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Neurodevelopmental impairments with necrotizing enterocolitis: Microbiome, gut and brain entanglements

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INTRODUCTION

Neurodevelopmental impairments in neonates, particularly those born prematurely, present significant challenges to paediatric healthcare. One condition that has garnered attention in this context is Necrotizing Enterocolitis (NEC) a severe gastrointestinal disorder that primarily affects premature infants. NEC not only poses immediate health risks but may also have long-term consequences for neurodevelopment. Understanding the interplay between the microbiome, gut health, and brain development is crucial for developing strategies to mitigate these impairments. Necrotizing enterocolitis is characterized by inflammation and necrosis of the intestinal tract, primarily occurring in preterm infants. The condition affects approximately 5-10% of very low birth weight infants and has a mortality rate that can exceed 20%. The etiology of NEC is multifactorial, involving a combination of factors such as intestinal immaturity, abnormal bacterial colonization, and compromised blood flow to the intestines [1].

Immature intestinal epithelial cells lead to increased permeability, allowing pathogens to breach the gut barrier. A disrupted microbiome, often characterized by a reduction in beneficial bacteria like Lactobacillus and Bifidobacterium and an increase in pathogenic organisms, is commonly observed in NEC cases. The condition is marked by an exaggerated inflammatory response [2], which can cause further tissue damage and necrosis. Neurodevelopmental impairments encompass a range of disorders that affect cognitive, motor, and social skills.

Infants with NEC may experience periods of reduced oxygen supply to the brain, leading to brain injury. The inflammatory cytokines released during NEC can have detrimental effects on brain development. The inability to feed adequately during episodes of NEC can result in malnutrition, affecting neurodevelopment. In neonates, especially preterm infants, the microbiome is still developing and is highly susceptible to environmental influences. Dysbiosis, characterized by an imbalance in the gut microbiota, is frequently observed in infants with NEC. Antibiotics can disrupt the normal microbiota, leading to overgrowth of harmful bacteria. Formula feeding has been associated with a less diverse microbiome compared to breastfeeding, which can promote the growth of beneficial bacteria [3].

DESCRIPTION

An increased incidence of cerebral palsy and other motor disorders has been observed. Children with a history of NEC may exhibit higher rates of attention-deficit hyperactivity disorder and anxiety disorders. Early intervention programs that focus on both nutritional support and neurodevelopmental care may help mitigate some of the adverse effects associated with NEC. Strategies to promote a healthy microbiome through targeted feeding practices. Early therapies to enhance motor and cognitive skills. Future research should focus on elucidating the mechanisms through which the microbiome influences both gut and brain health in neonates. Identifying specific bacterial profiles associated with both NEC and neurodevelopmental outcomes. Exploring the potential of probiotics and prebiotics to restore a healthy microbiome and improve outcomes. Translating research findings into clinical practice is crucial for improving outcomes in infants with NEC. Tailoring feeding strategies based on microbiome profiles to promote gut health and neurodevelopment. Implementing interventions that target the gut-brain axis to minimize neurodevelopmental impairments.

Emerging research suggests that the gut microbiome may influence brain development through the gut-brain axis, a bidirectional communication pathway between the gut and the brain. Short Chain Fatty Acids (SCFAs) produced by gut bacteria can have neuroprotective effects and promote brain health. The microbiome can influence the production of neurotransmitters, such as serotonin, which play critical roles in mood and behavior. A healthy microbiome can promote a balanced immune response, potentially reducing neuroinflammation. The vagus nerve provides a direct connection between the gut and the brain, transmitting signals that can influence mood and behavior. Hormones and neurotransmitters produced in the gut can affect brain function and development. Gut health can influence systemic inflammation, which is linked to neurodevelopmental outcomes [4].

The developing brain is sensitive to external stimuli and can be adversely affected by gut dysbiosis and inflammation. The stress associated with NEC can disrupt the delicate balance of the gut-brain axis, exacerbating neurodevelopmental impairments. Studies have shown that infants who experience NEC are at increased risk for long-term neurodevelopmental issues. Reduced IQ and learning difficulties are more prevalent in children with a history of NEC [5].

CONCLUSION

The intricate relationship between necrotizing enterocolitis, the microbiome, and neurodevelopmental impairments highlights the need for a holistic approach to neonatal care. By understanding the gut-brain axis and its implications for health, clinicians can develop targeted strategies to support the well-being of vulnerable infants. Future research will be essential in unlocking the complexities of this relationship and improving long-term outcomes for those affected by NEC. As we advance our understanding, we can better equip ourselves to mitigate the challenges posed by neurodevelopmental impairments in the wake of this serious condition.

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CONFLICT OF INTEREST

None.

REFERENCES

1. **Dominguez-Bello MG, Costello EK, Contreras M, et al.** Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns. Proc Natl Acad Sci U S A. 2010; 107(26):11971-11975.
2. **Kim G, Bae J, Kim MJ, et al.** Delayed establishment of gut microbiota in infants delivered by cesarean section. *Front Microbiol*. 2020; 11:2099.
3. **Shao Y, Forster SC, Tsaliki E, et al.** Stunted microbiota and opportunistic pathogen colonization in caesarean-section birth. *Nature*. 2019 Oct 3; 574(7776):117-121.
4. **Vu K, Lou W, Tun HM, et al.** From birth to overweight and atopic disease: Multiple and common pathways of the infant gut microbiome. *Gastroenterology*. 2021; 160(1):128-144.
5. **Chu DM, Ma J, Prince AL, et al.** Maturation of the infant microbiome community structure and function across multiple body sites and in relation to mode of delivery. *Nat Med*. 2017; 23(3):314-326.