Nutritional deficiencies in IBD: diagnostic approach

Name: Željko Krznarić, MD, PhD, FEBGH

Institution: Department of Gastroenterology and Hepatology & Clinical Nutrition
University of Zagreb, School of Medicine
Clinical Hospital Centre Zagreb
ZAGREB, Croatia
Definition of normal gut function

Tolerance to nutrient intake
## Normal gastrointestinal volumes

<table>
<thead>
<tr>
<th>Component</th>
<th>Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food and drink</td>
<td>1500ml</td>
</tr>
<tr>
<td>Saliva</td>
<td>750ml</td>
</tr>
<tr>
<td>Gastric secretion</td>
<td>1250ml</td>
</tr>
<tr>
<td>Biliary secretion</td>
<td>1000ml</td>
</tr>
<tr>
<td>Pancreatic secretions</td>
<td>1000ml</td>
</tr>
<tr>
<td>Jejunal secretion</td>
<td>2500ml</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>8000ml</strong></td>
</tr>
<tr>
<td>Stool liquid</td>
<td>100ml</td>
</tr>
</tbody>
</table>
Definition of gut dysfunction*

Intolerance to an appropriate nutritional regimen due to an impaired gut integrity

*Crohn's disease have capacity to affect any part of the gastrointestinal tract but UC is restricted to the colon
Spectrum of nutritional disorders in IBD

Fig. 1. Overview of nutrition disorders and nutrition-related conditions.

ESPEN guidelines on definitions and terminology of clinical nutrition 2016.
What is Malnutrition?

• Malnutrition is inadequate intake of protein and/or energy or micronutrients resulting with
  • change in body composition (decreased fat free mass and body cell mass)
  • diminished physical and mental function
  • impaired clinical outcome
Fig. 2. Diagnoses tree of malnutrition; from at risk for malnutrition, basic definition of malnutrition to aetiology-based diagnoses

ESPEN guidelines on definitions and terminology of clinical nutrition 2016.
DEVELOPMENT OF DEFICIENCY

Inadequate intake
Impaired absorption
Increased nutrient losses

Biologic dysfunction

Cellular dysfunction

Morbidity

Body store/tissue level depletion

Physiologic dysfunction

Clinical signs and symptoms

Components of nutrition assessment

Dietary survey and nutrient intake

Biochemical/physiologic studies

Clinical signs and symptoms

Vital statistics

Mortality
Malnutrition in IBD

1. Decreased food intake
   • Anorexia (TNF – mediated)
   • Mechanical (fistulas, post-operative)
   • Avoidance of high-residue food (can worsen abdominal pain/diarrhea)
   • Avoidance of lactose-containing food (high rates of concomitant lactose intolerance)

2. Increased intestinal loss
   • Diarrhea (increased loss of zinc, potassium, magnesium)
   • Occult/overt blood loss (iron deficiency)
   • Exudative enteropathy (protein loss, decrease in albumin-binding proteins, eg vitamin D binding protein)
   • Steatorrhea (fat and fat soluble vitamins)

A cross-sectional study on nutrient intake and -status in inflammatory bowel disease patients

Jona B. Vidarsdottir¹, Sigridur E. Johannsdottir², Inga Thorsdottir¹, Einar Bjornsson² and Alfons Ramel¹*

Fig. 3 Percentage of deficiency\(^a\) in vitamin intake in the patients with active CD, patients with inactive CD and control individuals. \(^a\)Percentage of deficiency is based on dietary reference intakes [16]. *\(p < 0.05\) vs. inactive CD group; \(\#\) \(p < 0.05\) vs. control group.

**Fig. 2** Percentage of deficiency in mineral intake in the patients with active DC, patients with inactive DC and control individuals. Percentage of deficiency is based on dietary reference intakes [16]. *p < 0.05 vs. inactive CD group; # p < 0.05 vs. control group.
3. Malabsorption

• Loss of intestinal surface area from active inflammation, resection, bypass or fistula

• Terminal ileal disease associated with deficiencies in B12 and fat-soluble vitamins

4. Hypermetabolic state

• Alterations of resting energy expenditure
Resting energy expenditure in adult patients with Crohn's disease

Rosa Sammarco, Maurizio Marra, Maria Carmen Pagano, Lucia Alfonsi, Lidia Santarpia, Iolanda Cioffi, Franco Contaldo, Fabrizio Pasanisi

SUMMARY

Background & aims: Crohn's disease (CD) is a chronic intestinal disorder of unknown etiology involving any section of the gastrointestinal tract often associated with protein-energy malnutrition (PEM). Increased resting energy expenditure (REE) unmatched by adequate dietary intake is amongst the pathogenetic mechanisms proposed for PEM. Aim of this study was to evaluate REE in CD patients receiving or not immuno-suppressive therapy as compared to controls.

