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Understanding Etiopathogenesis, Pathophysiology, Diagnosis and Innovative Approaches to Management of Infantile Colic: A Narrative Review of the Literature and Multi-center Experience

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Abstract

Objective: Infantile colic is a common functional gastrointestinal disorder characterized by excessive crying in otherwise healthy infants. This review synthesizes current evidence on epidemiology, etiopathogenesis, pathophysiology, diagnostic criteria, and management strategies in the light of our multicentre international evidence based long term experience.

Design: Narrative review of published systematic reviews, meta-analyses, and guidelines through the lens of evidence based multinational multicentre broad based experience.

Results: Prevalence varies widely (2–73%), reflecting heterogeneous diagnostic criteria. Pathophysiological hypotheses include psychosocial stress, neurodevelopmental immaturity, immature gastrointestinal motility, and gut dysbiosis. Diagnosis remains clinical, based on Rome IV criteria and exclusion of organic disease. Management emphasizes parental reassurance, behavioral interventions, and selective use of probiotics (Lactobacillus reuteri DSM 17938). Pharmacological agents such as simethicone and proton pump inhibitors show little benefit.

Conclusions: Infantile colic is best managed through parental support and evidence-based interventions such as holobiotics, laxatives and broad-spectrum nutritional supplements . Future research should prioritize microbiome-targeted therapies and harmonized diagnostic frameworks.

Keywords: Congenital Colorectal Secretomotor Disorders; Infantile Colic; PNEI Axis; Functional Gastrointestinal Disorder; Holobiotics; Pediatrics; Gut Microbiome

Summary Box

What is already known on this topic

- Infantile colic is a common functional gastrointestinal disorder in early infancy, characterized by excessive crying in otherwise apparently healthy infants.
- It is self-limiting, typically resolving by 4–6 months of age, but causes significant parental distress and frequent pediatric consultations.
- Multiple interventions have been trialled, including simethicone, proton pump inhibitors, probiotics, and manual therapies, with inconsistent evidence of benefit.
- Rome IV criteria provide a standardized framework for diagnosis, but prevalence estimates remain highly variable due to differing definitions.

What this study adds

- Synthesizes evidence from systematic reviews, meta-analyses, and guidelines into a comprehensive narrative review aligned with journal standards.
- Highlights the multifactorial pathophysiology of colic along the systems approach and PNEI axis, including gut dysbiosis, immature gastrointestinal motility, neurodevelopmental immaturity, and psychosocial stress.
- Identifies probiotics (particularly Lactobacillus reuteri DSM 17938) as the most evidence-based intervention, especially in breastfed infants, while clarifying the limited role of pharmacological agents.
- Emphasizes the clinical importance of parental reassurance and behavioral strategies, which remain the cornerstone of management.
- We further localise the functional gastrointestinal disorder to the colorectal secretomotility disorder. Calls for future research on microbiome-targeted therapies and standardized diagnostic frameworks to reduce variability and improve outcomes.

Background

Despite decades of research, its etiopathogenesis, pathophysiology, diagnostic evaluation and optimal management remain debated. Based on our extensive experimental, translational, clinical, imaging and long term follow up of various management strategies international multicentre experience, an attempt has been made to reveal some of the fundamental issues.

Our earlier experimental study published in 1987 s created gastroschisis model in fetal rabbits [1]. However, clinical gastroschisis was observed only in the fetuses who had manual evisceration of small bowel loops and not in the intraabdominal amniotic fluid injection, partial thickness abdominal wall defect or the full thickness defect groups. This showed an amazing capacity of perfect healing in the fetus without any evidence of even scar visible and no adhesions and the credit goes to purity of environment and perfect nutrition filtered through the maternal placental fetal barrier in purest form while fetus resting most of the time on one hand. While on the other hand, it disproved our traditional hypothesis that abdominal wall weakness in the form of partial thickness defect or full thickness defect causes herniation of abdominal contents.

