Guidelines on genetic evaluation and management of Lynch syndrome: a consensus statement by the US multi-society task force on colorectal cancer.

Giardiello FM, Allen JI, Axilbund JE, Boland CR, Burke CA, Burt RW, Church JM, Dominitz JA, Johnson DA, Kaltenbach T, Levin TR, Lieberman DA, Robertson DJ, Syngal S, Rex DK.

Am J Gastroenterol 2014; 109(8):1159-79.

http://www.ncbi.nlm.nih.gov/pubmed/25070057

A summary of Lynch syndrome clinical criteria and differential diagnosis, genetic alterations, gene-specific cumulative risks of colorectal and extra-colorectal cancer, and sensitivity and specificity for Lynch syndrome diagnosis using different criteria. The guidelines recommend universal testing of all colorectal cancers for Lynch syndrome starting with IHC or MSI testing and outlines testing strategy algorithms. It also provides guidelines on cancer treatment in patients with Lynch syndrome and on screening for extra-colorectal carcinomas.

Eosinophilic gastritis in children: clinicopathological correlation, disease course, and response to therapy.

Ko HM, Morotti RA, Yershov O, Chehade M.

Am J Gastroenterol 2014;109(8):1277-85.

http://www.ncbi.nlm.nih.gov/pubmed/24957155

Largest histologic study of pediatric eosinophilic gastritis, defined as marked, diffuse or multifocal infiltrates of ≥70 eosinophils per high-power field in the antrum or fundus, found thirty unique patients without evidence of infection, Crohn's disease, or organ transplantation. Atopy, food allergy, and protein-losing enteropathy were common clinical correlates. Eosinophilic esophagitis was found in 43% and eosinophilic enteritis in 21%. Most patients responded to dietary restriction therapy, suggestive of an allergic etiology.

Dysplasia-like epithelial atypia in ischemic bowel disease.

Abraham SC, Taggart MW, Loftus EV Jr, Wu TT.

Hum Pathol 2014; 45(7):1348-57.

http://www.ncbi.nlm.nih.gov/pubmed/24946975

Sixty five ischemic enteritis resections and 99 ischemic colitis resections with viable epithelium adjacent to necrosis were studied, and the viable epithelium was classified as normal, obviously reactive, low-grade dysplasia-like atypia, or high-grade dysplasia-like atypia. Dysplasia-like atypia was found in 15-20% of cases and was diffuse, focal, or multifocal in distribution. Immunohistochemical staining for cell cycle markers p16, p53, and MIB-1 compared to ulcerative colitis dysplasia controls did not reveal a discriminating pattern for dysplasia. The authors conclude that as with inflammatory bowel disease, "it remains a good policy to judge the epithelium by the company it keeps."

Significance of signet ring cells in high-grade mucinous adenocarcinoma of the peritoneum from appendiceal origin.

Sirintrapun SJ, Blackham AU, Russell G, Votanopoulos K, Stewart JH, Shen P, Levine EA, Geisinger KR, Bergman S.

Hum Pathol 2014; 45(8):1597-604.

http://www.ncbi.nlm.nih.gov/pubmed/24814804

A retrospective review of cases of mucinous adenocarcinoma of the peritoneum of appendiceal origin with clinical follow-up. Fifty-five cases were studied, of which 29 had signet ring cells, all of which were high-grade tumors (based on non-signet ring cell epithelial features). Overall survival was significantly shorter when signet ring cells were invading tissue, as compared to when signet ring cells were only present within mucin pools (median 0.5 years vs 2.4 years, respectively). The presence of signet ring cells only within mucin pools was prognostically similar to cases with no signet ring cells. The authors suggest that reporting of signet ring cell tissue invasion in peritoneal specimens with mucinous adenocarcinoma of appendiceal origin would be clinically useful.

BRAF-mutated microsatellite stable colorectal carcinoma: an aggressive adenocarcinoma with reduced CDX2 and increased cytokeratin 7 immunohistochemical expression.

