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Protein Kinase C (PKC) Analysis of Basic Experimental Studies

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ABSTRACT

Background: Appendicitis is an acute inflammatory disorder involving dysregulated cell signaling and intestinal barrier damage, and protein kinase C (PKC)—a key serine/threonine kinase—regulates inflammation, epithelial apoptosis, and barrier integrity.

Objective: To synthesize basic experimental evidence on PKC's role in appendicitis and explore nursing relevance.

Methods: Retrospective analysis of PubMed (2019–2024) using keywords "Appendicitis[MeSH] AND Protein Kinase C[MeSH] AND Basic Research[Filter]". Eligible studies were animal/cell models focusing on PKC in appendicitis.

Results: Ten studies were included. PKCα/PKCβ (dominant subtypes) activation (phosphorylation) was upregulated in appendiceal tissues of animal models (mouse/rat) and LPS-stimulated immune/epithelial cells, correlating with increased pro-inflammatory cytokines and epithelial apoptosis. PKC inhibition alleviated appendiceal inflammation and barrier damage.

Conclusion: PKC (PKCα/PKCβ) promotes inflammatory progression in appendicitis, providing a basis for nursing strategies in inflammation control and infection prevention.

Keywords: Appendicitis; Retrospective analysis; Cytokines; Epithelial apoptosis

INTRODUCTION

Appendicitis affects 7–15 per 100,000 individuals annually, with untreated cases leading to perforation (20–35%) and sepsis (5–12%)[1]. Aberrant cell signaling—particularly activation of pro-inflammatory kinase pathways—drives appendiceal tissue damage; PKC (10 subtypes, PKCα/PKCβ enriched in intestinal

epithelium/immune cells) mediates NF-κB activation, epithelial apoptosis, and tight junction disruption[2]. While PKC's role in inflammatory bowel disease and acute intestinal injury is documented, its dynamic changes and regulatory effects in appendicitis remain fragmented in basic research, and translation to nursing practice (e.g., pain management, barrier protection) is unaddressed. This analysis aimed to: (1) summarize PKC-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 2019 to December 2024 (to include recent findings).

Search Strategy

Search string: ("Appendicitis"[MeSH Terms] OR "Appendicitis"[All Fields]) AND ("Protein Kinase C"[MeSH Terms] OR "PKC"[All Fields] OR "PKCα"[All Fields] OR "PKCβ"[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: RAW264.7 macrophages, Caco-2 intestinal epithelial cells, IEC-6 cells); (2) studies investigating PKC expression, activation, or inhibition in appendicitis; (3) outcomes including inflammation, epithelial apoptosis, or barrier function.
- **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, PKC detection methods [Western blot, immunohistochemistry (IHC), qPCR, kinase activity assay], key results, nursing-related findings) using a standardized form. Discrepancies were resolved by a third reviewer.

RESULTS

Literature Retrieval Outcomes

Initial search yielded 44 articles. After removing duplicates (n=8) and screening titles/abstracts (n=17 excluded for non-basic research), 19 full-texts were assessed. Nine were excluded (3 reviews, 6 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 RAW264.7/Caco-2 cells). PKC (predominantly PKCα/PKCβ) was detected via Western blot (n=9, measuring total/phosphorylated PKC), IHC (n=7, localizing appendiceal PKC), kinase activity assay (n=6, quantifying PKC catalytic activity), and qPCR (n=5, measuring PKC mRNA).

PKC Activation in Appendicitis

In animal models, PKCα/PKCβ activation (phosphorylated PKC, p-PKC) increased 4–8 hours post-appendicitis induction, peaked at 24 hours: p-PKCα (2.5–4.2-fold increase vs. control), p-PKCβ (2.1–3.8-fold increase), and total PKC activity (2.3–3.9-fold increase)[3,5,7]. IHC showed p-PKCα localization in appendiceal epithelial cells and p-PKCβ in submucosal macrophages—both upregulated in inflamed tissues[4,6]. In LPS-stimulated cells, PKC activation increased in a dose-dependent manner (LPS 0.5–10 μg/mL), with maximum activity at 12 hours[11,12]. Concurrently, PKC-mediated downstream targets (p-NF-κB p65, cleaved caspase-3) increased by 2.2–3.7-fold[3,5].

PKC-Mediated Pathogenic Mechanisms

Eight studies linked PKC activation to inflammation: PKCα/PKCβ enhanced NF-κB signaling, increasing pro-inflammatory cytokines (TNF-α: 2.8–4.1-fold increase, IL-6: 2.3–3.6-fold increase)[3,5,8-10]. Seven studies reported epithelial damage: PKC activation promoted apoptosis (cleaved caspase-3: 2.1–3.5-fold increase) and reduced tight junction proteins (occludin: 1.7–2.4-fold decrease, zonula occludens-1: 1.5–2.2-fold decrease)[4,6,9,11].

PKC Intervention Effects

Four studies tested PKC inhibitors: (1) GF109203X (pan-PKC inhibitor, 1–5 μM) reduced p-PKCα/β by 45–65%, decreased TNF-α by 3.1–4.0-fold, and inhibited epithelial apoptosis[5,9]; (2) LY333531 (PKCβ-specific inhibitor, 5–10 mg/kg) suppressed PKCβ activity by 50–70%, alleviating appendiceal edema and bacterial translocation (E. coli count: 3.3-fold decrease)[10,12]; (3) PKCα siRNA transfection in Caco-2 cells increased occludin by 2.1-fold and reduced cell apoptosis[7]; (4) Curcumin (PKC inhibitor) upregulated anti-inflammatory IL-10 by 2.4-fold and relieved pain-related behaviors[8].

Nursing-Relevant Implications

Three studies provided nursing insights: PKC inhibition reduced abdominal pain (writhing tests: 2.9-fold decrease)[10]; GF109203X decreased sepsis markers (procalcitonin: 2.3-fold decrease)[9]; LY333531 improved intestinal barrier function, supporting early enteral nutrition to suppress PKC overactivation[12].

DISCUSSION

This analysis confirms PKC (PKCα/PKCβ) as a key pro-inflammatory mediator in appendicitis basic models. Consistent findings show PKC activation amplifies inflammation via NF-κB, induces epithelial apoptosis, and disrupts barriers—while inhibition mitigates these effects.

Translation to Nursing

PKC inhibition's pain-relieving effects [10] support nursing use of PKC-targeted adjuvants (e.g., curcumin) for pre-operative pain management. PKC's role in bacterial translocation [9] highlights monitoring of vital signs/procalcitonin in high-risk patients. PKC-mediated barrier damage [12] aligns with nursing guidance on early enteral nutrition to modulate PKC signaling.

LIMITATIONS

All studies used animal/cell models (limited human relevance); only 10 studies were included (small sample); few studies addressed PKC subtypes other than PKCα/PKCβ.

FUTURE DIRECTIONS

Basic research should explore PKC subtypes in human appendiceal cells; clinical nursing studies could test PKC inhibitors (e.g., low-dose GF109203X) on patient outcomes.

CONCLUSION

Basic experimental studies demonstrate PKC (PKCα/PKCβ) promotes inflammatory progression and tissue damage in appendicitis. PKC inhibition alleviates inflammation, pain, and infection risk—providing a molecular basis for nursing interventions (inflammation monitoring, pain management, barrier protection). Bridging basic PKC research and clinical nursing is critical for improving appendicitis care.

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