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Distinctive Molecular Drivers in Fibro-Inflammatory Pathologies: An Integrative Review of FAT10 in Cholestasis and Kisspeptin in Endometriosis

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Abstract

Background: Chronic inflammatory diseases characterized by aberrant tissue remodeling and fibrosis present significant clinical challenges. Two distinct pathologies, cholestatic liver disease and endometriosis, share underlying mechanisms of inflammation-driven tissue injury despite affecting different organ systems. This review explores the molecular underpinnings of these conditions, specifically focusing on the ubiquitin-like protein FAT10 in hepatic contexts and the metastasis-suppressor Kisspeptin in endometriosis.

Methods: We conducted a comprehensive review of recent literature focusing on the pathophysiology of cholestasis, the molecular functions of FAT10, the epidemiology of endometriosis, and the role of Kisspeptin signaling.

Results: In cholestatic liver disease, bile acid accumulation triggers inflammatory cascades leading to fibrosis. Recent evidence identifies FAT10 as a critical regulator in this process, influencing the TGF-β1/Smad and Erk/Egr-1 signaling pathways, which drive hepatic stellate cell activation and carcinogenesis. Conversely, endometriosis is characterized by the invasion of endometrial tissue outside the uterus. The expression of Kisspeptins and their regulation of Matrix Metalloproteinases (MMPs) appears crucial in modulating the invasive capacity of these ectopic lesions, acting similarly to metastasis suppressors in oncology.

Conclusion: While distinct in etiology, both cholestasis and endometriosis involve complex alterations in cell migration and survival signaling. FAT10 serves as a potential biomarker and therapeutic target for liver fibrosis and malignancy, while the Kisspeptin system offers novel insights into controlling the invasive phenotype of endometriosis. Understanding these

molecular drivers provides a foundation for developing targeted therapies that interrupt the progression of fibrosis and tissue invasion.

Keywords: Cholestasis, FAT10, Endometriosis, Kisspeptin, Fibrosis, Ubiquitin-like modifiers, Inflammation.

Introduction

The pathophysiology of chronic disease often converges on the dysregulation of cellular signaling pathways that govern inflammation, tissue repair, and cell migration. Across diverse organ systems, the failure to resolve acute injury leads to chronic inflammation, subsequent fibrosis, and, in many cases, a predisposition to malignancy or invasive growth patterns. This review seeks to bridge the conceptual gap between two seemingly disparate conditions—cholestatic liver disease and endometriosis—by examining specific molecular drivers that characterize their progression: the ubiquitin-like modifier FAT10 and the metastasis-suppressor peptide Kisspeptin, respectively.

Cholestatic liver diseases comprise a spectrum of disorders resulting from the impairment of bile formation or flow. This stagnation leads to the accumulation of toxic bile acids, initiating a cascade of hepatocellular necrosis and apoptosis. As noted by Jansen et al., understanding the ascending pathophysiology of these conditions is critical, as the chronic insult inevitably triggers fibrogenesis [1]. If left unchecked, this process advances to cirrhosis and liver failure. A primer for generalists emphasizes that early recognition of these cholestatic patterns is vital for intervention [2]. However, the molecular machinery driving the transition from simple cholestasis to fibrosis involves complex signaling. Recent attention has turned to the ubiquitin-proteasome system (UPS), specifically the role of Human Leukocyte Antigen-F Adjacent Transcript 10 (FAT10), a protein that becomes upregulated in inflammatory states and cancers [9].

Parallel to the invasive nature of hepatic fibrosis is endometriosis, a gynecological condition defined by the presence of endometrial-like tissue outside the uterus. Endometriosis affects a significant proportion of women of reproductive age and is associated with profound morbidity, including pain and infertility [14]. The epidemiology of endometriosis suggests a complex interplay of genetic, environmental, and hormonal factors [15]. A key feature of endometriotic tissue is its ability to migrate, invade, and establish vascularized lesions in ectopic sites, a behavior that mimics neoplastic metastasis. Here, the focus shifts to Kisspeptin, a peptide product of the KISS1 gene. Originally identified as a metastasis suppressor in melanoma [21], Kisspeptin has emerged as a crucial regulator in the reproductive system, potentially governing the invasiveness of endometrial cells [20].

By juxtaposing the role of FAT10 in the liver with Kisspeptin in endometriosis, this article aims to highlight how distinct molecular markers contribute to the broader themes of tissue remodeling and disease progression. We will synthesize current findings on how FAT10 mediates inflammatory signaling and fibrosis in the liver, and compare this with the regulatory role of Kisspeptin in the adhesion and invasion of endometriotic lesions.

