Necrotizing enterocolitis (NEC) is an inflammatory disease of the gut primarily affecting premature infants. NEC is a complicated disease. The clinical presentation of NEC may represent a spectrum of disease with varying causal mechanisms. Its pathogenesis is multifactorial, with a likely unique combination of individual factors. Age at diagnosis is inversely proportional to gestational age at birth, reflecting the accrual of risk factors such as dysbiosis and the developmental immune state of the intestine. At present, it is known that the main factors involved in the pathogenesis of NEC include prematurity, microbial dysbiosis (abnormal intestinal microbiota), feeding, and impaired intestinal oxygen delivery. Of these, prematurity is the strongest risk factor. Premature infants are particularly vulnerable to developing NEC because they have immature host, comprising 70% to 90% of total NEC cases. Measures focused on preventing the development of dysbiosis, including administration of probiotics, protocolized feedings focused on mother’s own milk, avoiding acid blockers, and judicious use of antimicrobials, remain the primary tools to prevent NEC. Neonatal diet is an important modifiable factor that can affect long-term cognitive outcome through a “human milk” factor, protecting against infection/NEC, and a “nutrient content” factor. Neonatal diet plays an important role in preventing infection and NEC in preterm infants. Intake of breast milk during the first days of life has been shown to be associated with decreased infection, morbidity, and mortality in very low birth weight infants. Preterm infants exclusively fed human milk were up to 6–10 times less likely to suffer from NEC than infants fed cow milk-based formula, with a strong dose-response relationship. In summary, identifying the risk factors related to NEC in premature infants and exploring preventive measures has been confirmed by a large number of relevant literature. Therefore, this article will not elaborate on this topic in detail. The focus of this article is on the degree of impact of human milk as an important protective factor.

The conclusion that donated milk can prevent NEC is confirmed, but there is still controversy over its effectiveness in preventing ROP, BPD, and promoting neurodevelopment. Donated milk can prevent NEC, not because the beneficial components of donated milk (freeze-thaw and bus disinfection kill many beneficial components in human milk) play a role, but because the use of donated milk avoids the direct toxic effect of formula milk on intestinal epithelial cells and reduces intestinal permeability. Furthermore, DHM may also contribute to reduction in NEC by enabling earlier enteral feeding and reducing the inflammatory impact of prolonged TPN. Thus, our clinicians introduce enteral feedings of DHM earlier post-birth instead of waiting for MOM (mother’s own milk) to become available.

Finally, regarding the fortification of donated milk, we clinicians will use individual fortification agents according to the growth and development curve of premature infants. As it is not closely relevant to this topic, how to use fortification agents is not described.
Disclosure
The authors report no conflicts of interest in this communication.

References