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Participating in the Webinar

All attendees will be muted and will remain in "Listen Only Mode"

Type your questions here so that the moderator can see them.
Not all questions will be answered but we will get to as many as possible.

A handout with the slides and room to take notes can be downloaded from your control panel.

Moderator:
Lindsey Russell, MD, MSc, CNSC, FRCPC

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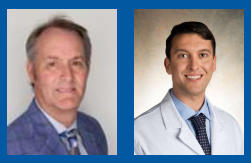
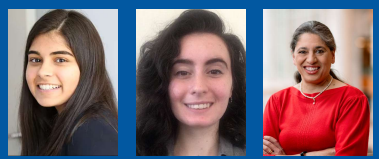
ACG Virtual Grand Rounds

Join us for upcoming Virtual Grand Rounds!

Special VGR - Week 35 – Tuesday August 26, 2025
Leveraging the Mentor-Mentee Relationship: Successes in Fellowship to Practice
Faculty: Lisa B. Malter, MD, FACG, Aileen Bui, MD, Lauren D. Feld, MD and Samir A. Shah, MD, FACG
Moderator: Nikki Duong, MD and Alana B. Persaud, MD
At 8pm Eastern



Week 35 – Thursday August 28, 2025
Short Bowel Syndrome/Intestinal Failure: Recognition, Complications, and Basic Management
Faculty: John K. DiBaise, MD, FACG
Moderator: Dejan Micic, MD, FACG
At Noon and 8pm Eastern



Week 36 – Thursday September 4, 2025
Patients Needs and Satisfaction in the IBD Transition to Adult Care
Faculty: Ms. Sneha Dave and Ms. Rosa Kelekian
Moderator: Sunanda V. Kane, MD, MSPH, MACG
At Noon and 8pm Eastern

Visit **gi.org/ACGVGR** to Register

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ACG ANNOUNCES
a New Book Series
Guide to the Guidelines
Volume 2
A vignette-based journal through the ACG clinical practice guidelines
BRENNAN SPIEGEL
HETAL A. KARSAN
AMERICAN COLLEGE OF GASTROENTEROLOGY
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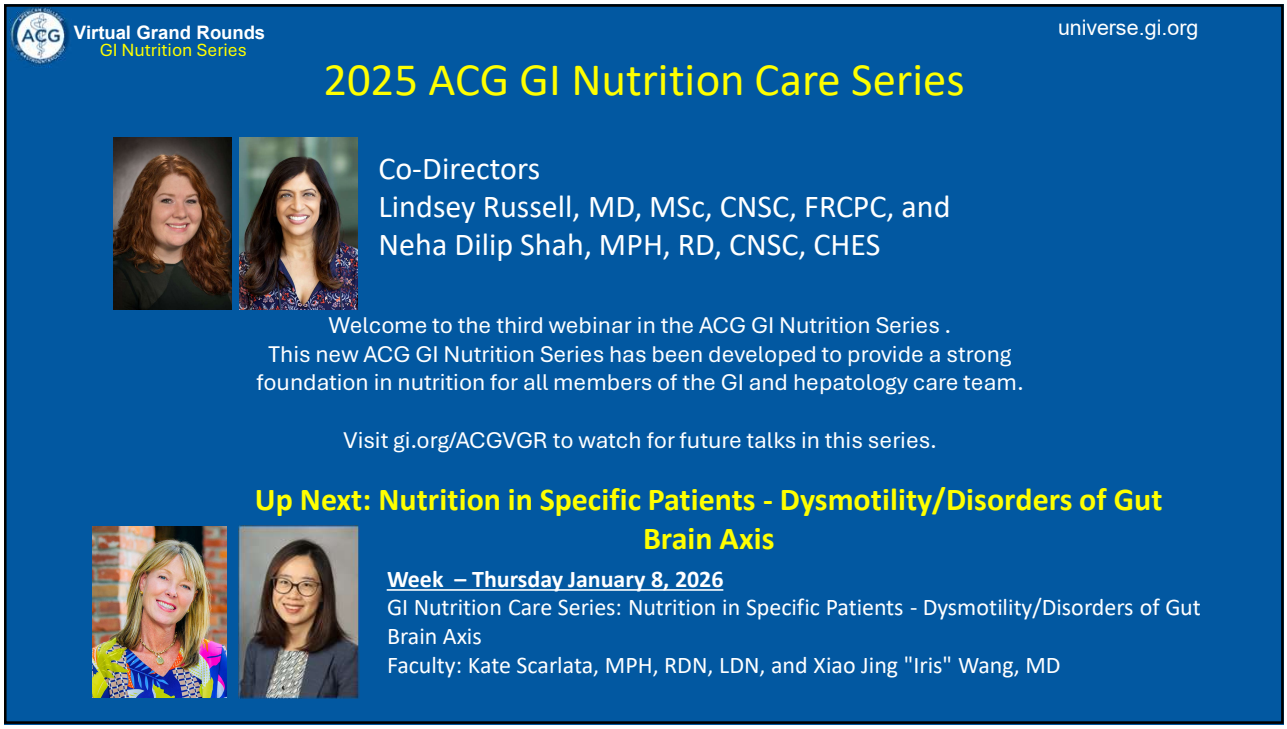
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GI Nutrition Series
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2025 ACG GI Nutrition Care Series
Co-Directors
Lindsey Russell, MD, MSc, CNSC, FRCPC, and
Neha Dilip Shah, MPH, RD, CNSC, CHES
Welcome to the third webinar in the ACG GI Nutrition Series .
This new ACG GI Nutrition Series has been developed to provide a strong foundation in nutrition for all members of the GI and hepatology care team.
Visit gi.org/ACGVGR to watch for future talks in this series.
Up Next: Nutrition in Specific Patients - Dysmotility/Disorders of Gut Brain Axis
Week – Thursday January 8, 2026
GI Nutrition Care Series: Nutrition in Specific Patients - Dysmotility/Disorders of Gut Brain Axis
Faculty: Kate Scarlata, MPH, RDN, LDN, and Xiao Jing "Iris" Wang, MD

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Up Coming 2025-26 ACG GI Nutrition Care Series

- Micronutrient Deficiencies- Malabsorption
- Nutrition in Specific Patients- Dysmotility/Disorders of Gut Brain Axis
- Nutrition in Specific Patients- Mucosal Diseases
- Nutrition in Specific Patients- Surgical Resections
- Nutrition Applications for a GI Practice

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Disclosures

Kristen Roberts, PhD, RDN, CNSC, FASPEN, FAND:
No relevant financial relationships with ineligible companies.

Holly Estes Doetsch, DCN, RD, LD:
No relevant financial relationships with ineligible companies.