Methods

To provide a comprehensive overview of these molecular mechanisms, a narrative review approach was utilized. Literature was sourced from major biomedical databases including PubMed, Scopus, and Web of Science. The search strategy prioritized peer-reviewed original research and systematic reviews published between 1988 and 2024, with a strong emphasis on literature from the last decade to ensure currency of molecular data.

Keywords included "Cholestasis," "FAT10," "Ubiquitin-like proteins," "Endometriosis," "Kisspeptin," and "Fibrosis." The selection criteria focused on studies that provided mechanistic insights into the signaling pathways involved, specifically those linking molecular expression to clinical pathology or animal model outcomes. Studies discussing the "ascending pathophysiology of cholestatic liver disease" [1] and the "epidemiology of endometriosis" [15] were used to establish the clinical context.

We excluded purely anecdotal case reports and focused on studies that offered quantitative data regarding gene expression, protein interaction, or epidemiological associations. The review is structured to first address the hepatic context involving FAT10, followed by the gynecological context involving Kisspeptin, before synthesizing these findings in a comparative discussion regarding therapeutic implications.

Results

The Pathophysiology of Cholestasis and Hepatic Injury

Cholestasis results from a failure in bile secretion or an obstruction of the biliary tract, leading to the retention of bile acids, bilirubin, and cholesterol. This retention is not merely a mechanical issue but a potent chemical insult to the liver parenchyma. The pathophysiology is described as "ascending," often involving cholangiocytes (bile duct epithelial cells) and hepatocytes [1]. The toxic accumulation of hydrophobic bile acids causes mitochondrial damage and oxidative stress, which in turn activates resident macrophages (Kupffer cells) and recruits circulating inflammatory cells.

Clinical management often relies on ursodeoxycholic acid (UDCA), a hydrophilic bile acid that displaces toxic variants. However, withdrawal of UDCA in patients with conditions like primary sclerosing cholangitis has been shown to lead to rapid biochemical deterioration, underscoring the delicate balance of bile acid homeostasis [4]. When this balance is disrupted chronically, the liver activates a repair response that, when excessive, results in fibrosis. A crucial finding in this domain is the involvement of specific ion transport mediators, identified through immunocytochemistry, which play roles in maintaining epithelial integrity in secretory tissues [3].

FAT10: A Critical Mediator in Liver Inflammation and Fibrosis

Central to the progression of liver injury is the ubiquitin-proteasome system (UPS). Within this system, FAT10 (also known as ubiquitin D or UBD) has emerged as a key player. Unlike ubiquitin, which targets proteins for degradation to maintain homeostasis, FAT10 expression is generally low in normal tissues but is massively induced by pro-inflammatory cytokines such as Tumor Necrosis Factor alpha (TNF-alpha) and Interferon-gamma (IFN-gamma) [6].

Research by Wang et al. (2022) demonstrated that Fluorofenidone, a novel pyridone agent, could ameliorate cholestasis and fibrosis in mice. The mechanism of action was directly linked to the inhibition of hepatic signaling pathways regulated by FAT10 molecules, specifically the Erk/Egr-1 signaling and the Tgfbeta1/Smad pathway [5]. This suggests that FAT10 is not just a passive marker of inflammation but an active participant in the fibrotic cascade. By targeting proteins for degradation or stabilizing them, FAT10 alters the cellular response to injury.

FAT10 itself is a proteasomal degradation signal. Buchsbaum et al. established that FAT10 is regulated by ubiquitination, creating a complex feedback loop within the protein degradation machinery [6]. Furthermore, FAT10 has been shown to interfere with other ubiquitin-like modifiers, such as SUMO (Small Ubiquitin-like Modifier). Aichem et al. found that FAT10 interferes with SUMO activation, thereby altering the sub-cellular localization and stability of SUMOylated proteins [7]. This interference is significant because SUMOylation is often involved in transcriptional repression and stress responses; by disrupting this, FAT10 may promote a more aggressive, proliferative cellular phenotype.

The relevance of FAT10 extends beyond fibrosis to carcinogenesis. In genome-wide integration studies, tissue-specific expression analysis has highlighted the unique profiles of proteins like FAT10 in pathological states [8]. In the context of cancer, FAT10 has been identified as a potential therapeutic target. Zhang et al. noted its role as a cardioprotective factor but also highlighted its overexpression in various cancers [9]. In breast cancer models, cytokines-activated nuclear IKKalpha-FAT10 pathways have been shown to induce tamoxifen resistance, indicating that FAT10 contributes to cell survival under stress [10]. Similarly, in glioma, increased UBD (FAT10) levels serve as diagnostic and prognostic biomarkers [11]. The malignant behavior of ovarian cancer cells has also been linked to the LIN28B/UBD axis, driven by ZNF384, further confirming FAT10's role in promoting aggressive cell behavior [12].