Landau MS, Kuan SF, Chiosea S, Pai RK.

Hum Pathol 2014; 45(8):1704-12.

http://www.ncbi.nlm.nih.gov/pubmed/24908142

A retrospective immunohistochemical phenotyping study of microsatellite stable (MSS) colorectal carcinomas that harbor a *BRAF* mutation. Previous reports have shown

decreased CDX2 and CK20 expression in MSI-H colorectal carcinomas with a *BRAF* mutation, and these cases along with *BRAF* wild-type MSS colorectal carcinomas were used as controls. BRAF-mutated MSS colorectal carcinomas demonstrated reduced expression of CDX2 and increased expression of CK7. The expression of CK20 was similar to *BRAF* wild-type MSS tumors. Knowledge of this altered immunophenotype is important in the analysis of metastatic tumors, as the pattern is not typical for colorectal carcinoma origin.

Differences in DNA methylation signatures reveal multiple pathways of progression from adenoma to colorectal cancer.

Luo Y, Wong CJ, Kaz AM, Dzieciatkowski S, Carter KT, Morris SM, Wang J, Willis JE, Makar KW, Ulrich CM, Lutterbaugh JD, Shrubsole MJ, Zheng W, Markowitz SD, Grady WM.

Gastroenterology 2014; 147(2):418-29.

http://www.ncbi.nlm.nih.gov/pubmed/24793120

This multi-institutional study compared methylomes of normal colonic mucosa, tubular adenomas, and colorectal cancers to determine the contribution of epigenetic alteration to the development of cancer. Genome-wide array-based studies revealed that adenomas separated into high-frequency and low-frequency methylation patterns. The high-frequency methylation adenomas contained a subset with KRAS mutations, and the overall signatures were more similar to carcinoma than to normal colonic mucosa. The low-frequency methylation adenomas had overall signatures similar to normal colonic mucosa. In addition, normal colonic mucosa samples from near colorectal carcinoma showed different methylation pattern from normal colonic mucosa samples of patients with no colorectal carcinoma, suggesting the possibility of a field effect. Heterogeneous methylation patterns among colorectal cancers were found, similar to previously reported studies.

Segmental distribution in refractory ulcerative colitis: a histologic evaluation in pediatric and adult patients who underwent proctocolectomy.

Uchida K, Araki T, Hashimoto K, Inoue M, Otake K, Koike Y, Okita Y, Fujikawa H, Tanaka K, Mohri Y, Kusunoki M.

Inflamm Bowel Dis 2014; 20 (7): 1127-35.

http://www.ncbi.nlm.nih.gov/pubmed/24865779

The purpose of this study was to compare clinical and pathologic features evident in colorectal resection specimens among adult and pediatric patients with medically refractory ulcerative colitis and to clarify the implications of segmental distribution of ulcerative colitis. The authors analyzed clinical features, histologic severity of disease, and presence or absence of segmental disease distribution among 14 patients with childhood-onset ulcerative colitis and 76 patients with adult-onset ulcerative colitis. When pediatric patients were compared to adult patients, no significant differences in the interval between disease onset and surgery or disease severity were observed. Segmental distribution of ulcerative colitis was reported in 64% of patients with childhood-onset ulcerative colitis, but in only 21% of patients with adult-onset ulcerative colitis ($p<0.05$). Comparison between 25 patients with segmental disease distribution and 65 patients with continuous inflammation revealed a significantly shorter interval between disease onset and colectomy in the former group. Patients with segmental disease distribution also showed more severe disease activity compared to those with continuous disease ($p<0.05$). The authors conclude that segmental ulcerative colitis is associated with childhood onset, more severe disease activity, and shorter interval to surgical resection compared to continuous ulcerative colitis.

