Colon Cancer Xenograft Models:

Subcutaneous, Orthotopic, And Metastatic Xenograft Tumor Models

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Xenograft and Allograft Models Advance Preclinical Research in Colon Cancer

Colon cancer or colorectal cancer (CRC) remains one of the most prevalent and lethal malignancies worldwide, representing a significant burden in both developed and developing nations. Despite advances in screening strategies and therapeutic regimens, CRC continues to pose challenges due to its molecular heterogeneity, frequent metastasis, and variable responses to treatment. A key barrier to therapeutic progress is the lack of reliable preclinical models that faithfully recapitulate the complexity of human CRC biology, including tumor-stroma interactions, metastatic dissemination, and treatment resistance mechanisms. Traditional in vitro systems offer limited insight into these dynamic tumor processes, underscoring the necessity for in vivo models that better simulate the human tumor microenvironment.

Among preclinical models, xenograft and allograft transplantation systems have emerged as essential tools for investigating CRC pathogenesis and evaluating novel therapeutics. Xenografts, typically generated by transplanting human CRC cell lines or patient-derived tumor fragments into immunodeficient mice, provide a platform to study human-specific oncogenic signaling, drug responsiveness, and tumor progression. Allografts, by contrast, involve the transplantation of murine-derived CRC cells into syngeneic hosts and enable the interrogation of tumor-immune system interactions within an immunocompetent context. These complementary models have contributed to delineating critical pathways involved in CRC growth and metastasis and have been instrumental in the preclinical validation of targeted therapies, immunotherapies, and combination regimens. However, persistent limitations such as inter-model variability, immune incompatibility in xenografts, and genetic divergence from human tumors in allografts highlight the need for ongoing refinement.

The objective of employing xenograft and allograft models in CRC research is to bridge the translational gap between molecular discovery and clinical application. This study aims to evaluate and characterize the utility of several colon cancer xenograft systems, including cell line-derived and patient-derived models, in recapitulating tumorigenic behavior and treatment response. The rationale for this approach lies in the pressing need to establish physiologically relevant models that more accurately reflect human disease, thereby enhancing the predictive validity of preclinical findings. By providing comparative insights into tumor growth dynamics, histopathological features, and therapeutic responsiveness, this research seeks to contribute to the optimization of in vivo models for colorectal cancer. The broader impact lies in enabling more precise and accelerated development of anticancer agents, ultimately informing clinical strategies and improving patient outcomes.

Subcutaneous Colon Cancer Xenograft Models

Subcutaneous xenograft transplantation remains one of the most widely utilized models in preclinical oncology due to its operational simplicity, reproducibility, and compatibility with a range of experimental manipulations. This model involves the injection of tumorigenic cell lines into the subcutaneous space, typically in immunodeficient rodents, enabling accessible and non-invasive monitoring of tumor growth and response to therapeutics. In colorectal cancer research, subcutaneous xenografts have proven indispensable for evaluating drug efficacy, resistance mechanisms, and tumorigenic capacity. A diverse array of human and murine colorectal cancer cell lines has been employed in this context, including COLO205, CT-26, DLD-1, HCT-15, HCT116, HT29, KM-12, LoVo, LS174T, MC38, RKO, SW480, SW620, and WiDr. These models allow researchers to interrogate distinct oncogenic pathways and therapeutic vulnerabilities across a spectrum of genetic backgrounds, enhancing the generalizability of preclinical findings.

Each of these cell lines offers unique characteristics relevant to colorectal cancer pathophysiology. For example, HCT116 and SW480 harbor activating mutations in KRAS, making them suitable for investigating resistance to EGFR-targeted therapies. DLD-1 and HCT-15 provide models for exploring alterations in DNA mismatch repair and APC signaling. HT29 and WiDr, both derived from well-differentiated adenocarcinomas, support studies on tumor proliferation and differentiation. LoVo and LS174T are frequently used to study metastatic potential, while KM-12 has been instrumental in modeling liver metastasis. MC38 and CT-26, as murine-derived lines, are applied in immunocompetent hosts to investigate immune responses, including checkpoint blockade and tumor vaccine approaches. RKO, with its microsatellite instability and wild-type KRAS and BRAF, offers a distinct model for testing responses to immunomodulatory agents. The utility of COLO205 and SW620 lies in their application in drug screening and metastatic research, respectively. Collectively, these subcutaneous xenograft models offer an adaptable and scalable platform for assessing therapeutic efficacy, biomarker expression, and resistance development. Although limited in their ability to replicate organ-specific microenvironments or immune-tumor interactions in human cancers, their integration with orthotopic, syngeneic, and humanized systems enables a comprehensive approach to colorectal cancer model development, supporting translational oncology efforts.

Orthotopic Colon Cancer Xenograft Models

Orthotopic xenograft transplantation has become a pivotal model in colorectal cancer research due to its ability to replicate the tumor's native anatomical environment. By implanting human or murine tumor cells directly into the colon or rectum of host animals, this method enables the study of site-specific tumor development, invasion, and metastasis with greater fidelity than subcutaneous models. It offers a more accurate representation of the tumor microenvironment, including local stromal interactions, angiogenesis, and immune cell recruitment. Orthotopic models also facilitate the observation of spontaneous metastatic spread to organs commonly affected in patients. Cell lines including DLD-1, HCT-15, HT29, SW480, and SW620 have been extensively used in this context to investigate oncogenic signaling pathways, therapy resistance, and metastatic behavior. RKO and COLO205 models have contributed to the understanding of microsatellite instability and BRAF-driven tumor dynamics, while CT26 and MC38, both murine-derived, are frequently used in syngeneic systems to evaluate immunotherapy agents in immunocompetent hosts.

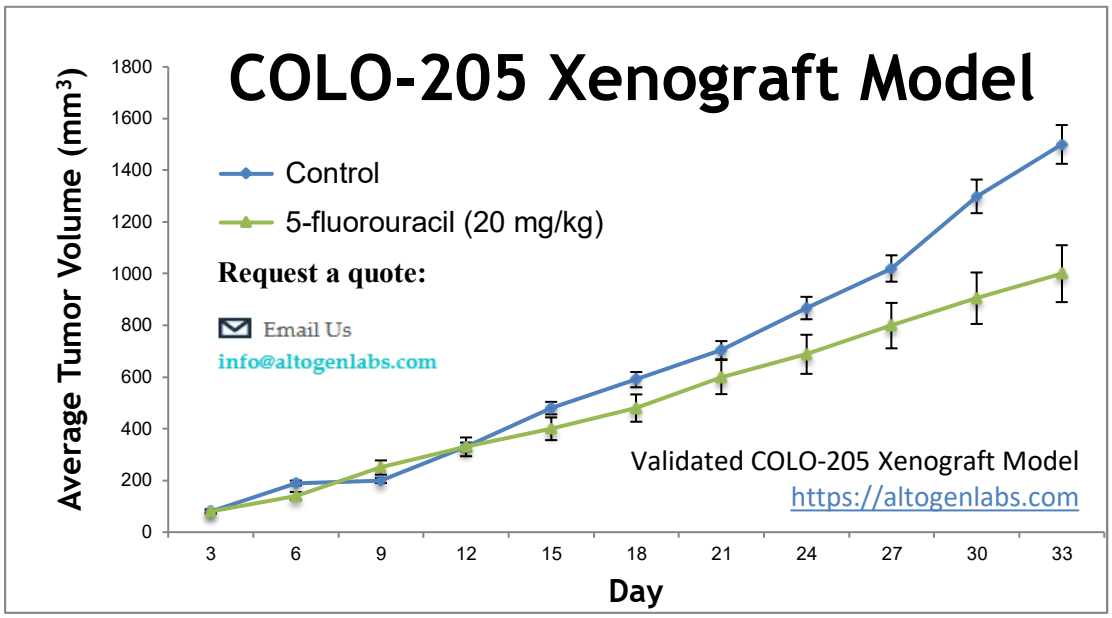
Recent advancements in orthotopic modeling have improved implantation techniques, enhanced tumor take rates, and enabled more robust monitoring through the use of bioluminescent or fluorescent reporters. These developments allow for non-invasive tracking of tumor growth and dissemination, as well as longitudinal assessment of drug efficacy. Orthotopic models are increasingly integrated with humanized mouse systems to better approximate human immune responses, providing a platform for testing immune checkpoint inhibitors and neoantigen-targeted therapies. Despite being more technically challenging than subcutaneous models, the orthotopic approach offers greater biological relevance and is critical for understanding the spatial and systemic dynamics of colorectal cancer progression. When used alongside subcutaneous and metastatic models, orthotopic xenografts form an essential component of a comprehensive preclinical strategy, supporting the development of more effective and clinically translatable cancer therapies.

Metastatic Colon Cancer Xenograft Models

Metastatic xenograft transplantation represents a critical advancement in preclinical modeling, providing a means to investigate the mechanisms underlying cancer dissemination and to evaluate therapeutic strategies targeting metastatic disease. Unlike subcutaneous or orthotopic models, metastatic xenografts are specifically designed to study the secondary spread of tumor cells to distant organs, a process that accounts for the majority of cancer-related mortality. These models can be established through intravenous, intrasplenic, intracardiac, or orthotopic inoculation of tumor cells, each method offering a distinct pattern of metastatic colonization. For colorectal cancer, liver and lung metastases are of particular clinical relevance, and xenograft systems have been developed to simulate these processes with increasing fidelity. The ability to track metastatic progression and therapeutic response in real time, especially using bioluminescent or fluorescent cell lines, has significantly enhanced the utility of these models in translational research.

