

SECTION OF GASTROENTEROLOGY, HEPATOLOGY & NUTRITION

# JOHN F. GILMORE SYMPOSIUM ON

# NUTRITION AND SMALL BOWEL DISORDERS

# **COURSE DIRECTORS**

Edwin K. McDonald IV, MD Dejan Micic, MD Carol Semrad, MD Elizabeth Wall, RDN-AP

# **UNIVERSITY OF CHICAGO**

Gleacher Center 450 Cityfront Plaza Dr Chicago, IL 60611

Course Credit: https://cme.uchicago.edu/JGS2025

Syllabus/Slides: https://cme.uchicago.edu/GilmoreSymposiumSyllabus2025







# JOHN F. GILMORE SYMPOSIUM ON

# **NUTRITION AND SMALL BOWEL DISORDERS**

The University of Chicago Gleacher Center

September 27, 2025

# **DESCRIPTION**

This activity will enhance clinicians' knowledge and skills to effectively deliver nutrition support and improve patient outcomes across diverse clinical settings. The training encompasses a comprehensive understanding of basic energy metabolism; macronutrient, and micronutrient requirements; nutrient digestion and absorption; and the optimal provision of nutrition support through both enteral and parenteral routes. By equipping healthcare professionals with this broad knowledge base, they will effectively manage the increasing complexity of patient cases and integrate dietary interventions into disease management, a priority for many patients.

#### **TARGET AUDIENCE**

This activity is designed for gastroenterologists, gastroenterology trainees, internists, advanced practice nurses, physician assistants, registered dietitians, nurses, and pharmacists dedicated to caring for patients with nutrition-related disorders.

### **LEARNING OBJECTIVES**

After this activity, participants will be able to:

- Identify evidence-based recommendations for the use of dietary fiber for patients with gastrointestinal diseases;
- Select the appropriate enteral feeding tube based on a patient's unique clinical condition;
- State the role of medical therapy in the novel field of metabolic dysfunction-associated steatotic liver disease (MASLD);
- Differentiate the clinical features and management approaches of short bowel syndrome (SBS) and intestinal failure (IF);
- Describe techniques for managing physiologic intolerances to mechanical complications of enteral access devices (e.g., leaking, clogged, and connection incompatibilities);
- Discuss the appropriate nutrition management techniques in patients presenting with chronic liver disease;
- Recommend suitable dietary options and hydration fluids tailored to the nutritional needs of patients with intestinal failure, taking into account their medical condition and treatment goals.

# ACCREDITATION AND CREDIT DESIGNATION

# PHYSICIAN CREDIT

The University of Chicago Pritzker School of Medicine is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The University of Chicago Pritzker School of Medicine designates this live activity for a maximum of 6.25 AMA PRA Category 1 Credits $^{\text{TM}}$ . Physicians should claim only the credit commensurate with the extent of their participation in the activity.

University of Chicago Medicine is accredited as a provider of nursing continuing professional development by the American Nurses Credentialing Center's Commission on Accreditation.

Participants who successfully complete the entire activity and complete an evaluation form will earn 6 contact hours.

#### **NURSING CREDIT**

University of Chicago Medicine is accredited as a provider of nursing continuing professional development by the American Nurses Credentialing Center's Commission on Accreditation.

Participants who successfully complete the entire activity and complete an evaluation form will earn 6.25 contact hours.

### **DIETICIAN CREDIT**

This program is approved for 6.25 continuing medical education credits. At the completion of this course Registered Dietitians can submit their CME credits to Commission on Dietetic Registration.

# AMERICAN BOARD OF INTERNAL MEDICINE MOC II CREDIT

Successful completion of this CME activity, which includes participation in the evaluation component, enables the participant to earn up to 6.25 MOC points in the American Board of Internal Medicine's (ABIM) Maintenance of Certification (MOC) program. Participants will earn MOC points equivalent to the amount of CME credits claimed for the activity. It is the CME activity provider's responsibility to submit participant completion information to ACCME for the purpose of granting ABIM MOC credit.

### OTHER PARTICIPANT CREDIT

Other participants will receive a Certificate of Participation. For information on the applicability and acceptance of Certificates of Participation for educational activities certified for *AMA PRA Category 1 Credit*<sup>TM</sup> from organizations accredited by the ACCME, please consult your professional licensing board.



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# **EDUCATIONAL GRANTS/COMMERCIAL SUPPORT**

Educational grant funding has been generously provided by::

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**NorthSea Therapeutics** 



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### **DISCLOSURE DECLARATIONS**

As a provider accredited by the ACCME, The University of Chicago Pritzker School of Medicine asks everyone in a position to control the content of an education activity to disclose all financial relationships with any ineligible companies. This includes any entity whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients. Financial relationships are relevant if a financial relationship, in any amount, exists between the person in control of content and an ineligible company during the past 24 months, and the content of the education is related to the products of an ineligible company with whom the person has a financial relationship. Mechanisms are in place to identify and mitigate any relevant financial relationships prior to the start of the activity.

Additionally, The University of Chicago Pritzker School of Medicine requires Authors to identify investigational products or off-label uses of products regulated by the US Food and Drug Administration at first mention and where appropriate in the content.

#### COURSE FACULTY

The following individuals have no relevant financial relationships with ineligible companies to disclose:

Alice Cheng, MD Neal Desai, PharmD Crystal Dowell-Green, BSN, RN Annette Jean Herlitz, RN, CNSC Omar Jamil, MD Simon Lal, MD, PhD Edwin McDonald, MD Elise Pajak, MS, RD, CNSC Shirley Paski, MD

Valerie Reynolds, MS, RD, LDN, CNSC

Ankita Sisselman, MD Sarah Vilt, RD Lori Welstead, RD

Dawn Adams, MD, has served as a consultant for Takeda, Ironwood, and Zealand.

Archita Desai, MD, has served as a consultant for Eli Lilly.

John Fang, MD, has served as a consultant for Merit and Circa Scientific.

**Dejan Micic, MD,** has served on an advisory board and speakers bureau for Takeda Pharmaceuticals and on an advisory board for Ironwood Pharmaceuticals.

George Ou, MD, has served as a consultant for Knight Therapeutics and Celltrion and as a speaker for Baxter.

Carol Semrad, MD, has received research funding from Zealand Pharmaceutical and Copenhagen.

Sonali Paul, MD, has received research funding from Gore.

Elizabeth Wall, RDN-AP, has served as a consultant for Ironwood.

The staff of the Center for Continuing Medical Education and the patient panelists have no relevant financial relationships with ineligible companies to disclose.

All of the relevant financial relationships listed for these individuals have been mitigated.

### **DISCLAIMER**

The views expressed in this activity are those of the individual speaker. It should not be inferred or assumed that they are expressing the views of any pharmaceutical or product/device manufacturer, provider of commercial services, or The University of Chicago. The drug selection and dosage information presented in this activity are believed to be accurate. However, participants are urged to consult the full prescribing information on any agent(s) presented in this activity for recommended dosage, indications, contraindications, warnings, precautions, and adverse effects before prescribing any medication. This is particularly important when a drug is new or infrequently prescribed.

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# JOHN F. GILMORF SYMPOSIUM ON

# **NUTRITION AND SMALL BOWEL DISORDERS**

The University of Chicago Gleacher Center

September 27, 2025

# SYMPOSIUM FACULTY

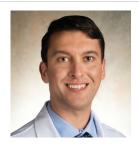
### **Course Directors**



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Assistant Professor of Medicine Director, Adult Clinical Nutrition

The University of Chicago Medicine



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Associate Professor of Medicine Chief, Division of Gastroenterology and Nutrition

Loyola University Medical Center



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Advanced Clinical Nutrition Support Dietitian The University of Chicago Medicine

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OptionCare Joint Venture Division of Gastroenterology. Hepatology and Nutrition

Vanderbilt University **Medical Center** 

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Center for Liver Diseases

Lori Welstead, MS, RD

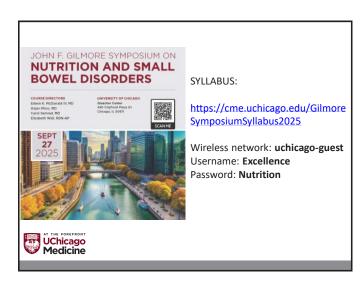
Dietitian IV

# SATURDAY, SEPTEMBER 27, 2025

	SATORDAY, SELTENBER 27, 2023
7:30 a.m.	REGISTRATION & BREAKFAST
8:00	Welcome Remarks
	CHALLENGES WITH ENTERAL ACCESS DEVICES MODERATOR: EDWIN MCDONALD
8:10	When You Think it is Not Possible: Enteral Access in Relative Contraindications John Fang, MD
8:30	Placement of Tubes in Surgically Altered Anatomy Edwin McDonald, MD
8:50	Tube Challenges in the Age of ENFit Shirley Paski, MD, MSc
9:10	Panel Discussion Session 1 Faculty
	BREAK
	DIETARY FIBER IN GI DISEASES
	MODERATOR: DEJAN MICIC
9:35	Evidence For Fiber In Gastrointestinal Disorders Dejan Micic, MD
9:55	Low FODMAP Diet: When Should It Be Tried? Lori Welstead, RD
10:15	Eating For Two: Updates On Fiber For The Gut Microbiome Alice Cheng, MD
10:35	Panel Discussion Session 2 Faculty
	KEYNOTE INTRODUCTION: CAROL SEMRAD
10:50	Clinical Approaches to the Management of Non-Short Bowel Dysmotility Leading to IF Simon Lal, MD
11:50	LUNCH
	MULTIDISCIPLINARY CARE OF INTESTINAL FAILURE PATIENTS MODERATOR: DAWN ADAMS
1:00 p.m.	A SB Pathway for Managing Short Bowel/Intestinal Failure Carol Semrad, MD
1:20	Recognizing Intestinal Failure and When To Intervene Ankita Sissleman, MD
1:40	The Rise of Nutrition Support Use in Disorders of Brain Gut Interaction George Ou, MD
2:00	Diet and Oral Hydration: Does It Make A Difference In IF? Elizabeth Wall, MS
2:20	Panel Discussion Session 3 Faculty
	BREAK
	METABOLIC DYSFUNCTION-ASSOCIATED LIVER DISEASE (MASLD) MODERATOR: OMAR JAMIL
2:40	Travel Through Time: NASH to MASLD/MASH Sonali Paul, MD
3:00	Diet and Lifestyle: The Initial Management for MASLD/MASH Sarah Kolb, RD
3:20	Approach to Drug Therapies for MASLD/MASH Archita Desai, MD
3:40	Panel Discussion Session 4 Faculty
4:00	ADJOURN



# Introduction







John F. Gilmore, Jr.
Symposium for Excellence
in Nutrition

# John F. Gilmore, Jr

- Grew up in Chicago's South Shore neighborhood
- Bachelor's Degree, History/Philosophy, John Carroll University Cleveland
- · Studied business at the University of Chicago
- VP at EF Hutton as a livestock analyst
- A founding partner of Goldman Sachs; industry leader investment banking
- Director Chicago Board of Trade
- Retired in 1994, diagnosed with head and neck cancer
- During therapy, obtained Master's Degree, Liberal Arts, U of Chicago
- In 2007, came to me as a patient with a tube feeding problem
- Diagnosing and treating his problem, improved his quality of life
- His motto was "You should never stop learning"
- Fitting motto for this Nutrition Symposium in his memory



# **Goals for this Symposium**

- There is a renaissance in nutrition as therapy to prevent and treat human diseases
- Severe shortage of physicians, dietitians, nurses and pharmacists trained in nutrition that impacts patient care
- Our goal is to close this gap by a premier symposium dedicated to nutrition
  - Stimulate interest in nutrition and small bowel disease
  - Identify gaps in knowledge, training, and research
  - Promote excellence in nutrition patient care



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Ironwood Pharmaceuticals
Nestlé HealthCare Nutrition, Inc.
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# We also thank our exhibitors:

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<u>Bronze Level</u> NorthSea Therapeutics A special THANK YOU to the Gilmore Family for your support!







# When You Think it is Not Possible: Enteral Access in Relative Contraindications

John Fang, MD

# When you Think it is Not Possible: Enteral Access in Relative Contraindications

John Fang M.D. University of Utah

# Difficult PEG case

- 38 y.o. male anesthesiologist with stage IV colon CA peritoneal mets with ascites admitted over Xmas holiday
  - NG tube placed for refractory N/V
  - IR consulted to place decompressive PEG
  - Declined to do procedure due to ascites and peritoneal involvement

# **Contraindications to PEG**

- Medical Futility
  - Limited life expectancy
  - Dementia
  - Terminal Cancer
- Ethical issues
- When expertise not available

# **Relative Contraindications**

- Massive ascites
- Coagulopathy
- Gastric varices
- Peritoneal dialysis
- Large hiatal hernia
- Peritoneal metastases or carcinomatosis
- Hepatomegaly
- · Morbid obesity
- Subtotal gastrectomy
- Gastric neoplasm
- Multiple abdominal surgeries
- Previous mesh repair

The role of endoscopy in enteral feeding.  $Gastrointest\ Endosc.\ 2011;74(1):7-12$ 

# More Difficult Situations

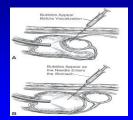
- Active upper aerodigestive cancer
- Unable to hold Anticoagulation
- Surgery or IR refused or failed
- Technical Issues
  - Inability to oppose anterior gastric wall to abdominal wall
  - Inability to advance endoscope through esophagus

# **Options for difficult PEG**

- · Devices/Methods
  - Introducer method (Russell, Push)
    - Gastropexy
  - Ultrathin Endoscope
  - EUS guided
  - Direct Percutaneous Endoscopic Jejunostomy
- · Delay and re-attempt
  - Place NET
- Non-endoscopic
  - IR
  - Surgery

# **Difficult PEG Tips**

- Change from CO2 to air insufflation
- Use safe track technique
- Spinal needle for obese patients
- · General anesthesia
- Ultrathin scopes
  - Significant co-morbidityOropharyngeal/ esophageal
  - Decreased or no sedation



# Potential Difficulties in PEG placement cirrhotic ascites

- Appostion gastric/abd walls more difficult
- Impaired tract formation
- Ascites
  - Reaccumulation
  - Leakage fluid
- · Increased risk infection
- · Increased risk bleeding
  - Coagulopathy
  - Abd venous collaterals

oagulopatny additional risk fac

ESGE 2021Guidelines:

- ascites should be considered to be additional risk factor for infection

583 cirrhosis

Ascitis 107 vs non-ascitis 476 P-value

2 (1.9%) 0.35

I (0.9%) 34 (23.8%) Odds ratio 95% CI

0.45-123

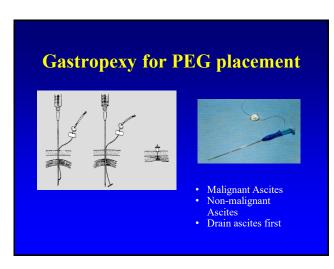
229 02-24 16 103-26

Table 2 Complication from percutaneous endoscopic gastrostomy tube placement

4 (0.8%) 2 (0.4%)

further preventive precautions must

Endoscopy 2021; 53:a-1303-7449 Clinical Experimental Gastro 2017:10 211–214

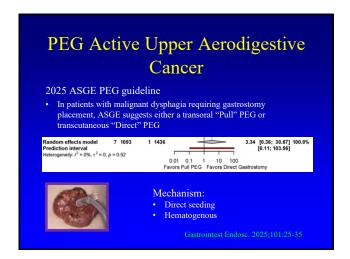


# PEG Method in cirrhotic ascites

- Drain ascites
  - Paracentisis ? U/S guided
- · Place gastropexys
  - Before/after PEG
  - Use Two or three
- PEG using standard method
- · Keep ascites drained
  - Until stoma tract mature
  - 7-10 day
- Extended antibiotic coverage