Most pertinently to the liver, FAT10 promotes chemotherapeutic resistance and epithelial-mesenchymal transition (EMT). Zhu et al. demonstrated that FAT10 stabilizes FOXM1 expression in pancreatic cancer, facilitating EMT [13]. This mechanism—EMT—is the biological process where epithelial cells lose their cell polarity and cell-cell adhesion, gaining migratory and invasive properties. This is identical to the process where hepatocytes or cholangiocytes transition into myofibroblasts during liver fibrosis.

Endometriosis: A Disease of Invasion

Switching focus to the reproductive tract, endometriosis represents a benign yet invasive condition. It is characterized by the presence of endometrial glands and stroma outside the uterine cavity. Vercellini et al. describe the pathogenesis as multifaceted, involving retrograde menstruation, coelomic metaplasia, and altered immunity [14]. It is a high-burden disease; Parazzini et al. reviewed the epidemiology, noting significant comorbidities [15].

The disease is not uniform; epidemiologic factors in specific populations, such as East Asia, show variations in presentation [16]. The impact on patients is severe, with systematic reviews highlighting the correlation between endometriosis and depressive/anxiety symptoms, severely affecting quality of life [17]. Furthermore, there is debate regarding whether endometriosis constitutes a systemic disease phenotype, identifying it as a "high-risk population" for other chronic major diseases [18].

Research advances in endometriosis-related signaling have identified multiple pathways involved in inflammation and pain [19]. However, the mechanism by which endometrial tissue invades the peritoneum or ovaries shares similarities with cancer metastasis. This brings us to the role of Kisspeptins.

Kisspeptin and Matrix Metalloproteinases

Kisspeptins are a family of peptides encoded by the KISS1 gene. Originally discovered as a metastasis suppressor gene in melanoma cells [21], its role has expanded. In the context of reproduction, the kisspeptin/kisspeptin receptor system is vital

for implantation and placentation [22]. It regulates the invasion of trophoblasts into the maternal decidua—a controlled invasive process.

In endometriosis, this regulation appears to be disrupted. Kleimenova et al. (2024) investigated the expression of Kisspeptins and Matrix Metalloproteinases (MMPs) in extragenital endometriosis [20]. MMPs are enzymes capable of degrading extracellular matrix proteins, a prerequisite for tissue invasion. The study suggests that an imbalance in Kisspeptin expression fails to suppress MMP activity effectively, thereby allowing the ectopic endometrial tissue to invade surrounding structures. This posits Kisspeptin as a "brake" on invasion that may be defective in women with severe endometriosis.

Discussion

Molecular Convergence in Tissue Remodeling

While cholestasis and endometriosis affect different organ systems, this review highlights a convergence in their molecular behaviors: the maladaptive regulation of cell stability and migration. In the liver, cholestasis creates an inflammatory microenvironment where FAT10 is upregulated. FAT10 then interacts with the 26S proteasome and interferes with SUMOylation [7], leading to the stabilization of pro-inflammatory and pro-fibrotic factors. The link between FAT10 and the TGF-β1/Smad pathway [5] is particularly telling. TGF-β is the master regulator of fibrosis; its unchecked signaling transforms quiescent hepatic stellate cells into collagen-producing myofibroblasts.

In endometriosis, the pathology is driven by the survival and invasion of tissue in a hostile environment (the peritoneum). Here, the failure of metastasis-suppressor mechanisms, specifically the KISS1 system, allows cells to migrate. Just as FAT10 promotes EMT in cancer [13], the lack of effective Kisspeptin signaling allows MMPs to degrade the matrix, facilitating the establishment of endometriotic lesions [20].

Detailed Analysis of the Ubiquitin-Proteasome System (UPS) in Inflammation

To fully appreciate the pathological impact of FAT10 in cholestatic liver disease, one must delve deeper into the mechanics of the Ubiquitin-Proteasome System (UPS). The UPS is the primary intracellular mechanism for the degradation of damaged or unnecessary proteins. Typically, a protein is tagged with a poly-ubiquitin chain, which is recognized by the 26S proteasome, leading to proteolysis. FAT10 (HLA-F adjacent transcript 10) acts as a ubiquitin-like modifier, but with distinct kinetic properties and regulatory outcomes. Unlike ubiquitin, which has a long half-life, FAT10 is rapidly degraded along with its substrate, acting as a "suicide" tag.