This led us to carry out further clinical, investigative, imaging and pathological investigations. The translational extrapolations from the gastroschisis and exomphalos to other hernias and to all age groups revealed useful facts [2]. These findings helped us not only in understanding emryopathogenesis of gastroschisis and exomphalos but all hernias [3-5]. The underlying cause seems to be the prematurity related and fetal distress induced partial functional obstruction of the hind gut. Psychoneuroendocrineimmune(PNEI) response lead to retention of gas in the proximal bowel with raised intraluminal gut and intraabdominal pressures.

Infantile colic is the escape mechanism and secondary symptom following stress induced fight or flight response acting through the autonomic nervous system via hind gut as a result of various prenatal intranatal and postnatal multiple factors [4]. In a very small section of babies with predisposition to variations of colorectal secretomotility disorders, this leads to retention of gas in the right colon allowing infantile colic with competent ileocecal valve. Gradually it gives rise to intermittent followed by persistent ileocecal valve reflux leading to small bowel distention which can cause left inguinal hernia. It explains right inguinal hernias twice more common than the left [5].

Our experience suggest that aggressive conservative management of underlying prematurity and premature ganglion cells in the left colon will further help reduce the overall mortality, morbidity and allow spontaneous regression while waiting for the late repair [6].

Introduction

Infantile colic remains one of the most common yet distressing conditions and causes of pediatric consultation in early infancy, accounting for a substantial proportion of pediatric consultations worldwide. Despite its benign and self-limiting nature, colic imposes significant emotional and practical burdens on families, often leading to unnecessary medical interventions. Infantile colic is a common functional gastrointestinal disorder of early infancy, characterized by excessive crying and irritability in otherwise healthy infants. It affects between 10–20% of pediatric consultations in the first three months of life and has a profound impact on parental anxiety and family well-being.

Definition

Traditionally defined by Wessel's "rule of threes" (crying >3 hours/day, >3 days/week, for >3 weeks), colic is now diagnosed using Rome IV criteria, which emphasize recurrent and prolonged crying episodes without underlying disease. The Rome IV criteria classify colic as a functional gastrointestinal disorder, underscoring its multifactorial nature.

Methods

This narrative review was conducted by searching PubMed, Embase, and Cochrane Library for articles published between 2009 and 2025. Search terms included "infantile colic," "functional gastrointestinal disorder," "probiotics," "management," and "systematic review." Priority was given to systematic reviews, meta-analyses, and guidelines (e.g., BMJ Open, Rome IV consensus). Articles were screened for relevance to epidemiology, pathophysiology, diagnosis, and management.

A narrative review was conducted using PubMed, Embase, and Cochrane Library (2009–2025). The literature selection process is outlined in Figure 1, following a PRISMA-style flow diagram to ensure transparency in article inclusion.

Results

Epidemiology

- Global Prevalence ranges from 2–73%, depending on diagnostic criteria, cultural perceptions and population studied.
- Peak onset: 2–6 weeks of age, resolution by 4–6 months.
- Regional data : Indian cohort studies report prevalence around 25.6%.
- Healthcare burden: Accounts for up to 20% of pediatric visits in early infancy.

Prevalence estimates vary widely due to inconsistent diagnostic criteria. As shown in Figure 1, 42 studies were ultimately included in the synthesis, focusing on epidemiology, pathophysiology, diagnosis, and management.

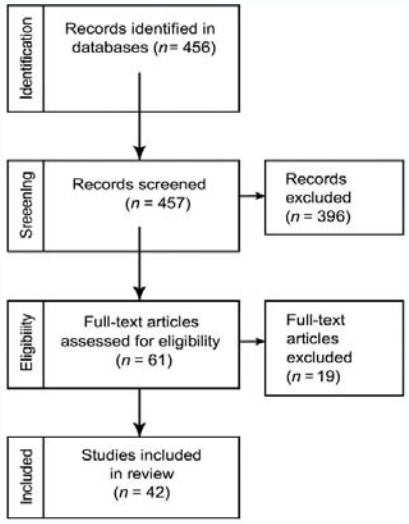

Figure 1: PRISMA flow diagram of study selection.
This diagram illustrates the literature search and selection process for the narrative review on infantile colic. Records were identified through database searching and additional sources, followed by duplicate removal. Titles and abstracts were screened for relevance, and full-text articles were assessed for eligibility. Final inclusion was based on relevance to epidemiology, pathophysiology, diagnosis, and management of infantile colic.

