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Follistatin-like Protein 3 in Colorectal Cancer: Linking Immune Evasion to Treatment Resistance

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ABSTRACT

Colorectal cancer (CRC) remains a significant global health challenge, characterized by high morbidity and mortality. Despite advances in surgical techniques, chemotherapy, targeted therapies, and immunotherapy, many CRC cases exhibit treatment resistance and immune evasion, necessitating the identification of novel therapeutic targets. Follistatin-like protein 3 (FSTL3) has recently emerged as a key regulator in CRC progression, influencing immune suppression and therapy resistance. FSTL3 modulates the tumor microenvironment by promoting epithelial-mesenchymal transition (EMT), sustaining β-catenin signaling, and stabilizing c-Myc, which collectively enhance tumor invasiveness and metastatic potential. Additionally, FSTL3 contributes to immune evasion by upregulating immune checkpoint molecules such as programmed death-ligand 1 (PD-L1) and indoleamine-2,3-dioxygenase 1 (IDO1), thereby suppressing cytotoxic T-cell activity. High FSTL3 expression correlates with poor prognosis and resistance to conventional chemotherapy, targeted agents, and immune checkpoint inhibitors. Given its pivotal role in CRC pathophysiology, FSTL3 represents a promising biomarker for disease prognosis and a potential therapeutic target. Future research should focus on developing FSTL3-targeted interventions, including monoclonal antibodies, small-molecule inhibitors, and combination strategies with immunotherapy. Understanding the precise molecular mechanisms underlying FSTL3-mediated tumor progression and immune escape will be essential for translating these insights into clinical applications.

Keywords: Colorectal cancer; Immune evasion; FSTL3; Treatment resistance; Tumor microenvironment

INTRODUCTION

Colorectal cancer (CRC) is one of the most common malignancies and is the second leading cause of cancer-related death worldwide.[1] This disease progresses through the accumulation of genetic mutations and

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complex interactions with the tumor microenvironment. In particular, the immunological tumor microenvironment plays a pivotal role in CRC progression and patient outcome.[2] Immunotherapy has emerged as a groundbreaking treatment modality for many cancers, including CRC, by harnessing the immune system to attack tumors.[3] Immune checkpoint blockade (ICB) therapies targeting inhibitory checkpoints such as programmed cell death protein 1 (PD-1), programmed death-ligand 1 (PD-L1), and cytotoxic T-lymphocyte-associated protein 4 (CTLA-4) have shown remarkable success in certain subsets of CRC patients.[4] However, only a minority of CRC cases—primarily those with high microsatellite instability (MSI-H)—derive substantial benefit from ICB, while microsatellite stable (MSS) tumors, which comprise ~85% of CRC, often do not respond. Even among MSI-H CRC patients, nearly half exhibit resistance to ICB therapy.[5-7] This resistance is thought to stem from immune evasion mechanisms such as poor tumor antigen presentation, T cell exhaustion, and immunosuppressive signaling within the tumor microenvironment.[2] Therefore, understanding how CRC tumors evade immune surveillance is critical for improving immunotherapy outcomes. It is equally important to identify factors driving resistance to standard treatments like chemotherapy and to find novel therapeutic targets.