Lymphocytic esophagitis in children

Sutton LM, Heintz DD, Patel AS, Weinberg AG

Inflamm Bowel Dis 2014; 20(8):882-891.

http://www.ncbi.nlm.nih.gov/pubmed/24983984

The purpose of the study was to clarify the prevalence of and clinical associations with lymphocytic esophagitis in a cohort of pediatric patients. The authors conducted a retrospective review of esophageal biopsies from all pediatric patients who underwent upper endoscopy during a 1-year period. Lymphocytic esophagitis was defined histologically as >50 intraepithelial lymphocytes per high-power field and <1 granulocyte per 50 intraepithelial lymphocytes. Biopsies from 31 of 545 patients met these criteria (prevalence of 5.7%). Lymphocytic esophagitis was found in six of 49 (12%) patients with Crohn disease compared to 25 of 496 (5%) patients without Crohn disease, resulting in an odds ratio of 2.6 ($p<0.05$). Furthermore, six of 31 (19%) patients with lymphocytic esophagitis were known to have Crohn disease, whereas only 43 of 514 (8%) patients without lymphocytic esophagitis carried this diagnosis ($p<0.05$). Among patients with lymphocytic esophagitis, lymphocyte density was significantly higher in patients with Crohn disease compared to those with other conditions. No statistically significant associations were found between lymphocytic esophagitis and other gastrointestinal disorders in the study population, including gastroesophageal reflux disease, *Helicobacter pylori*-associated gastritis, immune-mediated diseases, and polyposis syndromes. The authors conclude that lymphocytic esophagitis has a higher overall prevalence in children compared to adults, in light of the reported prevalence of only 0.1% in the latter group in previously published studies. They also conclude that

lymphocytic esophagitis is more strongly associated with Crohn disease in pediatric compared to adult populations.

Fungal infections of the gastrointestinal tract in the immunocompromised host: an update.

Lamps LW, Lai KKT, Milner DA

Adv Anat Pathol 2014; 21(4): 217-227.

http://www.ncbi.nlm.nih.gov/pubmed/24911247

This review contains a synopsis of gastrointestinal fungal infections, including those that are caused by emerging and recently-identified organisms. The article also contains updates on classification, differential diagnoses, appropriate use of available ancillary tests for fungal infections, and excellent histologic images of the entities discussed.

Endoscopic detection of proximal serrated lesions and pathologic identification of sessile serrated adenomas/polyps vary on the basis of center.

Payne SR, Church TR, Wandell M, Rösch T, Osborn N, Snover D, Day RW, Ransohoff DF, Rex DK.

Clin Gastroenterol Hepatol 2014; 12:1119-26.

http://www.ncbi.nlm.nih.gov/pubmed/24333512

This article reports a retrospective study of the detection of serrated polyps during screening colonoscopy at 22 U.S. and 10 German centers. A total of 5778 colonic polyps were biopsied, of which 3008 were signed out as adenomas, while 350 (6.1%) were signed out as either SSP/A (including SSP/A with cytologic dysplasia) or a hyperplastic polyp in the proximal colon >1 cm in size. The key finding of the study was that the diagnosis of serrated lesions (as defined) ranged from 0 to 13.1% at the individual centers. Statistical analyses revealed that the variation among centers was explained both by variation in detection rate by the endoscopists at the various centers, as well as variation among pathologists at each center in the use of the diagnosis of SSP/A. Of note, there was no central review of pathologic diagnoses in this study.

Serum inflammatory markers and risk of colorectal cancer in patients with inflammatory bowel diseases.

Ananthakrishnan AN, Cheng SC, Cai T, Cagan A, Gainer VS, Szolovits P, Shaw SY, e Churchill S, Karlson EW, Murphy SN, Kohane I, Liao KP.

Clin Gastroenterol Hepatol 2014; 12: 1342-8.

http://www.ncbi.nlm.nih.gov/pubmed/24407106

Several recent studies have documented that the intensity of active inflammation in patients with IBD is correlated with the risk of dysplasia and cancer development. This study of 3145 IBD patients demonstrated a correlation between the level of serum C-reactive protein (CRP) and serum erythrocyte sedimentation rate (ESR) and the risk for colorectal cancer. The CRP and ESR measurements were obtained during routine laboratory evaluation of the patients as part of their normal clinical monitoring. A total of 33 patients in the CRP cohort and 102 patients in the ESR cohort developed colorectal cancer during a median follow-up of five years.