Gastrointest Endosc, 2005;61(1):178-80









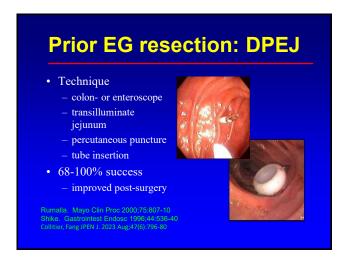
# Difficult PEG: Anticoagulation

- ASGE/ESGE guidelines
  - High risk procedure
    - 1.5-2.5% bleeding
  - Low risk pt's: hold antithrombotics
  - High risk pt's
    - Hold/Bridge anti-thrombotics
       When to re-start
    - Delay and use NET
  - Don't need to hold anti-plt agents
    - ASA/NSAIDs
    - Thienopyridines
    - DAPT

- Plts> 50K, INR<1.5
- When to stop
  - Nite before LMWH
  - ≥48 hr DOAC's
  - Warfarin 5 days
- - Warfarin: Evening of
  - LMWH: day after
  - DOAC 48 hrs

Gastrointest Endosc. 2025;101:25-35

# Inability appose gastric and abdominal wall: EUS guided PEG





# **Difficult PEG Conclusions**

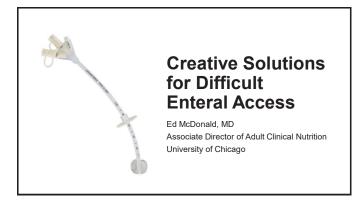
- Contraindications
  - Few absolute
  - Relative addressable
- When expertise available
  - Toolkit
    - Devices
    - Techniques
- Know your limitations
  - OK to try and fail
- Case outcome

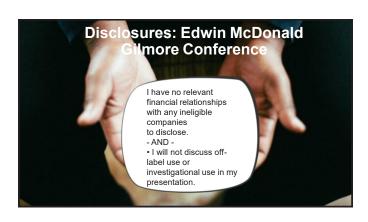




# **Placement of Tubes in Surgically Altered Anatomy**

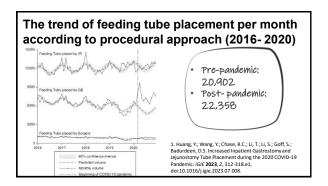
Edwin McDonald, MD

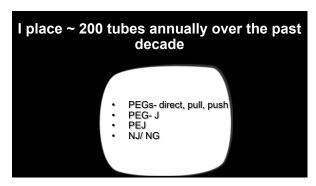






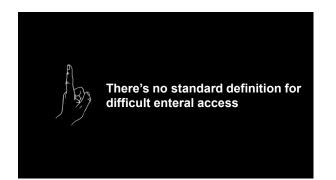




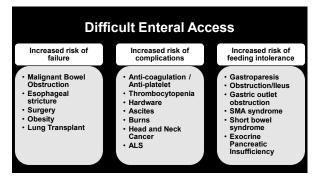




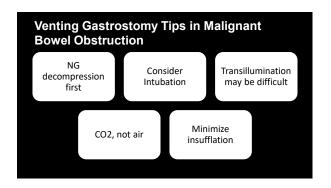


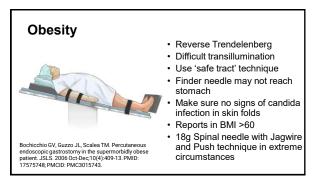


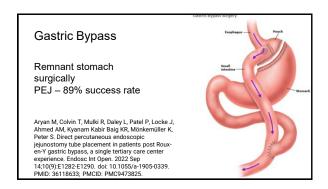




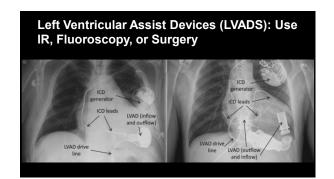


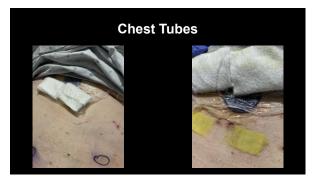


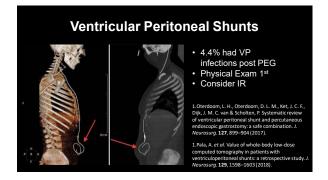


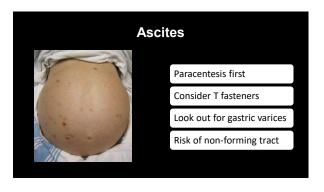


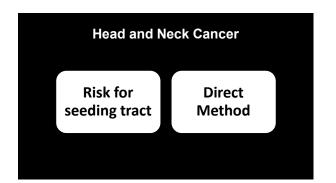
Increased Risk of Complications

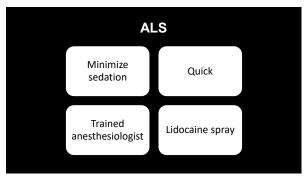


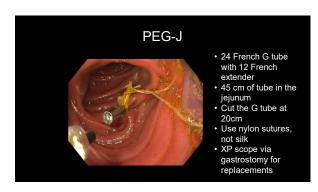


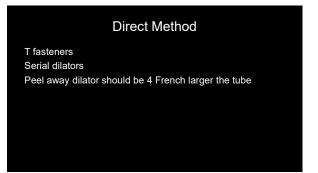












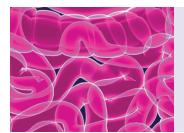
# PEJ

- Use 14 fr G tube
- Enteroscopy
- Otherwise same technique as PEG



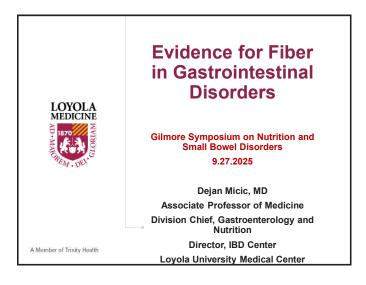
# **Tube Challenges in the Age of ENFit**

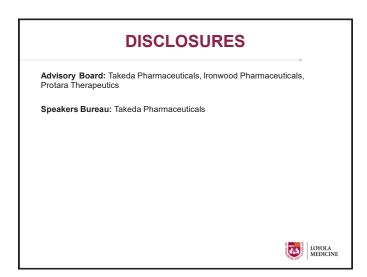
Shirley Paski, MD, MSc

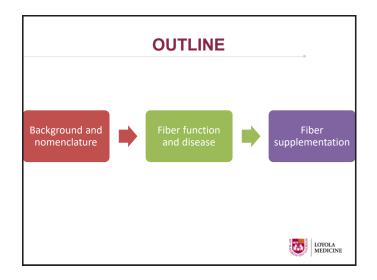


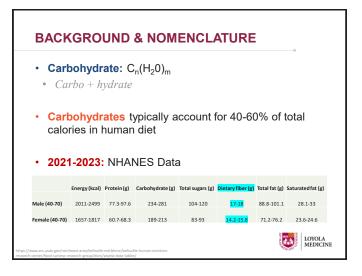
# **Evidence For Fiber In Gastrointestinal Disorders**

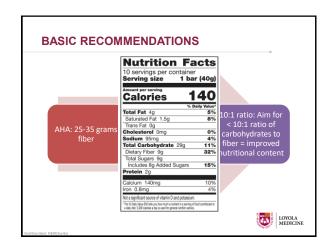
Dejan Micic, MD

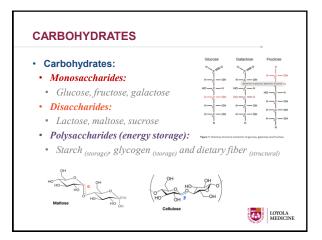


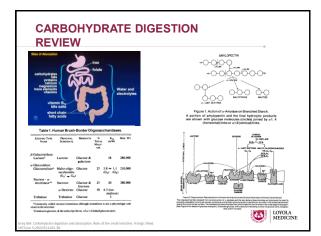


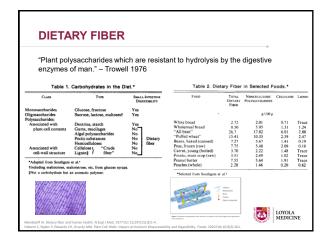


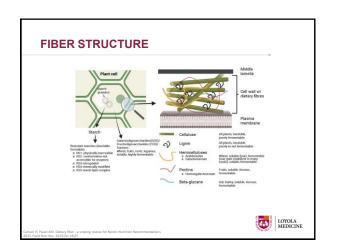


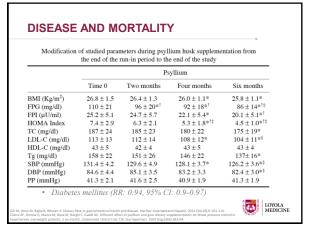


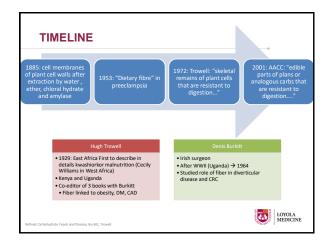


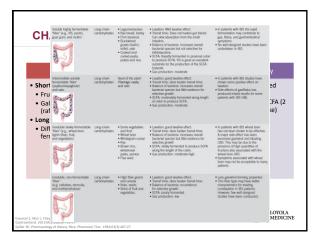


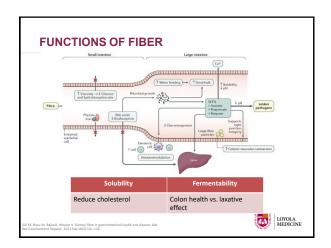


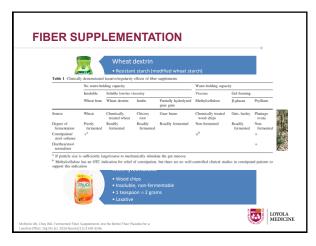


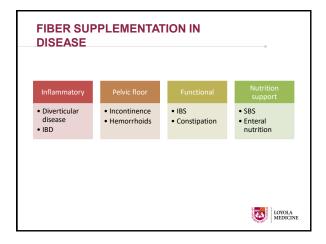


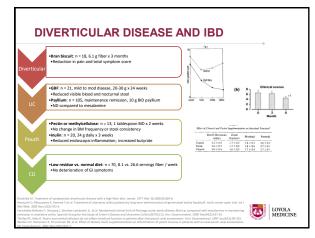


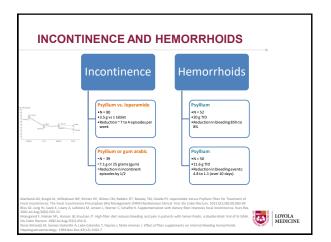


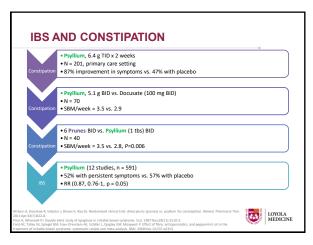


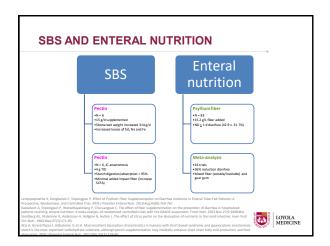












# **CONCLUSION**

- Understand difference in digestion of the polysaccharides: starch versus fiber
- · Goal intake: 25-35 grams per day; from diet
- · Solubility and fermentability (small bowel and colon)
- Limited data for strict supplementation
- First line options for incontinence, hemorrhoids, constipation





# Low FODMAP Diet: When Should It Be Tried?

Lori Welstead, RD

# **Low FODMAP diet:** when should it be tried?

Lori Welstead, MS, RDN University of Chicago Medicine Gastroenterology, Hepatology + Nutrition September 27, 2025

# Disclosures

- · I have no relevant financial relationships with any companies to disclose
- I will not discuss off label use or investigational use in my presentation

# Considerations

- Patient history and symptoms
- Constipation vs diarrhea
- Good or poor candidate for restrictive diet?
- Referral to Registered Dietitian

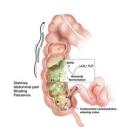
### RESEARCH

Fructose Malabsorption and Symptoms of Irritable Bowel Syndrome: Guidelines for Effective Dietary Management
SCHILD BOWEL ON PRES R. GESCH. NO. MR. PACP.

Shephard SJ, Gibson, PR. J Am Diet Assoc. 2006 Oct;106(10):1631-9.

# Fructose malabsorption

- Osmotic effect →water to distal small bowel & colon  $\rightarrow$  Speeds motility
- · Fructose and fructans rapidly fermented by bacteria in distal small bowel & proximal colon
- · Luminal distention; gas/bloat, abdominal pain, diarrhea



# Irritable Bowel Syndrome

- Rome I: 1994, Rome II: 2000, Rome III: 2006
- · Rome IV: 2016 Criteria:

Recurrent abdominal pain 1 day/week the last 3 months; with 2+ of the following:

- 1. Related to defecation
- 2. Associated with a change in frequency of stool
- 3. Associated with a change in form (appearance) of stool
- No "red flags":
   Weight loss, rectal bleeding, family history of cancer/IBD, unexplained hematologic or blochemical abnormalities, significant physical exam findings

https://theromefoundation.org/rome-iv/rome-iv-criteria/

# Low FODMAP Diet

- · Developed at Monash University
- 3 phases: temporary elimination → reintroduction → modified diet
- Carbohydrates that are poorly absorbed in small bowel or rapidly fermented in large bowel
- Effective treatment strategy for individuals with IBS



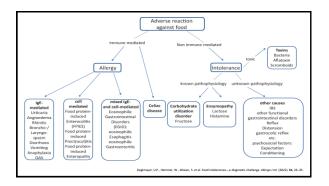
Patsy Catsos, RD

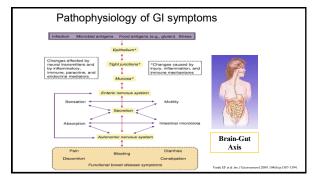
# Food Intolerance

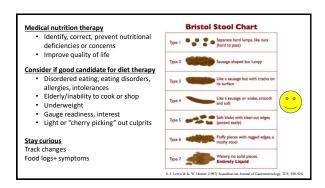
- Non-allergic food reactions that *do not* involve the immune system:
- Triggered at dose normally tolerated by the healthy population
   ~20% of the Westernized world's population suffers from a food intolerance<sup>2</sup>
- Might involve an organic pathophysiological process
- lactose intolerance due to lactase enzyme deficiency
- Food intolerances reported in irritable bowel syndrome (IBS)
  - 85-90% of IBS patients report symptoms with specific foods  $\ensuremath{^3}$

1. Turobull II., Adams 190, Gorard DA. The diagnosis and management of food allergy and food intolerances. Adment Pharmacol Ther. 2015;413–25 2. Tuck C. J., Bessekerski J.R., Schmid-Genedianier P., Polid D. Food Intolerances. Nationets. 2015;11:1084. doi: 10.3390/es11071684. 3. Patts at al Nutrals. Tood Intolerance Tood Margines and Essights and Shadows. 2012. 1:10(2):205.





