International Clinical and Medical Case Reports Journal
Research Article (ISSN: 2832-5788)

β-Catenin Signaling in Appendicitis: A Retrospective Analysis of Basic Experimental Studies and Nursing Implications

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Citation: *Chen W. β-Catenin Signaling in Appendicitis: A Retrospective Analysis of Basic Experimental Studies and Nursing Implications. Int Clinc Med Case Rep Jour. 2025;4(11):1-5.*

Received Date: 11 March, 2025; **Accepted Date:** 16 April, 2025; **Published Date:** 19 May, 2025

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ABSTRACT

Background: Appendicitis is a life-threatening acute abdominal disorder characterized by inflammatory injury and intestinal barrier dysfunction. β-Catenin, a core mediator of the Wnt pathway, regulates inflammation, epithelial proliferation, and tissue repair—key processes in appendicitis pathogenesis.

Objective: To synthesize basic experimental evidence on β-Catenin's role in appendicitis and explore its relevance to nursing practice.

Methods: A retrospective analysis of PubMed (2018–2024) using keywords "Appendicitis[MeSH] AND β-Catenin[MeSH] AND Basic Research[Filter]". Eligible studies were animal/cell models investigating β-Catenin in appendicitis.

Results: Ten studies were included. β-Catenin activation (nuclear translocation, upregulated expression) was observed in appendiceal tissues of animal models (mouse/rat) and LPS-stimulated intestinal epithelial cells, correlating with reduced pro-inflammatory cytokines (TNF-α, IL-6) and enhanced epithelial barrier repair. β-Catenin activation (via LiCl/Wnt3a) alleviated appendiceal damage.

Conclusion: β-Catenin modulates inflammation and tissue repair in appendicitis. Basic findings support nursing strategies for infection prevention and recovery promotion.

Keywords: Appendicitis; β-Catenin; Inflammation; Tissue repair

INTRODUCTION

Appendicitis affects 7–12 per 100,000 individuals annually, with perforation rates of 15–30% in untreated cases[1]. Beyond acute inflammation, appendicitis causes intestinal epithelial injury—an underrecognized driver of infection spread. β-Catenin, the central effector of the Wnt signaling pathway, governs intestinal homeostasis by regulating epithelial cell proliferation, apoptosis, and inflammatory responses [2]. While β-Catenin's role in inflammatory bowel disease and intestinal injury is well documented, its function in appendicitis remains fragmented in basic research, and translation to nursing practice (e.g., barrier protection, infection control) is unaddressed. This analysis aimed to: (1) summarize β-Catenin-related basic evidence in appendicitis; (2) identify nursing-relevant molecular targets; (3) highlight basic-clinical translation gaps.

MATERIALS AND METHODS

Study Design and Data Source

A retrospective review of basic experimental studies was conducted using **PubMed** (https://pubmed.ncbi.nlm.nih.gov/), covering January 2018 to March 2024 (to include recent advances).

Search Strategy

Search string: ("Appendicitis"[MeSH Terms] OR "Appendicitis"[All Fields]) AND ("β-Catenin"[MeSH Terms] OR "Beta-Catenin"[All Fields]) AND ("Basic Research"[Filter] OR "Animal Model"[All Fields] OR "Cell Culture"[All Fields]). No language restrictions; only full-text English studies were included.

Eligibility Criteria

- **Inclusion**: (1) Basic experiments (animal models: C57BL/6 mice, Sprague-Dawley rats; cell models: intestinal epithelial cells [Caco-2, IEC-6]); (2) studies investigating β-Catenin expression, activation, or intervention in appendicitis; (3) outcomes including inflammation, epithelial function, or histopathology.
- **Exclusion**: (1) Clinical studies (human subjects, trials); (2) reviews, case reports; (3) studies on non-appendicitis intestinal diseases.

Data Extraction

Two reviewers extracted data (study model, sample size, β-Catenin detection methods [Western blot, IHC, immunofluorescence], key results, nursing-related findings) using a standardized form. Discrepancies were resolved by a third reviewer.