Follistatin-like 3 (FSTL3) has recently gained attention as a potential mediator of immune evasion and treatment resistance in CRC. FSTL3 is a secreted glycoprotein (also known as follistatin-related gene, FLRG) that belongs to the follistatin family and can bind and neutralize activin, myostatin, and other members of the transforming growth factor-beta (TGF-β) superfamily.[8] It is produced by various normal tissues (including adipose tissue, reproductive organs, pancreas, liver, muscle, and placenta) and is involved in physiological processes such as reproductive aging and metabolic homeostasis.[9] For example, FSTL3 helps regulate glucose and lipid metabolism, and it can induce lipid accumulation and inflammatory cytokine release in macrophage-derived foam cells.[10] In the context of cancer, aberrant FSTL3 expression has been observed in multiple tumor types. Studies have shown that FSTL3 is overexpressed in lung, kidney, and gastric cancers, where it promotes tumor growth and angiogenesis and correlates with poor patient survival.[11-13] Emerging evidence indicates a similar pattern in colorectal cancer: FSTL3 levels are significantly higher in CRC tumors compared to normal tissues, and elevated FSTL3 is associated with advanced disease (such as lymph node metastasis) and worse prognosis in CRC patients.[14-16] These findings suggest that FSTL3 contributes to CRC aggressiveness. However, the specific roles of FSTL3 in modulating the immune microenvironment and influencing therapeutic responses in CRC have only begun to be elucidated. Below, we review how FSTL3 facilitates immune evasion and treatment resistance in colorectal cancer, and we discuss new opportunities to leverage this knowledge for improved therapy.

FSTL3 and Immune Evasion in CRC

An effective anti-tumor immune response requires adequate tumor antigen presentation, robust cytotoxic T cell activity, and the absence of overwhelming immunosuppressive signals. CRC tumors often develop ways to escape immune destruction, and recent studies implicate FSTL3 as a key player in this immune evasion. High FSTL3 expression in CRC has been linked to an immunosuppressive tumor microenvironment. For instance, bioinformatic and histological analyses have shown that FSTL3 overexpression correlates with increased infiltration of immunosuppressive cells and markers of T cell dysfunction. In one study, Yang et al (2021) found that CRC cases with elevated FSTL3 had a higher presence of certain stromal and immune components associated with immune suppression, including M2-polarized tumor-associated macrophages and cancer-associated fibroblasts, along with signs of T cell exhaustion.[15] This suggests that FSTL3-rich tumors create a microenvironment hostile to effective T cell activity. The same study noted that FSTL3 overexpression was linked to lymph node metastasis and a worse prognosis, underlining the connection between immune escape and disease progression.[15] Importantly, the authors proposed that FSTL3 might drive an "inhibitory immune microenvironment" by promoting macrophage and fibroblast polarization toward pro-tumoral phenotypes and inducing T cell exhaustion, thereby facilitating tumor immune evasion.[15]

Mechanistic insights into how FSTL3 suppresses anti-tumor immunity have come from recent laboratory studies.[16] Li et al (2024) provided direct evidence that FSTL3 expression by tumor cells enables immune escape and resistance to immunotherapy in CRC.[17] In CRC cell and animal models, hypoxia (a common feature of tumor cores) was shown to upregulate FSTL3

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via the hypoxia-inducible factor 1-alpha (HIF1α) pathway, linking FSTL3 to the hypoxic tumor microenvironment.[17] Once elevated, FSTL3 acts on tumor cells to enhance their immunosuppressive capabilities. Specifically, FSTL3 was found to bind directly to the transcription factor c-Myc within CRC cells, preventing c-Myc from being ubiquitinated and degraded. Stabilized c-Myc then upregulates expression of immune inhibitory molecules, notably PD-L1 (programmed death-ligand 1) and IDO1 (indoleamine-2,3-dioxygenase 1).[17] PD-L1 on tumor cells engages PD-1 on T cells to shut down T cell activity, and IDO1 catabolizes tryptophan to create a metabolically suppressive environment for T cells—both are well-known mechanisms of tumor immune evasion. By inducing PD-L1 and IDO1, FSTL3 effectively helps CRC cells turn off cytotoxic T lymphocytes and promote T cell exhaustion in the tumor milieu. Consistent with this, knockout of FSTL3 in CRC cells had the opposite effect: it led to increased infiltration of functional $CD8^+$ T cells in tumors, reduced levels of exhausted ($PD\text{-}1^+$) T cells and regulatory T cells, and an overall shift toward an anti-tumor immune microenvironment.[17] In vivo, tumors lacking FSTL3 were significantly more susceptible to immune clearance, especially when combined with PD-1 blockade therapy. In immunocompetent mouse models of CRC, loss of FSTL3 greatly improved the efficacy of anti-PD-1 treatment, indicating that FSTL3 was a major factor limiting the response to immunotherapy.[17] These findings clearly position FSTL3 as a mediator of immune evasion: it enhances immunosuppressive signaling (via PD-L1/IDO1 and immune cell reprogramming) and thereby allows tumor cells to escape immune attack.