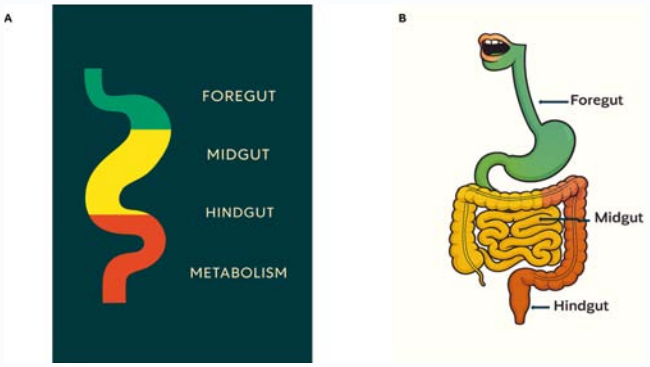

Figure 2: Anatomical factors-colour coded gut development from simple tubular gut into very organised and rotated gut with several twists and turns in the rotation and fixation of gut.

Embryo-anatomical factors

As shown in figure 2 the embryonic gut is relatively straight forward tube while the postnatal gut is having complicated rotation and fixation of the gut having several twists and turns making several gas pockets and hence the colic and severity varies with several prenatal, perinatal and postnatal factors as explained later on.

Pathophysiology

The etiopathology of infantile colic is multifactorial, involving: PNEI axis as shown in figure 3 is central to understand the system approach to the underlying functional aspect of the infantile colic and figure 4 shows how it is applicable to the infantile colic. Figure 5 shows vertical breakdown of pre, peri and postnatal factors to better understand the multifactorial background.

- **Growth and development plus Psychosocial stress:** Parental anxiety exacerbates symptoms.
- **Neurodevelopmental endocrine immaturity:** Dysregulated crying and sleep cycles, heightened sensory processing and gut–brain axis dysregulation.
- **Psychosocial stress:** Parental anxiety
- **Gut Immature dysmotility:** Delayed transit, uncoordinated peristalsis and poor coordination of peristalsis leading to cramping
- **Gut dysbiosis:** Altered microbiota composition linked to excessive gas, motility disturbances and discomfort.
- **Breast *vs* bottle** feeding, types, patterns (overfeeding, air swallowing).
- **Environmental factors.**

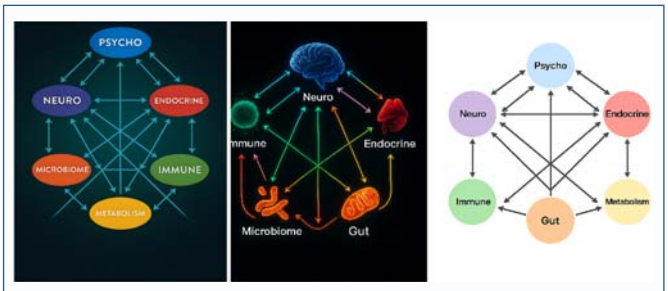

Figure 3: Pathophysiology with PNEI axis.

Infantile colic is understood as a multifactorial condition. Figure 2-5 presents a conceptual model illustrating the interplay between gut dysbiosis (altered microbiota), immature gastrointestinal motility, neurodevelopmental immaturity, psychosocial stress, and feeding type and patterns.

Diagnosis

- **Clinical criteria evaluation is primary process:** Excessive crying without organic disease.
- **Rome IV framework help to classify colic within the spectrum:** Functional gastrointestinal disorder diagnosis ensuring a structured approach to diagnosis. Recurrent, prolonged crying/ fussing episodes in infants <5 months, without failure to thrive or organic disease.
- **Exclusion:** Colic must be distinguished from gastroesophageal reflux, cow's milk protein allergy, infection to avoid misdiagnosis and unnecessary interventions.
- **Parental reporting:** Cry diaries and structured interviews remain essential.