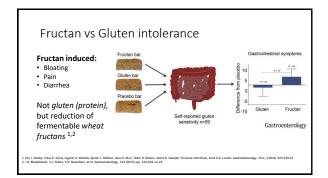


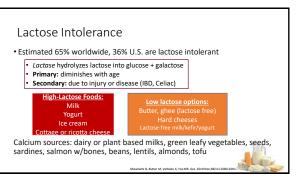


# Not all FODMAPs created equal Randomized, double-blind, FODMAP reintroduction study Patients reacted to average of 2 FODMAPs Fructans and galactans most likely to worsen abdominal pain and/or bloating Less restriction in diet is beneficial for patient's quality of life









# Sucrose Intolerance

- Low FODMAP diet does not restrict sucrose
- Not all patients benefit from a low FODMAP diet!
   Persistent gas, bloat, diarrhea? Consider other culprits
- • Sucrose intolerance or Congenital sucrase isomaltase deficiency (CSID) • Sucrose breath test
- Sucrase-isomaltase (SI) enzyme prominent intestinal disaccharidase that contributes to the final step of carbohydrate breakdown <sup>2</sup>
   Enzyme responsible for sucrase activity + 60%–80% of maltase activity in the intestine

Fernández-Bañares F, Esteve-Pando M, et al. Sugar malabsorption in functional bowel disease: clinical implications. Am J Gastroenterol. 1993.88(12):2044-50.
 Gerincke B et al. Biochimica et Biophysica Acta. 2017, Pages 817-826. Vol. 1865; pg 817-826.

### Histamine Intolerance

Presents as abdominal pain, flatulence, diarrhea, headache, pruritus, rhinorrhea, dysmenorrhea, respiratory obstruction, tachycardia + hypotension after consumption of histamine-rich foods

Food categories	Mg histamine/100 gram	Mg histamine/serving
Cheese: Gouda, Cheddar, Danish Bleu, Emmenthaler, goats cheese,	3-3-171	0.7-35 (20 gram)
Gorgonzola, Mascarpone, Parmesan		
Meat: fermented meat, hare, (dry) sausage, raw ham	3.0- 27	0.6-5.5 (20 gram)
Fish: herring, smoked mackerel, tinned fish (sardines), tuna fish,	0.8-16.5	0.6-11.5 (70 gram)
anchovy products	95-344	9.5-34 (10 gram)
Vegetables: egg plant, spinach, sauerkraut	2.5-11.5	5-23 (200 gram)
Alcoholics: beer and wine	0.6-1.6	0.6-1.6 (100 ml)
		1.2-2.2 (200 ml)
Fermented foods: Tamari, marmite, trassi, tempe	8.3-212	0.8-21 (10 gram)

# Mast Cells in IBS

- Role of mast cells in responding to therapies in IBS poorly understood
- • Mast cells produce **histamine**, **proteases**, **prostaglandins** + **cytokines** alter hypersensitivity + permeability defects, especially IBS-D  $^{\rm 1}$
- A low FODMAP diet in IBS-D improved colon barrier functions, symptoms and reduced mast cell activation<sup>3</sup>

Haisler, WL, Grabiuskiss, G, Singh, P, Owyang, C. Mast cell mediation of visceral sensation and permeability in initiable bowel syndrome. Neurogastroenterology & Motility. 2022; 34:e14339.
 Resea, Ballmer-Weber B, Beyer K, Fuchs T, Seien-Tebbe J, Ludger Klimekt, et al. German guideline for the management of advance reactions to injected histamine. Allergo J Int. 2017;26:72
 Schish B, al. A Sei-Comman (2017) (2

# Low FODMAP + microbiome changes

# Low FODMAP or control diet in IBD patients for 4 weeks

- •52% symptom relief vs 16% relief with control diet in IBD patients
- •Increased health related quality of life
- •No effect on inflammation
- Negative impact on fecal microbiome
   Decrease in beneficial bacteria (B longum, B adolescentis, F prausnitzii)

Goal to return to most varied diet; include back high FODMAP foods!

Cox SR, Lindsay JO, Fromentin S, Set al. Gastroenterology. 2020 Jan;158(1):176-188.e7. doi: 10.1053/j.gastro.2019.09.024.

# Nickel

- Systemic Nickel Allergy Syndrome (SNAS)
  - Systemic reactions upon ingesting nickel rich foods + nickel contact dermatitis
  - Gastrointestinal, respiratory, neurological  $^{\scriptsize 1}$
- Estimated to be 10-20% population worldwide<sup>2</sup>
  - Diagnosed by allergist and patch testing (48hr)
- · High prevalence of Nickel allergy in patients with IBS
  - Profound clinical response in IBS symptoms on low nickel diet for 3 months <sup>3</sup>
  - Low Ni diet allows many foods avoided in a low-FODMAP diet
    - Tramontana M, Bianchi L, Hansel K, Agostinelli D, Stingeni L. Nickel Allengy: Endocr Metab Immune Disord Drug Targets. 2020;20(7):992-100.
    - 3. Rizzi A, Nucera E, Laterza L, et al. Inritable Bowel Syndrome and Nickel Allergy: What is the Role of the Low Nickel Diet?. J Neurogastroenterol Motil. 2017;23(1):101-108

# Constipation considerations

- · What to ADD to the diet
- · Fluid, fiber in foods vs supplements, movement
- Kiwi: 2 per day
  - Consumption of fresh whole fruit has demonstrated clinically relevant increases in complete BM, improved measures of GI comfort in constipated populations compared to psyllium husk (1.2)
- Watermelon
- · Blackberries, cherries
- Prune/plum, peaches, nectarines
- 1. Bayer, S. B., Heenan, P., Frampton, C., et al. (2022). *Nutrients*, *14*(19), 4146 2. Gearry R, Fukudo S, Barbara G et al (2023) Am J Gastroenterol. 118(6):1058-1068

# Stay Curious

- Avoid unnecessary restrictions: most varied diet optimal
- Not one size fits all
   Tools in tool box
- All parties need to be able to pivot (patient + clinicians)
- May contribute to or worsen nutrient deficiencies
  - Monitor nutritional labs
  - Work closely with team (RD, GI Psych, Pelvic floor, MD, APN/RN)
- · Low FODMAP best in those with overt diarrhea
  - Avoid if history of eating disorders
  - Watch restrictions + goal of end point for diet

# Long-term considerations

- Lactose-free milk + lactase away from home
- Soaking/cooking/discarding water reduces FODMAP content of beans, portions matter
- Try varieties of apples to test tolerance
- · Broccoli crowns lower fructan than stems
- Consider gluten v. fructans in grainsSourdough
- · Trial of garlic/onion
  - · Chives, green onion, garlic infused oils
  - Garlic/onion powder high fructan

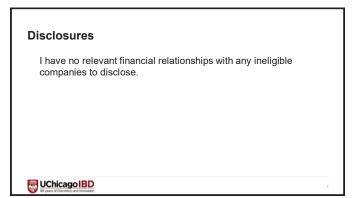




# **Eating For Two: Updates On Fiber For The Gut Microbiome**

Alice Cheng, MD

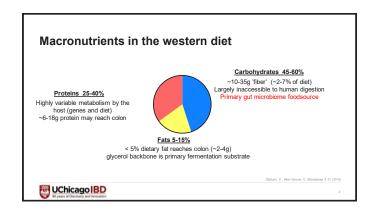
# Eating for two: fiber impacts on the gut microbiome and host Alice Cheng UChicago IBD

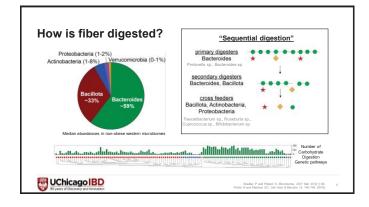


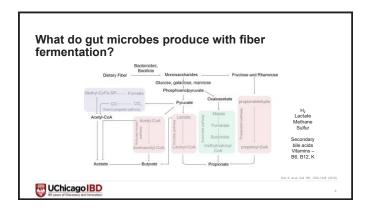
# **Objectives**

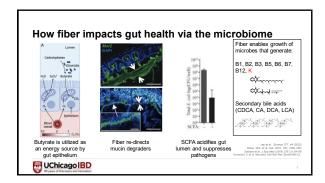
- · Understand how the gut microbiome digests fiber
- Understand the impact of digestion on metabolites and gut health
- · Appreciate the current and future applications of dietary fiber

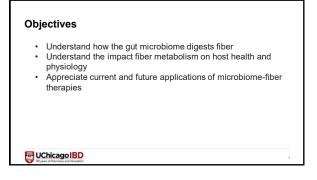


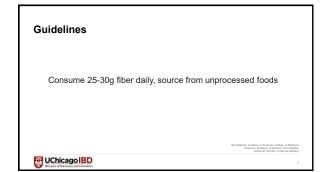


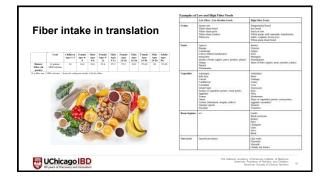


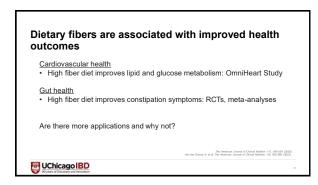


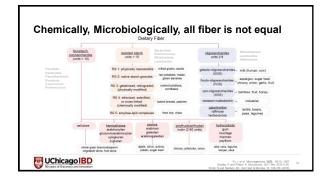


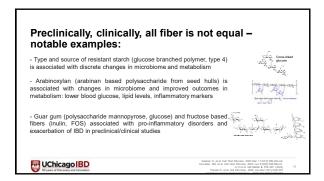


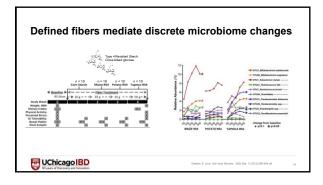


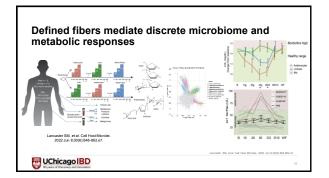


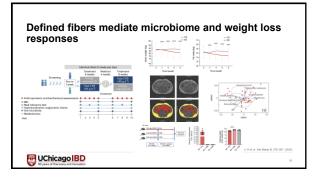


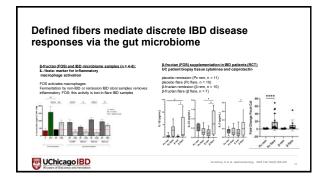


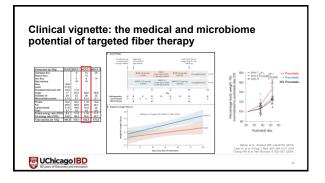












#### Where we are:

Fiber rich foods to promote gut health

Fiber rich foods to improve cardiovascular health and constipation

Defined fiber sources for constipation

- Psyllium husk (primarily arabinoxylan):
   Metamucil, Konsyl, generic psylium husk
   Methylcellulose: Citrucel

- Calcium polycarbophil: FiberCon
   Acacia Gum, Flaxseed

UChicago IBD

#### Where we are headed:

Defined food and defined fiber recommendations to promote gut health and metabolism

Defined food and defined fiber recommendations to improve disease outcomes

Metabolic syndrome: Arabinoxylan, RS2

Kwashiorkor: MDCF

#### **CME** question

Dietary fiber therapy improves human health through which following mechanisms?

- a. Increasing populations of butyrate-producing gut commensal microbes
- b. Suppressing populations of opportunistic gut commensals and pathogens
- c. Altering host lipid and glucose metabolism
- d. All of the above







### **KEYNOTE:** Clinical Approaches to the Management of Non-Short Bowel Dysmotility Leading to IF

Simon Lal, MD

Clinical Approaches to the Management of Non-Short Bowel Dysmotility leading to Intestinal Failure



Professor Simon Lal Clinical Director National Intestinal Failure Reference Centre Salford Royal & Manchester University



Clinical Approaches to the Management of Small Bowel 'Dysmotility?' leading to Intestinal Failure



Professor Simon Lal Clinical Director National Intestinal Failure Reference ( Salford Royal & Manchester Univer



### I have the following relevant financial relationships to disclose:

- Consultant for: Northsea, Ironwood, Vectiv Bio.
- Honoraria from: Takeda, Zealand, Fresenius Kabi, B Braun, Baxter.
- Grant/Research support from: Fresenius Kabi, Baxter and

I will not discuss off label use or investigational use in my presentation.

#### Salford National Intestinal Failure Reference Centre









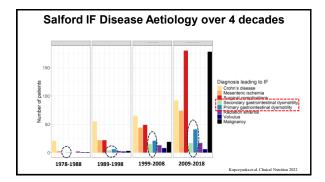


4 beds (1979)

13 beds (1996)

21 beds (2011)

Acute Severe (type 2) & Chronic (type 3) Intestinal Failure: 320 HPN patients (2025)





#### **ESPEN Chronic IF Registry**



travenous supplementation type and volume are sociated with 1-year outcome and major implications in patients with chronic testinal failure Gut 2020

> 1-year outcomes (2015-2016): 2194 patients with benign disease, from 51 centres in 22 countries.

> 2024 data:

	Benign	Malignant
Adult	10803	4265
Children	669	30

Underlying disease classification: IBD, CIPO (1º or 2º), Ischaemia, Radiation enteritis, Surgical Complications & 'other'.

#### Is the ESPEN Classification of 10 & 20 CIPO adequate for all forms of 'dysmotility' that lead to Intestinal Failure?

- $\textbf{A. YES,} \ primary \ \& \ secondary \ CIPO \ represent \ all \ types \ of \ small \ bowel$ dysmotility leading to Chronic IF.
- B. NO, there should be additional categories & considerations.

#### Example of a patient considered under ESPEN's 'CIPO' category: Case 1

- > 17-year-old male: transitioned from paediatric centre.
- > Intestinal Failure since birth.
  - Hollow Visceral Myopathy on small bowel histology.
  - Hydroureter & bilateral hydronephrosis.
- > Liver Histology (aged 15): mild periportal & perivenular
- ➤ Supra-cardiac venous thrombosis → femoral CVC in-situ.



#### Example of a patient considered under ESPEN's 'CIPO' category: Case 2

- 50-year-old female: 2-year history of nausea, intermittent vomiting & abdominal pain.
- Raynaud's but no other feature of connective tissue disease. No other co-morbidity.
- Endoscopies unremarkable, CT non-dilated small bowel.
- Delayed gastric emptying & small bowel contrast followthrough. No opioids or other medications affecting transit.
- $\succ$  Significant weight loss. Failed naso-gastric & naso-jejunal feeding  $\rightarrow$  commenced on HPN.
- > Antro-duodenal manometry requested......