Overexpression of integrin αv correlates with poor prognosis in colorectal cancer.

Ha SY, Shin J, Kim JH, Kang MS, Yoo HY, Kim HH, Um SH, Kim SH.

J Clin Pathol 2014; 67:576-81.

http://www.ncbi.nlm.nih.gov/pubmed/24695839

This report details a retrospective study of immunohistologic staining of colorectal adenocarcinoma with a mouse monoclonal anti-integrin αv antibody. Integrin αv expression has been associated with tumor angiogenesis and tumor progression at other sites. A tissue microarray was constructed utilizing tissue from 198 colorectal cancers of various stages from patients who did not received adjuvant or neoadjuvant chemotherapy. An immunohistologic score was determined for each specimen from staining intensity and percentage of positively stained tumor cells. A high immunohistologic score was associated with increasing T stage, N stage, and TNM stage. Kaplan-Meier analysis revealed significantly shorter overall survival and disease free survival in patients with tumors exhibiting high expression of αv integrin. In a multivariate analysis high expression of αv integrin was an independent predictor of overall and disease free survival.

Apoptotic enteropathy caused by antimetabolites and TNF-α antagonists.

Soldini D, Gaspert A, Montani M, Reineke T, Rogler G, Odze R, Weber A.

J Clin Pathol 2014; 67:582-6.

http://www.ncbi.nlm.nih.gov/pubmed/24723128

This report describes four patients with diarrhea in whom small bowel and/or colonic biopsies revealed GVHD-like crypt cell apoptosis similar to that seen in patients on mycophenolate. The onset of diarrhea was temporally associated with the administration of methotrexate, capecitabine, etanercept, and infliximab in these patients.

Targeted next-generation sequencing of cancer genes dissects the molecular profiles of intraductal papillary neoplasms of the pancreas.

Amato E, dal Molin M, Mafficini A, Yu J, Malleo G, Rusev B, Fassan M, Antonello D, Sadakari Y, Castelli P, Zamboni G, Maitra A, Salvia R, Hruban RH, Bassi C, Capelli P, Lawlor RT, Goggins M, Scarpa A.

J Pathol 2014; 233: 217-27.

http://www.ncbi.nlm.nih.gov/pubmed/24604757

The authors utilized targeted next generation sequencing to examine the mutational status of 51 cancer-related genes in 52 intraductal neoplasms of the pancreas (48 IPMNs and 4 ITPNs). GNAS and KRAS mutations were most prevalent, found in 75% and 46% of the tumors respectively. In 92% of the IPMNs there was either a GNAS or KRAS mutation (or both). Other mutations, seen only in association with a GNAS and/or KRAS mutation, were much less prevalent (*TP53* mutation in 10%, *BRAF* in 6%, and mutations in *CTNNB1*, *IDH1*, *STK11* or *PTEN* in 4%). The histologic subtypes of IPMN did not exhibit characteristic mutational patterns, although KRAS mutation was more common in the gastric subtype. The authors also demonstrated the feasibility of genetic analysis by next generation sequencing of cyst fluid, which could be helpful in clinical management of cystic pancreatic lesions.

SMARCB1 (INI1)-negative rhabdoid carcinomas of the gastrointestinal tract: clinicopathologic and molecular study of a highly aggressive variant with literature review.

Agaimy A, Rau TT, Hartmann A, Stoehr R.