A range of human and murine colorectal cancer cell lines have been employed to develop metastatic xenograft models. CT26 and MC38 are widely used in immunocompetent mice to examine immune-mediated regulation of metastasis and to evaluate immunotherapeutic interventions. HCT-116 and HT29 have been utilized to study hepatic metastasis following intrasplenic or portal vein injection, capturing key features of tumor cell extravasation, survival, and proliferation in the liver microenvironment. LoVo and SW620, derived from metastatic lesions, display inherent metastatic potential and are frequently applied in experimental metastasis models to investigate pathways involved in epithelial-mesenchymal transition, angiogenesis, and chemoresistance. SW480, though originally derived from a primary tumor, is often compared with SW620 to explore progression-associated genetic and phenotypic changes. WiDr, with its well-characterized growth kinetics, has been included in studies evaluating anti-metastatic compounds and metastatic niche interactions. Collectively, these models offer valuable insights into the molecular drivers of metastasis and provide a platform for assessing targeted therapies, combination regimens, and biomarkers predictive of metastatic progression. The integration of metastatic xenograft transplantation into preclinical workflows is essential for advancing therapeutic strategies aimed at preventing or eradicating disseminated disease.

Characterization and Preclinical Application of the COLO205 Xenograft Model in Colon Cancer Research



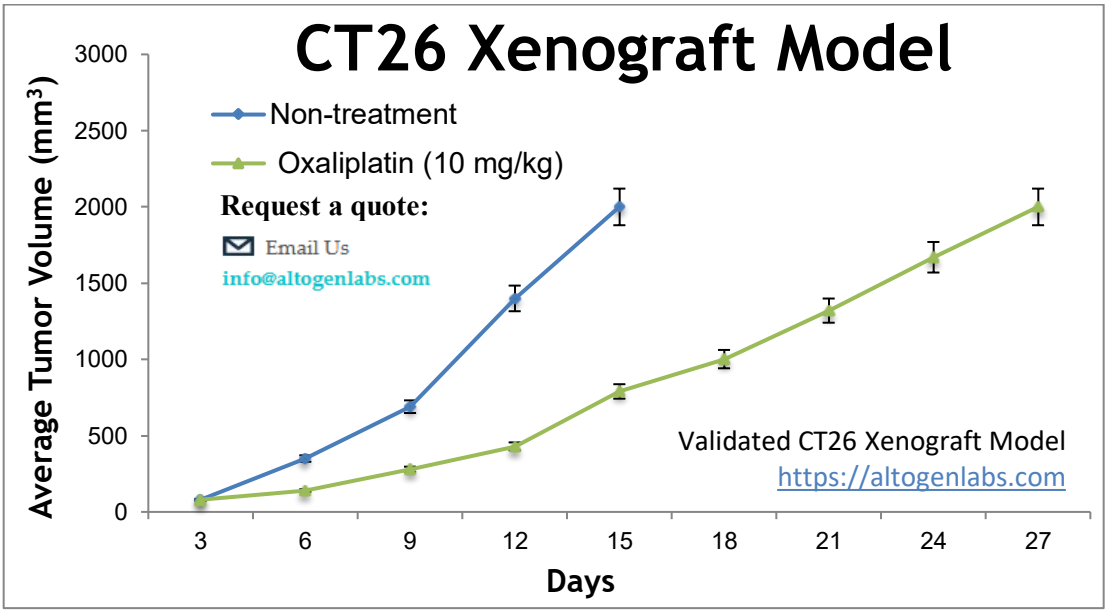
The COLO205 xenograft model, available from Altogen Labs, serves as a robust and highly reproducible platform for studying colorectal cancer (CRC) biology, therapeutic response, and resistance mechanisms. Derived from the ascitic fluid of a colorectal adenocarcinoma patient, the COLO205 cell line harbors a homozygous BRAF^V600E mutation and remains wild-type for KRAS and TP53. This genetic profile makes it particularly relevant for preclinical studies investigating BRAF-targeted therapies, EGFR inhibitors, and combination regimens that address MAPK pathway feedback activation and PI3K/AKT engagement. COLO205 cells exhibit strong apoptotic responses and are well-suited for dissecting mechanisms of programmed cell death, drug sensitivity, and metabolic adaptation.

Altogen Labs offers validated COLO205 subcutaneous and orthotopic xenograft models, performed in immunocompromised BALB/c nude mice under GLP-compliant and IACUC-regulated conditions. In subcutaneous models, 1×10^6 COLO205 cells suspended in Matrigel are injected into the flank, and tumors are measured using digital calipers until reaching a defined size for treatment initiation. Orthotopic models enable transplantation into the cecal submucosa, supporting localized tumor development that more accurately mimics the anatomical and microenvironmental features of human CRC. While distant metastases are infrequent, these models facilitate the study of early invasion and localized therapeutic responses. Collected tissues are preserved for histology, molecular profiling, and gene expression analysis.

This model has supported a wide array of translational research studies. Targeted TRAIL therapy using dimerized fusion proteins significantly enhanced apoptosis and tumor regression in COLO205-bearing mice, demonstrating up to 100-fold increased potency over conventional ligands. The model also enabled investigation of mitotic disruption using CENP-E inhibitors, revealing tight correlation between spindle checkpoint activation and antitumor efficacy. COLO205 xenografts have shown particular utility in anti-angiogenic studies; dual inhibition of VEGF-A and Ang-2 resulted in superior tumor suppression and reduced metastasis compared to monotherapies. Furthermore, combination treatment with 5-fluorouracil and Tanshinone IIA significantly decreased tumor burden and downregulated multiple resistance-associated proteins, including P-gp, VEGF, and MMP-7.

Folate receptor alpha (FRα)-mediated TP53 activation has also been demonstrated in this model, offering a unique approach for modulating oncogenic signaling and cell cycle arrest. COLO205's responsiveness to nutrient-regulated signaling further supports its value in metabolic and biomarker discovery. Altogen Labs provides comprehensive COLO205 xenograft study support, including tumor growth inhibition, pharmacokinetics, immunohistochemistry, survival analysis, and customized experimental design. Detailed information, including a downloadable technical datasheet, can be accessed at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/colo-205-xenograft-model/.

Characterization and Preclinical Application of CT26 Allograft Model in Colon Cancer Research



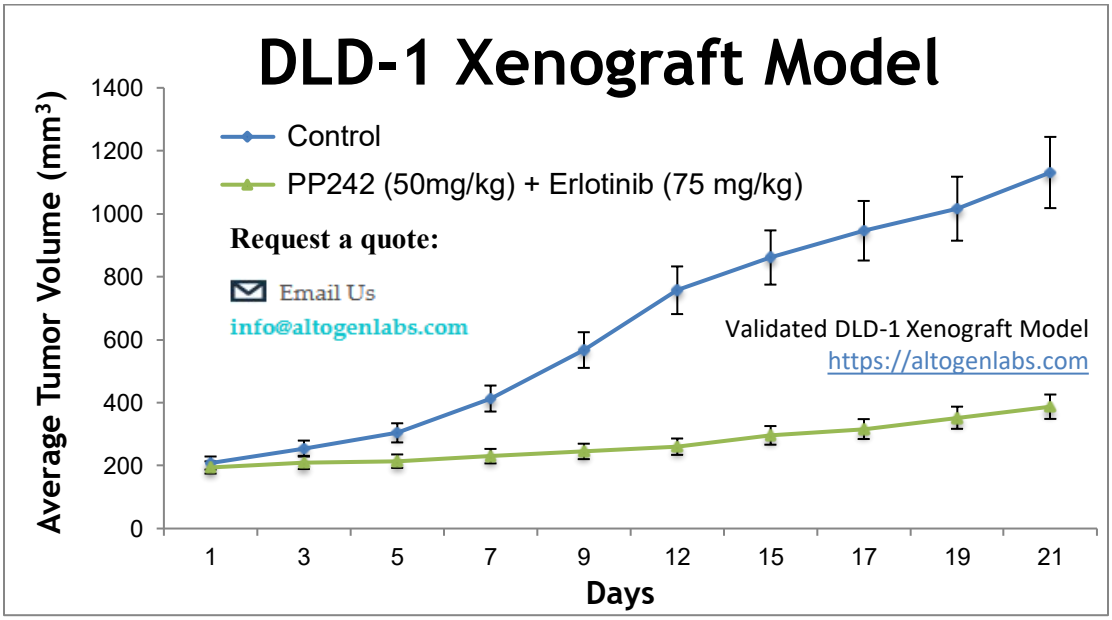
The CT26 murine colon carcinoma model offered by Altogen Labs is a well-established syngeneic allograft system used extensively in immuno-oncology research to evaluate the therapeutic efficacy of anti-cancer agents within an immunocompetent host. Originating from chemically induced colorectal tumors in BALB/c mice, CT26 cells exhibit a KRAS^G12D mutation and are characterized by a highly immunogenic tumor microenvironment. This includes robust infiltration of cytotoxic T lymphocytes, macrophages, and myeloid-derived suppressor cells. Unlike xenograft models in immunodeficient mice, the CT26 system preserves native immune-tumor interactions, enabling accurate preclinical assessment of immune checkpoint inhibitors, oncolytic virotherapies, and mRNA-based cancer vaccines.