#### Example of a patient considered under ESPEN's 'CIPO' category: Case 3

- ➤ 17-year-old female. 13-months in paediatric hospital. Intolerant of naso-enteral feeding due to pain → on PN as an in-patient.
- Endoscopies, MRI small bowel etc. unremarkable with normal calibre small bowel. Patient reports to be intolerant of contrast via naso-jejunal tube because of pain.
- Hypermobility, not fulfilling Ehlers Danlos Syndrome criteria. Wheelchair bound.
- Non-epileptiform seizures.
- Under investigation for Postural Orthostatic Tachycardia Syndrome.
- > IV Opioids & IV Cyclizine. NG for venting requesting a gastrostomy.
- > Declined psychological input, mother also not keen.

#### Which of the cases has underlying small intestinal dysmotility?

CASE 1 17-year-old male with hollow visceral myopathy since birth.



CASE 2 50-year-old female with delayed gastro-intestinal transit off opioids.



CASE 3 17-year-old female with hypermobility.

???

But all 3 would likely be included within the ESPEN classification of 'Primary CIPO'

#### Small Intestinal Dysmotility leading to Chronic Intestinal Failure

- > DEFINITION & CLASSIFICATION
- **➢ DIAGNOSIS**
- > TREATMENT
- > PROGNOSIS



#### Small Intestinal 'Dysmotility?' leading to Chronic Intestinal Failure

- > DEFINITION & CLASSIFICATION
- **➢ DIAGNOSIS**
- > TREATMENT
- **➢ PROGNOSIS**
- ➤ Patients with *possible* DGBI leading to HPN



#### Small Intestinal Dysmotility leading to Chronic Intestinal Failure

- > DEFINITION & CLASSIFICATION
- **➢ DIAGNOSIS**
- > TREATMENT
- > PROGNOSIS



The management of adult patients with severe chronic small intestinal dysmotility

Jeremy M D Nightingale , Peter Paine, John McLaughlin, Anton Emmanuel Loanne E Martin , Simon Lal on behalf of the Small Bowel and Nutrition Committee and the Neuropastroenterology and Motility Committee of the British Society of Gastroenterology

36 Recommendations

Classification 2
Diagnosis 5
Treatment 11

Strength of recommendation 36/36 'strong' Grade of recommendation

3/36: Level 4 Poor quality cohort/case series

33/36: Level 5 Expert opinion without critical appraisal

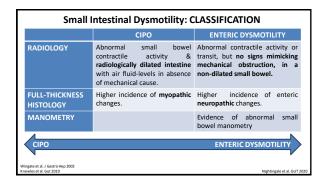
#### Small Intestinal Dysmotility: DEFINITION & CLASSIFICATION

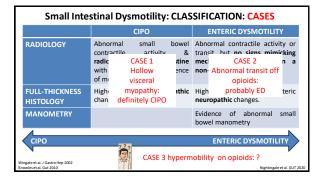
Small intestinal dysmotility occurs when there is a failure of coordinated intestinal propulsion, giving rise to the symptoms and signs of intestinal obstruction in the absence of a mechanical cause.

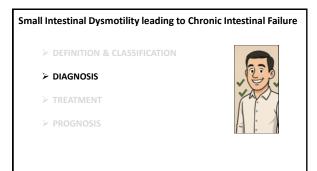
HISTOPATHOLOGICAL CLASSIFICATION			
MYOPATHY	NEUROPATHY	MESENCHYMOPATHY	
Smooth muscle	Enteric neurons	Interstitial cells of Cajal	
		<b>~</b>	
		RARE	

lightingale et al. GUT 20

Small Intestinal Dysmotility: CLASSIFICATION & CAUSES				
MYOPATHY		NEUROPATHY		
PRIMARY	SECONDARY	PRIMARY	SECONDARY	
Familial (e.g. hollow visceral myopathy, MNGIE)	Systemic Sclerosis & other connective tissue disorders.	Familial/congenital (e.g. Hirschprung's, mitochondrial disorders)	<b>Drugs</b> (e.g. opioids, anticholinergics)	
Acquired - Autoimmune - Diverticula	Muscular Diseases (e.g. muscular dystrophies, inclusion body myopathy, storage disorders)	Autoimmune (anti-neuronal antibodies, coeliac disease, ganglionosis)	General neurological (e.g. brainstem/spinal cord lesion, MS, Parkinson's, DM, hypothyroid)	
	Amyloidosis	Infective (e.g. Chagas, EBV, CMV)	Paraneoplastic (anti-Hu antibodies)	
Idiopathic	Radiotherapy	Idiopathic	Miscellaneous: MEN 2b, Porphyria	







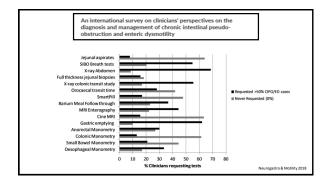
An international survey on clinicians' perspectives on the diagnosis and management of chronic intestinal pseudo-obstruction and enteric dysmotility Kasant<sup>k, g</sup> | Loris Pironi<sup>8</sup> | Giovanni Barbara<sup>8</sup> | Federico Bozzetti erda<sup>8</sup> | Francisca Johy<sup>8</sup> | Margevet Museh<sup>9</sup> | <sup>J.8</sup> | Michael Staun<sup>8</sup> | Kinga Szczepanek<sup>10</sup> | André Van Gossum<sup>11</sup> | 154 participants (82% European) – mostly IF Physicians & Neurogastroenterologists. ED N=154 (%) N-154 (%) time to > 95% felt CIPO/ED are difficult to diagnose diagnosis (particularly ED) because: <6mo 14 (9) 10 (6) Non-specific symptoms (70%) Lack of awareness among non-specialists (70%)
 Limitation in diagnostic tests (63%) 6-12 mo 45 (29) 24 (16) 1-5 yr 70 (45) 91 (59) Psychological co-morbidity (58%) Difficult in eliminating opioids as causal (47%) 5-10 yr 23 (15) 25 (16) 52% have patients with functional GI disorders > 10 yr 2 (1) 4 (3) that do not meet CIPO or ED criteria (especially UK at time of survey). Neurogastro & Motility 2018

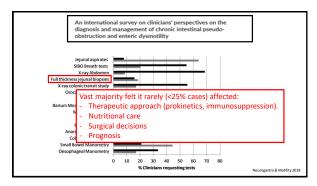
K. Twist<sup>1</sup> | J. Ablett<sup>2</sup> | A. Wearden<sup>1</sup> | P. Paine<sup>2</sup> | D. Vasant<sup>2</sup> | S. Laf<sup>2</sup> | S. Peten 20 patients, 13 on Home PN. Semi-structured interviews → thematic analysis. EXAMPLE QUOTE Feeling delegitimised 'Made to feel like it was in my head' 'These so-called doctors were convinced I had an eating disorder'. Lack of coherent  ${\it 'I think there should be more doctors out there what's trained up'}$ 'It's not put light at the end of the tunnel, it's put light in the tunnel' understanding Impact on 'My dad was like "well she's mental isn't she.." relationships  ${\it 'You feel guilty for dumping on them...} we wouldn't be able to do$ anything' Impact on mental 'I try and smile but inside I'm not happy' 'I understand how much physical and psychological health go together'. health Neurogastro & Motility 2018

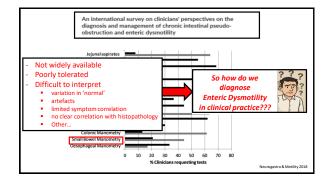
#### **Diagnostic Considerations in Small Intestinal Dysmotility** CONSIDERATION INVESTIGATION Mechanical Obstruction CT with oral contrast (MR, Contrast follow-through) Secondary Causes Thyroid, Coeliac, Diabetes. Auto-antibodies Scleroderma (anti-centromere, Sc170, M3R)/other connective tissue dis. Paraneoplastic CT Thorax, Abdomen, Pelvis & Auto-antibodies (Anti-Hu/other) Mitochondrial Disorder Thymidine & deoxyuridine, TYMP gene. Possible muscle biopsy. Contrast, Shape studies. Manometry Small bowel + other regions as indicated. Motility Gastric emptying, Smart Pill, Cine MRI. **Bacterial Overgrowth** Breath test (?), jejunal aspirate Full thickness small bowel. Other (e.g. amyloid). Histology Nightingale et al. GUT 2020

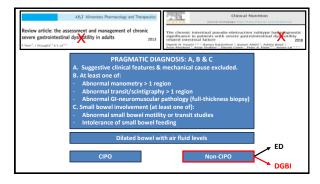
A. I regularly (>50%) request small bowel bacterial overgrowth breath tests.
B. I regularly (>50%) request small bowel manometry.
C. I regularly (>50%) request full thickness small bowel histology.
D. None of the above.

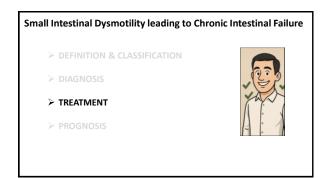
When investigating patients with small bowel dysmotility

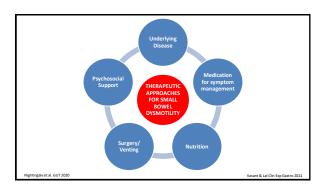


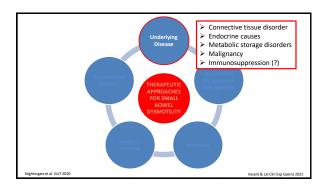


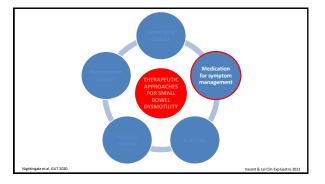




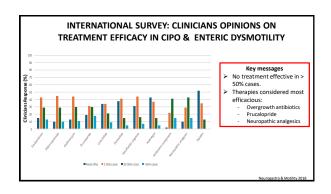


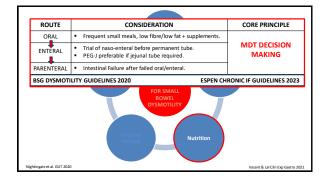


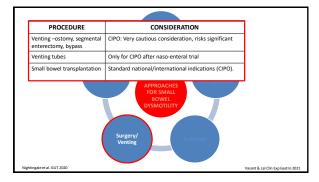


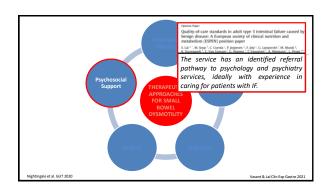


THERAPY	MEDICATION EXAMPLES/COMMENTS
LAXATIVES	Includes secretagogues such as linaclotide
PERIPHERALLY-ACTING MU-OPIOID RECEPTOR ANTAGONIST (PAMORA)	Naloxegol, Naldemidine.
PROKINETIC	Metaclopramide, domperidone, macrolides, prucalopride, pyrodostigmine, octreotide.
ANTIEMETIC	Ondansetron & D2 receptor antagonists, olanzepine. Avoid cyclizine.
ANALGESICS	Tricylics antidepressants, SNRI (duloxetine), pregabalin, (opioids).
ANTI-SECRETORY	Somatostatin analogues (scleroderma)
BACTERIAL OVERGROWTH	Rifaximin 1st line → cyclical if required

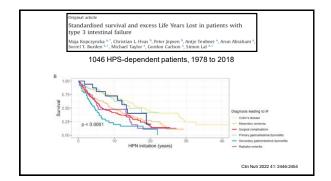


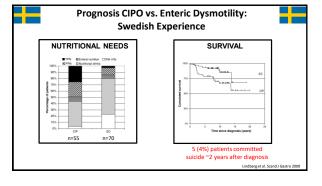


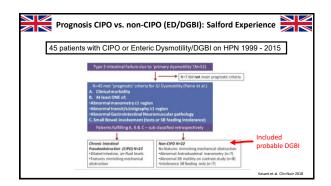


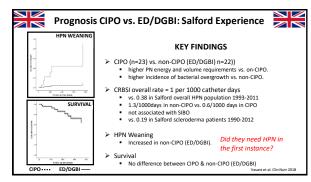


# Small Intestinal Dysmotility leading to Chronic Intestinal Failure > DEFINITION & CLASSIFICATION > DIAGNOSIS > TREATMENT > PROGNOSIS









## Small Intestinal 'Dysmotility?' leading to Chronic Intestinal Failure > DEFINITION & CLASSIFICATION > DIAGNOSIS > TREATMENT > PROGNOSIS

> Patients with possible DGBI on HPN

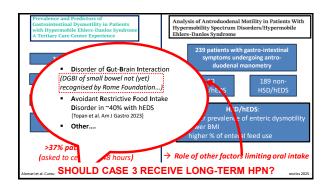
## A. I am seeing more patients with CIPO. B. I am seeing more patients with Enteric Dysmotility. C. I am seeing more patients with DGBI. D. I really don't know!

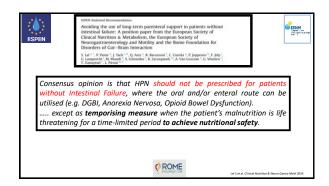
Patients referred for Home PN in recent years

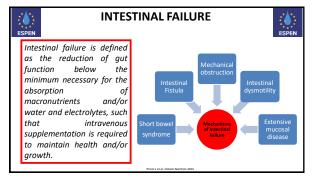
#### Example of a patient considered under ESPEN's 'CIPO' category: Case 3

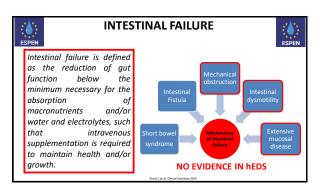
- > 17-year-old female. 13-months in paediatric hospital. Intolerant of naso-enteral feeding due to pain → on PN as an in-patient.
- Endoscopies, MRI small bowel etc. unremarkable with normal calibre small bowel. Patient reports to be intolerant of contrast via jejunal tube because of pain.
- Hypermobility, not fulfilling Ehlers Danlos Syndrome criteria. Wheelchair bound.
- Non-epileptiform seizures.
- Under investigation for Postural Orthostatic Tachycardia Syndrome.
- > IV Opioids & IV Cyclizine. NG for venting requesting a gastrostomy.
- Declined psychological input, mother also not keen.

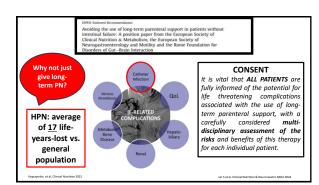
Does she have enteric dysmotility that may require long-term PN?











#### A multi-centre UK survey of patients with hypermobility disorders on intestinal failure units

- > Retrospective questionnaire 2012-2022; 7 centres.
- > 84 patients (98% female), mean age 35 (range 18-70). Only one patient from Salford!
- ▶ 65% POTS. Other diagnoses: Mast Cell Activation Syndrome, eating disorder, fibromyalgia, functional neurological disorder, bladder dysfunction, migraine.
- > 79% on opioids, 21% on parenteral cyclizine.
- 83% intolerance of oral/enteral nutrition; 26% weight loss.
- > CRBSI rate: 12.84/1000 catheter days.
- 37 (44%) ceased PN: CRBSI, endocarditis, poor quality of life on PN, non-compliance, factitious disorder.