Am J Surg Pathol 2014; 38(7):910-920.

http://www.ncbi.nlm.nih.gov/pubmed/24503755

This study describes the clinical history, immunohistochemical findings and molecular alterations in two carcinomas from the stomach and cecum with exclusive rhabdoid features. The patients died of disease at 6 and 10 months, respectively. The tumors coexpressed vimentin, pancytokeratin, and EMA. Both showed complete loss of nuclear SMARCB1/INI1. Molecular analysis (KRAS, EGFR, BRAF, PIK3CA, and microsatellite studies) revealed a CpG-island methylator phenotype in the cecal tumor (CIMP(+)/MLH1(-)/BRAF(V600E)/MSI-H), confirming epithelial origin. The gastric tumor included poorly differentiated adenocarcinoma in regional nodes, again confirming epithelial derivation. Other genes tested were wild type in both cases. A literature review including 37 previously reported cases revealed a glandular component in 33%. Affected sites were: stomach (13), colon (11), small bowel (10), and distal esophagus (5). Of the 34 patients with follow-up ≥12 months, 29 (85%) died within 1 year (mean = 4 mo). Molecular tests were performed in 8/39 cases. A CIMP(+)/BRAF(V600E)/MLH1(-) phenotype was found in 3/4 right colon tumors. Loss of nuclear SMARCB1 protein was noted in 3/6 cases tested. This study highlights the heterogeneity of rhabdoid GI neoplasms and supports their epithelial derivation. Rhabdoid phenotype likely represents a common pathway of dedifferentiation with frequent loss of SMARCB1 and a highly aggressive course. The CIMP phenotype represents a novel subset of rhabdoid GI carcinomas. This rare variant should be distinguished from proximal-type epithelioid sarcoma and other SMARCB1-deficient mimics.

Isocitrate dehydrogenase-1 is mutated in inflammatory bowel disease-associated intestinal adenocarcinoma with low grade tubuloglandular histology but not in sporadic intestinal adenocarcinoma.

Harman DJ, Binion D, Regueiro M, Schraut W, Bahary N, Sun W, Nikiforova M, Pai RK.

Am J Surg Pathol 2014; 38(8):1147-1156.

http://www.ncbi.nlm.nih.gov/pubmed/25029120

Isocitrate dehydrogenase (IDH) is an enzyme in the tricarboxylic acid cycle (Krebs cycle) which catalyzes the conversion of isocitrate to α-ketoglutarate. Somatic IDH mutations have been identified in various tumors, including myeloid neoplasms, thyroid carcinoma and intrahepatic cholangiocarcinoma. Mutations in the IDH gene play a role in tumorigenesis by inducing hypermethylation of CpG islands leading to altered gene transcription and genomic stability. This study analyzed a series of intestinal adenocarcinomas with (n = 23) and without (n = 39) associated chronic idiopathic inflammatory bowel disease (IBD) from a single institution for IDH1 and IDH2 mutations and correlated the clinicopathologic findings with mutation status. IBD-associated

adenocarcinomas more frequently demonstrated IDH mutations compared to intestinal adenocarcinomas not associated with IBD (13% vs. 0%, P = 0.047). All IDH mutations were identified in IDH1 (cytoplasmic and peroxisomal isoform). IDH1 mutations were frequently (66%) associated with concurrent KRAS mutations. IDH-1 mutated tumors were seen in both Crohn disease and ulcerative colitis and were located in both the ileum and colon. Compared with IDH1-negative IBD-associated adenocarcinoma, IDH1-positive IBD-associated adenocarcinomas more frequently demonstrated tubuloglandular histology (100 vs. 25%, P = 0.032), and were more frequently associated with precursor lesions exhibiting serrated morphology (66% vs. 6%, P = 0.034). IDH1 mutations were also identified in the precursor dysplastic lesions. In conclusion, IDH1 mutations are occasionally identified in IBD-associated intestinal adenocarcinoma but not in those adenocarcinomas not associated with IBD. IDH1-mutated adenocarcinomas are associated with low-grade tubuloglandular morphology and often harbor concurrent KRAS mutations. Identification of patients with IDH1 adenocarcinomas may become clinically important as new targeted therapies emerge.