Altogen Labs provides a validated CT26 model with subcutaneous, orthotopic, and metastatic configurations. In the subcutaneous setup, exponentially growing CT26 cells are injected into the hind flanks of BALB/c mice, and tumor volume is monitored using digital calipers until the desired size is reached for treatment initiation. Orthotopic models involve implantation into the cecal wall, offering anatomical relevance and enabling the study of local tumor invasion and immune modulation within the gastrointestinal tract. Metastatic variants utilize intravenous or orthotopic injection to facilitate secondary tumor formation, particularly in the lungs. Additionally, Altogen employs luciferase-expressing CT26 cells for real-time imaging and non-invasive monitoring of tumor progression.

The model has been instrumental in advancing targeted therapies, including the use of ATR inhibitors in mismatch repair-deficient (MMR-d) tumors, and demonstrating synthetic lethality when combined with immune checkpoint blockade. Studies have also shown the efficacy of decylubiquinone via SIRT2 upregulation, and innovative immunotherapy delivery systems using engineered bacterial vectors targeting the CD47/SIRPα axis. Furthermore, peptide inhibitors against KRAS^G12D mutations have demonstrated cytostatic effects in vivo, reinforcing the model's translational utility for early-stage drug development.

Altogen Labs supports a wide range of customizable preclinical studies using the CT26 model, including tumor growth delay, pharmacokinetics, survival analysis, and immune cell profiling. Services include multiple administration routes (e.g., intravenous, intraperitoneal, oral, intratumoral) and advanced imaging and molecular analysis techniques. The CT26 allograft model can be accessed on the Altogen Labs website at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/ct26-xenograft-model/.

Characterization and Preclinical Application of the DLD-1 Xenograft Model in Colon Cancer Research



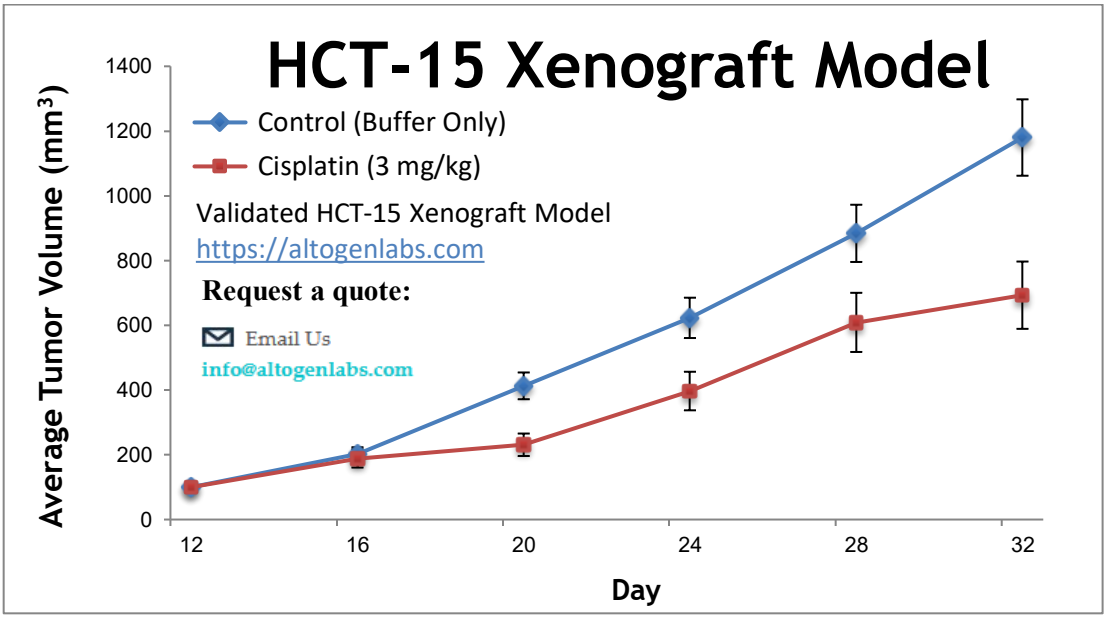
The DLD-1 xenograft model developed and validated by Altogen Labs represents a robust and translationally relevant system for the in vivo investigation of colorectal cancer. Originating from a human colorectal adenocarcinoma classified as Dukes' type C, the DLD-1 cell line harbors hallmark mutations in KRAS (G13D), TP53 (R273H), and APC, aligning it with the chromosomal instability subtype of colorectal cancer. This microsatellite stable line exhibits classical epithelial morphology and expresses key tumor-associated antigens such as carcinoembryonic antigen (CEA) and colon antigen 3. Widely employed in studies of drug resistance, oncogenic signaling, and therapeutic efficacy, the DLD-1 model is a cornerstone for evaluating targeted therapies, including MEK, PARP, ATR inhibitors, and combinations involving mTOR and EGFR pathway inhibitors.

Altogen Labs offers both subcutaneous and orthotopic configurations of the DLD-1 xenograft model, each enabling nuanced study designs tailored to specific experimental goals. Subcutaneous implantation permits reproducible tumor monitoring and high-throughput assessment of tumor growth inhibition, while orthotopic implantation into the cecal wall recapitulates native tumor–host interactions, enhancing the physiological relevance of therapeutic evaluation. Bioluminescent variants of DLD-1, such as DLD-1-luc, facilitate real-time imaging and longitudinal tracking of tumor progression. Experimental endpoints supported by Altogen Labs include tumor growth delay, survival analysis, blood chemistry, histopathology, and molecular profiling via RT-qPCR and Western blotting.

Recent case studies using the DLD-1 model have elucidated mechanisms of feedback resistance in kinase-targeted therapy and epigenetic silencing of WNT signaling via Withanolide F. Additionally, the model has been employed to validate apoptotic responses to novel compounds like MM-129 and investigate dual-pathway inhibition strategies involving AKT and WNT. These studies highlight the utility of DLD-1 for preclinical evaluation of combination therapies and transcriptional reprogramming strategies in KRAS-mutant, WNT-activated colorectal cancers.

Altogen Labs performs all in vivo procedures under IACUC guidelines and GLP compliance, with standardized protocols for animal acclimatization, cohort randomization, tumor measurement, and endpoint analyses. Researchers may select from multiple administration routes, including oral gavage, intravenous, intraperitoneal, and intratumoral injection, allowing for comprehensive modeling of therapeutic delivery. The DLD-1 xenograft model is accessible via the Altogen Labs website under the colon cancer xenograft section specifically at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/dld-1-xenograft-model/.

Characterization and Preclinical Application of the HCT-15 Xenograft Model in Colon Cancer Research

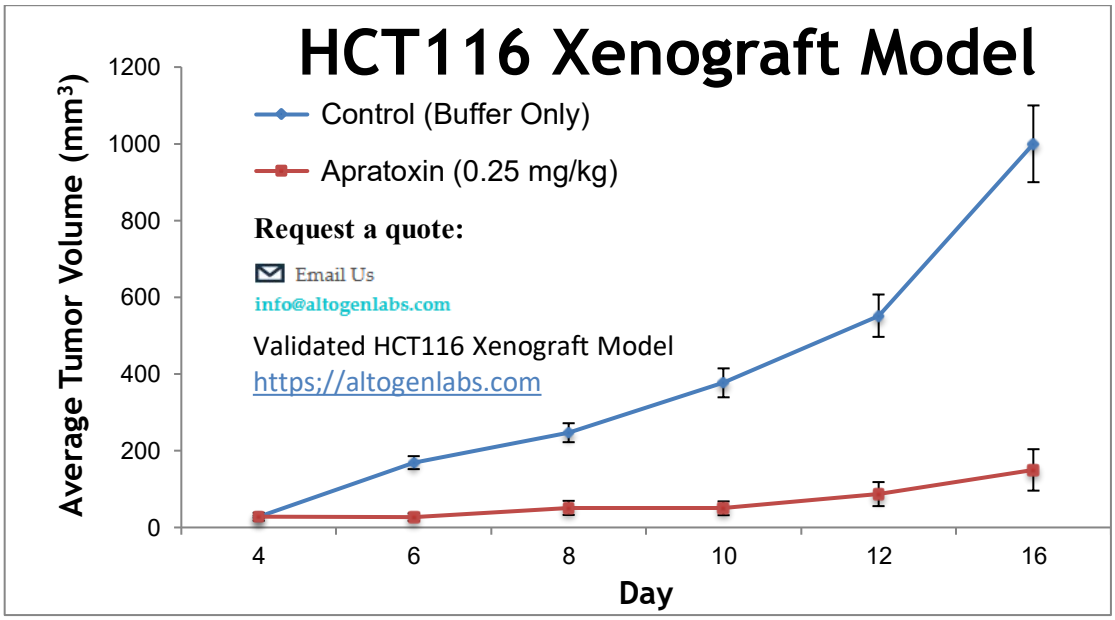


The HCT-15 xenograft model, developed and validated by Altogen Labs, represents a robust and physiologically relevant system for studying colorectal cancer in vivo. Derived from a human colorectal adenocarcinoma, the HCT-15 cell line is distinguished by its oncogenic KRAS mutation and inactivation of TP53, features that drive uncontrolled cell proliferation and resistance to standard therapies. These cells display a hyperdiploid karyotype with pronounced chromosomal aberrations and exhibit aggressive biological traits such as high tubulin expression, enhanced filopodia formation, and reduced actin stability. This phenotype contributes to increased motility, epithelial morphogenesis in 3D cultures, and adaptability under stress conditions. Preclinical models employing HCT-15 are particularly useful for investigating mechanisms of chemoresistance, including resistance to 5-fluorouracil and irinotecan. Moreover, recent data demonstrate that therapeutic agents like halofuginone and melatonin can reverse resistance by targeting non-coding RNAs or autophagy pathways, respectively. These insights position HCT-15 as a highly informative system for identifying biomarkers of resistance and testing combination therapies.