Lam et al. BSG Abstract 2025



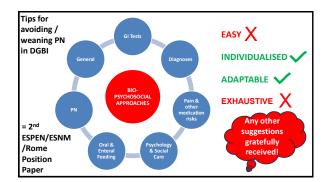


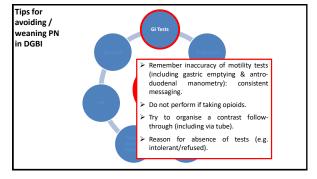
#### RISKS vs. BENEFITS: IF vs. non-IF

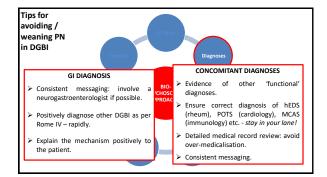
it is incumbent on clinicians not to commence long-term parenteral support in patients without IF, apart from in exceptional circumstances, where there may be a clear risk to life without it and where the patient has been fully informed of the life-threatening risks and burden associated with parenteral therapy.

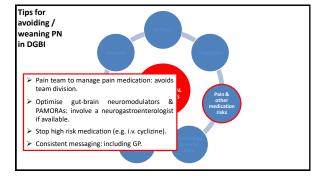
On the rare occasions that PN commencement is required to treat life-threatening malnutrition

in conditions such as DGBI, opioid bowel dysfunction and eating disorders, it should only be prescribed for a time-limited period to achieve nutritional safety, while the wider multi-disciplinary team focus on more appropriate biopsychosocial holistic and rehabilitative approaches to manage the patient's primary underlying condition.



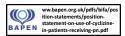






#### CASE EXAMPLE: Female with 'hEDS' transferred from another IF centre

- ➤ 3 weeks after discharge home → letter from Member of Parliament: 'Why have you stopped IV Cyclizine'.
- Response:
- •Engaged the GP & asked the hospital solicitor to respond on my behalf.
- IV Cyclizine cessation in keeping with BAPEN Recommendations 2019. On multiple oral tablets. We have commenced an alternative, safer oral anti-emetic.
- > Stop high risk medication (e.g. i.v. cyclizine).
- > Consistent messaging: including GP.



#### Tips for avoiding / weaning PN in DGBI

- > Early psychological/psychiatric involvement.
- > Medical record/GP review for previous psychological issues.
- > Support associated care needs (e.g. ADHD, PTSD).
- ➤ Identify associated diagnoses & issues e.g.
  - eating disorder, avoidant/restrictive food intake disorder (ARFID)/Restricted Intake Self Harm (RISH).
  - Fabricated or induced illness
  - Factitious disorder imposed on another with safeguarding need/maladaptive social attachments.
- Psychological therapies; psychoeducation around mind-body interactions; motivational interviewing; symptom validation & promoting self-management; family therapy....There's not enough room on this slide to include everything (and I should stay in my lane)!



#### Tips for avoiding / weaning PN in DGBI

- Promote effortful oral feeding with support.
- Positive risk management of low stable body weight and document adequate hydration.
- Avoid invasive/permanent feeding tube unless life-threatening malnutrition.
- ➤ Risk stratified approach if enteral tube feeding considered: NG→NJ (→PEG-J)
- ➤ IF PN started, avoid complete cessation of enteral feed (or even fluid) if tube in situ → build up at all opportunities.
- ➤ Blinded sham feeding?

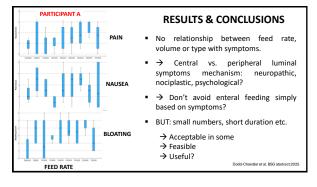


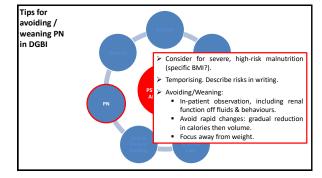
#### A pilot double-blinded randomized protocol to explore jejunal feeding intolerance in adults: feasibility, acceptability & utility

- Adults with jejunal feeding intolerance and no clear structural cause (tubes in situ).
- Up to 3 days of 10 hours of jejunal feed daily, via a double-blinded jejunal feeding system (a covered two pump design)
- Feed rate (active feeding pump) altered hourly via a randomization website, between 0-70ml/hr. Feed type also varied on subsequent days.
- Sham feeding pump was set at a constant rate of 50mls/hr for the full feeding period to create the noise of continuous feeding. Both pumps covered.
- Participants scored their perceptions of pain, nausea and bloating every 30 minutes using a Visual Analogue Scale of 0-10.
- 4 patients recruited over 9 months.

Dodd-Chandler et al. BSG abstract 2025







#### CASE EXAMPLE: Female (no structural GI cause) unable to tolerate any jejunal feeding → commenced on PN in referring hospital

- ➤ Admitted to Salford IFU.
- Noted to tolerate 60mL water flush after medications via tube.
- > Contrast via tube: 30 minutes to caecum.
- Perches on weighing scales.Weak handgrip strength but firm handshake.
- Discourages visitors but buys food from Marks & Spencers.
- > PN weaned (calories first and then volume with normal renal function)
- Discharged to referrer.

#### Avoiding/Weaning:

- In-patient observation, including renal function off fluids & behaviours.

  Avoid rapid changes: gradual reduction
- in calories then volume.
- Focus away from weight.

#### E-mail from Canadian colleague July 2025

I have noted similarities with the behavior of our EDS population. Most of them don't lose weight, furthermore, they gain weight on TPN...This is inconsistent with their reports of extremely low amount of food intake (~400 kcals x day).

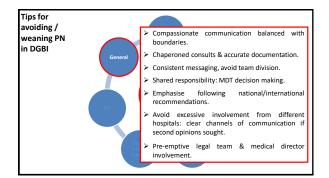
However, every single time we start the weaning process, becomes a fight with the patient (and family-often overprotective parent) because the patient "is not eating" and they fear the patient will become undernourished. This situation is becoming a frequent issue in these patients, and delays significantly the weaning process.

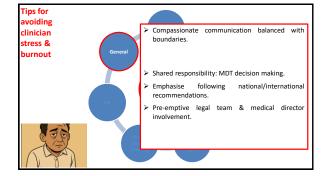
#### Case: 44 year old female on IV fluids only from another centre.

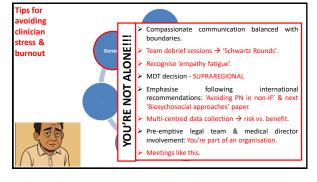
'I only chew & spit a cheese sandwich once/week'. BMI 28.

Case: 36 year old male on HPN.

'Unable to eat'. Admitted with CRBSI. Witnessed eating a Big Mac + large Coke. Approaches?

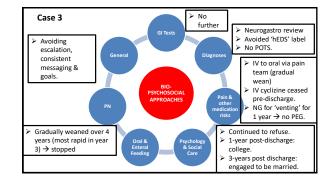




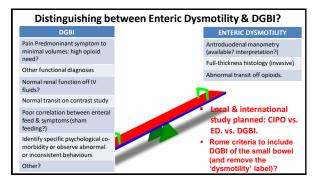


#### Example of a patient considered under ESPEN's 'CIPO' category: Case 3

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- Non-epileptiform seizures.
- Under investigation for Postural Orthostatic Tachycardia Syndrome.
- > IV Opioids & IV Cyclizine. NG for venting requesting a gastrostomy.
- > Declined psychological input, mother also not keen.
  - → Ultimately she was discharged on HPN



SUMMARY & FUTURE PERSPECTIVES		
DEFINITION & CLASSIFICATION	Small bowel dysmotility is defined as a failure of coordinated intestinal propulsion in the absence of a mechanical cause; this should not encompass patients with likely DGBI.	
DIAGNOSIS	Further work is required to help to positively diagnose Enteric Dysmotility and, in particular, distinguish it from patients with possible DGBI.	
TREATMENT	Current therapeutic options for intestinal dysmotility provide limited efficacy in most patients.	
PROGNOSIS	Distinguishing between CIPO & Enteric Dysmotility provides prognostic value.	
POSSIBLE DGBI	Avoiding & weaning PN in patients with likely DGBI requires a holistic, dynamic, multi-disciplinary & multi-faceted approach tailored to the individual's specific needs.	



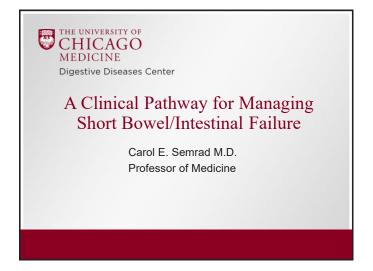


Thank-you.....
Questions?



#### A SB Pathway for Managing Short Bowel/Intestinal Failure

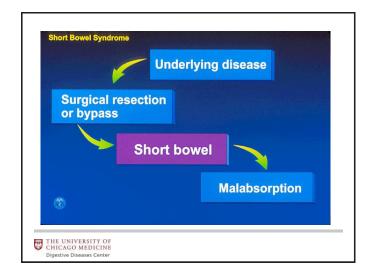
Carol Semrad, MD



#### Disclosures

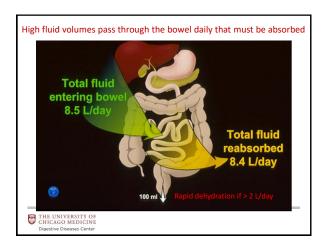
· Research support Zealand Pharmaceutical, Copenhagen

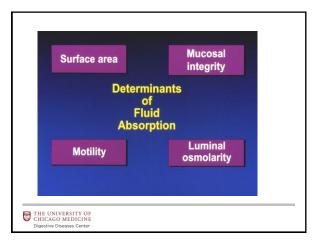


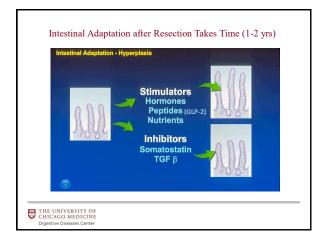


Short Bowel Pathophysiology
Important Concepts

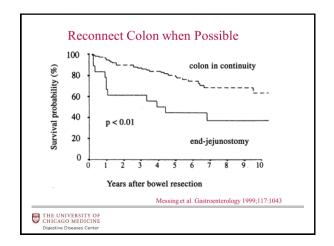


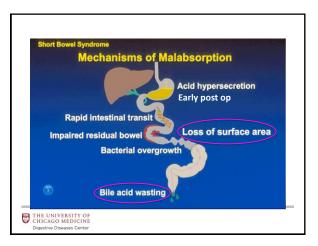


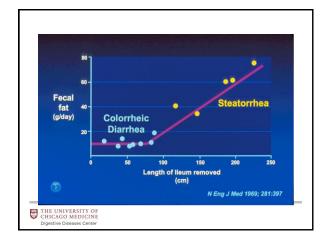


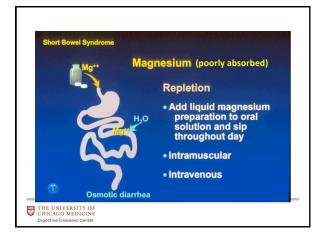


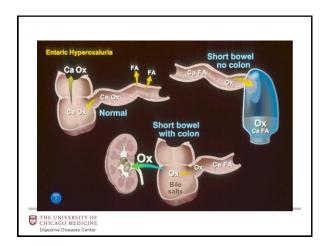






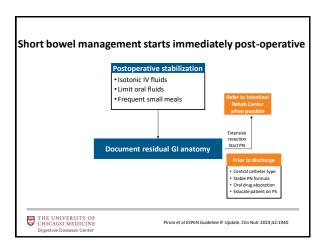


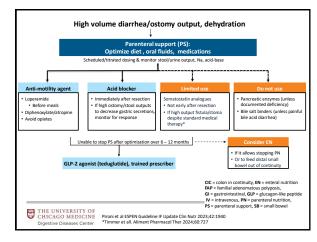


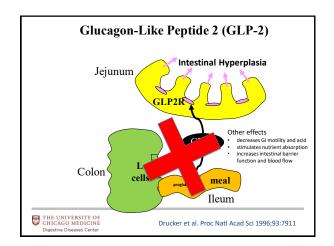


## Clinical Pathway for Management of Short Bowel Syndrome AGA sponsored multidisciplinary panel of experts 2024: lyer, Semrad, Issokson, Lal, Gabe, Nightingale, Culkin, Jeppesen, Wall, Micic, Paski, Ou, Tappenden, Winkler, Mercer, Borges, Kakani

THE UNIVERSITY OF CHICAGO MEDICINE Digestive Diseases Center

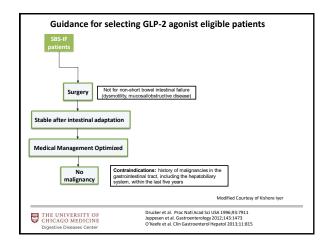


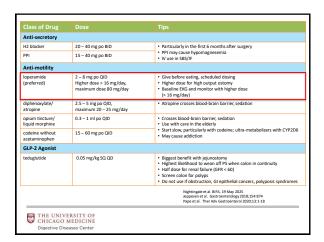


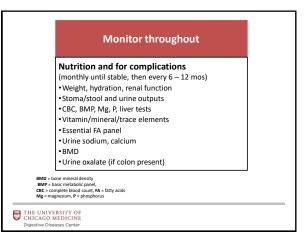


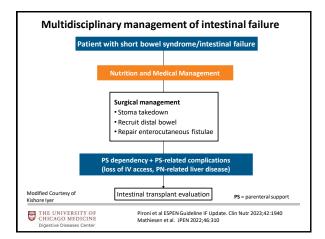


Study	Pt#	Dose	Time weeks	>20% reduction in PN volume	Impact
Teduglutide <sup>1,2</sup>	35 25	0.05 mg/kg/day SQ	24 52	46% (p=0.007) 68%	1 day off PN 4 stopped PN
Glepaglutide <sup>3</sup> Phase 3 Trial	35 35	10 mg once a week SQ 10 mg twice a week SQ	24 24	NS 66% (p=0.024)	5 stopped PN
Apraglutide <sup>4</sup> Phase 2 Trial Phase 3 Trial	8	5 mg SQ once a week 10 mg SQ once a week	4	not published	Increase in urine output
¹-lieppesen et al. Gastroenterology 2012;143:1473 ²-O'Keefie et al. Clin Gastroenterol Hepatol 2013;11:815 ³-lieppesen et al. Gastroenterology 2025;168:701 ⁴Eliasson et al. JPEN 2022;46:896					

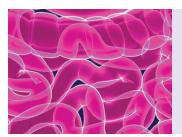








# Recognize and treat short bowel syndrome Starts with small bowel resection post-operative Block endogenous secretions, slow motility Start parenteral support when high stool/ostomy outputs Manage inpatient to outpatient setting Refer those with IF to a specialized nutrition support team/intestinal rehab center Maximize diet and medical therapy before considering a GLP-2 agonist Monitor closely for nutrition, hydration, vitamins and minerals, bone health Monitor urine oxalate for those with colon in continuity Monitor for polyps for those with colon on GLP-2 agonist Loss of venous access or PN-related liver disease refer for Intestinal Transplant



