thank you for that very nice introduction and thank you for showing up this afternoon to share some of what I think is interesting and new in gastroenterology. I had a bit of a challenge figuring out what you might think is interesting and new, I think there are other speakers in this series that voiced similar challenges of how do we determine what are the topics to cover in such a short period of time, and also right now I don't come close to covering all of the new and important things that have happened over the last year. But I did use a semi quasi-scientific method to try to come up with what might be important, and what I did was take the primary topics in research papers in Gastroenterology, our primary basic science journal; also taking all the hot topics in the American Journal of Gastroenterology Journal that over the last year that's really more of a clinically oriented journal; also pulling in up to date the last 6 months of what they call hot topics and the major presentations for our national - at our National GI Meeting in May of this year.

And I put it all together in a Word document, stripped away all the extraneous information and ran it through Tag Cloud software. Now you've probably seen this on different websites where the words relate to the significance based on their color and based on the font size and this is what I came up with running through these somewhat different journals and these various sources, so that filtered out a lot of things like that this audience may not be all that interested in or really what I wasn't all that interested in. And then I had to think well what do you need? What are the things that are troubling a primary care physician and probably a lot of the same things that are challenging for me as a general gastroenterologist, I'm really the only general gastroenterologist in the whole division, I don't

have a particular clinical speciality so I get a lot of the types of patients that you get, just the ones that after you get them and not sure exactly what to do and how to manage them.

So I get a lot of questions like these from patients and I imagine you are getting them too and I'd like to today try to focus around being able to answer some of these tough questions like you know what about avoiding glutin in your diet and is hepatitis C really curable now but how much is it going to cost? Should I be on a low carb diet? I'm sure you hear that one a lot. Do I really need a colonoscopy? Now they don't ask me that because it's too late by the time they are prepped and in the GI lab, but I'm sure you get that question once in a while and what are the alternatives here? Probiotics, antibiotics, lots of confusion out there. And then the number one question I've received in the last couple of weeks is can I still eat bacon? And the World Health Organization as many of you know made some announcements so we are going to touch on that too and help you answer some of these questions for your patients.

So taking that Tag Cloud I'm going to just start out with an area that I don't work in per se, hepatitis and hepatology in general, but is such big news that I just couldn't pass it up for this presentation. I'm sure you've heard more than one study about the ability for this new breed of antivirals to essentially cure hepatitis C, in particular genotype type 1 hepatitis C, but this is still huge, 3,2 million people in the United States suffer from this and in this particular study, and this is really one of the landmark ones, the combination drug was used in 865 people in four different arms. One arm was the drug along for either 12 weeks or 24 weeks, the other arm was combining it with Ribavirin

for 12 weeks or 24 weeks. The results really were quite dramatic in that all four arms did well with 97 to 99% sustained viral response. And that from that you can deduce that it's the shorter treatment of 12 weeks and the one without Ribavirin is going to be the cheapest and easiest with the least side effects. And this is going up against Interferon/Ribavirin which has far less of a response rate, 60% in the best studies and lots more side effects, and a duration of 48 weeks. And having treated patients in the past on this regimen it's really a relief to have an easier to take more effective regimen.

There are some criticisms of this particular study, there was no control group, but I don't think you really need to have one when you have this kind of results. And it was sponsored by Gilead, the company that stands to make quite a bit of money off of this drug. And that's where the problems come in. And I heard that this is - you heard a little bit about this just last week so I won't go into a lot of details, but just in the past week a Wall Street Journal article about the Federal Government going after the State Medicaid programs who are throwing up barriers and hurdles in front of the HCV patients to try to limit their access to this particular drug, and it's an expensive drug, $84,000 for one course of treatment, not counting labs and other medical visits. So it's a big burden at least in the short term on Medicaid programs and it's a real problem and you heard a lot about that last week. And the ability though to get this drug out to all the people that need it, not just in the United States but around the world, is really a challenge for us but something that we have to figure out a solution for. The company is starting to work with foreign governments and with the local state governments to try to figure out a way to get that drug to more people.

