Latest Nutritional Management strategies in treating Crohn's Disease

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Acknowledgments

- Dr Bhuvaneshwari Shankar
- Ms Lekha
- Ms Divya Lakshmi
- Dr S Srinivas
- No Conflicts of Interest
Graduated from GMKMCH, Salem

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MRCPCH-2005

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Alder Hey Childrens Hospital, Liverpool

Paediatric Endoscopy fellowship at Sheffield

Honorary Lecturer-University of Liverpool

Joined Apollo Family in November 2011.
My Alma Mater
My Alma mater
1904 & 1932
History of Crohns

• Described by two Doctors
• 1904-Antoni Lesniowski
• 1932-Burrill Bernard Crohn
• A series of terminal ileitis which was later described as what we now call as Crohns Disease
<table>
<thead>
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</thead>
<tbody>
<tr>
<td>Scotland</td>
<td>0.7–2.3*</td>
<td>1.5–1.9*</td>
<td>–</td>
<td>–</td>
<td>R</td>
</tr>
<tr>
<td>Norway</td>
<td>2.5</td>
<td>4.3</td>
<td>0</td>
<td>6.8</td>
<td>P</td>
</tr>
<tr>
<td>France</td>
<td>2.1</td>
<td>4.3</td>
<td>0.6</td>
<td>–</td>
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<td>Sweden</td>
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<td>1.9</td>
<td>0.7</td>
<td>5.3</td>
<td>P</td>
</tr>
<tr>
<td>Wales</td>
<td>2.2</td>
<td>0.7</td>
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<tr>
<td>Denmark</td>
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<td>0</td>
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<tr>
<td>Sweden</td>
<td>1.2</td>
<td>1.4–3.2†</td>
<td>2.2</td>
<td>4.6–7.0†</td>
<td>P</td>
</tr>
<tr>
<td>Wales</td>
<td>1.4</td>
<td>0.8</td>
<td>0.5</td>
<td>2.6</td>
<td>P</td>
</tr>
<tr>
<td>UK and Ireland</td>
<td>3.0</td>
<td>1.5</td>
<td>0.6</td>
<td>5.2</td>
<td>P</td>
</tr>
<tr>
<td>Scotland</td>
<td>2.5</td>
<td>1.3</td>
<td>–</td>
<td>–</td>
<td>R</td>
</tr>
<tr>
<td>Denmark</td>
<td>2.3</td>
<td>1.8</td>
<td>0.2</td>
<td>4.3</td>
<td>R</td>
</tr>
<tr>
<td>Norway</td>
<td>2.1</td>
<td>2.0</td>
<td>–</td>
<td>–</td>
<td>P</td>
</tr>
<tr>
<td>Australia</td>
<td>0.1–2.0‡</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>R</td>
</tr>
<tr>
<td>USA</td>
<td>4.6</td>
<td>2.1</td>
<td>0.3</td>
<td>7.0</td>
<td>P</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>4.8</td>
<td>2.7</td>
<td>1.8</td>
<td>0.3</td>
<td>R</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>2.1</td>
<td>1.6</td>
<td>3.6</td>
<td>5.2</td>
<td>P</td>
</tr>
<tr>
<td>Sweden</td>
<td>1.7–8.4§</td>
<td>3.3–1.8§</td>
<td>0.2</td>
<td>5.2–10.5§</td>
<td>P</td>
</tr>
</tbody>
</table>

IBD, inflammatory bowel disease; CD, Crohn disease; UC, ulcerative colitis; IC, indeterminate colitis; R, retrospective data collection; P, prospective data collection.


Adapted from van der Zaag et al. with permission.
IBD-Growing incidence!!