Lindsey Russell, MD, MSc, CNSC, FRCPC:
Cormedix: Grant/Research Support; Napo Pharmaceuticals: Grant/Research Support

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

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GI Nutrition Case Series: Micronutrient Deficiencies and Malabsorption

Kristen M Roberts, PhD, RDN
Holly Estes-Doetsch, DCN, RDN
August 21st , 2025

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Objectives

Review strategies for assessment, diagnosis and treatment of micronutrient deficiencies in malabsorptive conditions

Identify risk factors for micronutrient deficiency and approaches to management

Evaluate existing micronutrient literature in malabsorptive conditions

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Malabsorption and Micronutrients: Evaluating fat-soluble vitamin status in malabsorptive conditions

Kristen M Roberts, PhD, RDN

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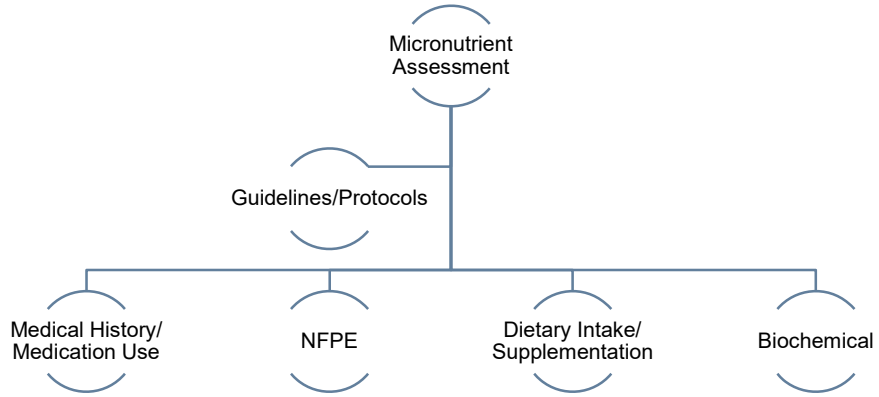
Foundations of Micronutrient Assessment[1]



[1]Roberts KM, Estes-Doetsch, H, Nahikian-Nelms M. Pocket Guide to Micronutrient Management. 1st edition. Academy of Nutrition and Dietetics; 2024.

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Micronutrient assessment strategies

How do we diagnose a micronutrient deficiency?

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Inflammation and Micronutrient Assessment[1]

- Serum/plasma levels tend to reflect recent dietary intake and are impacted by the acute phase response
- HPLC is assay of choice, but reference ranges vary

Fat-soluble Vitamin Laboratory	C-reactive Protein Cutoff (mg/L)
Serum retinol (Vitamin A)	>20
25-OH Vitamin D (Vitamin D)	>20
Alpha-tocopherol (Vitamin E)	>40
Phylloquinone (Vitamin K)	>10

[1]Berger et al. Nutr Clin Pract. 2023;38:56-69

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*Always with a C-reactive protein

Vitamin	Physical/Clinical Assessment	*Biochemical Assessments
Vitamin A	Ocular changes Xerophthalmia/Bitot spots/Corneal xerosis Iron deficiency/iron deficiency anemia Infectious complications/poor immune function Diarrhea Hyperkeratosis	Retinol, serum Retinol binding protein, serum Iron studies, serum Zinc, serum/plasma Complete blood count, serum
Vitamin D	Muscle weakness Impaired bone mineralization Decreased intestinal calcium absorption	25-OH vitamin D, serum/plasma Parathyroid hormone Total calcium, serum or ionized calcium, serum
Vitamin E	Peripheral neuropathy: Ataxia, myopathy, nystagmus, ophthalmoplegia Anemia Fatigue, weakness	Alpha-tocopherol, serum Cholesterol, serum Triglyceride, serum Alpha-tocopherol:Total Lipids(mg/g)→<0.8 mg/g suggests deficiency
Vitamin K	Bruising Bleeding Hemorrhage Impaired bone mineralization	Protein induced by vitamin K absence (PIVKA-II) Undercarboxylated osteocalcin Prothrombin time, INR Vitamin K, serum Triglyceride, serum

[1]Roberts KM, Estes-Doetsch, H, Nahikian-Nelms M. Pocket Guide to Micronutrient Management. 1st edition. Academy of Nutrition and Dietetics; 2024.

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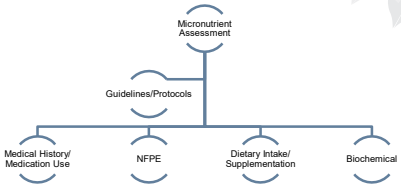