Altogen Labs offers both subcutaneous and orthotopic xenograft models using HCT-15 cells, enabling flexible modeling of localized or metastatic colorectal cancer. In the subcutaneous model, ten million viable HCT-15 cells suspended in Matrigel are injected into the flank of immunocompromised mice, with tumor growth monitored until experimental endpoints are reached. Orthotopic implantation into the cecum provides enhanced biological fidelity, supporting investigations of invasion, stromal interactions, and drug efficacy in a native microenvironment.

These models are integrated with advanced molecular analyses, including mRNA quantification by RT-PCR and protein expression profiling via the ProteinSimple WES system. Targeted inhibition of the IL-11/GP130/STAT3 signaling axis has demonstrated potent antitumor effects in HCT-15 models, particularly in combination with agents such as oxaliplatin. Detailed information, including a downloadable technical datasheet, can be accessed at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/hct-15-xenograft-model/.

Characterization and Preclinical Application of the HCT-116 Xenograft Model in Colon Cancer Research

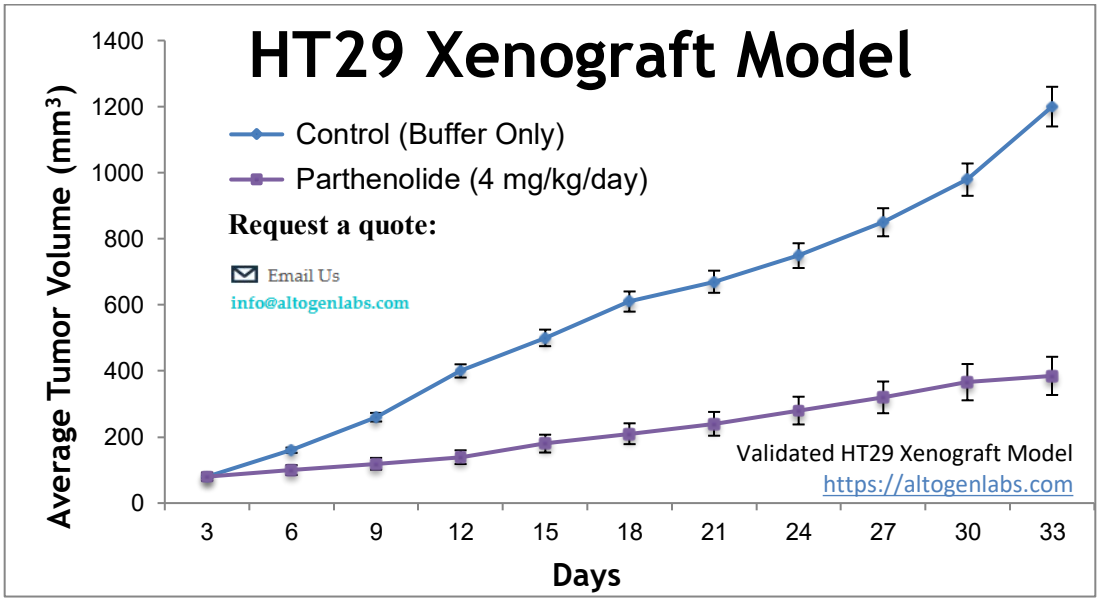


The HCT-116 xenograft model, validated and offered by Altogen Labs, is a versatile and physiologically relevant platform for investigating colorectal cancer progression, therapeutic response, and drug resistance. Derived from a human male with colorectal carcinoma, HCT-116 cells are defined by their microsatellite instability-high (MSI-H) phenotype and loss of mismatch repair function due to MLH1 inactivation. These cells harbor a KRAS G13D mutation while retaining wild-type p53, making them ideal for examining oncogenic signaling, apoptosis, and DNA damage response pathways. This model has significantly contributed to preclinical oncology by enabling the evaluation of chemotherapeutics, such as 5-fluorouracil, irinotecan, and oxaliplatin, and more recently, targeted agents including MEK and PARP inhibitors. However, limitations in traditional in vitro systems underscore the necessity of in vivo models to study tumor-stroma and immune interactions, particularly as MSI-H tumors have shown variable responses to immune checkpoint blockade.

Altogen Labs provides validated subcutaneous, orthotopic, and metastatic HCT-116 xenograft models under IACUC-compliant, GLP conditions. Subcutaneous transplantation involves the injection of one million viable cells mixed with Matrigel into immunodeficient mice, allowing for reproducible tumor formation within 7 to 10 days. Tumor progression is monitored using digital calipers, with defined endpoints based on tumor volume. Orthotopic implantation into the colonic wall enables the study of tissue-specific invasion, angiogenesis, and metastasis, while metastatic models facilitate the investigation of systemic tumor dissemination and therapeutic efficacy against secondary lesions. These models are augmented by advanced molecular analyses, including RT-PCR and capillary immunoassays using the ProteinSimple WES system. Additionally, Altogen Labs supports genetic manipulation of HCT-116 cells, including stable overexpression or RNA interference-based knockdown of genes of interest. The HCT-116 model is particularly valuable for assessing the impact of tumor suppressors such as SLC22A18 and evaluating the modulatory effects of botanical extracts like Ganoderma lucidum and Lagopsis supine.

For full details, the HCT-116 xenograft model page is available at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/hct116-xenograft-model/.

Characterization and Preclinical Application of the HT29 Xenograft Model in Colon Cancer Research

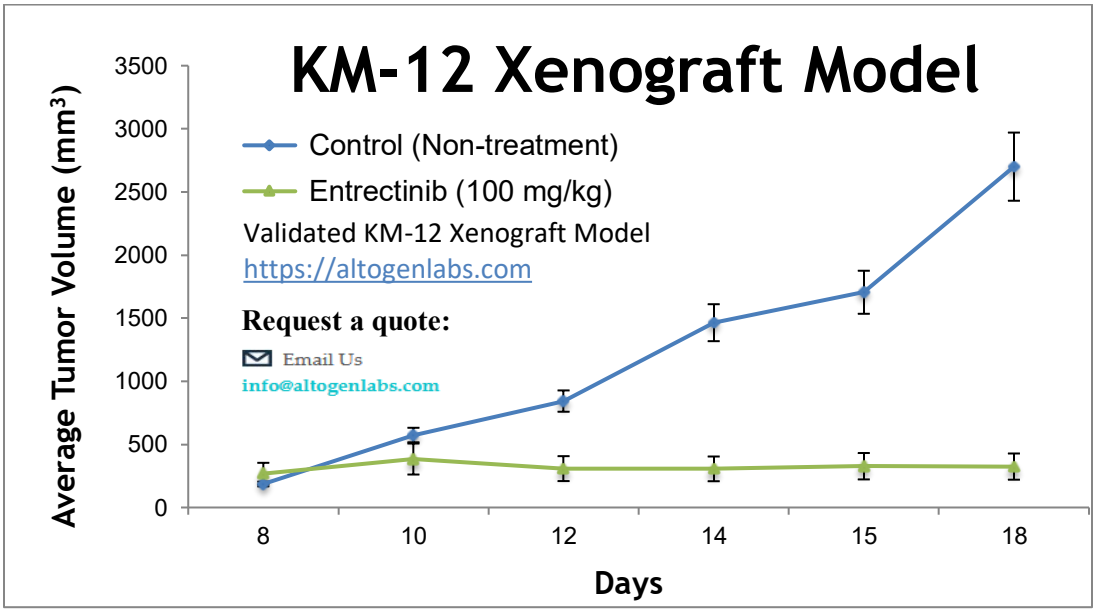


The HT29 xenograft model, validated by Altogen Labs, provides a comprehensive and translationally relevant platform for preclinical studies of colorectal cancer. Derived from a human colorectal adenocarcinoma, HT29 cells harbor a combination of oncogenic mutations, including APC, TP53, PIK3CA, and the BRAF V600E variant, while retaining wild-type KRAS. This distinct genetic profile contributes to the cell line's moderate responsiveness to conventional chemotherapeutics such as 5-fluorouracil, oxaliplatin, and irinotecan. HT29 cells are notable for their epithelial morphology, capacity for differentiation, and application in studies of drug delivery and epithelial barrier function. Despite their widespread use, limitations persist in fully modeling tumor microenvironmental factors, particularly those related to immune checkpoint regulation and hypoxia-driven therapy resistance. Recent investigations have focused on enhancing the model's translational utility by integrating immune and stromal elements, as well as evaluating its behavior in orthotopic and metastatic contexts.

