3 minority groups, with pancreatic cancer nationally has remained unchanged since 2002. Among all 5261 visits of minority patients to our medical center over the past 8 years, 1.3% (68) were for the Emergency Department, 11.1% (398) were in the inpatient setting, and 1.6% (84) were for surgery. These differences are statistically significant for the 3 sites of care (p<0.001 using chi-square analysis) when compared with White/Non-Hispanic patients (0.2%, 7.3%, and 2.6%, respectively). CONCLUSION: A large percentage of the patients seen at our medical center for pancreatic cancer belong to a minority group and the percentage of these patients is declining, in contrast with the unchanged incidence observed nationally. Minority patients have significantly greater visits to the Emergency Department and inpatient services but significantly lower surgical visits when compared with White/Non-Hispanic patients. This may reflect the younger age of the minority population in the minority group patient care but precluding surgery. The causes of these trends are likely multifactorial but in need of further study in order to understand and improve the management and outcomes of minority patients with cancer.

**Sa1960**
Mucosal Adherent Bacteria, Inflammation and Colorectal Adenomas
Felix Araujo-Perez, Nina Snapareddy, Amber N. McCoy, Biljana Jovov, Andrew K. Benson, Zaid Abdo, Anthony Fodor, Robert S. Sandler, Temoteo O. Koku

The gut microbiota is closely involved in normal host physiology and may contribute to the etiology of many diseases. Colorectal cancer (CRC) is the third cause of cancer death in the United States. Evidence from animal and human studies supports a link between inflammation and CRC. However, the contribution of the gut microbiota and local inflammation to sporadic adenomas and CRC in the absence of diagnosed inflammatory bowel disease is not defined. We hypothesized that altered mucosal adherent bacterial composition and increased local inflammation are associated with elevated risk of adenomas. We examined the relationship between adenomas, local mDNA expression of cytokines and adherent bacterial profiles in normal colorectal mucosa in a case control study. Methods: Participants were consenting patients undergoing a screening colonoscopy at UNC Hospitals with no history or diagnosis of inflammatory bowel disease. Subjects were defined as cases or controls based on the presence or absence of adenomas. High throughput deep sequencing method was used to assess bacterial diversity in normal colorectal mucosa of 80 cases and 87 controls. mDNA gene expression of inflammatory markers in normal colonic tissue were determined by real time RT-PCR. Gene expression relative to the housekeeping gene HMB2 was determined. Differences in bacterial profiles between cases and controls were evaluated by t-tests and analysis of similarity matrix. Diversity was measured by Shannon diversity. Logistic regression was used to compute odds ratio (OR) and 95% confidence intervals (CI). Results: Case subjects were more likely to be, male and have higher waist:hip ratio. Compared to the lowest quartile, subjects in the highest tertile of IL-10 gene expression levels were significantly less likely to have adenomas (OR 0.46, 95% CI 0.2-0.9). The most abundant bacteria in the mucosa were Firmicutes (54%), Bacteroidetes (19%), Proteobacteria (18%), and Cyanobacteria (6%). Compared to controls, cases had lower abundance of the Firmicutes (p=0.02) and higher abundance of Cyanobacteria (p=0.04): At the genus level, case subjects showed increased abundance of Acidovorax, Cloacibacterium, Acinetobacter and Lactobacillus and lower abundance of Streptococcus than controls. Increased local IL-17 and IL-23 expression positively correlated with bacterial diversity in cases (r=0.38, p=0.007, r=0.34, p=0.01 respectively) but not controls (r= -0.13, p=0.4, r= -0.03, p=0.8 respectively). IL-10 expression was inversely linked with bacterial diversity in cases (r= -0.39, p=0.05). At the genus level, increased IL-17 and IL-23 expression were associated with elevated abundance of Lactobacilllts and Helicobacter. These findings suggest that increased local inflammation is linked with bacterial dysbiosis and may contribute to the etiology of colorectal cancer.

**Sa1961**
Interaction Between Diet and Colonic Microbiota in Low and High Colon Cancer Risk Populations
Junho Ou, Kayelen Umekane, James DeLany, Anthony Fodor, Robert S. Sandler, Temoteo O. Koku

The observation that colon cancer is more common in developed than in developing nations suggests that the etiology of colorectal cancer.