Additional evidence supports the immunoregulatory role of FSTL3. High FSTL3 expression has been associated with lower numbers of infiltrating effector T cells in some analyses, and in other cancer types like gastric cancer, FSTL3 overexpression correlated with increased M2 macrophage infiltration in tumors.[13] These patterns reinforce the notion that FSTL3 helps skew the tumor microenvironment toward immune tolerance rather than immune activation. Together, current data indicate that FSTL3 contributes to CRC immune evasion by both cell-intrinsic mechanisms (upregulating immune checkpoint molecules) and cell-extrinsic effects (shaping the composition and behavior of immune and stromal cells in the tumor niche). This dual influence makes FSTL3 a particularly compelling target of interest in the context of tumor immunity.

FSTL3 and Treatment Resistance in CRC

Beyond its role in suppressing anti-tumor immunity, FSTL3 appears to promote resistance to various cancer therapies in CRC. Treatment resistance can refer to the failure of immunotherapies as well as conventional treatments like chemotherapy. Because immune evasion often underlies immunotherapy failure, there is some overlap between FSTL3's impact on immune escape and on resistance to ICB therapy. As described above, FSTL3 enables CRC cells to resist PD-1 checkpoint inhibition by maintaining an immunosuppressive microenvironment; correspondingly, patients with FSTL3-overexpressing tumors tend to have poorer responses to anti-PD-1 therapy. Indeed, retrospective analyses of clinical data suggest that CRC patients whose tumors have high FSTL3 expression are more likely to be non-responders to PD-1 blockade, whereas those with low FSTL3 have better chances of benefiting from immunotherapy.[17] This correlation underscores FSTL3 as a marker and mechanism of immunotherapy resistance.

In addition, emerging evidence links FSTL3 with resistance to chemotherapy and other standard treatments. Tumors that evolve to a more aggressive, mesenchymal state often become less responsive to chemotherapeutic drugs. FSTL3's functions in CRC, such as promoting EMT (epithelial-mesenchymal transition), invasion, and metastasis, can indirectly contribute to chemoresistance. Several studies have shown that FSTL3 drives EMT and cellular plasticity in CRC, changes that are known to confer resistance to apoptosis and drug-induced cell death. For example, Li et al. (2021) demonstrated that FSTL3 activation of the β-catenin signaling pathway leads to EMT and enhanced aerobic glycolysis in CRC cells, fueling their migratory and invasive capabilities.[14] Similarly, Liu et al. (2021) found that high FSTL3 expression promotes EMT through interactions with extracellular matrix components (such as fibronectin) and integrin signaling, further supporting metastasis and aggressive behavior.[16] These EMT-related effects of FSTL3 suggest that CRC cells with elevated FSTL3 might better withstand chemotherapy, as EMT is associated with reduced drug uptake and evasion of drug-induced senescence or death. In line with this, Yang et al (2021) noted an association between high FSTL3 levels and chemoresistance in

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CRC patients.[15] Although clinical data on FSTL3 and chemotherapy outcomes are still limited, the observation was that patients with tumors overexpressing FSTL3 had poorer responses to standard chemotherapeutic regimens, implying an innate or acquired resistance.[15] This could be due to the FSTL3-driven changes in tumor cell phenotype (like EMT and stemness) that make them less susceptible to cytotoxic drugs.