So enough said on that, you were promised some Thanksgiving advice. So I don't know about your Thanksgiving table, but this is what mine is starting to look like. We have a lot of medical professionals or medical professional want-to-bes in my family and everyone has an opinion about what the best diet you should on and a lot of particular diet requests, so here we've got the celiac patient who really should be on a glutin free diet, but then you have the glutin sensitive or the one that's on a low FODMAP diet or a Paleo diet or a low carb diet. Well while I'm going to talk about a lot of different GI diseases today you'll see that we'll be coming back to these same issues and I'm going to really try to tie together some of this diet preference information we are hearing about, some of the research that's coming out in the last year, how diet may affect the gut microbiome and how disease and health may be affected by all of those things.

So let me start with celiac disease. This is one that most of you have got a pretty good handle on. We have good diagnostic tools with antibody testing, we have confirmatory tools like small bowel biopsy and we have a not easy to do but a clearcut cure for it which is a glutin free diet for life. There is some new work being done on glutin blockers for the small bowel but that's pretty far off in the future and we won't go into that.

This graph though shows where celiac disease and particularly glutin itself is causing me a lot of stress and perhaps in your clinic too. Om one survey done by a marketing research group they found that 30% of the U.S. population states that they either eliminated or cutting back on glutin in their diet. Almost all of those are people that no doctor recommended that, they did ti - well no doctor

that they saw face to face, maybe one on cable or late night. But they've done it based on internet websites and advertisements and you know talking to each other and social networking. Another as much as 60% of the population believes that cutting back on glutin and a glutin free diet is a healthier diet. So this is you know whether your patients bring this up or not a lot of them are considering this.

Now from that group about 6% at the most are estimated to truly have something that's not celiac disease but a GI-like syndrome that is non-celiac glutin sensitivity and whose relatively objective measures could be considered a disease entity in and of itself. The University of Maryland's Celiac Research Group has done a lot of work on this and in their population it's 6%, other studies are putting it as low as .5%. But it's a real group of patients and this is an ara of a lot of intense discussion and research in GI right now. So does it really exist, because there are no biomarkers to prove that there is such a thing as non-celiac glutin sensitivity. There is a lot of studies that have been done, some good double blinded randomized controlled trials with large numbers of patients that suggest that yes this is an entity. The double blindedness is a little hard because if you've ever tried a glutin free muffin, which is what they use in a lot of these studies, it tastes a lot different than a muffin that has glutin in it.

So in order to kind of look at whether this is a real entity or not this interesting study came out this year where they subjected 61 people who claimed to have non-celiac glutin sensitivity, also they excluded people who had FODMAP sensitivity which we'll talk about a little bit too, and gave them

instead of a glutin free muffin as a intervention they used glutin-containing capsules with a control of a rice starch-containing capsule. They did a little study showing that they tasted exactly the same, you can't tell the difference. And with that small amount it really equates to about 2 slices of bread a day. They put them through a glutin free run in period, one week on the capsules, two week washout period and then crossed them over. And if you look at just the abstract and you look at the headline it says that the ones that the symptom score of the group that had glutin was significantly higher than during the glutin free period.

If you dig into the data carefully all the patients had about the same symptom score through both legs of the study except for 3. And 3 were so off the scale when they had glutin versus not having glutin that it threw the entire study and enough to get statistical significance. So you can conclude one of two things, either this was a really badly designed study and maybe there isn't any such thing as non-glutin sensitivity or perhaps these 3 patients really have an entity and the other ones don't. That's not proven by this study but it's certainly something worth looking at. So the jury is still out on this particular issue, but having treated - or having a lot of patients that are in this category there are some things that are a little bit different from your celiac patients. There tend to be primarily bloating complaints, abdominal pain complaints, foggy mind or brain fog is something that's fairly consistent in this group of patients and the development of stomatitis and aphthous ulcers are kind of hard to fake along with your symptoms. So I think there is something there, we just need better ways of testing for it and better ways of identifying the group of patients that fall into this category.

If you want to read a little bit about this entity of non-celiac glutin sensitivity there is a wonderful easy to read editorial in Clinical Gastroenterology and Hepatology and they pull together a lot of the recent studies and make some points like this brain fog symptom that seems to be unique to this group of patients, maybe more joint and muscle symptoms. The ones that are HLA-DQ2 or HLA-DQ8 like we see in virtually of the celiac disease patients have a much higher rate of non-glutin - non-celiac glutin sensitivity than the normal population, 50% versus 30%. But maybe there is other food intolerances in this and what I've always suspected is that you take glutin out of a diet you are actually taking a lot of other things out of a diet too. And in almost every study that I looked at you end up cutting back on the calories considerably, you take the carbohydrates which is a lot of the source of glutin is in our common carbohydrate based foods, you take that out of the diet and people are going to feel better and perhaps it has nothing to do with the glutin, that's just a bystander.