**Sa1962**
Acetlcarnitine Potentiates the Anticarcinogenic Effects of Butyrate on Colon Cancer Cells
Ernest G. Suidan, Serge Donnne, Ihsan El Imrani, Dan Saragosy, Emile Levy

AIMS: Butyrate, a short-chain fatty acid, is a potent anticarcinogenic compound for colon cancer cells. However, its rapid metabolism by colonic epithelial cells is hypothesized to limit its anticancer benefits. Carnitine is essential to fatty acid oxidation and carnitine depletion is implicated in the inability of mitochondria to maintain normal metabolism. The aims of this study were to evaluate the effect of carnitine and acetlcarnitine (ALCAR) on the response of color cancer cells to butyrate and to explore the underlying mechanisms. METHODS: SW480 cells were incubated with butyrate +/- carnitine or ALCAR. Cell viability was measured with an MTT and trypan blue exclusion. Apotosis was measured with the M30 assay. Carnitine uptake was assessed using [14C]-carnitine. Modulation of proteins implicated in dietary transport, as well as cell death and proliferation was assessed by Western blot. RESULTS: SW480 cells expressed higher levels of OCTN2 and displayed 8 fold higher carnitine uptake than unaltered Caco-2 cells (0.33 vs 0.04 pmol/min/mg protein, p<0.01). Butyrate induced SW480 cell death at concentrations of 2mM and higher. ALCAR (5 mM), but not carnitine, significantly increased cell death rate (30.2 vs 4.6 %, p<0.01). Cells treated with 3 mM butyrate combined with ALCAR displayed increased mortality (29 vs 21%, p<0.05). The effects of ALCAR could not be reproduced by acetate or acetyl + carnitine. Butyrate induced apoptosis in SW480 cells, while carnitine and its acetlyated congener had no independent effect. The addition of carnitine or ALCAR increased butyrate-induced apoptosis by 8-13% (p<NS). Butyrate increased levels of cyclin D1 and p21 (125 and 70% of control levels, p<0.01). PARP cleavage, a marker of caspase activity, was increased 6 fold, while Bcl-XL and survivin levels were decreased by butyrate (61% and 46%, respectively, p<0.01). Butyrate also downregulated phospho-β-catenin and increased acetylated histone H4 (2.6 fold, p<0.01). The effect of carnitine and ALCAR on these proteins was also determined. At butyrate concentrations of 2 mM and below, carnitine consistently decreased survivin levels by at least 25% (p<0.05). ALCAR independently induced a 20% decrease in p21 (p<0.05). Acetylation of histone H4 as well as dephospho-β-catenin levels were not affected by ALCAR and carnitine. CONCLUSIONS. These results demonstrate that butyrate and ALCAR are potentially beneficial anticarcinogenic nutrients that inhibit colon cancer cell survival. The combination of both agents may have superior anticarcinogenic properties than butyrate alone. Supported by a grant from the Dairy Foundation of Canada and NSERC.

**Sa1963**
Increased Levels of Serum Glucose-Dependent Insulinotropic Polypeptide as a Novel Risk Factor for Human Colorectal Adenoma
Yu Sasaki, Hiroaki Takeda, Takashi Ashihara, Tomohiko Orii, Shoichi Nishise, Ko Nagino, Daisuke Iwamoto, Takao Yaita, Kazuya Yoshizawa, Sumio Kawata

Objectives: Obesity and insulin resistance are thought to be risk factors for colorectal adenoma. Glucose-dependent insulitotropic polypeptide (GIP) stimulates insulin secretion from the pancreas and promotes fat accumulation in adipocytes. The association between serum GIP and the risk of colorectal adenoma has not been examined previously. Methods: We investigated this association in 375 subjects who underwent total colonoscopy during thorough physical check-ups between January and December, 2008. We employed a cross-sectional design, and classified the subjects into a colorectal adenoma group and a control group without adenoma, according to their endoscopic findings. Serum GIP concentrations in samples of venous blood obtained after an overnight fast were measured using a sandwich ELISA kit. Results: The mean levels of fasting GIP (34.9 ± 49.5 pmol/ml vs 25.0 ± 20.1 pmol/ml; p < 0.047), triglyceride, glucose, insulin and values of homeostasis model assessment-insulin resistance in the colorectal adenoma group were significantly higher than those in the control group. When restricted to male subjects, the adenoma group (36.0 ± 47.7 pmol/ml) had a significantly higher fasting GIP concentration than the control group (23.7 ± 15.6 pmol/ml; p < 0.027). In contrast, in female subjects, no significant difference in GIP concentration was observed between the groups. Multiple logistic regression analysis showed that the top tertile of fasting GIP levels was associated with a significantly high risk of colorectal adenoma (odds ratio 5.1, 95% confidence interval 1.08-29.0, p < 0.01) in comparison with the bottom quartile. Quartile analysis demonstrated that increased levels of GIP were related to increased levels of fasting insulin and values of homeostasis model assessment (HOMA) β-Cell function. Conclusions: This suggest that increased level of fasting insulin and glucose are associated with an increased risk of colorectal adenoma, and may offer a new insight into the role of GIP in the development of human colorectal adenoma.

**Table 2: Stool bacteria levels (copies/perm g feces)**

<table>
<thead>
<tr>
<th>Total Bacteria</th>
<th>Faecalibacterium prausnitzii</th>
<th>Clostridium IV</th>
<th>Bifidobacterium longum</th>
<th>Methanobrevibacter</th>
<th>Subtilis reducing bacteria</th>
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<td>NA</td>
<td>627807439</td>
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<td>5057215</td>
<td>68339</td>
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<td>8292760</td>
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<td>301511</td>
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</tbody>
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