Interestingly, the same study by Yang et al. also suggested that tumors with high FSTL3 might be more "immunotherapy-sensitive," meaning those tumors exhibited molecular features that could potentially make them respond to immunotherapy despite being chemoresistant.[15] This initial prediction was somewhat surprising and contrasted with the later experimental evidence by Li et al, which indicated FSTL3 actually impairs immunotherapy efficacy.[17] One possible interpretation is that FSTL3-high tumors in Yang et al.'s cohort had significant immune infiltration (hence appearing immunotherapy-responsive on a genomic level), but the quality of that immune infiltrate was suppressive rather than effective. Thus, while bioinformatic indicators might have pointed to immunotherapy sensitivity, functional assays revealed that FSTL3 was, in fact, facilitating immune escape. These seemingly conflicting observations highlight the complexity of FSTL3's role and suggest that more research is needed to fully decipher how FSTL3 status affects different treatment modalities.

Overall, current evidence indicates that FSTL3 not only helps CRC tumors evade immune destruction but also contributes to a more treatment-resistant phenotype. By driving metastasis and possibly maintaining cancer stem cell-like properties (via EMT and metabolic reprogramming),[14,16] FSTL3-overexpressing tumors are harder to eliminate with conventional therapies. Additionally, by inducing an immune-cold environment, FSTL3 causes resistance to immunotherapies. Therefore, FSTL3 serves as a nexus between tumor aggressiveness and therapy resistance in CRC. Recognizing FSTL3's influence on these processes opens up new avenues to counteract resistance mechanisms.

New Opportunities and Future Directions

The recognition of FSTL3's multifaceted role in CRC progression, immune evasion, and treatment resistance provides several new opportunities to improve patient outcomes. First, FSTL3 itself is a promising biomarker for risk stratification and therapeutic decision-making. Given the correlation of high FSTL3 with advanced disease and poor prognosis, testing for FSTL3 expression in CRC tumors could help identify patients with more aggressive biology.[14-16] These patients might benefit from closer monitoring or adjuvant therapies. Moreover, since FSTL3-rich tumors are associated with immunosuppressive microenvironments and ICB resistance, FSTL3 could serve as a predictive biomarker for immunotherapy responsiveness. For instance, patients with MSS CRC (typically not ICB candidates) but low FSTL3 might have a more inflamed tumor microenvironment and could potentially be considered for immunotherapy trials, whereas those with high FSTL3 might need combination approaches to render immunotherapy effective. In the study by Li et al., tumor FSTL3 levels had a strong predictive value for anti-PD-1 treatment outcomes, raising the possibility that FSTL3 expression could be used alongside MSI status and other markers to guide immunotherapy use.[17] Validation in clinical cohorts will be important, but this represents a new diagnostic opportunity.

Beyond its value as a biomarker, FSTL3 is an attractive therapeutic target in its own right. As a secreted protein that influences both cancer cells and the surrounding stroma, FSTL3 can potentially be targeted by drugs (such as neutralizing antibodies or ligand traps) to disrupt its function. If FSTL3 is neutralized, one would expect reversal of its pro-tumor effects: restored activin/TGF-β family signaling, reduced c-Myc activity, lower PD-L1 and IDO1 levels, and a reinvigorated immune response against the tumor. While no FSTL3-specific inhibitor is yet available in routine practice, preclinical studies provide proof-of-concept that inhibiting FSTL3 can suppress cancer progression. In CRC models, silencing or knocking out FSTL3 led to markedly slower tumor growth and fewer metastases, and it enhanced the efficacy of PD-1 blockade therapy as discussed earlier.[17] Likewise, experiments in other cancer types have yielded encouraging results: In renal cell carcinoma, for example, blocking FSTL3 (via short hairpin RNA [shRNA]-mediated knockdown) significantly reduced cancer cell proliferation and metastatic potential by affecting the glycogen synthase kinase-3 beta (GSK-3β)/β-catenin pathway.[11] In lung cancer models, downregulation of FSTL3 (achieved by targeting an upstream long noncoding RNA that

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increases FSTL3) curbed tumor cell proliferation and migration.[12] These findings across cancer types underline a general principle that targeting FSTL3 can inhibit tumor progression and suggest that therapies against FSTL3 might have broad anti-cancer applications.