Altogen Labs offers a validated HT29 xenograft model utilizing subcutaneous, orthotopic, and metastatic transplantation strategies to simulate various stages of colorectal cancer. Subcutaneous models involve injection of one million viable HT29 cells suspended in Matrigel into immunodeficient mice, followed by regular monitoring of tumor progression using digital calipers. Orthotopic implantation into the cecum or rectal wall supports anatomically accurate tumor formation and spontaneous metastasis, while metastatic models allow assessment of tumor dissemination to the liver and lungs via intrasplenic or intravenous routes. These models enable rigorous evaluation of antitumor agents, mechanistic studies of metastasis, and analysis of tumor-intrinsic signaling pathways. Altogen Labs supports these studies with comprehensive services, including histology, gene and protein expression analysis, and generation of RNAi or overexpression cell lines.

The HT29 model has been pivotal in validating therapeutic candidates such as ENMD-2076 and lactoferrin, as well as uncovering oncogenic functions of ZNF277 and resistance mechanisms involving HER3 and redundant downstream signaling. Additional technical resources include in vivo imaging, toxicology profiling, and molecular biomarker discovery. The HT29 xenograft model is available through Altogen Labs at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/ht29-xenograft-model/.

Characterization and Preclinical Application of the KM-12 Xenograft Model in Colon Cancer Research

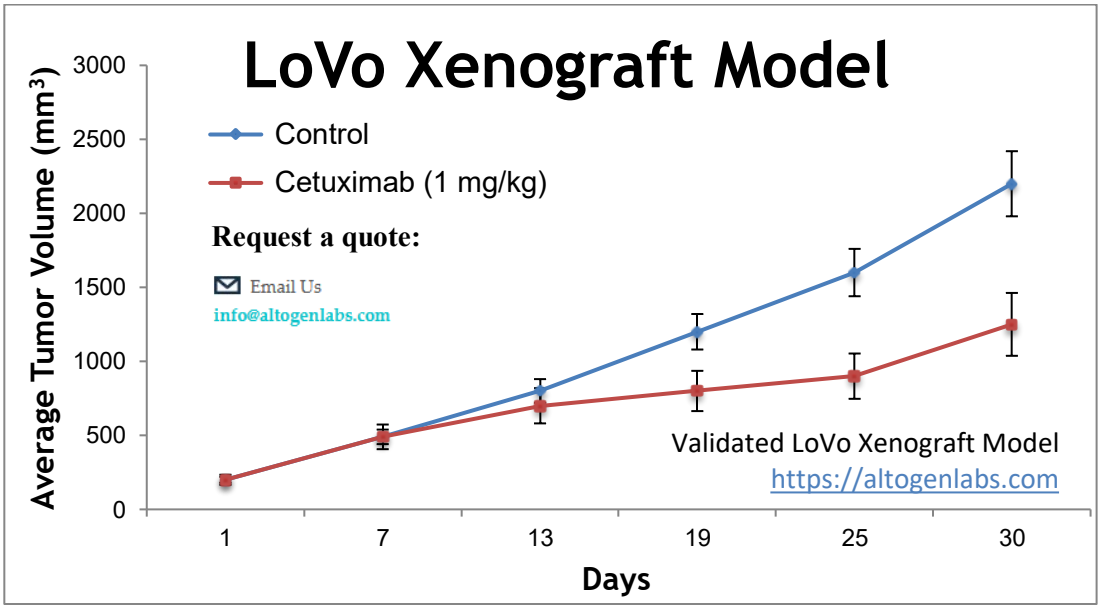


The KM-12 xenograft model, validated and offered by Altogen Labs, represents a high-value platform for the preclinical study of BRAF V600E-mutant, KRAS wild-type colorectal cancer. KM-12 cells originate from a human colorectal adenocarcinoma classified as Dukes B2 and exhibit hallmark oncogenic features, including constitutive activation of MAPK signaling, TPM3-NTRK1 gene fusion, and aberrations in cell migration and PI3K/AKT signaling. These attributes make the model particularly well-suited for evaluating the efficacy of targeted therapies such as BRAF, MEK, and TRK inhibitors, as well as identifying mechanisms of resistance, including those driven by metabolic rewiring through the mevalonate pathway. While traditionally underutilized in 3D and orthotopic systems, KM-12 xenografts remain foundational for in vivo drug screening, biomarker development, and mechanistic investigations.

Altogen Labs conducts KM-12 xenograft studies under IACUC-regulated, GLP-compliant protocols using subcutaneous implantation of one million viable cells suspended in Matrigel into immunodeficient mice. Tumor growth is tracked by caliper measurement until reaching predefined volumes, at which point animals are randomized into treatment cohorts. Tumors are subsequently analyzed for weight, histopathology, and molecular profiles. This model has been pivotal in evaluating compounds such as entrectinib, merestinib, AZD4547, and zurletrectinib, which target TRK fusions and overcome acquired resistance mutations.

Additional investigations have demonstrated the potential for dual-inhibition strategies and metabolic co-targeting with statins to improve treatment durability. KM-12 xenografts have also been applied in imaging studies using [18F]TRACK PET tracers to quantify TRKA activity in vivo. Altogen Labs supports these studies with a full suite of services including RNAi-mediated gene silencing, RT-PCR and WES-based protein analysis, and optional endpoints such as blood chemistry and immunohistochemistry. The KM-12 xenograft model is accessible on the Altogen Labs website at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/km-12-xenograft-model/.

Characterization and Preclinical Application of the LoVo Xenograft Model in Colon Cancer Research

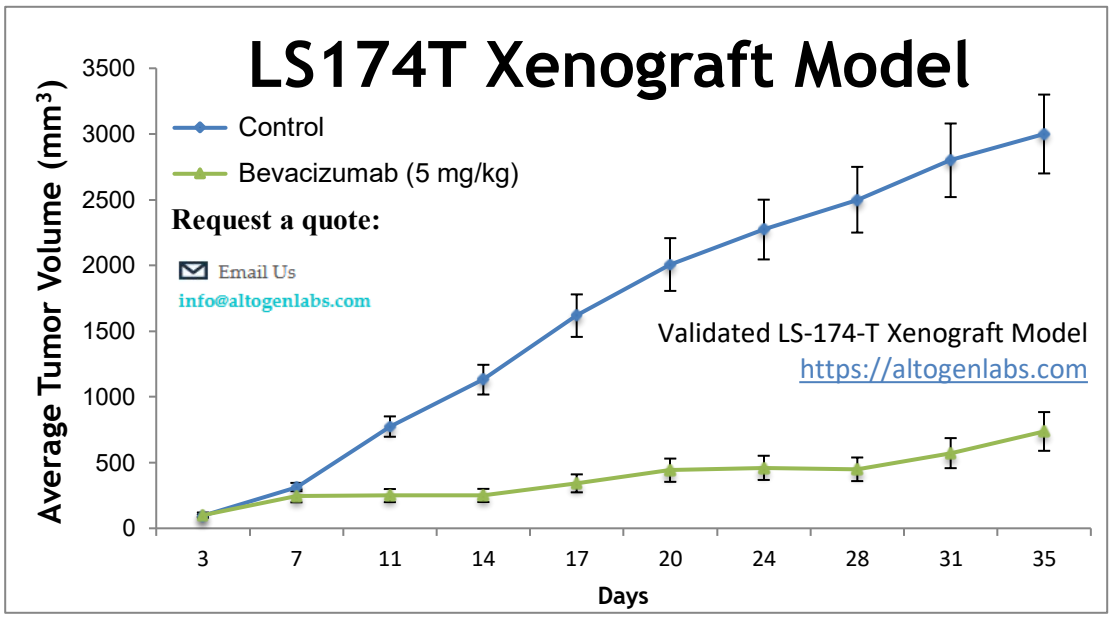


The LoVo xenograft model, validated by Altogen Labs, is a clinically relevant platform for preclinical studies of colorectal cancer, particularly in the context of chemoresistance, immune modulation, and tumor metastasis. Derived from a metastatic colorectal adenocarcinoma, LoVo cells exhibit microsatellite instability-high (MSI-H) status, a KRAS G13D mutation, and wild-type BRAF, enabling mechanistic investigations into therapeutic resistance and immune evasion. LoVo tumors demonstrate overexpression of thymidylate synthase and ATP-binding cassette transporters following exposure to 5-fluorouracil, and display epithelial-mesenchymal transition features in response to TGF-β, suggesting dynamic phenotypic plasticity. These characteristics, combined with their aggressive angiogenic potential and variable response to anti-angiogenic therapies, make LoVo xenografts an indispensable model for studying vascular biology and tumor heterogeneity in colorectal cancer.

Altogen Labs offers validated subcutaneous and metastatic LoVo xenograft models under IACUC-compliant and GLP-certified conditions. In the subcutaneous model, one million viable LoVo cells are suspended in 50% Matrigel and injected into athymic BALB/c mice. Tumor progression is measured using digital calipers, and experimental treatments are administered based on predefined tumor volume thresholds. Tumor tissues are collected at endpoint for histology, gene and protein expression profiling, and pathway analysis. The metastatic model involves intravenous injection of LoVo cells to simulate hematogenous dissemination, particularly to the lungs, although immune-deficient conditions limit study of immune responses. This model is suitable for testing agents that target metastatic progression and resistance pathways. LoVo xenografts have been extensively applied in therapeutic evaluation of cetuximab, VEGFR2 inhibitors such as apatinib, and nanoparticle-based delivery systems like PEG-PBLG-encapsulated hydroxycamptothecin. Additionally, LoVo studies have elucidated mechanisms of action involving apoptosis induction through survivin suppression, MET-STAT3 pathway activation following LGR5 loss, and the role of TELO2 in mTORC2 signaling.