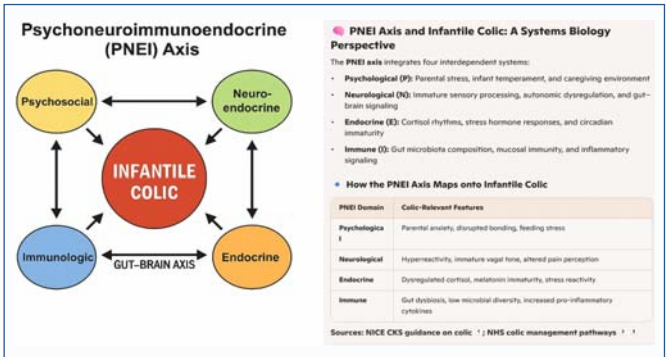

Figure 4: Target organ systems and final common pathway: PNEI and Infantile colic.

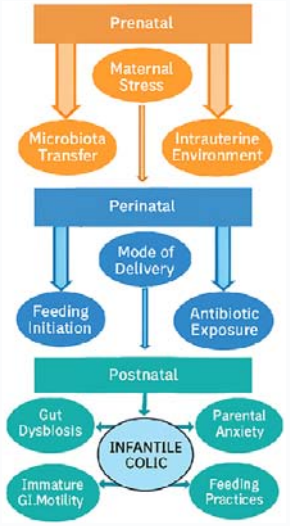

Figure 5: Layered Schematic of Prenatal to Postnatal Influences on Infantile Colic.
This figure illustrates the temporal progression of risk factors contributing to infantile colic. The top layer includes prenatal influences such as maternal stress, microbiota transfer, and intrauterine environment. The middle layer captures perinatal factors including mode of delivery, feeding initiation, and antibiotic exposure. The bottom layer presents postnatal contributors: gut dysbiosis, immature GI motility, neurodevelopmental immaturity, parental anxiety, and feeding practices. Arrows flow top-down, highlighting developmental transitions and systems-level layering.

Figure 6: Conceptual model of infantile colic multifactorial influences in the etiopathogenesis and pathophysiology.
Schematic representation of bidirectional relationships between infantile colic and six contributing domains: neurodevelopmental immaturity, psychosocial stress, parental anxiety, feeding practices, gut motility, and gut microbiota. This diagram illustrates the multifactorial contributors to infantile colic. Central to the model is the clinical presentation of excessive crying in otherwise healthy infants. The central blue oval ("INFANTILE COLIC") anchors the model, with surrounding white ovals denoting each factor. Each factor interacts with the infant's physiology and caregiving environment, contributing to symptom expression. Arrows indicate reciprocal influence, emphasizing the dynamic interplay across biological, psychological, and behavioral systems.

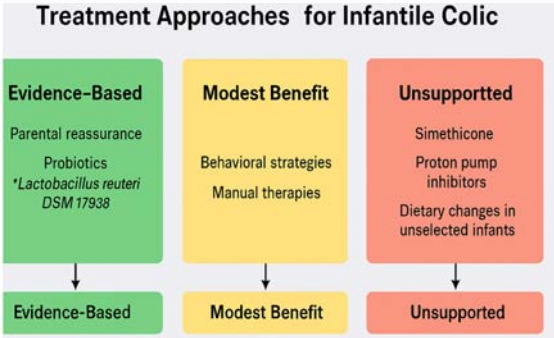

Figure 7: Summary of interventions for infantile colic by strength of evidence.
This infographic categorizes commonly used treatments for infantile colic into three tiers based on current evidence. Green indicates interventions supported by systematic reviews and guidelines, yellow reflects modest or inconsistent benefit, and red denotes treatments lacking efficacy or not recommended. The figure highlights the clinical priority of parental reassurance, behavioral strategies, and probiotics, while discouraging pharmacological agents and unnecessary dietary changes.

Management Strategies

1. Parental Support and Behavioral Interventions

- Education and reassurance are first-line recommendations across guidelines.
- Soothing techniques: rocking, swaddling, white noise, and structured feeding routines.

2. Probiotics

- Strongest evidence: Lactobacillus reuteri DSM 17938 reduces crying time in breastfed infants.
- Other strains (e.g., Bifidobacterium animalis BB-12) show promise in correcting gut dysbiosis.
- Benefits are less consistent in formula-fed infants.