So what about these food intolerances, the FODMAP diets? How many people here use the FODMAP diets in their clinical interaction with patients? So a few. I think if there is more gastroenterologists a lot of us really jumped on this bandwagon a couple of years ago when it came out. There were some pretty good studies, this one by Emma Halmos that showed that in a well designed study there is a group of patients with irritable bowel syndrome who have a positive response to a diet low in these fermentable oligo/di-saccharides, monosaccharides and polyols which comes down to lactose and fructose and sorbitols and beans, fruit, some vegetables. And in this particular study they showed a decrease in symptom score both intestinal and extra-intestinal symptoms in the group that was in a low FODMAP diet, a score of 22.8 versus a score of 44.9 with a

statistical significance. And again bloating, pan, flatus were the primary complaints and those were the ones that also show statistical significance in change in symptoms with and without FODMAP in their diet, so again there probably is something here for your IBS patients and in this study they actually measured the stool on these patients for the 21 days of their study. In the IBS diarrhea predominant patients the stool frequency went down. So encouraging for a group of patients that as you know and I know that are difficult to manage.

So when you pull out that instruction sheet on low FODMAP diets you usually have something like this, and this is just what you are supposed to avoid not what you can eat. Of course those are kind of hard to read and not meant for that, it's meant to show you how complex it is. And I really doubt that that many patients stay on this type of diet for a very long period of time. Now there are simpler charts, this one says just follow this simple chart and give it a try. And you know I've seen small icons but this is - and trying to - actually it's kind of fun to look at this and try and figure out what all these icons are supposed to be, different types of strawberries and fruits and bananas. The green is where the good and the red is the bad stuff, I'm not sure that this is that much easier for anybody.

So is there really something to this FODMAP and is there an easier way to approach recommending low FODMAP diets to patients? So this is a study done and published this year in Switzerland where they instead of controlling the diet in this group of 75 patients that had IBS (Rome III) criteria they made dietary recommendations to them. Similar to what you just saw they went through what is a low FODMAP diet and made verbal recommendations just like we would do in the clinic, so trying

to mimic real life a little bit more and also gave as a control the usual diet recommendations that they give to all their IBS patients which is a diet low in insoluble fibers, cut back on the caffein, more beans, cabbage, onions, things we all know to tell IBS patients, particularly the ones with the bloating and abdominal discomfort. And they found there was really no difference between the two groups.

Now there was some changes in both groups. Both groups showed a fairly significant decrease in the amount of calories they consumed each day. Also surprising was they actually followed the diet, so at least the diaries looked like they followed the diets pretty well. And they had improvement in symptoms in both groups, about 50% or so, but you know clearly I'll take 50% in IBS patients any day. So some of the predictors that they saw that may help you select out which patients to suggest dietary changes and maybe simpler dietary changes than the typical low FODMAP diet would be females, ones with particular bloating, pain and flatulence. And I find that in my own clinic that that's a patient that seems to respond to the dietary changes the best.

All right let's stick with irritable bowel syndrome for a little bit but onto a different treatment. The TARGET-3 study was announced this year at our national meeting just a few weeks ago and as you may know this also coincided with the FDA approval of Rifaximin to be used with - to be used for non-IBSC or non-constipation predominant IBS patients. It's kind of an interesting story, I won't go into too much detail but the TARGET-1 and TARGET-2 studies both showed improvement in patients with IBS but the FDA wanted to confirm that it was safe to give IBS patients these antibiotic

and it wasn't developing resistance. They wanted to know whether or not retreatment because that was inevitable was safe and whether it was effective. And that's - and how long the effect really lasts if you give somebody an antibiotic that has IBS. And that's what the TARGET-3 study answered all of those questions and answered in a fairly positive way that a 2 to 4 week treatment of Rifaximin gives you on average 10 weeks or more relief in the patients who do respond to this and that retreatment usually for a shorter period of time, maybe just a few days, works well and they didn't find any increase in the number of resistant bacteria in the patients' stool or any incidence in higher rates of infections of any in the study patients over the study period.