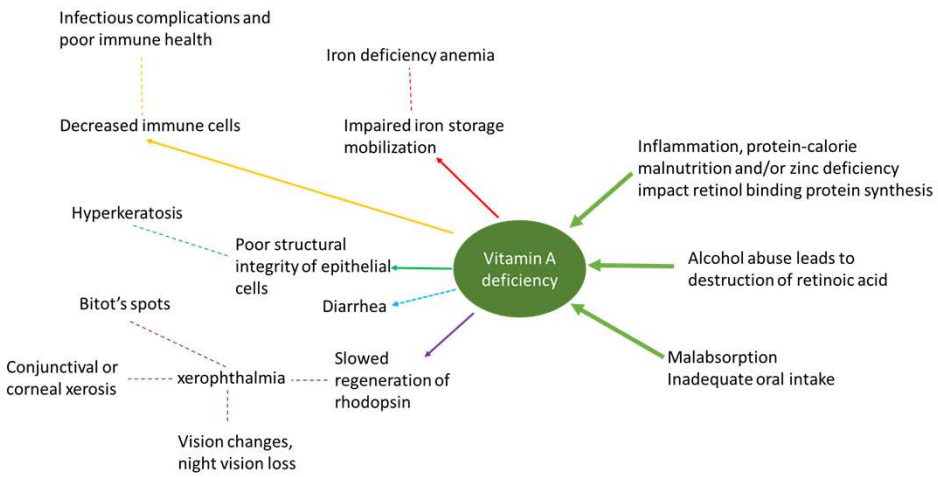

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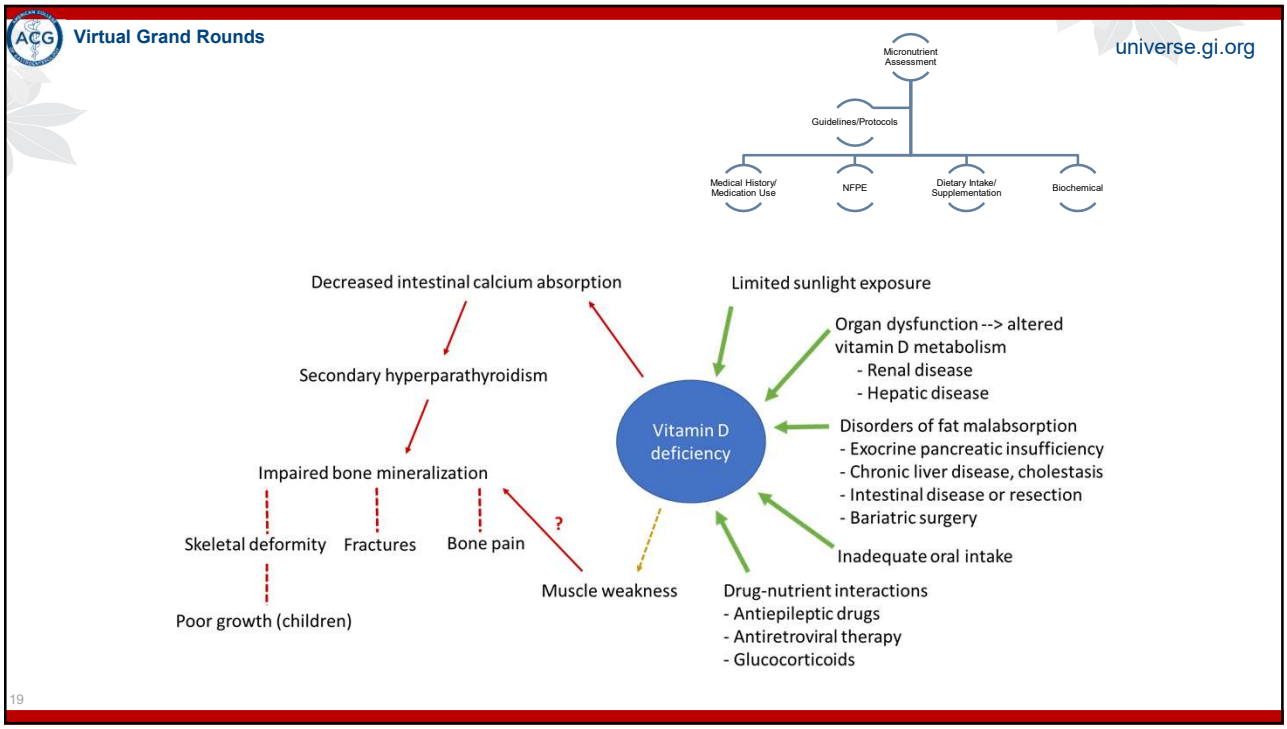
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Micronutrient Assessment
Guidelines/Protocols
Medical History/ Medication Use
NFPE
Dietary Intake/ Supplementation
Biochemical
Decreased intestinal calcium absorption
Limited sunlight exposure
Secondary hyperparathyroidism
Organ dysfunction --> altered vitamin D metabolism
- Renal disease
- Hepatic disease
Vitamin D deficiency
Disorders of fat malabsorption
- Exocrine pancreatic insufficiency
- Chronic liver disease, cholestasis
- Intestinal disease or resection
- Bariatric surgery
Impaired bone mineralization
Skeletal deformity
Fractures
Bone pain
?
Inadequate oral intake
Muscle weakness
Drug-nutrient interactions
- Antiepileptic drugs
- Antiretroviral therapy
- Glucocorticoids
Poor growth (children)
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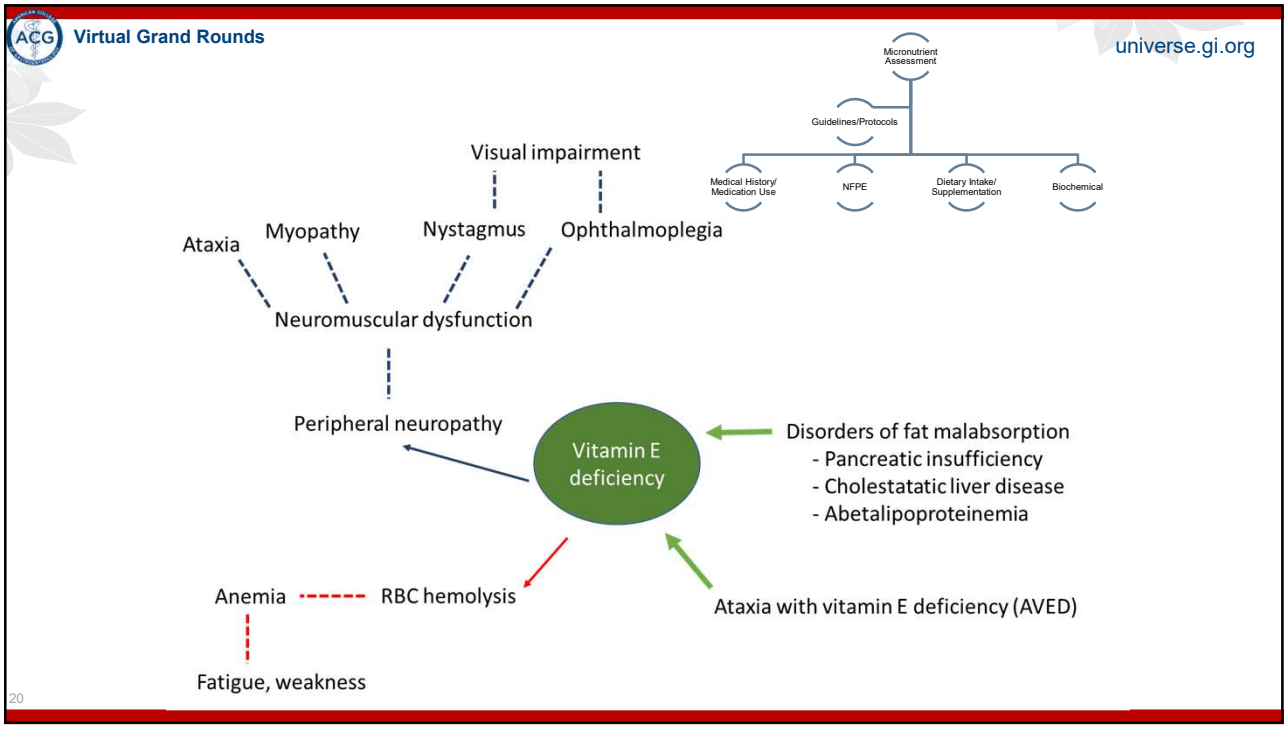
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Micronutrient Assessment
Guidelines/Protocols
Medical History/ Medication Use
NFPE
Dietary Intake/ Supplementation
Biochemical
Visual impairment
Ataxia
Myopathy
Nystagmus
Ophthalmoplegia
Neuromuscular dysfunction
Peripheral neuropathy
Vitamin E deficiency
Disorders of fat malabsorption
- Pancreatic insufficiency
- Cholestatatic liver disease
- Abetalipoproteinemia
Anemia
RBC hemolysis
Ataxia with vitamin E deficiency (AVED)
Fatigue, weakness
20

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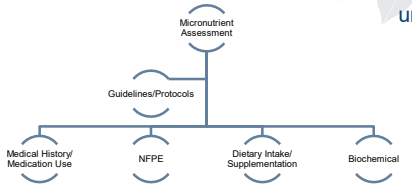