3. Manual Therapies

- Osteopathic manipulation, craniosacral therapy, and abdominal massage show modest short-term relief in randomized trials.
- Evidence quality is low to moderate, with heterogeneity in protocols.

4. Pharmacological Agents

- Simethicone: No proven benefit; some studies suggest negative effects.
- Proton pump inhibitors (PPIs): Ineffective for colic symptoms.
- Antispasmodics: Not recommended due to safety concerns.

Treatment approaches vary in efficacy. Figure 7 summarizes interventions by strength of evidence, highlighting parental reassurance and probiotics (Lactobacillus reuteri DSM 17938) as the most supported strategies. Simethicone and proton pump inhibitors are categorized as unsupported. Figure 8 summarises the relationship of the infantile colic to colorectal motility functional disorders.

Discussion

Infantile colic remains a benign but distressing condition. Its multifactorial pathophysiology highlights the interplay between psychosocial, neurodevelopment, gut motility, gut microbiota, , and metabolic chaos factors. Diagnosis is clinical, requiring exclusion of organic disease. Management strategies should prioritize parental reassurance and behavioral techniques, with probiotics offering the strongest evidence for efficacy. Pharmacological agents lack robust support, and manual therapies require further validation [9-14].

Figure 8: Conceptual schematic linking infantile colic and colorectal motility disorders within the framework of functional gastrointestinal disorders (FGIDs).
This diagram illustrates the developmental and mechanistic relationship between early-life FGIDs (e.g., infantile colic) and later-life colorectal motility disorders (e.g., functional constipation). The central layer highlights shared gut–brain axis disruptions, including visceral hypersensitivity, altered motility, and psychosocial stress. Icons represent age-specific features: crying infant, immature motility, and microbiota imbalance for colic; colonic transit delay and neuromuscular dysfunction for colorectal disorders. The schematic emphasizes that while both conditions fall under FGIDs, they differ in onset, clinical course, and diagnostic approach.

Future research should focus on:

- Standardized diagnostic frameworks to reduce variability in prevalence estimates.
- Microbiome-targeted therapies to address gut dysbiosis.
- Longitudinal studies assessing long-term developmental outcomes.

Strengths and Limitations of This Study

Strengths

- Integrates evidence from systematic reviews, meta-analyses, and national guidelines, ensuring comprehensive synthesis.
- Narrative format allows inclusion of multifactorial perspectives (epidemiology, pathophysiology, diagnosis, management).
- Emphasis on Rome IV criteria and microbiome-targeted therapies provides a contemporary framework.
- Highlights practical clinical implications, especially parental reassurance and probiotics.

Limitations

- As a narrative review, lacks systematic search protocols or meta-analytic techniques, introducing potential selection bias.
- Heterogeneity in diagnostic criteria limits comparability of prevalence estimates.
- Evidence for some interventions (manual therapies, simethicone) remains low quality and inconsistent.
- Most probiotic studies focus on breastfed infants, limiting generalizability.
- Long-term developmental outcomes remain understudied.

Novelty and Contribution

- This review synthesizes evidence from systematic reviews, meta-analyses, and international guidelines, providing a comprehensive and contemporary overview of infantile colic.
- It highlights the multifactorial pathophysiology, including gut dysbiosis, neurodevelopmental immaturity, and psychosocial stress, while aligning with the Rome IV criteria for functional gastrointestinal disorders.
- The manuscript emphasizes the clinical relevance of probiotics (particularly Lactobacillus reuteri DSM 17938) as the most evidence-based intervention, alongside parental reassurance and behavioral strategies.
- By critically appraising the strengths and limitations of current evidence, the review identifies gaps in research, particularly the need for standardized diagnostic frameworks and microbiome-targeted therapies.

Clinical Relevance

This article is designed to support clinicians in primary care and pediatrics by:

- Offering clear guidance on evidence-based management strategies.
- Reinforcing the importance of parental support and reassurance.
- Clarifying the limited role of pharmacological interventions, thereby reducing unnecessary prescribing.