The comprehensive utility of the LoVo model is further supported by Altogen Labs' integrated services including gene editing, RT-PCR, WES-based proteomics, and custom in vivo study design. The LoVo xenograft model is accessible through Altogen Labs at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/lovo-xenograft-model/.

Characterization and Preclinical Application of the LS174T Xenograft Model in Colon Cancer Research

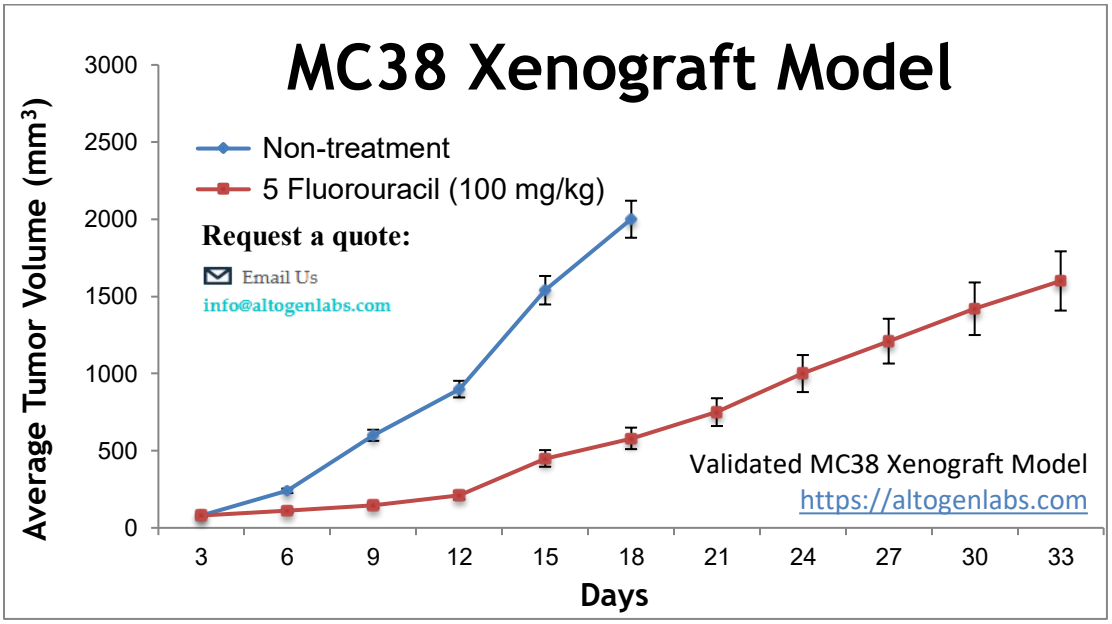


The LS174T xenograft model, offered by Altogen Labs, is a validated preclinical tool for investigating mucinous colorectal adenocarcinoma and evaluating the efficacy of anticancer therapeutics in vivo. Originating from a human colorectal tumor, LS174T cells are characterized by high MUC2 secretion, a KRAS G12D mutation, inactivation of TP53, and dysregulation of Wnt signaling due to APC mutations. These molecular features contribute to their partially differentiated epithelial phenotype and relevance in studying intestinal mucosal biology, epithelial plasticity, and therapeutic resistance. LS174T tumors recapitulate mucin-rich architecture in vivo, providing an appropriate context for examining drug penetration, immune exclusion, and microenvironmental barriers in colorectal cancer.

In the Altogen Labs subcutaneous xenograft model, one million viable LS174T cells are suspended in Matrigel and injected into immunodeficient mice, with tumor formation monitored via digital calipers. Once tumors reach 50 to 150 mm³, animals are randomized into treatment groups. Tumor tissue is collected for histopathological and molecular analyses including mRNA and protein expression profiling. The model supports a range of therapeutic evaluations, including monoclonal antibodies, small molecule inhibitors, and combination therapies. Notably, studies utilizing bevacizumab have demonstrated selective inhibition of tumor growth, while investigations into antibody distribution highlight the structural limitations of mucinous tumors in achieving uniform therapeutic exposure. Imaging and biodistribution studies using engineered anti-CEA scFv-SNAP-tag fusion proteins further illustrate the potential for pretargeted molecular imaging and therapeutic targeting in this model.

Altogen Labs conducts all xenograft studies in GLP-compliant and IACUC-regulated facilities, providing clients with customizable protocols, comprehensive reports, and optional services such as RNA/protein extraction, immunohistochemistry, and toxicity profiling. The LS174T xenograft model is particularly suitable for examining mucin-related immune evasion, drug delivery challenges, and molecular response signatures, making it a valuable system for advancing colorectal cancer therapeutics. For further information, the LS174T model is available on the Altogen Labs website at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/ls174t-xenograft-model/.

Characterization and Preclinical Application of the MC38 Xenograft Model in Colon Cancer Research

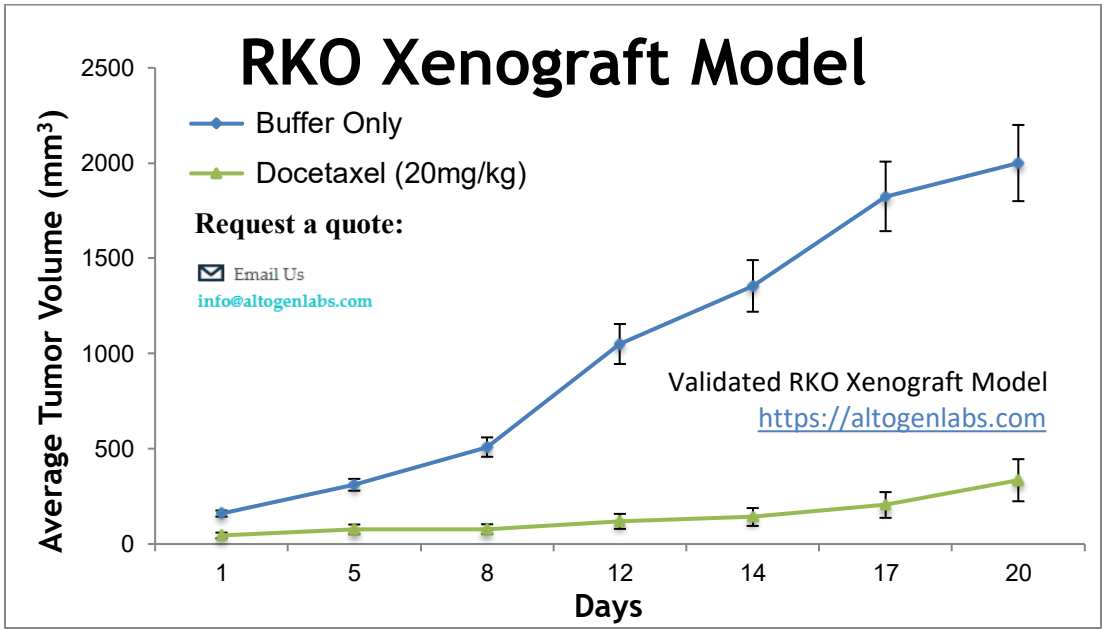


The MC38 xenograft model, offered by Altogen Labs, is a robust and extensively validated platform for studying colorectal cancer within an immunocompetent context. Derived from murine colon adenocarcinoma in C57BL/6 mice, MC38 cells are highly immunogenic, making them exceptionally valuable for immuno-oncology research. This model is widely used for evaluating immune checkpoint inhibitors, such as PD-1 and CTLA-4 blockade, as well as for exploring combination therapies that include radiotherapy, chemotherapeutics, metabolic inhibitors, or cytokine modulation. MC38 tumors demonstrate significant infiltration by cytotoxic $CD8^+$ T cells and are responsive to various immunotherapeutic strategies, enabling detailed analyses of immune activation, tumor metabolism, and resistance mechanisms.

Altogen Labs provides subcutaneous, orthotopic, and metastatic MC38 xenograft models under GLP-compliant and IACUC-regulated protocols. Subcutaneous implantation involves injecting one million viable MC38 cells, suspended in Matrigel, into athymic BALB/c mice, with tumor growth monitored by caliper measurements. For metastasis studies, intrasplenic injection is employed to establish hepatic lesions, while orthotopic implantation into the colonic wall replicates primary tumor formation and natural metastatic spread. Each model supports longitudinal assessment of tumor growth, immune cell infiltration, cytokine expression, and treatment efficacy. Additionally, the MC38 model has been instrumental in demonstrating immune-dependent chemotherapeutic effects, such as those mediated by oxaliplatin, and in identifying synergistic immune activation through combined cytokine therapy with IL-2 and IL-33.

Comprehensive support services are available through Altogen Labs, including molecular characterization using real-time qPCR and WES-based protein quantification, as well as development of engineered or RNAi-modified cell lines. The MC38 model is a critical resource for examining costimulatory immune therapies, metabolic-immune interactions, and long-term immune memory, especially within orthotopic and metastatic tumor environments. The MC38 xenograft model can be accessed on the Altogen Labs website at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/mc38-xenograft-model/.