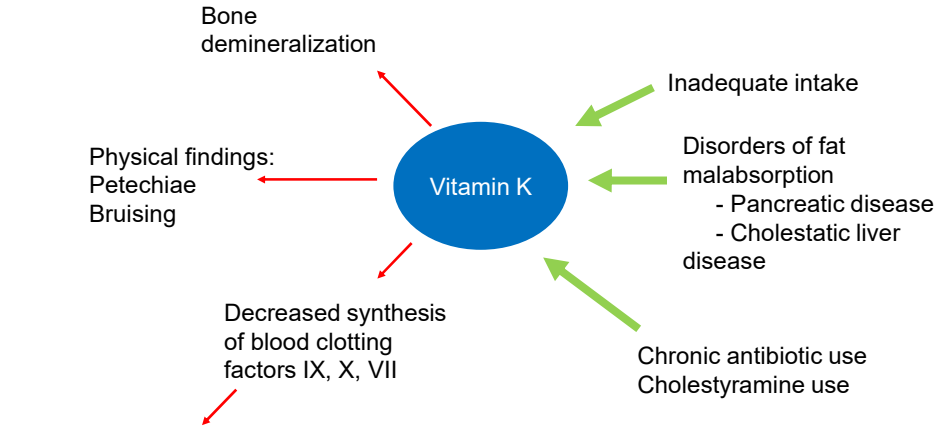

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How are we doing with micronutrient assessment?

Taking a look at exocrine pancreatic insufficiency…

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There are no studies in acute or chronic pancreatitis that account for all factors necessary to understand micronutrient deficiency prevalence and therefore, the impact on outcomes of interest.

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Submit a Manuscript: https://www.f6publishing.com
World J Gastroenterol 2021 September 14; 27(34): 5775-5792
DOI: 10.3748/wjg.v27.i34.5775
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

META-ANALYSIS

Dietary intake in patients with chronic pancreatitis: A systematic review and meta-analysis

Qurat Ul Ain, Yasir Bashir, Linda Kelleher, David M Bourne, Suzanne M Egan, Jean McMahon, Laura Keaskin, Oonagh M Griffin, Kevin C Conlon, Sinead N Duggan

Table 4 Studies analysing micronutrient intake in patients with chronic pancreatitis and controls

Ref.		Vitamin A (SD), IU	Vitamin D (mcg)	Vitamin E (IU/mg)	Vitamin K (mg)
Nakamura *et al*[6]	CP	2337.9 (3018.9)	NM	NM	NM
	Control[1]	4016.3 (4902.7)	NM	NM	NM
Tinju *et al* [37]	CP	M: 3659.7 (6546.8); F: 3888.4 (3936.1)	NM	NM	NM
	Control	UC	UC	UC	UC
Turner *et al* [32][2]	CP	278 (μg/d)	NM	NM	NM
	Control	UC	UC	UC	UC
Roberts *et al* [24][3]	CP	744.4 (RAE)	5.88	11.77	64
	Control[2]	651.3 (RAE)	4.03	11.54	99.5

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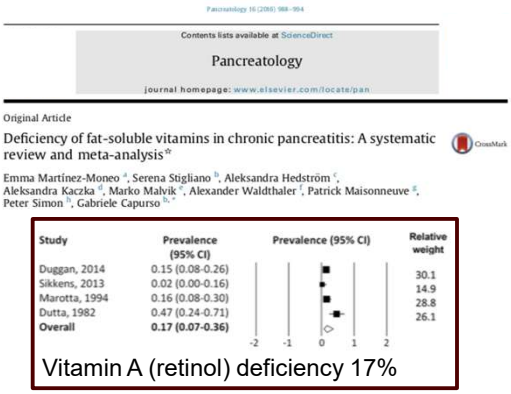

Contents lists available at ScienceDirect

Pancreatology

journal homepage: www.elsevier.com/locate/pan

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Study	Prevalence (95% CI)	Prevalence (95% CI)	Relative weight
Duggan, 2014	0.15 (0.08-0.26)		30.1
Sikkens, 2013	0.02 (0.00-0.16)		14.9
Marotta, 1994	0.16 (0.08-0.30)		28.8
Dutta, 1982	0.47 (0.24-0.71)		26.1
Overall	**0.17 (0.07-0.36)**		

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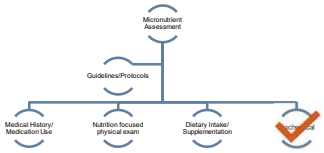
Vitamin A (retinol) deficiency 17%



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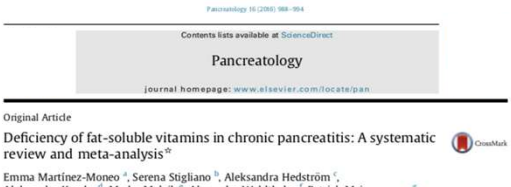

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Marotta, 1994	0.55 (0.40-0.68)		26.4
Dutta, 1982	0.67 (0.41-0.85)		24.1
Overall	**0.29 (0.09-0.64)**		

-2 -1 0 1 2

Vitamin E (tocopherol) deficiency 29%

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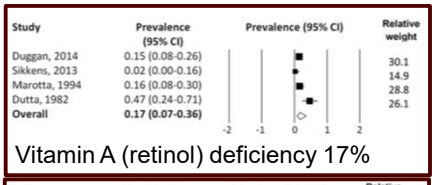

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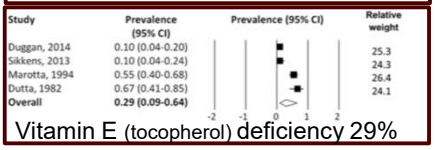

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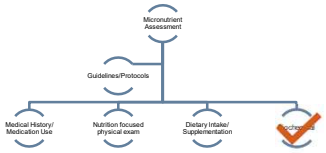

Duggan et al "Vitamin E deficiency was more prevalent when expressed as a ratio of blood lipid" (data not shown)

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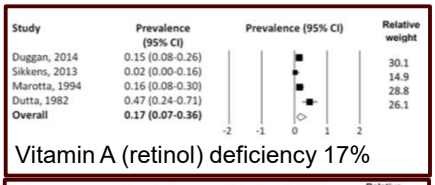

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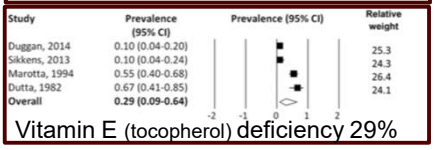

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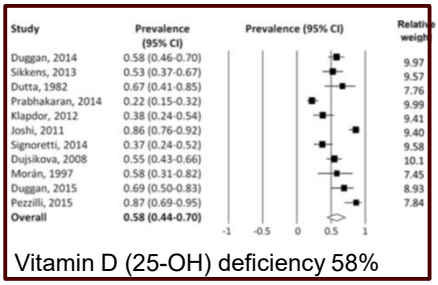