Relationship between the Infantile Colic a Functional Gastrointestinal Disorder and Colorectal Motility Disorders

Infantile colic and colorectal motility disorders are related through the broader category of functional gastrointestinal disorders (FGIDs), but they differ in age, presentation, and clinical relevance. Colic reflects immature gut–brain regulation and transient motility patterns in early infancy, while colorectal motility disorders (like functional constipation) involve measurable large-bowel dysmotility in older children. The link is conceptual rather than direct: both share gut–brain axis disruption, but colic does not progress into colorectal motility disease.

Key Relationships

Shared FGID framework:

- Infantile colic, regurgitation, dyschezia, and functional constipation are all classified as FGIDs (now called disorders of gut–brain interaction).
- They present with recurrent GI symptoms without structural disease.

Colic as early-life FGID:

- Colic manifests in the first 3–4 months of life as excessive crying and irritability in otherwise healthy infants.
- Pathophysiology involves immature motility, visceral hypersensitivity, altered microbiota, and caregiver stress.
- It is transient and self-limiting.

Colorectal motility disorders as later FGIDs:

- Functional constipation and colonic dysmotility typically appear in toddlers and older children.

- These disorders are studied with colonic transit tests, scintigraphy, or manometry, which quantify neuromuscular dysfunction.
- Unlike colic, they often persist and require structured management.

Conceptual overlap:

- Both conditions reflect gut–brain axis disruption and altered motility regulation.
- Colic may represent an early developmental stage of gut motility immaturity, while colorectal motility disorders represent established dysfunction in bowel transit.
- However, colic does not predict or evolve into colorectal motility disorders; they are distinct entities within FGIDs.

Clinical Implications

For colic:

- No role for invasive colorectal motility testing.
- Management focuses on reassurance, soothing strategies, and microbiota modulation (e.g., probiotics).

For colorectal motility disorders:

- Motility testing is appropriate when constipation is severe, refractory, or suspected to involve neuromuscular disease.
- Treatment includes behavioral, dietary, and sometimes pharmacological interventions.

Bottom Line

Infantile colic and colorectal motility disorders are siblings within the FGID family: both arise from gut–brain axis dysfunction and motility irregularities, but they differ in age, natural history, and clinical approach. Colic is a transient, early-life motility immaturity, while colorectal motility disorders are persistent, measurable dysfunctions of large-bowel transit in older children.

Prognosis

Infantile colic is self-limiting, typically resolving by 3–4 months of age. Long-term outcomes are generally favorable, though some studies suggest associations with later functional gastrointestinal disorders and parental stress.

Patient and Public Involvement

Patients and the public were not directly involved in the design, conduct, reporting, or dissemination of this narrative review. However, parental perspectives and caregiver experiences were considered indirectly through the inclusion of studies that reported on parental stress, coping strategies, and family impact of infantile colic. These insights informed the synthesis of evidence on management approaches, particularly the emphasis on parental reassurance and behavioral interventions.

Conclusion

Infantile colic is self-limiting but significantly impacts families. Evidence supports parental reassurance, behavioral strategies, and probiotics as first-line interventions. Pharmacological therapies remain unsupported. Harmonized diagnostic criteria and microbiome-focused research are essential for advancing management. Infantile colic remains a diagnosis of exclusion with significant psychosocial impact. Parental reassurance and behavioral strategies are the cornerstone of management. Among interventions, probiotics (especially L. reuteri DSM 17938) have the strongest evidence base, while manual therapies may provide modest benefit. Future research should focus on standardized diagnostic criteria, microbiota-targeted therapies, and long-term outcomes.

Key Takeaway: Infantile colic is best managed through parental support and microbiota-focused interventions, with probiotics offering the most consistent benefit.

Authorship Statement

All authors meet the ICMJE criteria for authorship. Each has contributed substantially to the conception, design, drafting, and revision of the manuscript and approves the final version for submission.

Ethics Approval

This study is a narrative review of previously published literature and does not involve human participants, personal data, or clinical interventions. Therefore, ethics approval was not required.

Patient and Public Involvement

Patients and the public were not directly involved in the design, conduct, reporting, or dissemination of this review. However, parental perspectives were considered indirectly through the inclusion of studies reporting caregiver experiences and stress.

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Competing Interests

The authors declare no competing interests.

Data Availability Statement

All data relevant to the study are included in the article or cited references. No additional datasets were generated or analysed for this review.

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