Characterization and Preclinical Application of the RKO Xenograft Model in Colon Cancer Research



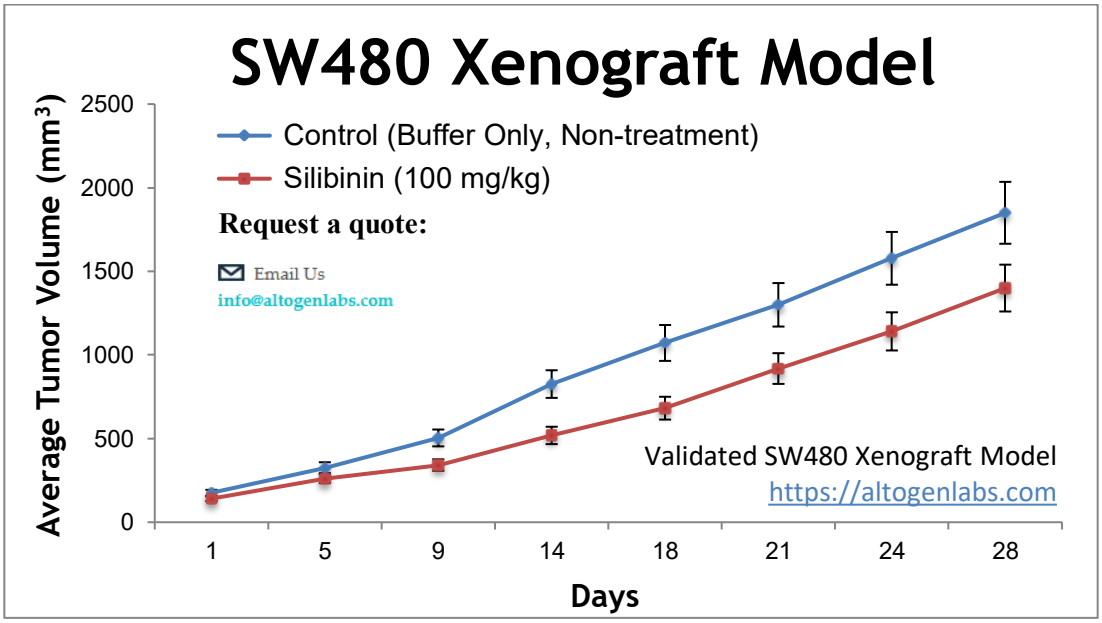
The RKO xenograft model offered by Altogen Labs represents a highly valuable preclinical platform for investigating colorectal cancer, particularly the microsatellite instability-high (MSI-H) molecular subtype. Derived from a poorly differentiated human colorectal carcinoma, the RKO cell line exhibits a wild-type TP53 genotype and lacks common oncogenic mutations in KRAS and BRAF. It is also characterized by epigenetic silencing of MLH1, leading to mismatch repair deficiency. These features enable the RKO model to be used for mechanistic studies of p53-mediated apoptosis, DNA damage responses, epigenetic regulation, and emerging therapeutic strategies targeting MSI-associated vulnerabilities.

Altogen Labs has developed both subcutaneous and orthotopic xenograft models using RKO cells under IACUC-compliant and GLP-aligned conditions. In the subcutaneous model, one million RKO cells suspended in Matrigel are injected into the hind leg of immunodeficient BALB/c mice. Tumor growth is monitored via digital caliper measurement, with treatment initiated at 50–150 mm³ tumor volumes. At study endpoint, tumors are harvested for downstream analyses such as histopathology, mRNA profiling, and protein expression assays. Orthotopic transplantation involves direct injection of RKO cells into the colonic wall and recapitulates native tumor microenvironmental interactions and metastatic progression to organs such as the liver and pancreas. Both models are suitable for efficacy evaluations of DNA methyltransferase inhibitors, checkpoint inhibitors, and targeted therapies.

Numerous studies using the RKO xenograft model have contributed to understanding oncogenic pathways and therapeutic resistance mechanisms. Recent investigations include characterization of NEDD8-activating enzyme inhibitors (e.g., SOMCL-19-133), deubiquitinase inhibitors targeting USP28/USP25, and combination therapies involving Akt inhibitors with cetuximab. These studies have highlighted the RKO model's sensitivity to agents that promote apoptosis, disrupt protein stability, or reverse epigenetic silencing. In addition, gene knockdown strategies targeting NOB1 and RNF6 have demonstrated the model's utility in dissecting post-translational regulatory networks. Given its defined genetic background and tumorigenic consistency, the RKO model continues to support translational research and precision oncology initiatives.

The RKO xenograft model is accessible at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/rko-xenograft-model/.

Characterization and Preclinical Application of the SW480 Xenograft Model in Colon Cancer Research



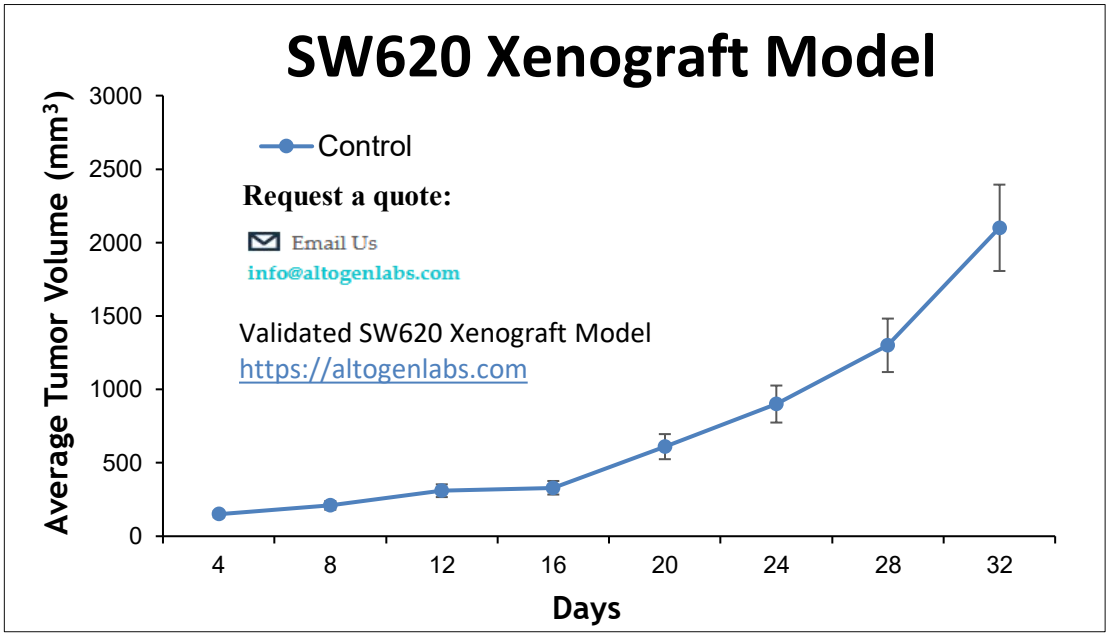
The SW480 xenograft model, available from Altogen Labs, is a rigorously validated preclinical platform for evaluating therapeutic strategies targeting early-stage colorectal cancer. Derived from a primary colon adenocarcinoma, SW480 cells possess pathogenic mutations in KRAS (G12V), TP53, and APC, resulting in constitutive Wnt/β-catenin pathway activation and impaired apoptotic signaling. This model is widely employed in translational oncology to study chemoresistance, oncogene function, epithelial-to-mesenchymal transition (EMT), and immune modulation. Despite its limited metastatic potential compared to SW620 cells, SW480 retains strong tumorigenicity, making it highly suited for early-stage intervention and mechanistic studies.

Altogen Labs offers subcutaneous, orthotopic, and metastatic SW480 xenograft models conducted in a GLP-compliant, IACUC-regulated facility. In the subcutaneous model, 10,000 cells/μL suspended in Matrigel are injected into the flanks of immunodeficient mice. Tumor volumes are monitored with digital calipers, and animals are randomized once tumors reach 75–125 mm³. Tissues are collected at endpoint for histopathology, RNA/protein extraction, and gene expression profiling. Orthotopic models involve implantation into the colonic or rectal wall, providing a physiologically relevant microenvironment that supports studies of angiogenesis, drug penetration, and host-tumor interactions. Metastatic models using vascular injection routes enable the evaluation of systemic spread and therapeutic efficacy against secondary lesions.

The SW480 xenograft model has been pivotal in recent case studies. Zymogen granule protein 16 (ZG16) overexpression was shown to suppress PD-L1 and enhance innate immune activation in SW480 tumors, highlighting its role as a candidate immunotherapeutic target. Similarly, PRDM5 and miR-145 were demonstrated to inhibit tumor growth and EMT, respectively, while CRMP-4 knockdown suppressed proliferation. These studies underscore the model's versatility in investigating both genetic and epigenetic regulators of tumor behavior.

Altogen Labs supports over 90 cell line-derived xenograft models and provides comprehensive services including tumor growth delay (TGD), growth inhibition (TGI), pharmacokinetics, survival studies, and biodistribution. Customizable protocols and advanced imaging technologies enhance the utility of the SW480 model in drug development. For more information, the SW480 xenograft model can be accessed at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/sw480-xenograft-model/.