So it appears to be safe, you can use it for shorter durations as time goes on and what I do in my clinic is if somebody does respond to - and this is particularly IBSD, patients with diarrhea, they've usually been through a few other empiric attempts at treating their symptoms and if they do respond to Rifaximin then titrate the repeated doses. So go as long as you can, if symptoms recur have hem take the Rifaximin until the symptoms go away. Wait as long as you can before the next one, try to shorten the duration and eventually you can come up with a few days every few months type of approach that you know from a drug resistant standpoint, from a cost standpoint seems like a reasonable approach in a group of patients that respond quite well when you do identify the ones with IBS that have this.

So again if you want to read something that kind of summarizes not just this study but a number of other ones and updates you on the recent advances in understanding the pathophysiology and

treatment for IBS I recommend you to this review article done by Lacy, Chey and Lembo in this year's Gastroenterology and Hepatology. They still recommend you know things like lifestyle modifications like exercise and sleep and diet changes like we talked about, and then kind of going through a low FODMAP diet, trying something similar to that, trying a glutin free diet, trying antidiarrheals, Rifaximin, and kind of tailoring and selecting based on the patient's symptoms.

In that same article though they do suggest that the future is the gut microbiome and they have a nice diagram that shows it. This balance between well to put it simply good bacteria and bad bacteria that they suggest is probably affected much by our diet. And then this diagram shows that a red meat and high fat diet low in vegetables will promote one type of bacteria and the opposite will give you more of the good bacteria. So kind of keep that one in your mind as we talk about a few other conditions that aren't IBS.

So I'm going to jump into cancer and colon cancer specifically and some diet things related to that. This is our new enemy I guess, these cured red meats. Ironically this is a photo I took in Lyon, France, they have some of the best cured red meats in the world; but that's also where the World Health Organization decided to make their announcement about the risk of consuming red meat on a regular basis. I suspect a few of the people on the committee tried some of Lyon's good products anyway.

So this was announced just in October of this year and I took a direct quote. And it is interesting that this group of 22 experts said that probably - red mean is probably carcinogenic to humans and it's based on limited evidence, and that the consumption of red meat causes cancer in humans based on strong mechanistic evidence supporting a carcinogenic effect. This is actually something that a lot of us have known for a long time. There is a lot of in vitro studies showing the carcinogenic effects of red meat, carcinogenic effects of meat that is overcooked and burned and some rat studies that also show the increase in colon cancer. So it's a recommendation we've been making for a long time. This was mainly seen in colon cancer so unfortunately what I thought was a ideal meal for me to be consuming a few months ago which is kale and peas and tomatoes but with a little bacon on top to make it taste good is no longer allowed and that perhaps that bacon is going to increase my chances of getting colon cancer. Or it is really?

I'm not going to ask who in here has cut back on their red meat and bacon consumption but you do need to make that decision and your patients will come to you with that. So I went into the studies that they cited and the actual recommendations and looked at well what is the - what is the risk? So they said 50 grams a day will increase your risk of colorectal cancer by 18%. So I don't measure my meat in grams and most of the menus I read in this country at least don't show how many grams, so I went and got some pictures that I had from a cruise we took a few years ago. This is the first time I got any benefit from cruise food. So the small steak is a 6 oz steak and the large steak is a 16 oz porterhouse, and if you look at the recommendation you are allowed about 12 oz a week. Or to put it another way, if you consume 12 oz a week you are increasing your risk by 18%, so 2 little steaks or

you know 2/3 of a big steak. Perhaps that's not very much, but how much is 18%? And if you look at say cigarette smoking, cigarette smoking increases your risk by 25-fold, that's a lot more than 18%. Again you know personal decisions you and your patients need to make about that, but you know it's a little bit hard to make based on just their recommendations.

So I looked at another recent review of this whole question, it's probably what this group based a lot of their decisions on because Song and her group published this monumental paper looking at all the factors that affect your risk of colorectal cancer with 558 references and really comprehensively put all this information together, and not only said don't eat red meat but outlined what put you at higher risk and this coincided pretty well with what the WHO said. What may put you at lower risk and so the association of people that have diets high in calcium, fiber and milk and whole grains have a lower risk of colon cancer and that certain foods may actually be preventive in your patients who are either at higher risk because of a family history of personal history of high risk lesions.