In CRC specifically, there are several potential strategies to exploit FSTL3 for therapy. One approach is to develop a monoclonal antibody or soluble receptor that binds FSTL3, preventing it from interacting with its partners (like activin or cell-surface receptors on tumor cells). By neutralizing FSTL3, such a therapy could dismantle the protective niche that CRC tumors build – for instance, decreasing immunosuppressive macrophage polarization and fibroblast activation, and allowing T cells to penetrate and destroy the tumor. Combining an FSTL3 inhibitor with immune checkpoint blockade might be particularly powerful: the FSTL3 inhibitor would "release the brakes" on the immune system by mitigating tumor-induced immune suppression, while anti-PD-1 or anti-CTLA-4 would further unleash T cell activity. This combination could convert currently unresponsive CRC tumors into ones that can be attacked by the immune system. Another strategy is to target the upstream regulators or downstream effectors of FSTL3. The Yes-associated protein 1 (YAP1) oncogenic pathway was identified as a driver of FSTL3 expression in CRC; thus, using YAP1 inhibitors (some of which are in development) could indirectly suppress FSTL3 and thereby reduce metastasis and therapy resistance.[14] Downstream, since FSTL3 boosts c-Myc and consequently PD-L1/IDO1, conventional approaches like c-Myc inhibitors or IDO1 inhibitors might partly mimic FSTL3 blockade effects and are already being explored in clinical trials for cancer.[17] For example, small-molecule IDO1 inhibitors have been tested to overcome immunotherapy resistance; combining those with checkpoint blockade in FSTL3-high tumors might improve outcomes. Additionally, given FSTL3's role in binding activins and myostatin, modulating the TGF-β family signaling might offer another avenue: freeing these ligands could potentially restrain tumor cell growth or re-differentiate tumor cells, although careful consideration is needed since TGF-β family members can have tumor-promoting or suppressing effects depending on context.

Finally, the insights into FSTL3's role open up new research directions. Future studies should evaluate FSTL3 in clinical biospecimens to correlate its expression with patient responses to various treatments, beyond the initial findings. It will be important to test emerging FSTL3-targeted therapies in preclinical CRC models, especially combination regimens (for instance, FSTL3 inhibition plus chemotherapy or immunotherapy) to see if they can prevent metastasis or overcome drug resistance. There is also interest in exploring whether circulating FSTL3 (since it's secreted, it might be detectable in blood) could serve as a minimally invasive biomarker for monitoring disease status or response to therapy. In summary, targeting the FSTL3 pathway represents a novel opportunity to intervene in the deadly interplay of immune evasion and treatment resistance in colorectal cancer.

CONCLUSION

FSTL3 has emerged as a significant contributor to colorectal cancer malignancy, bridging two critical challenges in oncology: immune evasion and therapy resistance. By sculpting an immunosuppressive microenvironment and promoting tumor-intrinsic survival pathways, FSTL3 allows CRC cells to escape immune attack and endure standard treatments. These discoveries, supported by multiple lines of evidence,[15,17] not only advance our understanding of CRC pathogenesis but also reveal actionable vulnerabilities. FSTL3 stands out as both a biomarker and a potential therapeutic target–its high expression signals aggressive, treatment-refractory disease, yet also offers a focal point for novel interventions to dismantle tumor defenses. Ongoing and future research will clarify how best to inhibit FSTL3 or its network, and clinical trials will be needed to test the safety and efficacy of such strategies. Optimistically, therapies targeting FSTL3 (or integrating FSTL3 status into treatment planning) could improve responses to immunotherapy, prevent metastasis, and ultimately enhance survival for CRC patients who currently face limited options. Harnessing this new knowledge about FSTL3 could open a fresh chapter in the fight against colorectal cancer, converting an agent of immune escape into a vulnerability that clinicians can exploit for better patient outcomes.

STATEMENT OF ETHICS

Not applicable.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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