Study	Prevalence (95% CI)	Relative weight
Duggan, 2014	0.58 (0.46-0.70)	9.97
Sikkens, 2013	0.53 (0.37-0.67)	9.57
Dutta, 1982	0.67 (0.41-0.85)	7.76
Prabhakaran, 2014	0.22 (0.15-0.32)	9.99
Klapdor, 2012	0.38 (0.24-0.54)	9.41
Joshi, 2011	0.86 (0.76-0.92)	9.40
Signoretti, 2014	0.37 (0.24-0.52)	9.58
Dujsikova, 2008	0.55 (0.43-0.66)	10.1
Morán, 1997	0.58 (0.31-0.82)	7.45
Duggan, 2015	0.69 (0.50-0.83)	8.93
Pezzilli, 2015	0.87 (0.69-0.95)	7.84
Overall	**0.58 (0.44-0.70)**	

Vitamin D (25-OH) deficiency 58%

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Contents lists available at ScienceDirect

Clinical Nutrition ESPEN

journal homepage: http://www.clinicalnutritionespen.com

Original article

Nutritional parameters following first episode of pediatric acute pancreatitis

Sarah Orkin [a, b, *], Phillip Holovach [a], Tyler Thompson [b], Peter Farrell [a, b], Alexander Nasr [b], David Vitale [a, b], Sherif Ibrahim [b], Nicole Kotha [b], James Estes [b], Lindsey Hornung [c], Maisam Abu-El-Haija [a, b]

Baseline — 3 Months — 12 Months

N=181

- Vitamin A (retinol) in 0%
- Vitamin D (25-OH) in 56%
- Vitamin E (alpha-tocopherol without lipid adjustment) deficiency is 6%
- Vitamin K (INR) deficiency in 4%

- Vitamin A deficiency in 8%
- Vitamin D in 56%
- Vitamin E deficiency in 5%
- Vitamin K deficiency in 9%

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Submit a Manuscript: http://www.wjgnet.com/esps/
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DOI: 10.3748/wjg.v20.i48.18432

World J Gastroenterol 2014 December 28; 20(48): 18432-18438
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

OBSERVATIONAL STUDY

Pancreatic exocrine insufficiency, diabetes mellitus and serum nutritional markers after acute pancreatitis

Miroslav Vujasinovic, Bojan Tepes, Jana Makuc, Sasa Rudolf, Jelka Zaletel, Tjasa Vidmar, Maja Seruga, Bostjan Birsa

- N=100 AP (75% 1 episode, 25% 2+ episodes)
- No difference in fat-soluble vitamin deficiency between those with and without EPI.
- In EPI:
  - Vitamin A deficiency in 6.3%
  - Vitamin D deficiency in 68.8%
  - Vitamin E deficiency in 8%
  - Vitamin K deficiency not included

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Improving People's Lives
Through Innovations in
Personalized Health Care
Treatment of micronutrient deficiencies
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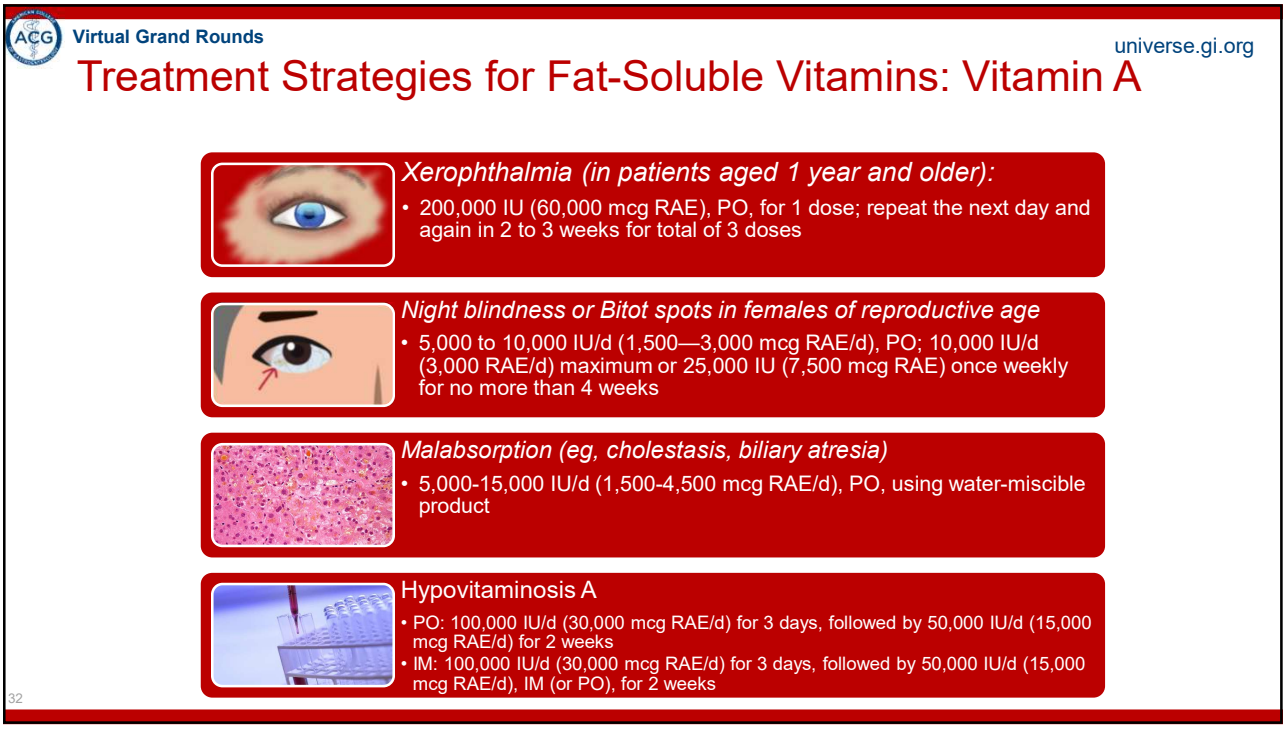
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Treatment Strategies for Fat-Soluble Vitamins: Vitamin A
Xerophthalmia (in patients aged 1 year and older):
• 200,000 IU (60,000 mcg RAE), PO, for 1 dose; repeat the next day and again in 2 to 3 weeks for total of 3 doses
Night blindness or Bitot spots in females of reproductive age
• 5,000 to 10,000 IU/d (1,500—3,000 mcg RAE/d), PO; 10,000 IU/d (3,000 RAE/d) maximum or 25,000 IU (7,500 mcg RAE) once weekly for no more than 4 weeks
Malabsorption (eg, cholestasis, biliary atresia)
• 5,000-15,000 IU/d (1,500-4,500 mcg RAE/d), PO, using water-miscible product
Hypovitaminosis A
• PO: 100,000 IU/d (30,000 mcg RAE/d) for 3 days, followed by 50,000 IU/d (15,000 mcg RAE/d) for 2 weeks
• IM: 100,000 IU/d (30,000 mcg RAE/d) for 3 days, followed by 50,000 IU/d (15,000 mcg RAE/d), IM (or PO), for 2 weeks
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Treatment Strategies for Fat-Soluble Vitamins: Vitamin D

Hypovitaminosis D

- 1,250 mcg (50,000 IU) once weekly for 8 to 12 weeks.
- Increase daily maintenance dose for patients with malabsorption, intestinal resection, Roux-en-Y gastric bypass, obesity, or concurrent use of cytochrome P450 (CYP3A4) enzyme–inducing drugs to prevent redeveloping deficiency. As much as 150 to 250 mcg/d (6,000-10,000 IU/d) may be required. Closely monitor 25-hydroxyvitamin D concentrations.