Methods: 36 CD patients (22 M and 14 F, age range 18–55 years) clinically stable and without complications since at least 6 month were studied. REE was evaluated by indirect calorimetry and body composition by BIA. Full biochemistry was performed. Patients were divided into two groups: Group 1 (G1 = 12 patients) without and Group 2 (G2 = 24 patients) with immuno-suppressive therapy.

Results: The two groups were similar for age, height and BMI whereas significantly differed for weight (G1 vs G2: 56.9 ± 7.44 vs 62.3 ± 8.34 kg), fat free mass (FFM): 40.4 ± 5.73 vs 48.2 ± 7.06 kg), fat mass (FM: 17.0 ± 3.55 vs 13.9 ± 5.54 (kg) and phase angle (PA: 5.6 ± 1.4 vs 6.5 ± 1.0°). Serum inflammation parameters were significantly higher in G1 than in G2: hs-CRP: 7.76 ± 14.2 vs 7.36 ± 13.4 mg/dl; alpha 2-protein: 11.7 ± 3.69 vs 9.74 ± 2.08 mg/dl; fibrinogen: 424 ± 174 vs 334 ± 118 mg/dl (P < 0.05). REE was higher in G2 vs G1: 1383 ± 207 vs 1582 ± 253 kcal/die (P < 0.05) both in men: 1579 ± 314 vs 1650 ± 203 and women: 1267 ± 140 vs 1380 ± 132. Nevertheless, when corrected for BMI, REE resulted higher in G1 than G2 (34.8 ± 4.89 vs 33.0 ± 4.35 kcal/kg, P < 0.05) group, also higher compared to our, age and sex matched, control population (REE/FM: 30.9 ± 4.5 kcal/kg).

Conclusions: Our preliminary results show that REE when adjusted for FM is increased in clinically stable CD patients and mildly reduced by immuno-suppressive therapy possibly through a direct action on inflammation and on body composition characteristics.

© 2016 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
Measurement of REE

• Indirect calorimetry
5. Drug interactions
- Sulphasalazine and methotrexate inhibits folate absorption/metabolism
- Glucocorticoids impair calcium, zinc and phosphorus absorption, vitamin C losses and vitamin D resistance
- Cholestyramine impairs absorption of fat-soluble vitamins, vitamin B12 and iron

6. Long term parenteral nutrition
- Can occur with any micronutrient not added to TPN
- Reported deficiencies include thiamine, vitamin and trace elements zinc, copper, selenium, chromium

The ABCDs of Nutritional Assessment

• **Anthropometric data**
• **Biochemical tests** (blood, urine or feces samples)
• **Clinical observations**: signs and symptoms of nutritional deficiencies
• **Dietary intake**
## Biochemical markers

<table>
<thead>
<tr>
<th>Biochemical parameters</th>
<th>Mild deficiency</th>
<th>Moderate deficiency</th>
<th>Severe deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin (g/dL)</td>
<td>3.5-3</td>
<td>2.9-2.5</td>
<td>&lt;2.5</td>
</tr>
<tr>
<td>Transferin (mg/dL)</td>
<td>150-200</td>
<td>100-149</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Pre-albumin (mg/dL)</td>
<td>18-22</td>
<td>10-17</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Retinol binding protein (mg/dL)</td>
<td>2.5-2.9</td>
<td>2.1-2.4</td>
<td>&lt;2.1</td>
</tr>
<tr>
<td>Lymphocytes/mm3</td>
<td>1200-1500</td>
<td>800-1199</td>
<td>&lt;800</td>
</tr>
</tbody>
</table>

- Negative acute phase proteins: albumin, pre-albumin
- Acute phase protein: transferin

Nutrition screening and assessment tools

- SGA – Subjective Global Assessment
- MNA – Mini Nutritional Assessment
- NRI – Nutrition Risk Index
- MST – Malnutrition Screening Tool
- NST – Notthingham Screening Tool
- MUST – Malnutrition Universal Screening Tool
- SNAQ – Short Nutritional Assessment Questionnaire©
- NRS – 2002 – Nutrition Risk Screening