#### **Recognizing Intestinal Failure and When To Intervene**

Ankita Sissleman, MD

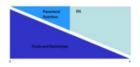


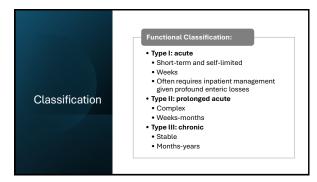


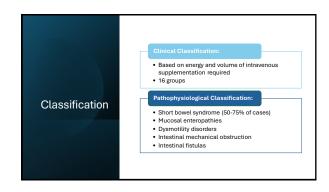
# Define Intestinal Failure Identify different classification systems Recognizing signs and symptoms Determine timing of intervention

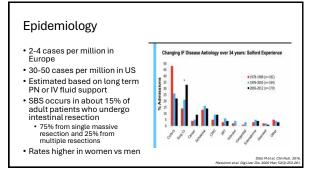
## Intestinal Failure • Inability to maintain one's nutrition and hydration without intravenous or enteral support

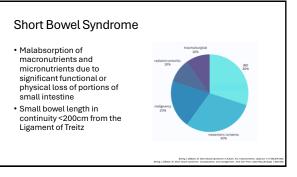
- Multiple classification systems
- Early recognition improves outcomes and reduces complications

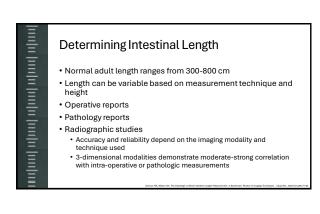


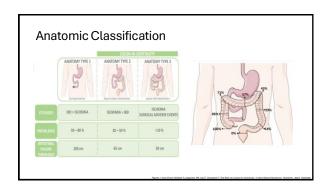


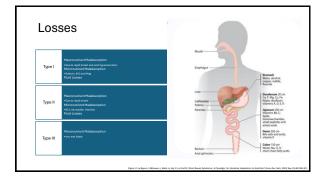






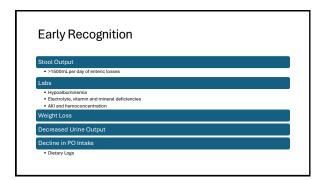


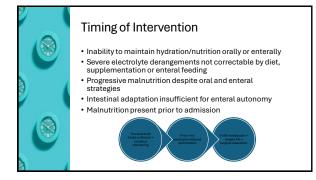


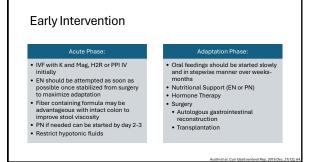


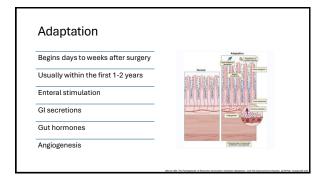








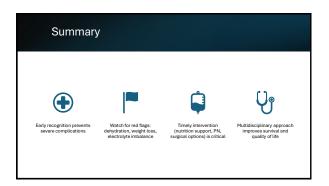




## Malnutrition Infection Wound healing Renal function Length of stay Morbidity and mortality

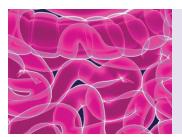






#### MOC Question

- Which clinical scenario most strongly suggests progression to chronic intestinal failure?
- A. A patient with mild diarrhea after small bowel resection B. A patient with high-output jejunostomy C. A patient with Crohn's disease controlled on biologics D. A patient with intermittent partial small bowel obstruction



### The Rise of Nutrition Support Use in Disorders of Brain Gut Interaction George Ou, MD

### The Rise of PN Use in Disorders of Brain Gut Interaction

George Ou, MD, MPH Gilmore Symposium 2025 September 27, 2025

#### Disclosure

- Consulting honorarium from Knight Therapeutics & Celltrion
- Speaker honorarium from Baxter
- I will not be discussing off-label use of medications

#### Objectives

- To describe the types of disorders of gut brain interactions (DGBI) that may be referred for advanced nutrition intervention
- To outline a systematic approach to the nutrition management in patients with DGBI and severe symptoms resulting in malnutrition.
- To appreciate why parenteral nutrition support is not an ideal nutrition intervention for this patient population.

#### Disorders of Gut-Brain Interaction (DGBI)

- Defined in ROME IV Criteria (fka Functional GI Disorders)
- GI symptoms due to complex interactions among factors
  - Microbial dysbiosis within the gut
  - Altered mucosal immune function
  - Altered gut signaling (visceral hypersensitivity)
  - CNS dysregulation of the modulation of gut signaling and motor function

#### **DGBI**

- Esophageal
- Gastroduodenal (e.g. functional dyspepsia)
- · Bowel (e.g. IBS)
- Centrally mediated disorders of GI pain (e.g. narcotic bowel)
- Gallbladder
- Anorectal

#### Nutrition implications of DGBI

- $\bullet$  Avoidant/restrictive eating is more common in pts with DGBI
  - 22.8% vs 18.2 % (Blomsten et al. Am J Gastroenterol 2025)
- Rarely leads to significant malnutrition unless there is truly an underlying dysmotility, which is difficult to differentiate from DGBI
- Nutrition management usually through oral diet modification, ideally with assistance from dietitians.

#### Case study

- ~20F w/ chronic symptoms of N/V, pain, diarrhea/constipation
- PMH: **POTS**, **hEDS**, **impaired gastric emptying**, Migraines, Complex regional pain syndrome, PTSD, somatization, asthma, lap chole, urinary retention
- SH/FH: Non contributory. No prior cannabis use.
- Investigations: EGD, Colonoscopy, MRE, CTA, SBFT, serum tryptase levels all within normal

#### Case study

- Admitted to hospital with **worsening oral intolerance** since lap cholecystectomy.
- Nutrition support escalated from  $\mathbf{NG}$  to  $\mathbf{ND}$  to  $\mathbf{GJ}$  feeding.
- Also required outpatient IV fluids to support POTS.
- Referred for consideration of home PN.

#### Familiar sounding cases in your practice?

• You are not alone.

#### Recent increase in hEDS/POTS/MCAS

- Increasing incidence vs better recognition
  - Better defined diagnostic criteria for clinicians
  - Increased information sharing among patients and HCP
- Often referred to gastroenterology for GI symptoms and/or feeding
  - Hypermobile joint syndrome screening is now recommended (AGA 2025)

Visant et al. Neurogastroenterol Motil. 2020 Fikree et al. Clin Gastroenterol Hepatol. 2014

#### Ehler-Danlos syndrome (EDS)

- Connective tissue disorder multiple subtypes
- Hypermobile EDS (hEDS) = most common (80-90%)
  - Clinical diagnosis based on 2017 criteria (Malfait et al. 2017)

  - High prevalence of DGBI
     98% of hEDS met ROME IV criteria vs 47% in controls (Lam et al. CGH 2021)
  - Inquire about joint hypermobility in patients with DGBI (AGA 2025)
  - Association with abnormal GI transit changes in collagen structure

#### Overlapping syndromes with hEDS

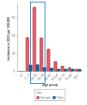
- Reports of association with POTS and MCAS
  - Further testing should be guided by compatible systemic symptoms
  - Lack of robust clinical data with rigorous diagnostic criteria for linkage



Raj et al. CMAJ 2022 Aziz et al. Clin Gastroenterol Hepatol 2025

#### Postural orthostatic tachycardia syndrome (POTS)

- Incidence of 24.8 (0.02%) per 100,000
- Prevalence of 100 (0.1%) per 100,000
- Higher rates of health care utilization
- Post-viral onset and/or immunological



#### POTS diagnosis

- . ↑ HR ≥ 30bpm w/in 10min of upright posture w/out orthostatic hypotension + orthostatic symptoms that resolve upon recumbency
  - Sustained HR increase ≥ 4-5 minutes

  - SBP drop by <20mmHg
     More sensitive in morning
  - Head-up tilt table test more sensitive
- · 24h Holter to detect inappropriate sinus tachycardia
- · Association with abnormal GI transit dysautonomia

Raj et al. CMAJ 202

#### Mast cell activation syndrome (MCAS)

- Episodic surge of mast cell mediators without mastocytosis
- Flushing, urticaria, dyspnea, headache, **nausea, vomiting, diarrhea, abdominal pain** significant overlap with IBS
- Screening with tryptase level at baseline and during 1-4h of flare

Where does parenteral nutrition support fit in the management?

#### PN use is clearly indicated in intestinal failure

- Most accepted / accurate definition of Intestinal Failure (ESPEN 2015)
  - 1. Reduced gut function below the minimum necessary for the absorption of macronutrients and/or water and electrolytes
  - 2. Such that intravenous supplementation is required to maintain health and/or growth
- PN Benefit (survival) > PN Risk (CVC, metabolic, psych, QoL)

Lal et al. Clinical Nutrition 2024

#### PN use in non-intestinal failure is less certain

- Risk vs Benefit less clear than in that in intestinal failure
- ? Survival benefit, quality of life (QoL)
   Complications: CVC (DVT, infectious), metabolic, QoL of patient and caregiver
- There is increasing referral of hEDS patients for PN (Vasant et al. Neurogastroenterology & Motility. 2020)
- ~ ~10% with POTS required IV fluids and/or PN in one retrospective cohort (Tseng et al. JPEN 2018)
   30% had concomitant EDS
   Concomitant WCAS (24%) more likely to have PN dependence

  - healthcare utilization, impaired gastric emptying and opioid use

#### ESPEN Endorsed Recommendation

Avoiding the use of long-term parenteral support in patients without intestinal failure: A position paper from the European Society of Clinical Nutrition & Metabolism, the European Society of Neurogastroenterology and Motility and the Rome Foundation for Disorders of Gut-Brain Interaction

S. Lal \*\*, P. Paine \*, J. Tack \*-, Q. Aziz \*, R. Barazzoni \*, C. Cuerda \*, P. Jeppesen \*, F. Joly \*, G. Lamprecht \*, M. Mundi \*, S. Schneider \*, K. Szczepanek \*\*, A. Van Gossum \*, G. Wanten \*, T. Vanuytsel \*, L. Pironi \*\*, Q. Wanten \*, T. Vanuytsel \*, L. Pironi \*\*, Q. Wanten \*, T. Vanuytsel \*, L. Pironi \*\*, Q. Wanten \*, T. Vanuytsel \*, L. Pironi \*\*, Q. Wanten \*, Q. W

"As there is also no established evidence that patients with HD/hEDS have small intestinal malabsorptive failure, PN should be avoided other than again in life threatening extremis as a temporary bridge to pain management and rehabilitative MDT therapies. This may be particularly important since anecdotal clinical experience suggests that HPN-related CRBSI are more frequent in this patient cohort."

#### Management of GI symptoms = supportive

- · Lack of clinical trials in managing GI symptoms
- Target most prominent GI symptoms and abnormal results of testing (e.g. Gastric emptying test)  $\,$
- · Setting realistic goals as symptom resolution may be unattainable
- Consider medication side-effects on the bowel (e.g. CCB, anti-cholinergics)
   Avoid opioids dysmotility, hyperalgesia, (risk of CRBSI in PN)
- · Psych support (brain-gut behavioral therapy)

#### Recommendations

- Early diagnostic clarification is key to future success
   Assess for impaired gastric emptying in hEDS + POTS with UGI symptoms
   Gastric emptying and bowel motility can be impaired in severe malnutrition
  - · Assess for functional defecation disorders in hEDS with LGI symptoms
- Avoid premature/"over" diagnosis unless a clear cause identified.
   Risk of mis-label perpetuating in electronic medical record
   May lead to mistrust and confusion for patient and other HCP
- Rule out eating disorder (e.g. anorexia vs bulimia) different from disordered eating (ARFID)

#### Recommendations

- Timely referral to specialist (clinical nutrition) teams for management to build therapeutic rapport.
- Involve multidisciplinary team
   Rheum, Cardio, Neuro, Gl, Allergy/immunology, Radiology, Surgery...etc
   Psychiatry, Complex pain
- · Limit / withdraw opioid use

  - Opioid contributes to dysmotility & hyperalgesia
     Opioid associated with worse outcomes on HPN / ↑ CRBSI (Dibb et al Clin Nutr 2017)
- Minimize medical institutionalization avoid unnecessary testing / intervention (e.g. multiple endoscopies/colonoscopies, CT scan...etc)

#### Recommendations

- · Nutrition support is similar to that for patients w/ intestinal dysmotility
- Oral nutrition > enteral nutrition > parenteral nutrition, but they are not mutually exclusive and may be complementary
- Better outcomes with oral intake in patients with intestinal dysmotility on long-term  $\ensuremath{\mathsf{PN}}$
- CRBSI risk may be higher vs other IF's (Vasant et al. Clinical Nutrition 2018)
- · Long-term goal is to resume PO and/or EN to reduce M&M risk

Lehmann et al. Nutr Clin Pract. 202

#### Case study (the outcome)

- Meds tried: Prucalopride, metoclopramide, domperidone, erythromycin, prochlorperazine, ondansetron, diMENhydrinate, CBD oil, nabilone, pantoprazole, esomeprazole, mitrazapine, bupropion, duloxetine, sumatriptan, topiramate, pregabalin, senna, PEG3350, linaclotide, plecanatide, tenapanor, ivrabidine, midodrine
- Multiple disciplines involved: internal medicine, neurology, rheumatology, complex pain, psychiatry, urology, cardiology, gastroenterology, radiology, surgery, outpatient dietitian, physiotherapy
- . CLABSI with candida sp required IVAD replacement
- GJ converted to G tube for meds and surgical J tube for feeds.
   Complicated by J tube site infections and tube falling out (not replaced after 3 months)

#### Case study (the outcome)

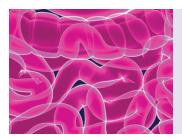
- Addressing main bothersome symptoms = Constipation.
  - Multiple pharmacological agents for constipation: PEG3350, linaclotide, plecanatide, tenapanor, prucalopride
  - Defecography generalized pelvic floor descent on straining-defecation and small anterior rectocele and there was inappropriate sensation of incomplete voiding, despite total evacuation → Pelvic floor physio
- Patient remains on combination of PO, EN and outpatient IV fluids

#### Summary

- hEDS / POTS / MCAS may present with symptoms of DGBI
- Management of GI symptoms is largely supportive
   Focus on the patient's most bothersome symptom.
   Avoid opioid and anticholinergic
- Multidisciplinary involvement may be more successful
- Long-term parenteral nutrition support is rarely needed and should be carefully evaluated in this patient population

#### Thank you

- Questions?
- Email: george.ou@phc.ca



#### Diet and Oral Hydration: Does It Make A Difference In IF?

Elizabeth Wall, MS



#### **Disclosures**

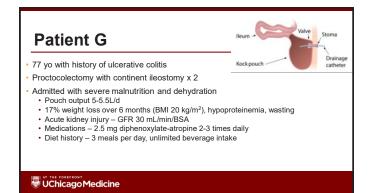
 I have the following relevant financial relationships to disclose: Consultant for Ironwood Pharmaceuticals and Zealand Pharma