But does it all matter as long as we do colon cancer screening? Maybe not. But as long as we do colon cancer screening we probably are catching any of these 18% or so increase, and I think we've all kind of internalized and accepted now that we have made an impact on colon cancer over the last few years that the 30% decrease in the rate of colon cancer according to the American Cancer Society's data this year, but is it due to colonoscopies? And I get asked that by some of the people in this room, is it really the colonoscopies or is it changes in diet or changes in environment or just I

don't know changes in the microwaves in the air or something that are showing us a decrease in colon cancer?

Well this really impressive study done by the Germans this year draws a nice thick line between colonoscopy and decrease in colon cancer rates. They have a registry of 4.4 million Germans over the last - the 10 year period of 2003 to 2012 where they detected 180,000 cancers - or I'm sorry they prevented 180,000 cancer or 1 per 28 colonoscopies that were done in Germany. And these were purely screening colonoscopies. They detected 40,000 colorectal cancer or 1 out of 121 colonoscopies detected a cancer. Now one criticism is well what about all the people that had tumors detected or precancerous lesions that ended up not living long enough for it to really matter or over diagnosis? And they did find that 4500 people in the registry it wouldn't have mattered if they found their precancerous or cancerous lesions. But it was clear that you know in the under 75 age group 97% had cancer prevented, 89% were in the under 75 group where cancer was detected.

And then one other question that came up was well you know when do we stop screening? This study doesn't really answer that but it does say that the over diagnosis or finding lesions in people that it ends up not being a factor in their long term survival is a small factor and it's not necessarily weighted towards the older patients either. It was 28% of the over 75. So we are - you know this may affect some of the guidelines that we are currently following but does help us feel better about recommending the colonoscopy for screening purposes.

Despite that a lot of patients still don't get screened, for whatever reasons. Many times it's because they don't want the colonoscopy. So a lot of work has been done recently and over the last few years really on DNA testing of the stool. And this suggests one more piece of data and I found it kind of interesting that this particular multitarget DNA assay of stool is getting a little bit better. And when you compare it to colonoscopy its ability to detect colon cancer is 92.3% of the ones that were detected at colonoscopy. Unfortunately it drops down a good bit in detecting precancerous lesions, lesions with high grade dysplasia or sessile serrated polyps, which are kind of at the high end of those precancerous lesions as far as their risk of evolving into colon cancer. So still not quite there yet, this did show that it's clearly better than fecal immunochemical testing and may become an adjunct to your recommendations and especially in patients that are unwilling to go through colonoscopy.

So I mentioned a couple of things about the microbiome and diet and inflammatory conditions and I'm sure you've heard about it related to cardiac disease and maybe some rheumatologic disease. And at this year's GI meeting we had a lot of these pictures of fat mice and skinny mice and a lot of studies that were presented relating things like this one here from the GI group and published in Nutrients where a low carbohydrate diet improved the lipid profile and insulin sensitivity in rats, and you probably heard a lot of that stuff. But the fact that it's showing up at GI meetings is important because we are also finding that it relates to things like inflammatory bowel disease and it's additives like emulsifiers that they use to keep oil and water from separating in a lot of the food products they

- this one study in mice showed that it promoted the development of colitis by changing the microbiome in the gut.

Well a few human studies now coming out these were also recently published basically mostly on children where isocaloric fructose restriction resulted in improvements in metabolic syndrome and obesity. This one in particular I wanted to point out and I found kind of interesting, so a large group of investigators, 20 or more from all over the world, U.S., U.K. and Germany and China did a study, no surprise, on patients in China who were hospitalized and either with Prader-Willi Syndrome or simple obesity and placed them on a low carbohydrate diet. And no surprise they lost weight in both groups and there was a decrease in the circulating inflammatory markers, a decrease in the proinflammatory metabolites coming from their gut in both groups. But what is really fascinating is that they took stool specimens prior to putting them on a low carb diet and put those into germ free mice and those mice developed obesity. And took the stool samples after being on a low carb diet and losing some weight, put those in the same type of mice and they did not gain weight. So there is something going on here and this isn't just genetic Prader-Willi Syndrome, this also applies to a larger group of subjects that had simple obesity.

So if I was to give advice to a young and up and coming researcher in GI and like these gentlemen, GI Fellows over here, I might give advice kind of like you saw in the movie The Graduate. These guys probably have no idea what this is. But the microbiome was a big topic this year at our national

meeting and I think it's going to be something that ties together a lot of the more challenging GI diseases that all of us are trying to deal with.