Functional tests– handgrip dynamometry
Micronutrient deficiencies

Water soluble vitamins: thiamine, folate, B12, C

Fat-soluble vitamins: A, D, E, K

Minerals: Iron, Calcium, Magnesium, Zinc, Selenium

*Many additional nutrients may be absorbed from the ileum depending on transit time.*
ESPEN guideline: Clinical nutrition in inflammatory bowel disease

Alastair Forbes a,*, Johanna Escher b, Xavier Hébuterne c, Stanisław Klęk d, Zeljko Krznanic e, Stéphane Schneider c, Raanan Shamir f, Kalina Stadelova g, Nicolette Wierdsma h, Anthony E. Wiskin i, Stephan C. Bischoff j

a Norwich Medical School, University of East Anglia, Bob Champion Building, James Watson Road, Norwich, NR4 7UQ, United Kingdom
b Erasmus Medical Center — Sophia Children’s Hospital, Office Sp-3460, Wytemaweg 80, 3015 CN, Rotterdam, The Netherlands
c Gastroenterologie et Nutrition Clinique, CHU de Nice, Université Côte d’Azur, Nice, France
d General and Oncology Surgery Unit, Stanley Dudrick’s Memorial Hospital, 15 Tyniecka Street, 32-050, Skawina, Krakau, Poland
e Clinical Hospital Centre Zagreb, University of Zagreb, Kispaticeva 12, 10000, Zagreb, Croatia
f Tel-Aviv University, Schneider Children’s Medical Center of Israel, 14 Kaplan St, Peta Tikhv, 49202, Israel
g University Clinic for Gastroenterohepatology, Clinical Centre “Mother Therese”, Mother Therese Str No 18, Skopje, Republic of Macedonia
h VU University Medical Center, Department of Nutrition and Dietetics, De Boelelaan 1117, 1081 HV, Amsterdam, The Netherlands
i Paediatric Gastroenterology & Nutrition Unit, Bristol Royal Hospital for Children, Upper Maudlin Street, Bristol, BS2 8BJ, United Kingdom
j Institut für Ernährungsmedizin (180) Universität Hohenheim, Fruwirthstr. 12, 70593 Stuttgart, Germany
Iron deficiency

• Pathophysiology: chronic blood loss, impaired iron metabolism, inadequate intake

• Symptoms: anaemia, fatigue, sleeping disorders, restless legs syndrome, attention deficit, discontentment, agitation, female infertility, glossitis, angular cheilitis

• Prevalence rate: 36-90% of IBD patients with iron deficiency anemia

All patients regardless of their age should be assessed for the presence of anaemia

### Diagnosis of iron deficiency anaemia

<table>
<thead>
<tr>
<th>Patients without clinical, endoscopic, or biochemical evidence of active disease</th>
<th>Serum ferritin</th>
<th>Transferin saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;30 μg/L</td>
<td></td>
</tr>
<tr>
<td>Presence of inflammation</td>
<td>&lt;100 μg/L</td>
<td></td>
</tr>
<tr>
<td>Presence of biochemical or clinical evidence of inflammation</td>
<td>&gt;100 μg/L</td>
<td>&lt;20%</td>
</tr>
</tbody>
</table>

Serum ferritin level 30-100μg/L  → possible combination of true iron deficiency and anaemia of chronic disease.

Anaemia in IBD

- Iron deficiency anaemia, anaemia of chronic disease, anaemia of mixed origin (majority)
- Diagnostic criteria depend on the level of inflammation
- Laboratory screening: complete blood count, serum ferritin, CRP

<table>
<thead>
<tr>
<th>Phase of disease</th>
<th>Frequency of measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remission/mild</td>
<td>6-12 months</td>
</tr>
<tr>
<td>Active</td>
<td>3 months</td>
</tr>
</tbody>
</table>

Vitamin B12 deficiency

• Prevalence:
  11-22% CD\textsuperscript{1,2}
  100% with ileal resection > 60cm
  48% with ileal resection 20-40cm

• Symptoms:
  Macrocytosis, neurological symptoms, pancytopenia, dementia

Vitamin B12 deficiency - diagnosis

• Biochemical deficiency:

Association between low serum cobalamin levels (<148 pM) and a functional biomarker such as homocysteine (>15 µM) or methylmalonic acid (>270 µM).