Treatment Strategies for Fat-Soluble Vitamins: Vitamin D

Hypovitaminosis D

- For every 25 mcg (1,000 IU) increase in oral vitamin D intake, the corresponding increase in plasma 25-hydroxyvitamin D is estimated to be ~7 ng/mL in 3 to 4 months.

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Treatment Strategies for Fat-Soluble Vitamins: Vitamin E

Hypovitaminosis E

- 15-25 IU per kilogram of body weight daily for documented clinical deficiency.
- Dose requirements are higher in patients with fat malabsorption, chronic pancreatitis, cholestatic hepatobiliary diseases, or intestinal resection.

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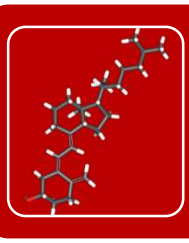
Treatment Strategies for Fat-Soluble Vitamins: Vitamin K

Prevention of hypovitaminosis K:

- *Typical regimen: up to 1 mg/d, PO, or 5 mg once weekly*



Treatment of hypovitaminosis K

- 1 to 5 mg/d, SC or PO
- In patients with severe malabsorption or who are unable to take oral medications, consider SC and IV administration.

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Summary

- We need to order accurate assays for determining biochemical nutrient deficiency
- Inflammation must be incorporated into the assessment to prevent over and under-reporting of fat-soluble vitamin deficiencies.
- Treatment strategies are similar amongst malabsorptive conditions and other populations likely due to the lack of data on optimal treatment strategies.
- Treating malabsorption is essential for prevention of fat-soluble vitamin deficiency.

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Malabsorption and Micronutrients: Evaluating iron, copper and zinc status in malabsorptive conditions

Holly Estes-Doetsch, DCN, RDN

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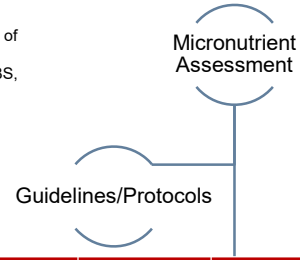
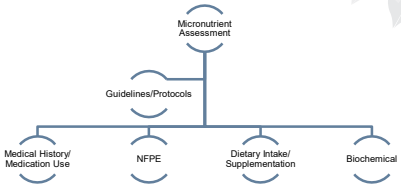
Iron Screening Guidelines

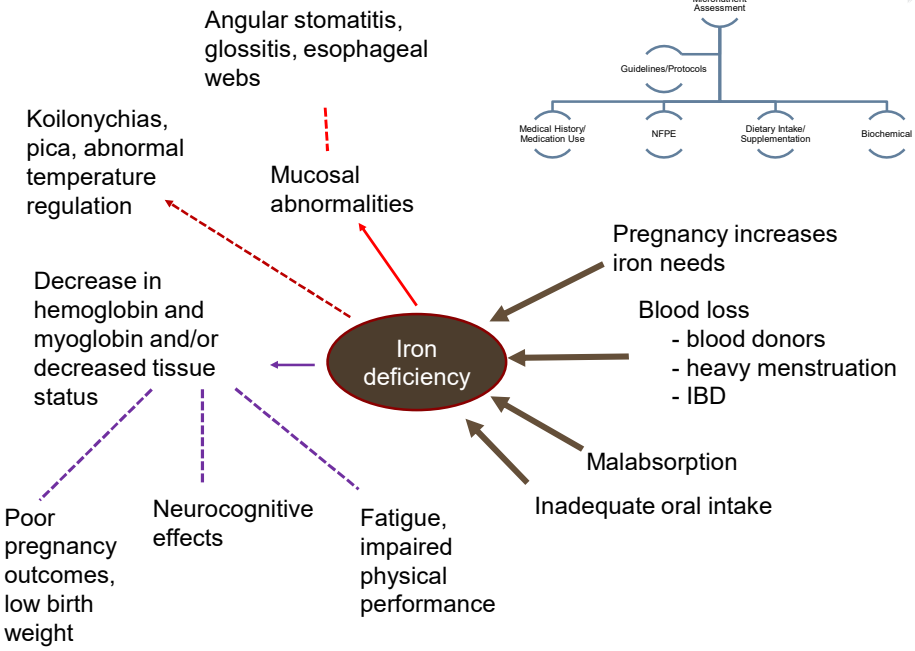
Abbreviations: ACG, American College of Gastroenterology; AGA, American Gastroenterological Association; ASMBS, American Society for Metabolic and Bariatric Surgery; ECCO, European Crohn's and Colitis Organization



1. Rubio-Tapia A, et al. Am J Gastroenterol. 2013;108(5):656-677.
2. Dignass AU, et al. J Crohn's Colitis. 2015;9(3):211-222.
3. Parrott J, et al. Surg Obes Relat Dis. 2017;13(5):727-741.
4. Iyer K, DiBaise JK, Rubio-Tapia A. Clin Gastroenterol Hepatol. 2022;20(10):2185-2194.e2.

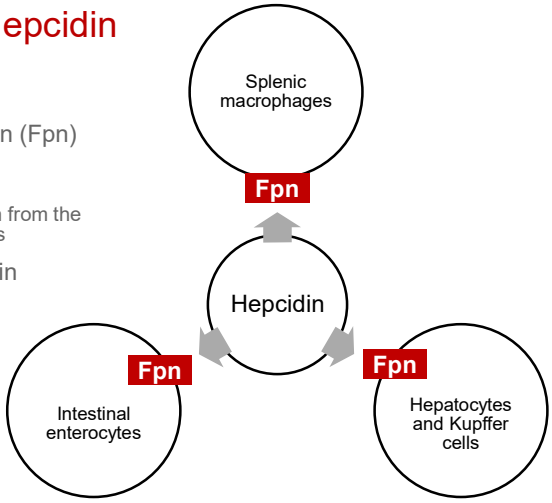
Gastrointestinal condition	Organization	Guideline
Celiac disease[1]	ACG	Measure at baseline and continue monitoring if previous values abnormal.
Inflammatory bowel disease[2]	ECCO	Measure every 6 to 12 months in patients with quiescent or mild disease. If active disease, measure every 3 months.
Metabolic and bariatric surgery[3]	ASMBS	Measure prior to surgery. Measure within 3 months after weight loss surgery and repeat every 3 to 6 months until 12 months, then measure annually.
Short bowel syndrome[4]	AGA	Measure at baseline and at least annually





Hormonal Regulation of Iron Homeostasis by Hepcidin

- Hepcidin acts on ferroportin (Fpn)
  - Impedes iron absorption from gastrointestinal tract
  - Controls release of stored iron from the liver and splenic macrophages
- Factors influencing hepcidin production
  - Physiologic iron status
  - Iron demands
  - Inflammatory cytokines



1. Lanser L, Fuchs D, Kurz K, Weiss G. *Nutrients*. 2021;13(11):3732.
2. Murawska, N, Fabisiak, A, Fichna, J. *Inflamm bowel dis*. 2016; 22(5):1198–1208.