And the Human Microbiome Project is really what got this started. They've studied for the last few years now what's in our gut and what's on our surface of our skin and other places and the colon represents the largest concentration of the microbiota that lives on our body with more cells in the colon than there are in our entire body and more DNA in the colon than we have in our own body. And a lot of research going on in this are, 1200 papers published so far this year, a lot of them on related to cancers of all kinds but in particular colon cancer and irritable bowel syndrome. And some kind of forward-thinking researchers are saying you know we thought that we developed an immune system to combat and control the organisms inside our gut but perhaps those organisms are controlling us more than we realize.

So what can you do to gain back some of that control and alter the microbiome and change disease and promote health? So back to the FODMAP idea and this is the same author a year later, Emma Halmos publishing that FODMAP, low FODMAP diets alter the colonic luminal microenvironment. And in a nice update done on fecal transplantation there is this group looked carefully at the current literature as to where we get some benefit in C. Diff colitis, especially the patients who have recurrent C. Diff that's not responding to other measures are an excellent group to use fecal transplant in, but safety and regulations have kind of limited its use. There is a couple of early studies that are kind of not - kind of weak data but intriguing looking at using fecal transplant in

patients with ulcerative colitis where there was a modest improvement in one study of 97 patients and the other study by Rossen had 50 subjects in it, was terminated early but because Rossen really thought everybody was going to respond he set the parameters very high so the safety monitors pulled it because not enough people were responding. But of the ones who did respond the ulcerative colitis patients who got better all of them adopted the flora of the host donor, I'm sorry developed the donor's flora.

So there is something going on with the food and immunity and the microbiome. I recommend this Gastroenterology article for this year that tries to tie a lot of this together related to not just GI disease but atherosclerosis and type II diabetes has a lot of good explanations of how the host gene expression may be modulated by the microbiome. The protein synthesis by the bacteria in our gut may have a big factor in disease and health and might explain some of these diet based approaches that I've covered in the last 40 minutes or so and why they work or don't work and perhaps the microbiome is that kind of common factor.

This cover shot from Gastroenterology has a nice diagram that shows you know diet and antibiotics and xenobiotics, the products of this bacteria all affect the makeup of the bacteria that are in our gut. Those interact with the colon mucosa primarily changing and inducing or suppressing inflammatory reactions and inflammatory conditions underneath the mucosa but then also producing a lot of metabolites that pass through the colonic mucosa and into the circulation and perhaps this, this balance of bacteria within the gut has a lot to do with the promotion of disease and some of these

other entities that we've discussed and maybe can help us answer some of these questions for our patients. So just to kind of wrap it up I wanted to see if we've answered some of these questions. Now I'm going to take these one by one and ask each of the Residents up here - just kidding. That's a nervous laugh up there.

So should you be avoiding glutin? Well as much as I try to tell my relatives and friends and I know a couple that are going to be at the Thanksgiving table that there's not a lot of good data suggesting that a glutin free diet in general is a healthy diet and that maybe there is a small percentage of people who don't have celiac disease that would benefit from it, but not generally recommended. They will all tell me I'm wrong and cite lots of social networking sources and friends and rumors despite the fact that I am a gastroenterologist.

Is there anything new for IBS? Well I think there is and if you are a primary care physician I'm sure you take care of a lot of patients with IBS and they are a troubling, frustrating group of patients to manage but I think we have some new tools now.

Hepatitis C is clearly curable at least for the genotype 1s. There is lots of new research that we didn't have time to go through today on for the other genotypes and we are getting better at that type of antiviral therapy too.

Hopefully I've introduced you to the microbiome a little bit and made you think about it. I think you'll hear about it in other specialty talks too.

Low carbohydrate diets for cholesterol and diabetes control and metabolic syndrome I think you already know about. My own doctor tells me about it.

So do you really need a colonoscopy, yes or no? Okay, hopefully everybody is going to agree here. In the patients who - now despite that a good third, probably more, of the population is not getting it. And probably and only not getting it because they don't want to have a colonoscopy for whatever reason. So having other tools like genetic testing of stool may help fill-in some of those gaps.

Probiotics, antibiotics still a little - the jury is a little out. I think Rifaximin is a good option for your IBS patients after they've failed some kind of general diet recommendations.

And can you still eat bacon? Well I'm not going to tell anybody that they can't, so yeah I think if you get your screening done and it's done in moderation that you don't have to eliminate red meat entirely from your diet. But again in moderation.

So thanks for your time and I appreciate any questions that you may have.