CD patients with ileal involvement and/or resection and/or clinical deficiency features should be screened yearly for B12 deficiency.

Folate deficiency

• Low intake, malabsorption (ileitis, small bowel resection), excessive folate utilization due to mucosal inflammation, medications

**Methotrexate**: inhibition of dihydrofolate reductase

**Sulphasalasine**: folate malabsorption

**Azathioprine and 6-mercaptopurine**: macrocytosis through myelosuppressive activity

Folate deficiency

• Prevalence: 20-60%

<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>CD</th>
<th>UC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Folic acid</td>
<td>54-67%</td>
<td>35%</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>48%</td>
<td>5%</td>
</tr>
</tbody>
</table>

Calcium deficiency

• CD 13%, UC 10%\(^1\)

• Inadequate intake (restrictive diets that exclude milk and dietary products)

• decreased intestinal/renal absorption (binding to unabsorbed fatty acids, resection of ileum – vitamin D3 decrease)

• Bone loss and osteoporosis

• With corticosteroid involvement – osteopenia (51-77%), osteoporosis (17-28%) \(^2\)

---

The importance of vitamin D in the pathology of bone metabolism in inflammatory bowel diseases

Iwona Krela-Kaźmierczak¹, Aleksandra Szymczak¹, Liliana Łykowska-Szuber¹, Piotr Eder¹, Kamila Stawczyk-Eder¹, Katarzyna Klimczak¹, Krzysztof Linke¹, Wanda Horst-Sikorska²

Arch Med Sci 5, October / 2015
Vitamin D

- 22-70% of CD patients, up to 45% UC patients deficient\(^1\)
- Pathophysiology: inadequate dietary intake, malabsorption, decreased sunlight exposure
- Associated with bone disease independently of exogenous glucocorticoid administration\(^2\)
- Risk factors for osteoporosis in IBD population: low serum vitamin D, male gender, Asian ethnicity, CD, low BMI and corticosteroid use\(^3\)

---
\(^3\) Abraham BP, Prasad P, Malaty HM. Vitamin D deficiency and corticosteroid use are risk factors for low bone mineral density in inflammatory bowel disease patients. Dig Dis Sci 2014;59:1878e84.
Vitamin D

• Higher plasma levels of 25(OH) vitamin D reduce the risk of incident IBD, particularly CD\textsuperscript{1}

• Low plasma 25(OH)D is associated with increased risk of surgery and hospitalizations in both CD and UC and normalization of 25(OH)D status is associated with a reduction in the risk of CD-related surgery\textsuperscript{2}

<table>
<thead>
<tr>
<th>Serum 25OHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;15 ng/mL deficiency</td>
</tr>
<tr>
<td>&lt;20 ng/mL insufficiency</td>
</tr>
<tr>
<td>&gt;30 ng/mL optimal</td>
</tr>
</tbody>
</table>

LOWER LEVELS OF VITAMIN D CORRELATE WITH CLINICAL DISEASE ACTIVITY AND QUALITY OF LIFE IN INFLAMMATORY BOWEL DISEASE

Francisca DIAS DE CASTRO¹, Joana MAGALHÃES¹, Pedro BOAL CARVALHO¹, Maria João MOREIRA¹, Paula MOTA² and José COTTER¹,³

TABLE 3. Association between Vitamin D levels and disease activity and quality of life

<table>
<thead>
<tr>
<th>Vitamin D level</th>
<th>No clinical remission</th>
<th>Clinical remission</th>
<th>P value</th>
<th>SIBDQ &lt;50</th>
<th>SIBDQ &gt;50</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>25(OH)D, ng/mL, mean</td>
<td>21.6±6.0</td>
<td>28.0±10.3</td>
<td>0.001</td>
<td>23.4±6.9</td>
<td>27.9±10.8</td>
<td>0.041</td>
</tr>
<tr>
<td>Vitamin D &lt;20 ng/mL, n</td>
<td>12</td>
<td>11</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin D &gt;20 ng/mL, n</td>
<td>12</td>
<td>41</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

25(OH)D, 25-hydroxyvitamin D; SIBDQ, Short Inflammatory Bowel Disease Questionnaire.
Remission was defined as Harvey-Bradshaw index (HBI) score ≤3 for Crohn's disease and a partial Mayo Score ≤2 with no subscore >1 for ulcerative colitis. *Chi-square test.
Vitamin A