The induction of FAT10 by cytokines such as TNF-alpha and IFN-gamma creates a direct link between systemic inflammation and intracellular protein handling. In the context of cholestasis, where bile acids induce a sterile inflammation, the upregulation of FAT10 serves to accelerate the degradation of specific tumor suppressors or cell-cycle regulators. For instance, the stabilization of FOXM1 by FAT10 [13] is a critical insight. FOXM1 is a transcription factor that promotes cell proliferation and resistance to apoptosis. In a healthy liver, hepatocytes must proliferate to repair damage, but this proliferation is tightly controlled. When FAT10 is overexpressed due to chronic cholestatic inflammation, it may cause persistent stabilization of FOXM1, pushing the cell toward an unregulated, fibrotic, or even malignant phenotype.

Furthermore, the interference of FAT10 with SUMOylation [7] represents a sophisticated level of deregulation. SUMOylation usually does not lead to degradation but alters a protein's interaction partners or subcellular localization. By competing with SUMO, FAT10 effectively rewires the cell's stress response. In the bile-acid-injured liver, this could mean that proteins meant to be sequestered in the nucleus for DNA repair are instead targeted for degradation or mislocalized, exacerbating genomic instability and accelerating fibrosis.

The TGF-beta Signaling Nexus

The connection between FAT10 and the TGF-beta/Smad pathway, as elucidated by Wang et al. [5], serves as a cornerstone for understanding hepatic fibrosis. TGF-beta (Transforming Growth Factor-beta) is widely recognized as the most potent pro-fibrogenic cytokine. Upon binding to its receptor, it phosphorylates Smad2 and Smad3, which then complex with Smad4 and translocate to the nucleus to transcribe genes related to extracellular matrix (ECM) production, such as collagens and fibronectin.

In a healthy resolution of injury, this pathway is turned off via negative feedback loops. However, the data suggests that FAT10 interferes with these negative regulators. By inhibiting the degradation of the TGF-beta receptor or by stabilizing the phosphorylated Smads, FAT10 ensures that the fibrogenic signal remains "on." This results in the continuous activation of hepatic stellate cells (HSCs). HSCs are the primary source of collagen in the liver. Under the influence of sustained TGF-beta signaling—bolstered by FAT10—these cells undergo transdifferentiation into myofibroblasts. This is the cellular mechanism that physically scars the liver, leading to the architectural distortion seen in cirrhosis.

The observation that Fluorofenidone can inhibit this axis provides a promising therapeutic template. By disrupting the FAT10-mediated reinforcement of TGF-beta signaling, it is

possible to uncouple inflammation from fibrosis. This implies that future therapies need not necessarily eliminate the inflammation (which is a necessary defense response) but rather target the FAT10-dependent coupling that translates inflammation into permanent scar tissue.

Kisspeptin: Beyond Reproduction to Metastasis Suppression

While FAT10 accelerates pathology in the liver through protein stabilization, the loss or dysfunction of Kisspeptin in endometriosis represents a failure of suppression. The KISS1 gene product was initially identified in melanoma cell lines that had lost the ability to metastasize, hence its classification as a metastasis suppressor. In the context of endometriosis, we must view the endometrial cell as a "quasi-malignant" entity—not in the sense of cancer lethality, but in its behavioral profile of migration and invasion.

The study by Kleimenova et al. [20] highlighting the interplay between Kisspeptins and Matrix Metalloproteinases (MMPs) is pivotal. MMPs are zinc-dependent endopeptidases capable of degrading all kinds of extracellular matrix proteins. For an endometrial cell to implant on the peritoneal wall or the ovary, it must first digest the connective tissue of the host site. In normal physiology, MMPs are tightly regulated by Tissue Inhibitors of Metalloproteinases (TIMPs). Kisspeptin appears to function upstream of this balance.

High levels of Kisspeptin signaling generally result in the downregulation of MMP-9 and MMP-2, two enzymes heavily involved in basement membrane degradation. In women with aggressive endometriosis, it is hypothesized that Kisspeptin expression is reduced or its receptor (KISS1R) is desensitized. This loss of the "molecular brake" allows MMPs to function unchecked, facilitating the deep infiltration of endometriotic lesions. This deep infiltrating endometriosis (DIE) is the most severe form, causing extensive pain and organ dysfunction.

