk, which could lead to transient neonatal zinc deficiency [6-9]. Hence, future knowledge about membrane transporters involved in nutrient transfer into human milk will enable us to identify and characterize those variations, which may serve as predictive maternal risk factors in infant development.

The above are examples of an essential vitamin and mineral for which transport and synthesis within the mammary gland are not clearly understood. The same pattern is true for nearly all other essential nutrients. With molecular tools available today these issues can be addressed.

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