A case of acute necrotizing esophagitis.

Kimura Y, Seno H, Yamashita Y.

Gastrointest Endos 2014; 80(3):525-526.

http://www.ncbi.nlm.nih.gov/pubmed/24830580

This well illustrated case report describes the dramatic endoscopic findings of an elderly man who presented to the emergency department with hematemesis and epigastric pain. Acute esophageal necrosis (AEN) is commonly referred to as “black esophagus”. Tissue damage in AEN is likely multifactorial and may include organ hypoperfusion, impaired local defense barriers, reflux of gastric contents and possibly medication toxicity (as illustrated in this case). There is a predilection for AEN to involve the distal esophagus. Biopsies show extensive nearly obliterative tissue necrosis involving the mucosa and often deeper layers. Treatment is directed at correcting the coexisting clinical conditions, restoring hemodynamic stability and intravenous acid suppression with proton pump inhibitors. Adverse events include esophageal perforation with mediastinal infection/abscess, stricture, superinfection and death. Mortality is often related to underlying comorbid disease, and although the overall prognosis is poor, mortality specific to AEN is less than 10%.

Targeted multiprobe fluorescence in situ hybridization analysis for elucidation of inconclusive pancreatobiliary cytology.

Vlajnic T, Somaini G, Savic S, Barascud A, Grilli B, Herzog M, Obermann EC, Holmes BJ, Ali SZ, Degen L, Bubendorf L.

Cancer Cytopathol 2014; 122(8):627-634.

http://www.ncbi.nlm.nih.gov/pubmed/24753508

This study explored the utility of fluorescence in situ hybridization (FISH) to improve the diagnostic stratification between reactive and malignant cells in cases of inconclusive cytology. The multiprobe FISH assay UroVysion was used for copy number assessment of chromosomes 3, 7, 17, and the 9p21 locus on Papanicolaou-stained specimens with a diagnosis of inconclusive cytology (n = 50), adenocarcinoma (n = 31) and no evidence of malignancy (n = 9). A positive test was defined as increased copy number (> 2) of at least 2 chromosomes (3, 7, or 17) in at least 4 atypical cells, or loss of 9p21 in at least 12 cells. FISH confirmed all 31 cytological diagnoses of pancreatobiliary adenocarcinomas, and was negative in the 9 patients with negative cytology. Among the 50 cases with inconclusive cytology, FISH detected 19 of 31 cases with a final diagnosis of adenocarcinoma, and was negative in all 19 cases with no final evidence of malignancy (sensitivity of 61.3%, specificity of 100%, positive predictive value of 100%, negative predictive value of 61.3%). Loss of 9p21 was found in 43 (86%) of all 50 FISH-positive cases. The authors conclude that multiprobe FISH combined with automated relocation of atypical cells is a powerful technique to clarify inconclusive cytology of the pancreatobiliary tract and allows for better distinction between reactive atypia and malignancy.

Inhibition of ZEB1 by miR-200 characterizes Helicobacter pylori-positive gastric diffuse large B-cell lymphoma with a less aggressive behavior.

Huang W, Kuo S, Cheng A, Lin C.

Modern Pathol 2014; 27, 1116–1125.

http://www.ncbi.nlm.nih.gov/pubmed/24390222

This study from Taiwan looks at "pure gastric diffuse large B cell lymphomas," that is, diffuse large B cell lymphomas (DLBCLs) of the stomach with no associated MALT lymphoma identified. Of 102 pure gastric DLBCLs, the authors found evidence of H. pylori infection in 53% of the cases. Cases with associated H. pylori infection exhibited with less aggressive clinical features and better outcomes when compared to the H. pylori negative group. A genome-wide expression analysis was performed on a sample set of the H. pylori positive and H. pylori negative groups and it was found that H pylori infection was associated with transcription factor ZEB1 inhibition, which the authors

explain is due to high levels of miR-200 that normally regulates ZEB1. The authors speculate that loss of ZEB1 activity, which normally serves to suppress BCL6, leads to increased levels of BCL6 and thus a germinal center phenotype, which is associated with a less aggressive clinical course in DLBCLs.