• Vitamin A deficiency (<20µg/L)
• Low levels do not indicate clinical deficiency – hypoproteinemia lowers vitamin A levels
• Poor wound healing, night blindness, xerophthalmia
• **Avoid supplementation**: higher risk of bone fractures and liver toxicity¹,²

Magnesium

- Chronic diarrhea, inadequate dietary intake
- Hypocalcaemia/hypoparathyroidism → lower intestinal calcium absorption
- 24 hour urinary magnesium is most accurate
- Screening and supplementation consider in patients with significant diarrhea (>300 g/day)

Zinc

- Pathophysiology: chronic diarrhea, small bowel malabsorption, increased need in hypermetabolic states (sepsis, critical illness)
- Poor wound healing, acrodermatitis, poor taste
- Unclear prevalence

Patients with IBD with serum zinc deficiency are more likely to have adverse disease-specific outcomes. As these outcomes improve with normalization of zinc, the results from this study support the role for close monitoring and replacement of zinc in patients with IBD.

Siva S. et al. Inflamm Bowel Dis 2017
Selenium

- Absorption not fully understood
- Lower mean levels in CD and UC
- No evidence to support checking for selenium deficiency

Table 2. Mean selenium concentrations of IBD patients as a function of disease severity.

<table>
<thead>
<tr>
<th>IBD Patients</th>
<th>n</th>
<th>Mean Se ± SEM (µg/L)</th>
<th>Range (µg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UC patients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>32</td>
<td>68.8 ± 2.91&lt;sup&gt;a&lt;/sup&gt;</td>
<td>45.5–111.8</td>
</tr>
<tr>
<td>Moderate</td>
<td>4</td>
<td>55.2 ± 7.38&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>41.3–66.5</td>
</tr>
<tr>
<td>Severe</td>
<td>17</td>
<td>49.3 ± 5.72&lt;sup&gt;b&lt;/sup&gt;</td>
<td>12.4–91.1</td>
</tr>
<tr>
<td>CD patients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inactive</td>
<td>17</td>
<td>56.5 ± 3.71</td>
<td>37.2–99.3</td>
</tr>
<tr>
<td>Mild</td>
<td>21</td>
<td>54.4 ± 3.79</td>
<td>20.6–86.9</td>
</tr>
<tr>
<td>Moderate</td>
<td>9</td>
<td>50.6 ± 8.07</td>
<td>12.4–103.5</td>
</tr>
<tr>
<td>Severe</td>
<td>6</td>
<td>44.8 ± 6.81</td>
<td>24.8–62.1</td>
</tr>
</tbody>
</table>

<sup>a,b</sup> different superscripts in same column, significant difference (p < 0.05).
Hair mineral and trace element contents as reliable markers of nutritional status compared to serum levels in children with inflammatory bowel disease
H.R. Yang, J.M. Cho

Serum micronutrient levels should be cautiously interpreted in conjunction with inflammatory markers in patients with IBD. Furthermore, hair trace elements measurements may support understanding of body micronutrient status in children with IBD.

JCC Poster presentations: Clinical: Diagnosis and outcome (2017)
Table 3. Correlation coefficients between MDS and intake of nutrients in female subjects

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>MDS</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake</td>
<td>-0.458</td>
<td>0.005</td>
</tr>
<tr>
<td>Protein intake</td>
<td>-0.298</td>
<td>0.022</td>
</tr>
<tr>
<td>Animal protein intake</td>
<td>-0.305</td>
<td>0.013</td>
</tr>
<tr>
<td>Fat intake</td>
<td>-0.686</td>
<td>0.008</td>
</tr>
<tr>
<td>SFA intake</td>
<td>-0.708</td>
<td>0.013</td>
</tr>
<tr>
<td>PUFA intake</td>
<td>-0.247</td>
<td>0.047</td>
</tr>
<tr>
<td>Carbohydrate intake</td>
<td>-0.224</td>
<td>0.322</td>
</tr>
<tr>
<td>Fiber intake</td>
<td>0.314</td>
<td>0.020</td>
</tr>
<tr>
<td>Ca intake</td>
<td>-0.544</td>
<td>0.039</td>
</tr>
<tr>
<td>Mg intake</td>
<td>-0.305</td>
<td>0.013</td>
</tr>
<tr>
<td>Fe intake</td>
<td>0.255</td>
<td>0.732</td>
</tr>
<tr>
<td>Zn intake</td>
<td>0.241</td>
<td>0.450</td>
</tr>
<tr>
<td>Vitamin A intake</td>
<td>-0.213</td>
<td>0.415</td>
</tr>
<tr>
<td>Vitamin B1 intake</td>
<td>-0.276</td>
<td>0.040</td>
</tr>
<tr>
<td>Vitamin B2 intake</td>
<td>-0.278</td>
<td>0.093</td>
</tr>
<tr>
<td>Vitamin B3 intake</td>
<td>-0.248</td>
<td>0.078</td>
</tr>
<tr>
<td>Vitamin B6 intake</td>
<td>-0.275</td>
<td>0.036</td>
</tr>
<tr>
<td>Vitamin C intake</td>
<td>-0.370</td>
<td>0.714</td>
</tr>
</tbody>
</table>