RESULTS

Literature Retrieval Outcomes

Initial search yielded 38 articles. After removing duplicates (n=7) and screening titles/abstracts (n=16 excluded for non-basic research), 15 full-texts were assessed. Five were excluded (2 reviews, 3 off-topic), resulting in **10 eligible studies**[3-12].

Study Characteristics

All studies used animal models (n=8: mouse/rat appendicitis induced by surgical ligation [n=5], E. coli inoculation [n=2], or LPS intraperitoneal injection [n=1]) or cell models (n=2: LPS-stimulated Caco-2/IEC-6 cells). β-Catenin was detected via Western blot (n=9, measuring total/phosphorylated β-Catenin), IHC (n=6, localizing nuclear β-Catenin), and immunofluorescence (n=3, epithelial β-Catenin distribution).

β-Catenin Activity in Appendicitis

In animal models, β-Catenin activation was detected 12 hours post-appendicitis induction, peaking at 24–48 hours (nuclear β-Catenin increased by 2.1–3.5-fold vs. control)[3,5,7]. IHC showed β-Catenin localization in appendiceal epithelial cells and submucosal stromal cells—consistent with epithelial repair[4,6]. In LPS-stimulated cells, β-Catenin expression increased in a dose-dependent manner (LPS 0.1–5 μg/mL), with nuclear translocation observed at 6 hours[11,12].

β-Catenin-Mediated Mechanisms

Seven studies reported β-Catenin's anti-inflammatory role: activated β-Catenin reduced TNF-α (1.8–2.9-fold decrease vs. appendicitis model) and IL-6 (1.5–2.4-fold decrease) via downregulating NF-κB activit[y3,5,8-10]. Five studies linked β-Catenin to epithelial repair: β-Catenin activation increased tight junction proteins (occludin, zonula occludens-1) by 1.7–2.3-fold, reducing intestinal permeability (assessed via FITC-dextran)[4,6,11].

β-Catenin Intervention Effects

Four studies tested β-Catenin modulators: (1) LiCl (Wnt/β-Catenin activator) increased β-Catenin expression by 40–60%, reduced appendiceal wall edema, and improved epithelial integrity[5,9]; (2) Wnt3a (recombinant protein) suppressed LPS-induced IL-1β by 35% in Caco-2 cells[12]; (3) β-Catenin siRNA transfection exacerbated appendiceal inflammation (TNF-α increased by 2.2-fold)[8].

Nursing-Relevant Implications

Three studies provided nursing insights: LiCl intervention reduced bacterial translocation (a sepsis risk factor) by 45% [9]; β-Catenin-mediated tight junction repair correlated with reduced peritoneal infection[6]; and Wnt3a improved epithelial barrier function—supporting nursing focus on early intestinal barrier protection[12].

DISCUSSION

This analysis confirms β-Catenin as a key regulator of inflammation and epithelial repair in appendicitis basic models. Consistent findings show β-Catenin activation mitigates appendiceal damage via anti-inflammatory and barrier-protective effects.

Translation to Nursing

β-Catenin's role in reducing bacterial translocation[9] highlights nursing need for sepsis monitoring (e.g., procalcitonin, vital signs) in high-risk appendicitis patients. Its epithelial repair function[6,12] supports early enteral nutrition (a known Wnt/β-Catenin activator) to enhance barrier integrity—aligning with pre-operative nursing care.

LIMITATIONS

All studies used animal/cell models (limited human relevance); only 10 studies were included (small sample); few studies explicitly addressed nursing outcomes.

FUTURE DIRECTIONS

Basic research should use human primary appendiceal cells; clinical nursing studies could test β-Catenin-targeted interventions (e.g., optimized enteral nutrition) on patient recovery.

CONCLUSION

Basic experimental studies demonstrate β-Catenin activates to regulate inflammation and epithelial repair in appendicitis. β-Catenin modulation alleviates tissue damage and reduces infection risk—providing a molecular basis for nursing interventions (sepsis monitoring, intestinal barrier protection). Bridging basic β-Catenin research and clinical nursing is critical for improving appendicitis care.

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