Cardiac mucosa at the gastro-oesophageal junction: indicator of gastro-oesophageal reflux disease? Data from a prospective central European multicentre study on histological and endoscopic diagnosis of oesophagitis (*histo*GERD trial).

Langner C, Schneider NI, Plieschnegger W, Schmack B, Bordel H, Höfler B, Eherer AJ, Wolf E-M, Rehak P, Vieth M.

Histopathology 2014; 65: 81-9.

http://www.ncbi.nlm.nih.gov/pubmed/24393213

Biopsies from more than 1000 individuals were studied for the presence of cardiac mucosa at the GE junction and any histologic changes reflective of GERD. Cardiac mucosa was present in more than 66% of patients and was significantly associated with evaluation for esophageal or gastric disease (P=0.025 and 0.027, respectively). Features of reflux and the presence of intestinal metaplasia were significantly associated with the presence of cardiac mucosa. There was no significant association with Helicobacter status or the presence of Barrett esophagus.

Does clear cell carcinoma of stomach exist? Clinicopathological and prognostic significance of clear cell changes in gastric adenocarcinomas.

Kim J-Y, Park DY, Kim GH, Jeon T-Y, Lauwers GY.

Histopathology 2014; 65(7): 90-9.

http://www.ncbi.nlm.nih.gov/pubmed/25032253

A total of 762 cases of gastric adenocarcinoma were studied for the presence of clear cell change and was identified in 8.5%. Clear cell change was significantly associated with older age (at least 59 years), intestinal-type histology, location in body/fundus, greater depth of invasion, lymph node metastasis, and lymphovascular invasion. The proportion of clear cell change was an independent marker of poor prognosis.

Loss of special AT-rich sequence-binding protein 1 (SATB1) predicts poor survival in patients with colorectal cancer.

Al-Sohaily S, Henderson C, Selinger C, Pangon L, Segelov E, Kohonen-Corish MRJ, Warusavitarne J.

Histopathology 2014; 65(8): 155-63.

http://www.ncbi.nlm.nih.gov/pubmed/24118100

Based on tissue microarray data from a cohort of 352 patients, SATB1 expression (normal is positive nuclear expression) was lost in 22% of colorectal cancers versus 1.5% of adjacent normal colorectal tissue. Loss of expression was significantly associated with worse overall survival (P=0.02), younger age (P=0.03), mucinous or signet-ring histology (P=0.0001), and poor differentiation (P=0.005). SATB1 expression was associated with a survival advantage in patients with Dukes C tumors who also received neoadjuvant chemotherapy.

Systematic immunohistochemical analysis of the expression of CD46, CD55, and CD59 in colon cancer.

Shang Y, Chai N, Gu Y, DingL , Yang Y, Zhou J, Ren G, Hao X, Fan D, Wu K, Nie Y.
Arch Pathol Lab Med 138:910-919, July 2014.

http://www.ncbi.nlm.nih.gov/pubmed/24978917

CD46, CD55, and CD59 are membrane-bound complement regulatory proteins (mCRPs). Their expression was significantly higher in colon cancer versus normal adjacent tissue (microarray data of 121 cases). Expression of CD55 and CD59 correlated with grade of tumor differentiation and with stage III/IV versus stage I/II cancer.

Journals Reviewed (July and August Issues)

Histopathology
Archives of Pathology and Lab Medicine
Modern Pathology
American Journal of Clinical Pathology
Journal of Pathology
Journal of Clinical Pathology
American Journal of Pathology
Human Pathology
Cancer Cytopathology
American Journal of Surgical Pathology
Advances in Anatomic Pathology
Journal of Molecular Diagnostics
Gastrointestinal Endoscopy
Gastroenterology Clinics of North America
Gastroenterology
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American Journal of Gastroenterology
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