<table>
<thead>
<tr>
<th>Disease</th>
<th>1973</th>
<th>2003</th>
</tr>
</thead>
<tbody>
<tr>
<td>CROHNS</td>
<td>0.1</td>
<td>4.6</td>
</tr>
<tr>
<td>UC</td>
<td>0.5</td>
<td>3.2</td>
</tr>
</tbody>
</table>
Incidence in the UK

- 5.2 per 100,000
- Boys > Girls
- CD > IC > UC
- Asian > Ethnic
- Mean 11.9 years
- 5% Under 15
- 15% over 60
- Certainly increasing 25% in 20 years
- UC > CD
Etiology of Crohn's Disease

- Infection
- Drugs
- Smoking
- Very much unknown
Explored or being explored areas

- Epidemiological
- Gut/environmental interface
- Inflammatory process
- Genetics/Mutations
- Chromosome 16 (CARD 15/NORD 2)
Genetics

• Long known that Crohn’s / UC is commoner in families / twins

• Not simple inheritance

• Sibling with CD/UC means 15-30x the risk

• 1 in 7 patients have a relative with the illness
Genetics (2)

THE HUMAN GENOME PROJECT

- 1996: Oxford group
- Showed Crohn’s and UC share some susceptibility genes
- Chromosomes 3, 7 and 12
SMOKING!

- Increased risk of:
  - Getting it in the first place
  - Aggressive disease
  - Relapse
  - Hospital admissions
  - Surgery
  - Cancer
An Infective Cause for Crohn’s?

- M. Paratuberculosis
- E. Coli
- Viruses eg: measles
- Post-infective bacteria
- Clostridium
- Bacteroides

- Toothpaste
- Cornflakes
- Hygiene
- “Allergy”
- Refined sugars
- Trauma
- Pollutants
Description
Aetiology
Pathophysiology
Predisposing factors

**Symptoms**
Signs
Investigations
Complications
Alternatives
Management
Prognosis
Symptoms
-depend on site of disease

- Abdominal pain
- Weight loss
- Diarrhoea +/- blood
- Obstructive symptoms
- Complications of fistulae
- Complications of malabsorption
  - B12, Ca/Vit D, Zn, etc
What do children present with?

- "Classical Triad"
- Abdominal pain, diarrhoea, weight loss
- Toronto - 1980-89: 80% presented
- UK - 98-99: 25%
- 44% - no diarrhoea but abdo pain in 72%
- Extra-intestinal - 10% Erythema nodosum
Many children with CD present with vague complaints such as lothargy, anorexia and abdominal discomfort or with isolated growth failure. A significant minority have markedly impaired final adult height [17, 18]. Neglect to record growth parameters, particularly for those not presenting to a paediatrician, has been identified [7, 17, 20]. Other symptoms may include fever, nausea, vomiting, delayed puberty, psychiatric disturbance and erythema nodosum [7]. The clinical course of CD is characterised by exacerbations and remission. CD tends to cause greater disability than UC.

**Table 1**
Presenting symptoms and signs of children in UK with CD; data from the national study [7]

<table>
<thead>
<tr>
<th>Patients</th>
<th>CD (n = 379)</th>
<th>IC (n = 72)</th>
<th>UC (n = 172)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Common symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>274 (72%)</td>
<td>54 (75%)</td>
<td>106 (62%)</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>214 (56%)</td>
<td>56 (78%)</td>
<td>127 (74%)</td>
</tr>
<tr>
<td>Bleeding</td>
<td>84 (22%)</td>
<td>49 (68%)</td>
<td>145 (84%)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>220 (58%)</td>
<td>25 (35%)</td>
<td>53 (31%)</td>
</tr>
<tr>
<td>Lothargy</td>
<td>103 (27%)</td>
<td>10 (14%)</td>
<td>20 (12%)</td>
</tr>
<tr>
<td>Anorexia</td>
<td>94 (25%)</td>
<td>9 (13%)</td>
<td>11 (6%)</td>
</tr>
<tr>
<td><strong>Other symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arthropathy</td>
<td>28</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>22</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Constipation/soiling</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychiatric symptoms</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary amenorrhoea</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Signs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anal fistula</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Growth failure/delayed puberty</td>
<td>14</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Anal abscess, ulcer</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erythema nodosum/rash</td>
<td>6</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Liver disease</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Appendicectomy</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toxic megacolon</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 1. Presence of symptoms at diagnosis in 623 children with inflammatory bowel disease. Adapted from Sawczenko et al. (20) with permission.
Description
Aetiology
Pathophysiology
Predisposing factors
Symptoms