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Iron Biomarkers

	Absolute iron deficiency (normal CRP)	Absolute iron deficiency and FID (elevated CRP)	FID (elevated CRP and adequate iron stores)
Ferritin	Low (< 30 ng/mL)*	Low/normal (≤ 100 ng/mL)	Elevated (> 100 ng/mL)
Serum iron	Low	Low	Low
TIBC	Elevated	Low	Low
Tsat	Low	Low	Low

*Suggested cut-offs as general lack of consensus

Abbreviations: CRP, C-reactive protein; ng/mL, FID, functional iron deficiency; nanograms per milliliter; TIBC, total iron binding protein; Tsat, transferrin saturation

1. Camaschella C. *Blood*. 2019;133(1):30-39.
2. Al-Naseem A, et al. *Clin Med (Lond)*. 2021;21(2):107-113.
3. Murawska, N, Fabisiak, A, Fichna, J. *Inflamm bowel dis*. 2016; 22(5):1198–1208.
4. Thongkhao-On S, Estes-Doetsch H, Roberts K. *Pract Gastro*. 2025; 49(8): 36-44.

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Oral Iron Treatment

Iron Formulation	Elemental Iron
Ferrous sulfate	20%
Ferrous gluconate	12%
Ferrous fumarate	33%

- 65 mg elemental iron daily
- Best absorbed on an empty stomach[1]
- Interactions[1]
  - Drugs – e.g., levothyroxine, tetracyclines, fluoroquinolones, acid suppressants
  - Nutrients – e.g., calcium, zinc
- Risk for gastrointestinal side effects increases with daily iron dose and frequency of oral iron therapy[1]
- Alternate day iron or reduction of daily iron dose may reduce risk for side effects[2,3]

1. Alleyne M, Horne MK, Miller JL. *Am J Med*. 2008;121(11):943-948.
2. Camaschella C. *Blood*. 2019;133(1):30-39.
3. Al-Naseem A, et al. *Clin Med (Lond)*. 2021;21(2):107-113.

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Intravenous Iron Treatment

- Indications
  - Elevated inflammatory markers
  - Severe anemia
  - Poor tolerance and/or response to oral iron
  - Reduced absorption

IV iron formulations in the United States

Iron sucrose
Ferric carboxymaltose
Low molecular weight iron dextran
Ferric derisomaltose
Ferumoxytol
Ferric gluconate

Camaschella C. *Blood*. 2019;133(1):30-39.

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Iron Monitoring

- Hemoglobin increases 1-2 g/dL within 2-3 weeks
- Ferritin may take up to 6 months to normalize
- Patients with IBD experience high rate of iron deficiency recurrence

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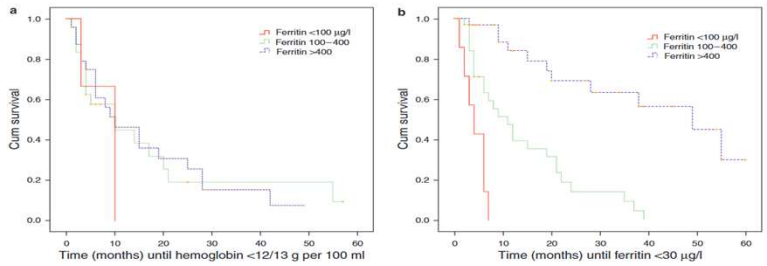
ORIGINAL CONTRIBUTIONS: INFLAMMATORY BOWEL DISEASE

Rapid Recurrence of IBD-Associated Anemia and Iron Deficiency After Intravenous Iron Sucrose and Erythropoietin Treatment

Kulnigg, Stefanie MD[1]; Teischinger, Lena[1]; Dejaco, Clemens MD[1]; Waldhör, Thomas PhD[2]; Gasche, Christoph MD[1,3]

Author Information

American Journal of Gastroenterology 104(6):p 1460-1467, June 2009.



- Median time to:
  - anemia: 10 months (95% CI, 8-12 months)
  - ferritin < 30 ug/L: 19 months (95% CI, 11-28 months)
  - ferritin < 100 ug/L: 11 months (95% CI, 8-14 months)

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Pediatric Crohn's disease, iron deficiency anemia and intravenous iron treatment: a follow-up study

Sara Valério de Azevedo, Catarina Maltez & Ana Isabel Lopes

To cite this article: Sara Valério de Azevedo, Catarina Maltez & Ana Isabel Lopes (2017) Pediatric Crohn's disease, iron deficiency anemia and intravenous iron treatment: a follow-up study, Scandinavian Journal of Gastroenterology, 52:1, 29-33, DOI: 10.1080/00365521.2016.1224381

- N = 19 patients
- Received either iron sucrose or ferric carboxymaltose
- After a median follow-up of 15.5 months, iron deficiency anemia recurred in 6 patients
- Patients in clinical remission

Prevalence of Zinc and Copper Deficiencies

- Zinc
  - Inflammatory bowel disease: overall prevalence of 54% (95% CI 0.51- 0.56) in Crohn's disease and 41% (95% CI 0.38 - 0.45) in ulcerative colitis[1]
  - Metabolic and bariatric surgery: pooled prevalence of 18.3% (95% CI 11.4 – 26.3%)[2]
  - Celiac disease: 59.4% in retrospective study of 309 patients with new diagnosis[3]
- Copper
  - Metabolic and bariatric surgery: pooled prevalences of 16%, 28%, 21%, and 16% at 1-, 2-, 3-, and 4-years after surgery[4]

1. Zupo R, Sila A, Castellana F, et al. *Nutrients*. 2022;14(19):4052. 2. Cao L, Liang S, Yu X, et al. *Obes Surg*. 2023;33(12):3907-3931. 3. Bledsoe AC, King KS, Larson JJ, et al. *Mayo Clin Proc*. 2019;94(7):1253-1260. 4. Xu B, Xiao T, Li J, et al. *Obes Surg*. 2025;35(2):602-613.