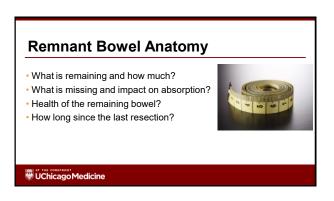
UChicago Medicine

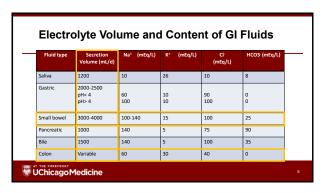
#### Learning objectives

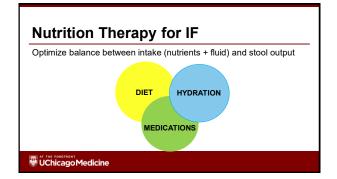
- 1. Role of diet for patients with intestinal failure (IF)
- 2. Utility of oral rehydration solutions (ORS) in short bowel/IF

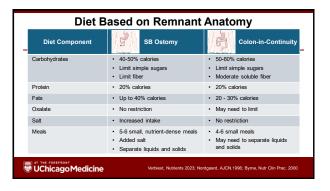
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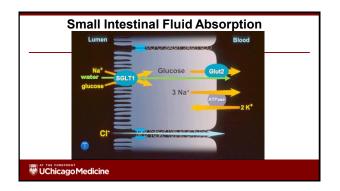


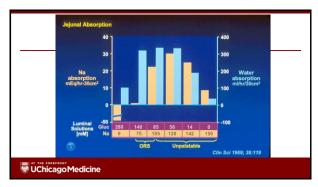


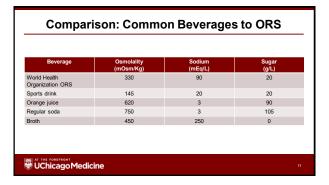








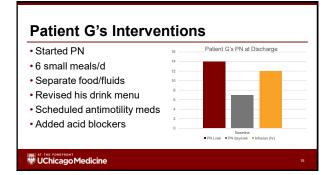


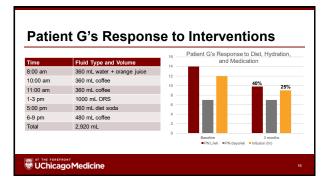




# How Much ORS is Enough? • Start with 2 cups (~500 mL) daily • Monitor urine (and stoma) output – goal > 1L/d • If urine output increases →slowly increase ORS volume > Sip, sip, sip • Barriers – taste and tools to mix/consume • Help patients create a daily "drink menu"

Time	Fluid type and volume
8:00 am	360 mL orange juice
10:00 am	480 mL coffee
11:00 am	480 mL coffee
1-3:00 pm	1000 mL sports drink
5:00 pm	360 mL diet soda
6-9:00 pm	1000 mL ounces coffee
Total	3,680 mL

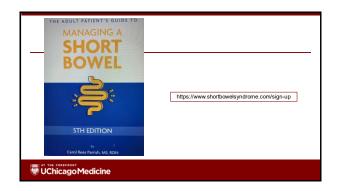




#### **Summary**

- · Know the remnant bowel anatomy and function to guide management
- · Optimize synergy between diet, hydration, and medications
- Engage the patient in their care and provide the tools needed for success
- · Provide consistent and longitudinal monitoring ➤ Adjust each sphere of care
  - » Diet
  - » Hydration oral and parenteral
  - » Medications
  - » Micronutrient supplements





#### Sources

- Byrne TA, Veglia L, Camelio M, et al. Beyond the prescription: Optimizing the diet of patients with short bowel syndrome. Nutr Clin Pract 2000; 15:306-311.
  Wall, E: ORS: The Solutions to Optimize Hydration in Short Bowel Syndrome; Pract Gastroent. 2020;24-31.
  Nordgaard I, Hansen BS, Mortensen PB. Importance of colonic support for energy absorption as small-bowel failure proceeds. Am J Clin Nutr. 1996;46(2):222-231. proceeds. Am J Clin Nutr. 1996;64(2):222–231.

  Pironi L Definition, classification, and causes of short bowel syndrome. Nutr Clin Pract. 2023;38:S9-S16.

  Verbiest A, Jeposeen PB, July F, Yanutysel T. The Role of a Colon-in-Continuity in Short Bowel Syndrome. Nutr Clin Pract. 2023;15:528.

  Roberts K, Shah ND, Parrish CR, Wall E. Navigating nutrition and hydration care in the adult patient with short bowel syndrome. Nutr Clin Pract. 2023;38:S59-S75.

  Lakananurak N, Wall E, Catron H, Delgado A, Greff S, Herlitz J, et al. Real-world management of high stool output in patients with short bowel syndrome. An international multicenter study. Nutrients. 2023; Cuerda C, Piron L, Arends J, Boczett E, Gillanders L, Jeposeen PB, et al. ESPEN practical guideline: Clin Clin Nutr. 2021;40:5196-5220.

  Staden GE, Dawson AM. Internationality all setween the Absorptions of Glucose, Sodium and Water by the Normal Human Jejunum. Clin Sci., 1969;38:119-132.



#### **Travel Through Time: NASH to MASLD/MASH**

Sonali Paul, MD



#### A Wrinkle in Time From NASH to MASH

Sonali Paul, MD MS Associate Professor of Medicine Center for Liver Diseases

#### **Disclosures**

• Research support from Gore



#### **Objectives**

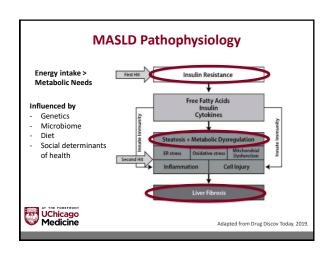
- Review updated steatotic liver disease terminology
- Explore underlying disease mechanisms
- Recognize varied clinical presentations
- Define a clinical practice algorithm for MASLD evaluation

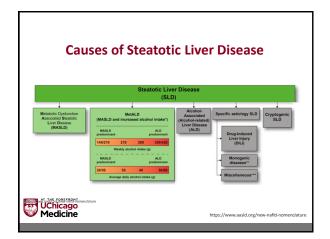


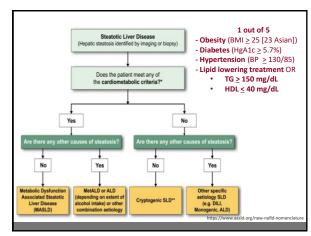
NAFLD / NASH
It's more than just "no alcohol" ...

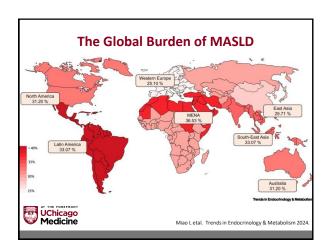


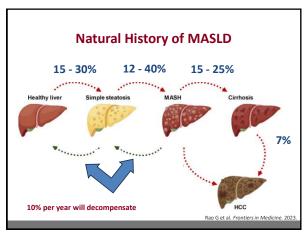
# NAFLD / NASH It's more than just "no alcohol" ... Insulin Resistance is Key MASLD Metabolic Dysfunction-Associated Steatotic Liver Disease MASH Metabolic Dysfunction-Associated Steatohepatitis UChicago Medicine

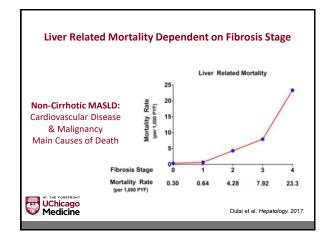


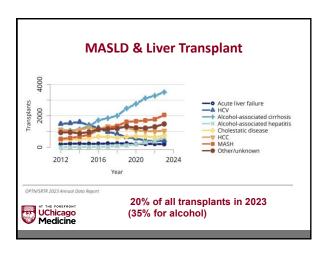












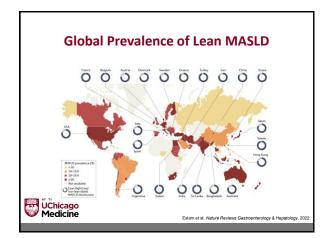


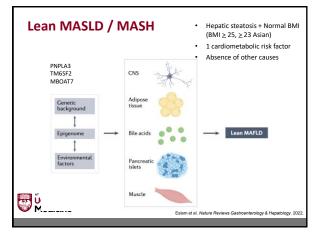
#### **Lean MASLD: Beyond Obesity**

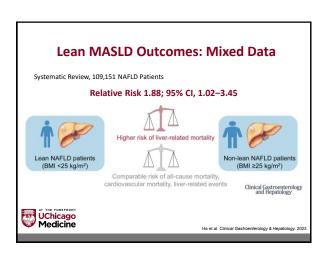


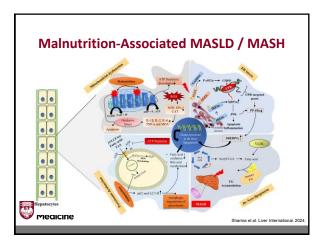
- 7–20% of MASLD (BMI <25 non-Asians, <23 Asians)
- 5% general population
- More common in Asian populations, but global
- Lack classic metabolic syndrome features
- · Risk factors
  - Visceral adiposity
  - Sarcopenia
  - Subtle insulin resistance

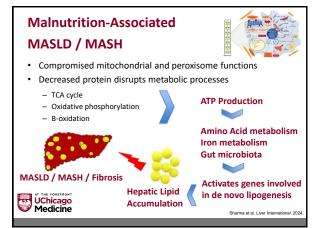












#### **General Approach To MASLD**

· Identify Metabolic Risk Factors

· Exclude other causes of steatosis

sis 5-FU Tamoxifen

- Medications

- Atypical Causes

Methotrexate Steroids

Amiodarone

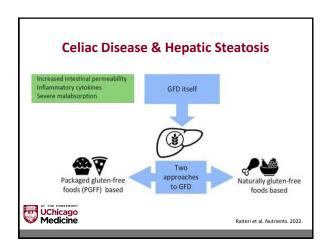
- Alcohol

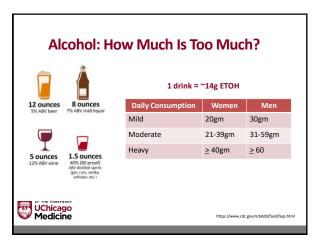
Wilson disease Celiac disease

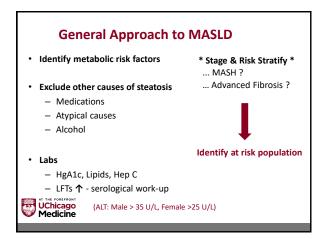
Nutrient deficiency (choline, carnitine) Lysosomal acid lipase deficiency Hypobetalipoproteinemia

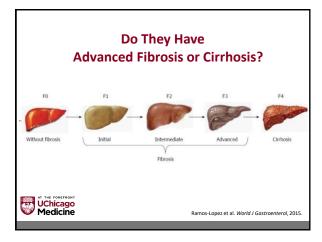


Chalassani et al. Hepatology. 2018 Rinella et al. Hepatology, 2023.









#### Liver Biopsy used to be the Gold Standard

- · Small pieces
  - 1.5cm length, 1.2-2mm diameter
- Mis-staging in 40% cases
  - ~1/50,000 of the liver
  - Sampling variability
- Bleeding (0.35%) and Death (0.14%)
- Expensive (\$1-\$4.3k)



"liver shank

THE FOREFRONT

UChicago
Tsal and Lee, Clin Liver Dis, 2018.

Myers RP, et al, Liver Int, 2008.

#### **Fibrosis Scoring Systems**

- FIB-4 Score
  - Age, AST, ALT, platelets

≤ 1.3 Advanced Fibrosis unlikely (Likely > 2.67)

≥ 3.48 Rules-In Cirrhosis (< 1.67 Rules-Out)

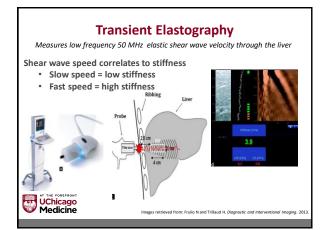
- Best at identifying patients without advanced fibrosis (<1.3)</li>
- Enhanced Liver Fibrosis (ELF)

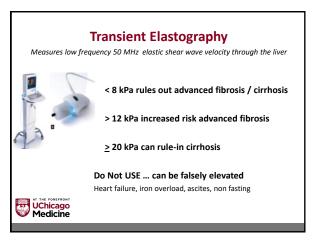
≥ 9.8 ↑ progression risk to cirrhosis / liver events

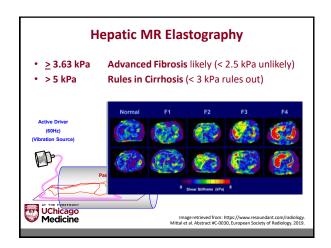
≥ 11.3 Rules-In Cirrhosis (< 7.7 Rules-Out)

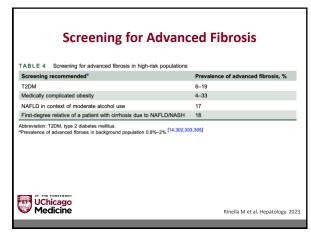
High ELF and > change over time can predict decompensation

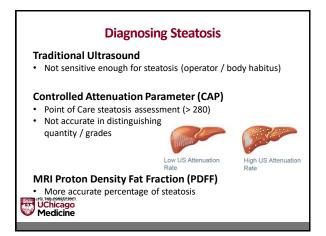


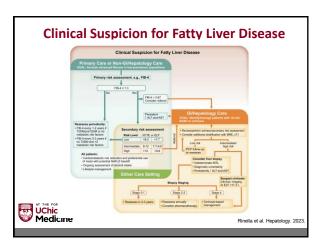


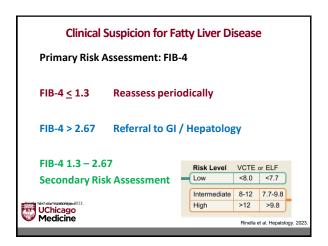


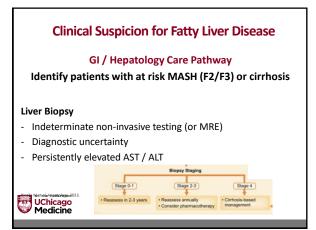


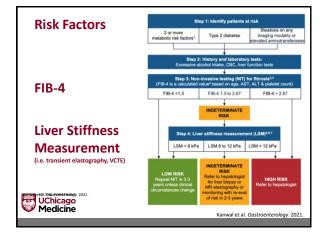












#### **Take Home Points**

- NAFLD is now MASLD
- · Insulin resistance is key
- Treat metabolic co-morbidities
- Need to identify advanced fibrosis



#### Questions?

spaul@bsd.uchicago.edu





#### Diet and Lifestyle: The Initial Management for MASLD/MASH

Sarah Kolb, RD



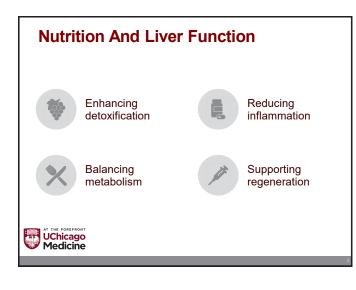
## The Link Between Nutrition And Liver Health