**Signs**
Investigations
Complications
Alternatives
Management
Prognosis
Oral apthous ulcers-OFG
Uveitis

- Recurrent red Eye
- Red eye
- Associated with GI Symptoms
Erythema Nodosum

IBD

TB/ Sarcoid

OCP, sulphonamides

Streptococcal infections

Yersinia, psitticosis

Lymphogranuloma venereum

Connective tissue disorders

Tuleraemia
Pyoderma Gangrenosum
Other manifestations

- Arthropathy with effusion
- Sacro-ileitis
- Failure to thrive
- Weight loss
- Nocturnal stooling
- Recurrent Diarrhoea
- List is endless............
Description
Aetiology
Pathophysiology
Predisposing factors
Symptoms
Signs

Investigations
Complications
Alternatives
Management
Prognosis
How do you diagnose?

- Clinical-History, History, History
- Biochemical
- Endoscopic
- Radiological
- Histological
- +/- nuclear medicine
What Bloods – are they useful?

- FBC
- ESR
- LFT-esp albumin
- CRP
- Stool
- TB and C difficile
<table>
<thead>
<tr>
<th>Blood test</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin &amp; Platelets</td>
<td>90.8%</td>
<td>80%</td>
</tr>
<tr>
<td>ESR (Known already)</td>
<td>82</td>
<td>78</td>
</tr>
<tr>
<td>CRP</td>
<td>60% (Poor sensitivity)</td>
<td></td>
</tr>
<tr>
<td>2 out of 3</td>
<td>85.7%</td>
<td>89.8%</td>
</tr>
<tr>
<td>1 out of 2 (PLT+Hb)</td>
<td>90.8%</td>
<td>80%</td>
</tr>
<tr>
<td>Albumin</td>
<td>Poor correlation</td>
<td></td>
</tr>
<tr>
<td>Beattie et al 1995</td>
<td></td>
<td></td>
</tr>
<tr>
<td>39 (26 cd/13 uc) 37 c</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelets</td>
<td>88% (CD)</td>
<td>70% (UC)</td>
</tr>
<tr>
<td>CD at least one abnormal</td>
<td>UC-8% All normal</td>
<td></td>
</tr>
<tr>
<td>Albumin was reduced</td>
<td>Not significant</td>
<td></td>
</tr>
</tbody>
</table>

ESR inessential predictor in combination with platelets and Hb. Only 3 patients have elevated ESR.
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<td></td>
<td></td>
</tr>
<tr>
<td>Albumin was reduced</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Results</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Results**