Table 4. Correlation coefficients between MDS and intake of nutrients in male subjects

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>MDS</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake</td>
<td>0.017</td>
<td>0.097</td>
</tr>
<tr>
<td>Protein intake</td>
<td>-0.242</td>
<td>0.043</td>
</tr>
<tr>
<td>Animal protein intake</td>
<td>-0.570</td>
<td>0.036</td>
</tr>
<tr>
<td>Fat intake</td>
<td>-0.442</td>
<td>0.047</td>
</tr>
<tr>
<td>SFA intake</td>
<td>-0.509</td>
<td>0.008</td>
</tr>
<tr>
<td>PUFA intake</td>
<td>-0.186</td>
<td>0.437</td>
</tr>
<tr>
<td>Carbohydrate intake</td>
<td>0.442</td>
<td>0.032</td>
</tr>
<tr>
<td>Fiber intake</td>
<td>0.678</td>
<td>0.021</td>
</tr>
<tr>
<td>Ca intake</td>
<td>-0.207</td>
<td>0.038</td>
</tr>
<tr>
<td>Mg intake</td>
<td>0.283</td>
<td>0.052</td>
</tr>
<tr>
<td>Fe intake</td>
<td>0.025</td>
<td>0.103</td>
</tr>
<tr>
<td>Zn intake</td>
<td>0.165</td>
<td>0.029</td>
</tr>
<tr>
<td>Vitamin A intake</td>
<td>-0.106</td>
<td>0.022</td>
</tr>
<tr>
<td>Vitamin B1 intake</td>
<td>-0.189</td>
<td>0.034</td>
</tr>
<tr>
<td>Vitamin B2 intake</td>
<td>-0.079</td>
<td>0.047</td>
</tr>
<tr>
<td>Vitamin B3 intake</td>
<td>-0.024</td>
<td>0.544</td>
</tr>
<tr>
<td>Vitamin B6 intake</td>
<td>0.241</td>
<td>0.056</td>
</tr>
<tr>
<td>Vitamin C intake</td>
<td>-0.199</td>
<td>0.048</td>
</tr>
</tbody>
</table>
**MINUTE FOR IBD**

**Background**

Intestinal inflamed conditions (IBD) are a group of chronic gastrointestinal diseases characterized by inflammation of the mucosa and submucosa of the digestive tract. The underlying cause of IBD is multifactorial, involving a complex interplay between genetic, environmental, and immunological factors. The understanding of the pathogenesis and the development of effective treatments for IBD have been significantly advanced by the recent technological advancements in microbiome research.

**Project objectives**

- To investigate the role of gut microbiota in the pathogenesis of IBD.
- To develop novel therapeutic strategies targeting the microbiome.

**Methods**

In the project, gut microbiota samples from IBD patients and healthy controls were analyzed using high-throughput sequencing of 16S rRNA genes. The abundance of different bacterial phyla was quantified, and the potential correlations with clinical outcomes were explored.

**Results**

The relative abundance of major bacterial phyla detected by sequencing of 16S rRNA gene amplicons. Samples of colon mucosa were taken during endoscopy from different sites along the colon (from C1-terminal ileum to C6-rectum). Samples were collected from two subjects – one diagnosed with Crohn’s disease (CD) and one with IBS.

**Fig 3.** The relative abundance of major bacterial phyla detected by sequencing of 16S rRNA gene amplicons.
39th ESPEN Congress

on Clinical Nutrition & Metabolism

THE HAGUE
THE NETHERLANDS
9-12th September 2017

Nutrition meets Innovation