Characterization and Preclinical Application of the SW620 Xenograft Model in Colon Cancer Research



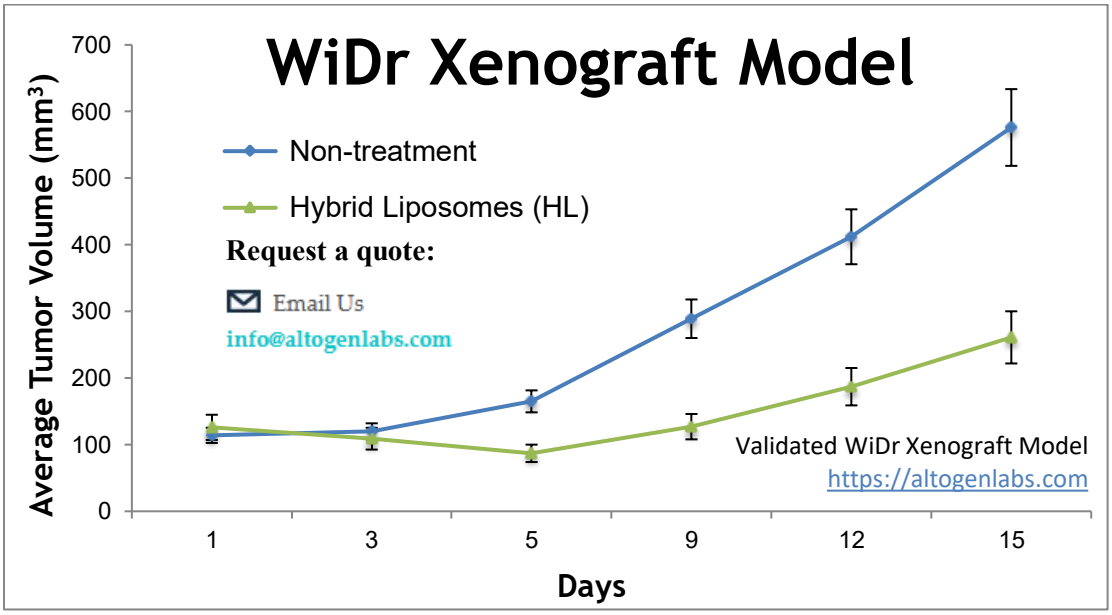
The SW620 xenograft model, available from Altogen Labs, is a well-characterized and widely adopted in vivo system for investigating metastatic colorectal cancer (CRC). Derived from a lymph node metastasis of a Duke's stage C adenocarcinoma, the SW620 cell line exhibits a mesenchymal phenotype marked by loss of E-cadherin, elevated vimentin expression, and upregulation of metastasis-associated genes including MMP7, CXCR4, and CD44. These cells harbor activating mutations in KRAS (G12V) and TP53, as well as loss-of-function alterations in APC, resulting in aberrant Wnt/β-catenin signaling. SW620 xenografts are highly tumorigenic and display enhanced invasive and chemoresistant features, making them highly suitable for modeling metastatic progression and therapeutic resistance.

Altogen Labs offers validated subcutaneous, orthotopic, and metastatic SW620 xenograft models conducted in immunodeficient mice under GLP-compliant and IACUC-regulated conditions. Subcutaneous implantation involves suspending 1 × 10^6 viable SW620 cells in Matrigel and injecting them into the hind flanks of BALB/c nude mice. Tumor volumes are measured by caliper until reaching 100–150 mm^3, followed by treatment initiation. Orthotopic implantation into the colonic or rectal wall allows for more accurate modeling of the tumor microenvironment and metastatic spread. Metastatic models utilizing tail vein or intrasplenic injections replicate systemic dissemination, with secondary tumor formation in organs such as the liver and lungs. These models facilitate preclinical testing of antineoplastic agents, exploration of epithelial-to-mesenchymal transition, and identification of regulators of immune evasion and stromal remodeling.

SW620 xenografts have been instrumental in evaluating molecular drivers of CRC metastasis. Recent studies demonstrated that focal adhesion kinase (FAK) inhibitors targeting the Y397 phosphorylation site significantly reduce SW620 tumor growth in vivo. Additional work has shown that AKT3 uniquely regulates mitochondrial metabolism and cancer stem cell properties in this model. Baicalin and genistein, plant-derived compounds, were also shown to induce ROS-dependent apoptosis and suppress tumor growth selectively in SW620 xenografts. These findings highlight the model's value in dissecting metastatic signaling pathways and identifying candidate therapeutic agents.

Researchers can find the SW620 xenograft model listed under Altogen Labs' colon cancer xenograft portfolio. Detailed service information is available at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/sw620-xenograft-model/.

Characterization and Preclinical Application of the WiDr Xenograft Model in Colon Cancer Research



The WiDr xenograft model offered by Altogen Labs represents a highly valuable preclinical platform for investigating colorectal cancer (CRC) biology and treatment responses. Derived from a human colorectal adenocarcinoma, the WiDr cell line is defined by its epithelial morphology, BRAF V600E mutation, wild-type KRAS, and deregulated Wnt/β-catenin and EGFR signaling. It is particularly suited for modeling CRC with constitutive MAPK activation and has been extensively used to evaluate targeted therapies, immune modulators, and combination chemotherapeutics. WiDr cells also express high levels of COX-2 and EGFR, contributing to their relevance in studies targeting inflammatory and growth signaling pathways.

Altogen Labs provides validated WiDr subcutaneous and metastatic xenograft models, with studies conducted under IACUC regulations and GLP-compliant conditions. In the subcutaneous model, one million WiDr cells suspended in Matrigel are injected into the flanks of immunocompromised BALB/c or NOD/SCID mice. Tumor progression is tracked by digital caliper measurement until tumors reach 100–150 mm³, at which point treatment regimens are initiated. Metastatic modeling has also been achieved using intrasplenic or portal vein injection, resulting in clinically relevant liver metastases. This capacity allows researchers to explore systemic dissemination, metastatic colonization, and the efficacy of anti-metastatic agents in vivo. Comprehensive post-mortem analysis includes tumor weight, histopathology, and preservation for molecular profiling.

Preclinical research using the WiDr model has yielded critical insights into therapeutic resistance and oncogenic signaling. Investigations have shown that Fn14-targeted antibody BIIB036 triggers apoptosis and tumor regression via NFκB activation and ADCC. Metabolic modulation studies reveal that insulin and IGF-1 significantly influence chemotherapeutic sensitivity, altering cellular responses to 5-FU and oxaliplatin. Additionally, CK2 inhibition via TBB reduces tumor growth and enhances DNA damage responses, although without synergistic radiosensitization. The WiDr model's sensitivity to mitogenic and survival pathway modulation underlines its suitability for mechanistic studies of BRAF-driven CRC.

Altogen Labs supports a full range of in vivo pharmacology services with this model, including tumor growth delay (TGD), tumor growth inhibition (TGI), and evaluation of multi-route drug administration (intravenous, oral, intraperitoneal, and others). Detailed service information is available at https://altogenlabs.com/xenograft-models/colon-cancer-xenograft/widr-xenograft-model/.

Preclinical Oncology Research Using Validated Colon Cancer Models from Altogen Labs

Altogen Labs offers a comprehensive suite of preclinical in vivo services specializing in xenograft and allograft tumor models, with a focus on the advancement of oncology drug development. The extensive portfolio includes over 90 validated xenograft models and several syngeneic models that collectively capture the heterogeneity of human cancers. Each model is thoroughly characterized for key oncogenic mutations, histopathological features, and therapeutic response profiles, enabling high-fidelity recapitulation of clinical disease phenotypes. The company's robust infrastructure supports subcutaneous, orthotopic, and metastatic tumor establishment, as well as tumor cell line engineering for specific research needs, such as stable knockdown, gene overexpression, or fluorescent and luminescent labeling.

Altogen Labs operates under stringent IACUC guidelines and GLP-compliant protocols, ensuring ethical and reproducible study execution. Each study is customized to align with the client's experimental goals, whether they involve early-stage screening of novel therapeutics, mechanistic interrogation of tumor signaling pathways, or late-stage preclinical validation of drug candidates. Available services include tumor growth delay and inhibition studies, survival analysis, biodistribution, pharmacokinetics, and toxicology assessments. Tumor and tissue samples are analyzed using state-of-the-art platforms such as RT-qPCR, Western blotting, capillary electrophoresis-based proteomics, and immunohistochemistry, ensuring robust molecular insight into therapeutic responses.

By offering an array of molecularly diverse colon cancer models such as HCT116, HT29, KM-12, LoVo, LS174T, MC38, RKO, SW480, SW620, WiDr, COLO205, CT26, and DLD-1, Altogen Labs addresses a broad spectrum of oncogenic drivers and resistance mechanisms. These models facilitate the evaluation of small molecules, monoclonal antibodies, immune checkpoint inhibitors, RNA therapeutics, and antibody-drug conjugates. The availability of both human tumor xenografts in immunodeficient mice and murine tumor allografts in immunocompetent hosts positions Altogen Labs as a critical partner in both traditional pharmacology and immuno-oncology studies.

Altogen Labs is committed to accelerating the pace of cancer research by delivering high-quality, data-driven, and customizable in vivo testing services. The integration of advanced imaging technologies, tailored therapeutic dosing regimens, and precise biomarker analysis ensures that each project generates actionable insights to inform clinical translation. With a foundation in scientific rigor and a reputation for reliability, Altogen Labs continues to support biopharmaceutical companies and academic researchers in the pursuit of innovative cancer therapeutics studies.