Trace element	Clinical manifestations	Laboratory assessment
Zinc	Alopecia, acrodermatitis enteropathica, hypogeusia, dysgeusia, hyposmia, anorexia, diarrhea, growth failure (pediatrics), hypogonadism, poor wound healing	Serum or plasma zinc, CRP, albumin Considerations: Zinc is a negative acute phase reactant and may be falsely low when CRP is > 20 mg/dL. Zinc status may appear falsely low in hypoalbuminemia. If prolonged zinc use (> 3 months), assess copper status.
Copper	Hair and skin depigmentation Myeloneuropathy Gait ataxia Vision loss	Serum or plasma copper < 8 umol/L, CRP, CBC Considerations: Copper is a positive acute phase reactant. If CRP > 20 mg/L, use < 12 umol/L. Rule out folate and B12 deficiencies if neurological symptoms. Include iron studies if anemia is present.

1. Berger MM, et al. *Clin Nutr*. 2022;41(6):1357-1424. 2. Berger MM, et al. *Nutr Clin Pract*. 2023;38(1):56-69. 3. Roberts, K., Estes-Doetsch, H., and Nelms, M., eds. (2024). *Pocket Guide to Micronutrient Management*. Academy of Nutrition and Dietetics

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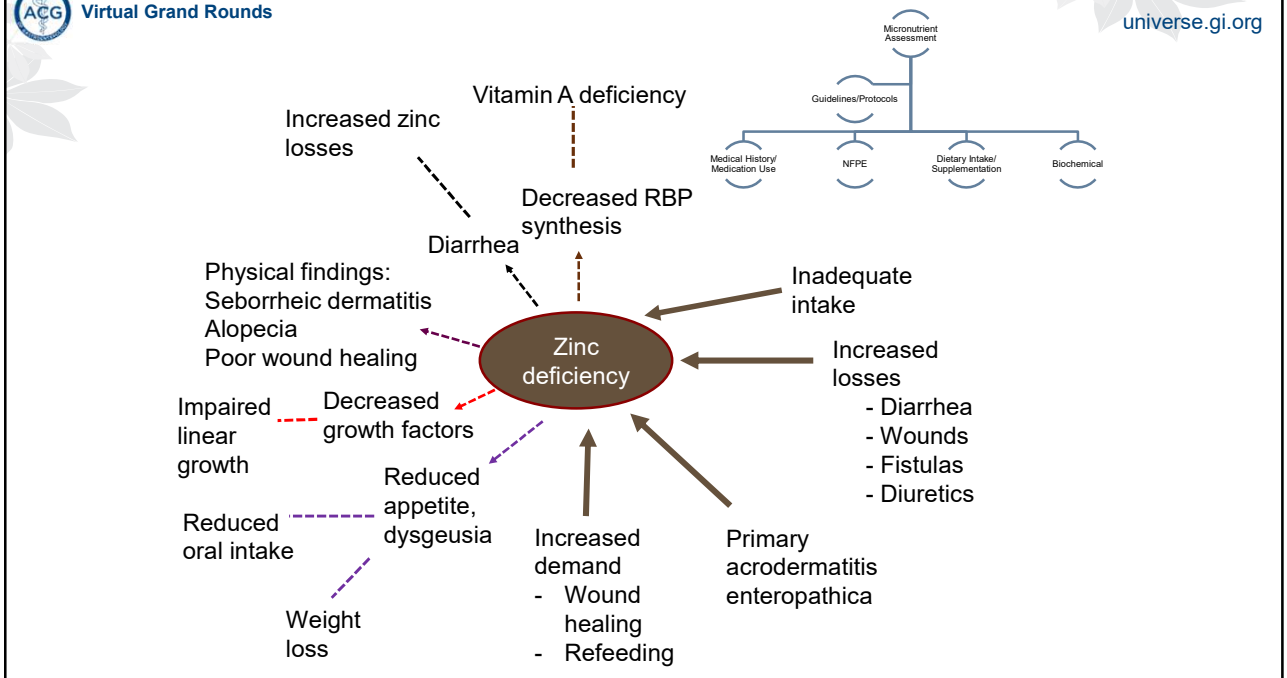

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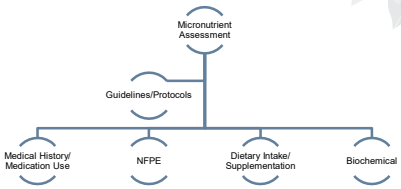

Decreased functional capacity

Myeloneuropathy

Optic neuropathy

Physical findings: Depigmentation of hair and skin

Increased risk for infection

Exclusive jejunal feeds

Inadequate intake

Increased losses
- Diarrhea
- RRT
- Wounds

Copper deficiency

Leukopenia
Neutropenia
Pancytopenia

Impaired hematopoiesis

Altered iron metabolism

Anemia

Chronic zinc therapy

Menkes disease

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Prolonged Zinc Therapy as an Overlooked Cause of Copper Deficiency

- Case series of 12 hospitalized patients with liver cirrhosis receiving zinc therapy
- ≥ 50 mg elemental zinc for an average of 21.5 months
- Mean copper level: 5.03 umol/L
- Signs/symptoms: paresthesia, altered hematological profile

Makepeace S., Schnicke E, Roberts K, Estes-Doetsch H. Food and Nutrition Conference and Expo; Oct. 2025.

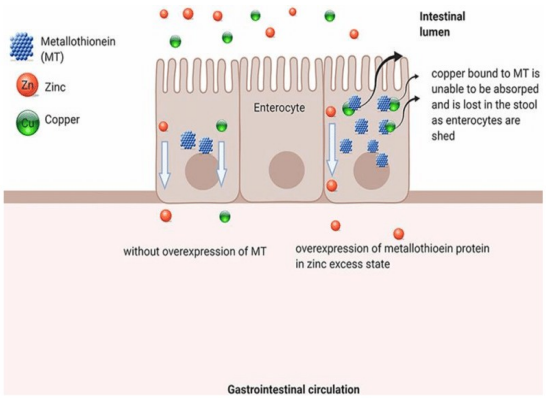

Image source: Munie S, Pintavorn P. *Case Rep Nephrol Dial.* 2021;11(2):167-175.

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Treatment of Zinc and Copper Deficiencies

- Elemental zinc, 0.5-1 mg/kg/day, up to 150 mg/day in divided doses
- Elemental copper, 2-8 mg/day, PO in 2-3 divided doses for 2-3 weeks
- Intravenous replacement in cases of overt symptoms
- If zinc therapy used > 3 months, consider supplementing zinc and copper in a ratio of 10:1 (e.g., 10 mg zinc for every 1 mg copper)

Roberts KM, Estes-Doetsch, H, Nahikian-Nelms M. Pocket Guide to Micronutrient Management. 1st edition. Academy of Nutrition and Dietetics; 2024.

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Thank you!

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Questions

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