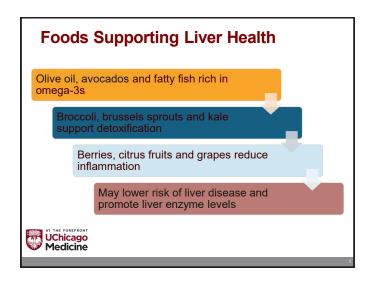
Sarah Kolb MS, RD, LDN

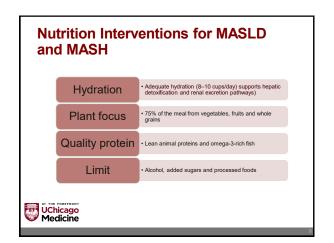
#### **Disclosure Information**

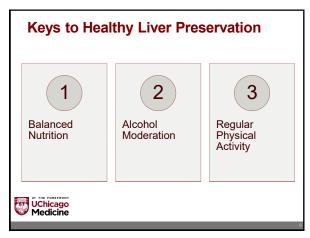
- I have no relevant financial relationship with any ineligible companies to disclose
- I will not discuss off label use or investigational use in my presentation

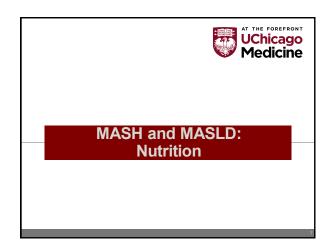


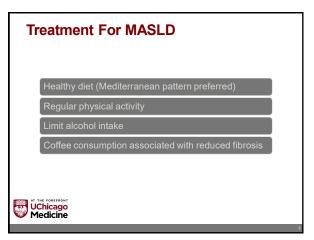


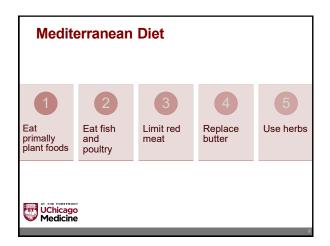


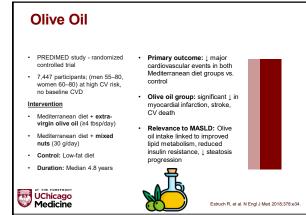


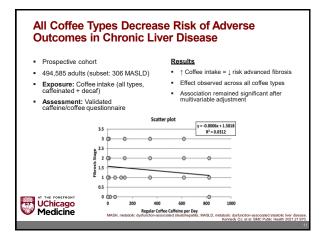














#### **Intermittent Fasting**



- Time-Restricted Feeding (12h eating / 12h fasting)
  - •Validated in human and animal studies
  - •Reduces adiposity and hepatic steatosis
  - •Improves lipid profile, glycemic control, and insulin sensitivity



Rothschild J, et al. Nutr Rev 2014;72:308-31



#### **Carbohydrate Counting**

#### 30 g carbohydrate per meal

Starch-based sources (e.g., pasta, rice, potatoes, cereal)

15 g carbohydrate per snack

#### ≥25 g dietary fiber daily

- · Emphasize fiber-rich carbohydrate sources
- Examples: whole grains, legumes, fruit





#### Summary

- Liver health strongly influenced by nutrition
- Coffee, olive oil, fiber are protective
- Mediterranean diet is the best dietary pattern
- Limit sugars, refined carbs
- Sustainable lifestyle changes > short-term fixes



#### References

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#### Approach to Drug Therapies for MASLD/MASH

Archita Desai, MD

### Metabolic Dysfunction—Associated Steatotic Liver Disease

#### **Drug Therapies**

Archita P. Desai, MD
Associate Professor of Medicine
Indiana University School of Medicine

GASTROENTEROLOGY AND HEPATOLOGY

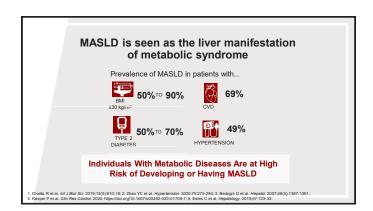
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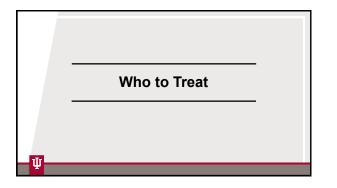
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- · Consultant for: Eli Lilly
- I will not discuss off label use or investigational use in my presentation.

GASTROENTEROLOGY AND HEPATOLOGY

#### **Objectives**

- 1. Review how to identify individuals who will benefit treatment
- 2. Review currently approved pharmacotherapy for MASLD
- 3. Identify the role of non-hepatic metabolic syndrome management

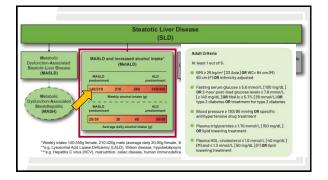


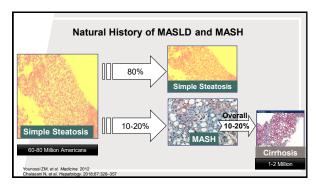


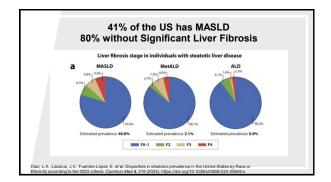
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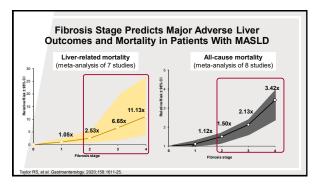
What can she expect?

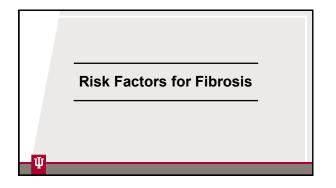
SH: 1-2 standard drinks, 2-3 times per week
Labs: PLTs 150, AST 42 ALT 51, ALP 110, T bili 0.4

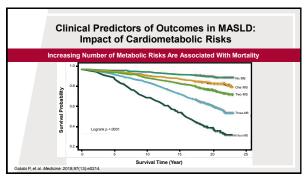


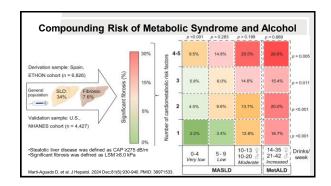






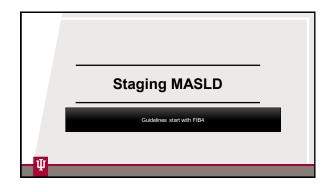


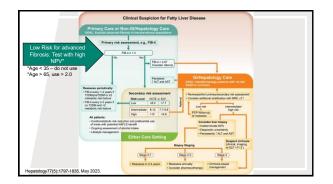


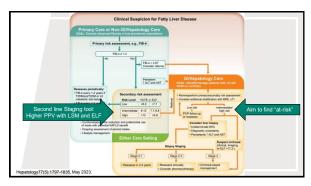


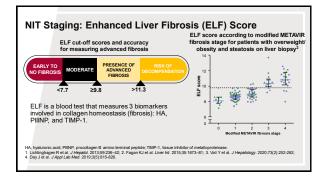


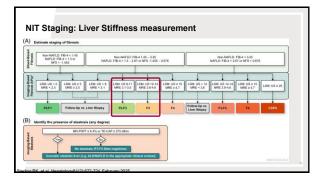
# Back to Sue • 51-year-old woman with known MASLD diagnosed after an US in the ER 10 years ago when she presented with symptomatic cholelithiasis. She was last seen 3 years ago and presents for re-assessment • PMHx: T2DM, HTN, OSA, HLD • PE: BMI 42, obese abdomen, acanthosis nigricans, mild LE edema • Meds: ACEi, Metformin, Glipizide, Atorvastatin • Labs: PLTs 150, AST 42 ALT 51, ALP 110, T bili 0.4 What can she expect?

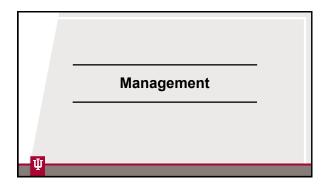


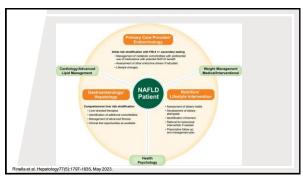


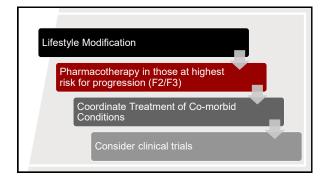


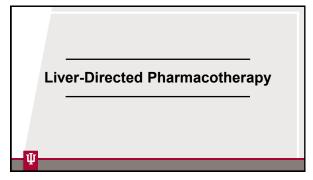


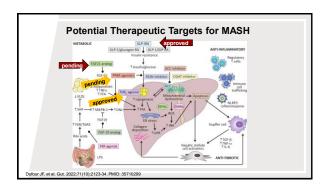


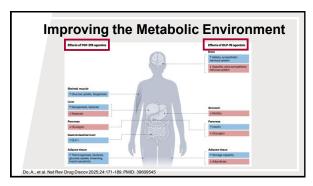


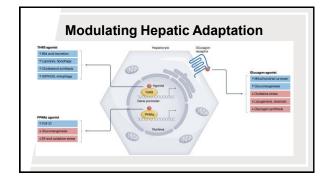


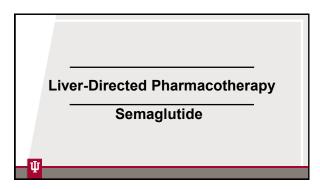


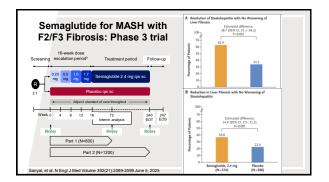


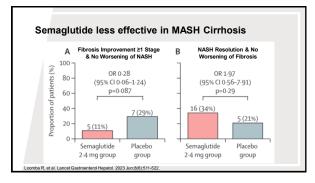


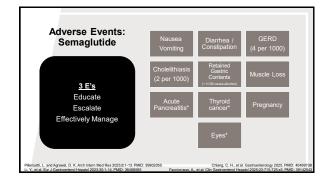


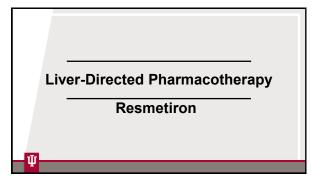




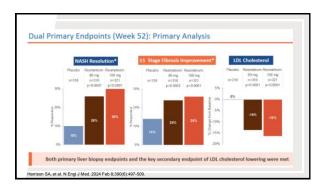


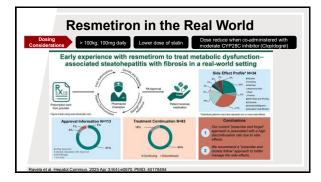


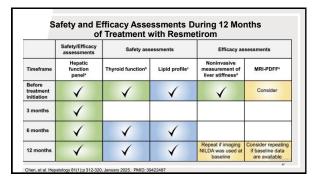


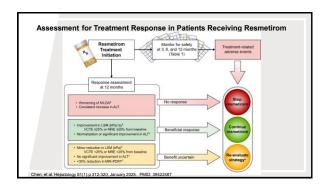


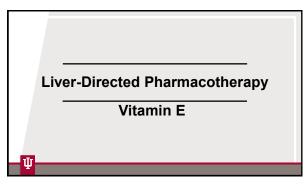


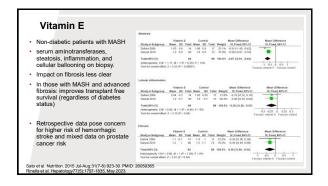




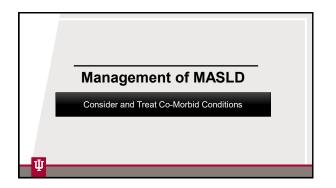


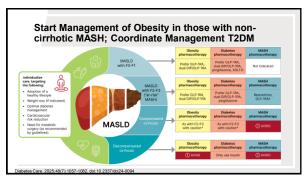


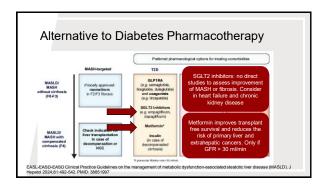


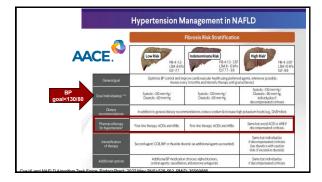


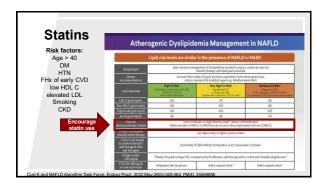




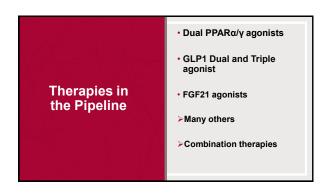


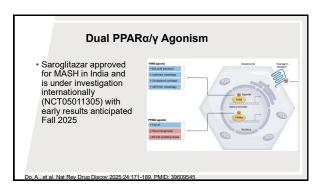


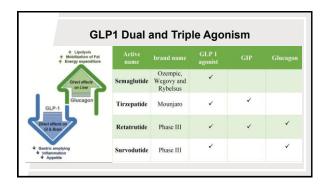


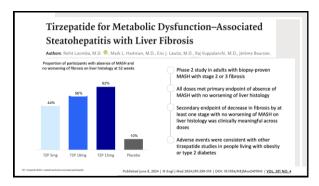


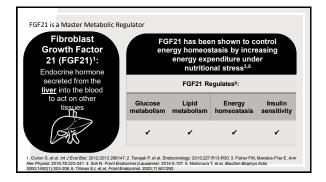


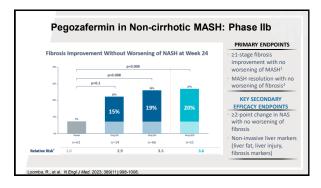


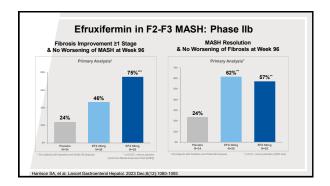


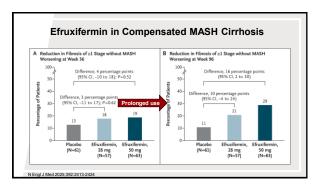


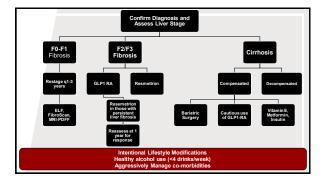








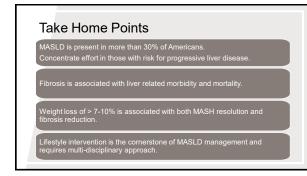


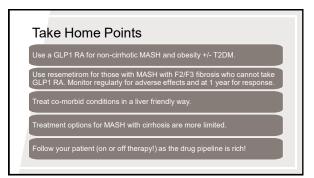


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- 51-year-old woman with known MASLD
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- PE: BMI 42, obese abdomen, acanthosis nigricans, mild LE edema
- · Meds: ACEi, Metformin, Glipizide, Atorvastatin
- Labs: PLTs 150, AST 42 ALT 51, ALP 110, T bili 0.4
- VCTE: Liver stiffness of 6 kPa, CAP 300 (<F2 fibrosis with steatosis)

Started GLP1 to address her many metabolic comorbid conditions. She plans to have repeat staging in a year.









## JOHN F. GILMORE SYMPOSIUM ON NUTRITION AND SMALL BOWEL DISORDERS

**SEPTEMBER 27, 2025**