Furthermore, the role of Kisspeptin in placentation [22] offers a developmental parallel. During pregnancy, trophoblasts must invade the uterus to form the placenta—a process that mimics cancer invasion but is physiologically controlled. Kisspeptin is a key regulator that stops this invasion from going too far (which would result in placenta accreta). In endometriosis, this developmental program is reactivated in the wrong place (ectopic tissue) and without the proper regulatory stops. Therefore, therapeutic strategies that re-introduce Kisspeptin agonists could theoretically restore this braking mechanism, limiting the spread of lesions without requiring surgical excision.

Clinical Implications and Therapeutic Horizons

The juxtaposition of these two molecular systems—FAT10 and Kisspeptin—opens new avenues for "cross-disciplinary" therapeutic logic.

1. FAT10 Inhibitors for Fibrosis and Cancer: Given the clear role of FAT10 in stabilizing pro-tumorigenic and pro-fibrotic factors [12, 13], developing small molecule inhibitors of the FAT10-proteasome interaction is a high-priority target. If a drug can block the covalent attachment of FAT10 to its substrates, it could sensitize cancer cells to chemotherapy (reversing the resistance mechanisms described by Chen et al. [10]) and halt the progression of liver fibrosis by dampening TGF-beta signaling. Currently, proteasome inhibitors like bortezomib exist, but they are non-specific and have high toxicity. A FAT10-specific inhibitor would offer a targeted approach for chronic liver disease and inflammation-associated cancers.

2. Kisspeptin Agonists for Endometriosis: Current treatments for endometriosis rely heavily on hormonal suppression (inducing a pseudo-menopause) or surgery. These have significant side effects and recurrence rates. If the invasiveness of endometriosis is driven by low Kisspeptin signaling, then the administration of stable Kisspeptin analogs could serve as a non-hormonal (in terms of estrogen suppression) method to limit disease progression. By upregulating the metastasis-suppressor function, the lesions would lose their ability to invade new tissue, potentially leading to regression or stabilization of the disease. This is supported by the findings on KiSS-1 suppression of metastasis in breast carcinoma [21], suggesting a broad applicability of this pathway in controlling invasive cell phenotypes.

3. Biomarker Utility: Both molecules offer diagnostic potential. FAT10 levels in serum or tissue biopsy could serve as a predictor of how fast a patient with cholestasis might progress to cirrhosis, or as a prognostic marker in glioma [11]. Similarly, profiling Kisspeptin and MMP levels in menstrual effluent or peritoneal fluid could help stratify endometriosis patients by risk of deep infiltration, allowing for more personalized surgical planning.

Limitations

It is important to acknowledge the limitations in the current body of literature. Much of the detailed molecular work on FAT10 is derived from murine models [5] or in vitro cancer cell lines [13]. While these models are essential for dissecting pathways, the translation to human liver disease is complex due to the heterogeneity of human genetic backgrounds and environmental exposures. Similarly, endometriosis research is hampered by the lack of a perfect animal model that spontaneously develops the disease as humans do. The retrograde

menstruation theory [14] explains peritoneal implants but struggles to explain distant sites, suggesting that stem cell trafficking or lymphatic spread may also be involved, mechanisms that need further exploration in the context of Kisspeptin signaling.

Additionally, the dual role of inflammation must be considered. While chronic inflammation drives these pathologies, acute inflammation is necessary for repair. Complete suppression of inflammatory mediators (like NF-kappaB, which regulates FAT10) can lead to immunodeficiency. Therefore, therapies targeting these pathways must be nuanced, aiming to modulate rather than ablate the signal.

Conclusion

The molecular landscapes of cholestatic liver disease and endometriosis, while anatomically distinct, share a common narrative of aberrant tissue remodeling driven by dysregulated signaling. In the liver, the ubiquitin-like modifier FAT10 acts as an accelerant, linking bile acid toxicity to the fibrotic TGF-beta pathway and promoting cell survival in a way that predisposes to cancer. In endometriosis, the failure of the Kisspeptin signaling system allows for unchecked enzymatic degradation of the extracellular matrix, facilitating the invasive nature of the disease.

This comparative review underscores the importance of looking beyond the organ of origin to the molecular machinery of the cell. Both pathologies represent a loss of cellular discipline—hepatocytes refusing to die and instead becoming fibrotic, and endometrial cells refusing to stay in place and instead invading the peritoneum. Targeting these specific molecular drivers—FAT10 and Kisspeptin—offers the promise of precision medicine that moves beyond symptom management to address the root causes of disease progression. As research advances, the integration of these molecular insights into clinical practice holds the potential to significantly improve outcomes for patients suffering from these chronic, debilitating conditions.

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