ESR inessential predictor in combination with platelets and Hb. Only 3 patients has elevated ESR.
<table>
<thead>
<tr>
<th>Degree</th>
<th>UC</th>
<th>CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (all 4 normal)</td>
<td>21%</td>
<td>54%</td>
</tr>
<tr>
<td>Moderate/Severe (all 4)</td>
<td>3.8%</td>
<td>4.3%</td>
</tr>
<tr>
<td>ESR</td>
<td>26% Normal</td>
<td>18% Moderate/Severe</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>32% Normal</td>
<td></td>
</tr>
<tr>
<td>platelets</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>60%</td>
<td></td>
</tr>
<tr>
<td>Endoscopy (and visualization of oral and/or perianal regions)</td>
<td>Crohn disease</td>
<td>Ulcerative colitis</td>
</tr>
<tr>
<td>------------------------------------------------------------</td>
<td>---------------</td>
<td>--------------------</td>
</tr>
<tr>
<td>Ulcers (aphthous, linear, or stellate)</td>
<td>Ulcers</td>
<td></td>
</tr>
<tr>
<td>Cobblestoning</td>
<td>Erythema</td>
<td></td>
</tr>
<tr>
<td>Skip lesions</td>
<td>Loss of vascular pattern granularity</td>
<td></td>
</tr>
<tr>
<td>Strictures</td>
<td>Friability</td>
<td></td>
</tr>
<tr>
<td>Fistula</td>
<td>Spontaneous bleeding</td>
<td></td>
</tr>
<tr>
<td>Abnormalities in oral and/or perianal regions</td>
<td>Pseudopolyps</td>
<td></td>
</tr>
<tr>
<td>Segmental distribution</td>
<td>Continuous with variable proximal extension from rectum</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Histology</th>
<th>Submucosal (biopsy with sufficient submucosal tissue) or transmural involvement (surgical specimen)</th>
<th>Mucosal involvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcers, crypt distortion</td>
<td>Crypt distortion</td>
<td></td>
</tr>
<tr>
<td>Crypt abscess</td>
<td>Crypt abscess</td>
<td></td>
</tr>
<tr>
<td>Granulomas (non-caseating, nonmucin)</td>
<td>Goblet cell depletion</td>
<td></td>
</tr>
<tr>
<td>Focal changes (within biopsy)</td>
<td>Mucin granulomas (rare)</td>
<td></td>
</tr>
<tr>
<td>Patchy distribution (biopsies)</td>
<td>Continuous distribution</td>
<td></td>
</tr>
</tbody>
</table>

Histology for both Crohn disease and ulcerative colitis included acute and chronic inflammation with architectural changes, loss of glands, and branching of crypts. Crohn disease abnormalities in oral region included lip swelling, gingival hyperplasia, aphthous ulcers; Crohn disease abnormalities in perianal region included tags, fissures, fistulae, and abscess.
Endoscopy and upper GI endoscopy histology of multiple biopsies

Fig. 2

Conclusive

Small Bowel Follow Through (SBFT)
This publication has been produced by the IBD Working Group of BSPGHAN with the financial support of CICRA - Crohn’s in Childhood Research Association and NACC - National Association for Colitis & Crohn’s Disease
Treatment-Considerations

- No available Surgical or pharmacological cure!
- Be open and honest
Considerations

• Induction of remission and treating relapse
• Growth-Measure, treat suppression
• Nutrition +/- gastrostomy or NGT
Enteral nutrition

- Liquid formula: Elemental (single amino acids), semi-elemental (small peptides of 4/5 amino acids), polymeric (whole protein)
- Calorie density of most feeds is between 0.7 and 1.5 kcal/mL
- Oral, NG, gastrostomy tube

<table>
<thead>
<tr>
<th>Exclusive EN</th>
<th>Partial EN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sole dietary source</td>
<td>+ plus normal diet</td>
</tr>
<tr>
<td>Induce remission</td>
<td>Maintain remission Nutritional support</td>
</tr>
<tr>
<td>Duration of 6-12 weeks followed by introduction of new food over 2-4 weeks</td>
<td>No defined duration, usually prolonged</td>
</tr>
</tbody>
</table>
History of EN in Crohn’s disease

- Efficacy suspected when patients awaiting surgery (nil orally and TPN) showed improvement
- ? possible role of luminal antigens in triggering acute attack and avoiding further damage by 'total bowel rest'
- Initial studies: elemental diets- amino acids (reduced antigenicity) and low fat (MCT-require little luminal lipolysis and micellar solubilization before absorption) to provide 'bowel rest'
- Elemental diets as effective as corticosteroids in remission
- Later, due to better nitrogen absorption and reduced osmotic load of peptide or whole protein diets than amino acid diet, polymeric enteral diets tried and found equally effective.
Mechanism of action?

• Restoration of altered intestinal permeability
• Decreased antigenic effects of food proteins
• Avoidance of pro-inflammatory trigger factors such as food additives
• Improvement of nutritional status and repletion of nutrient, trace element or vitamin deficiency implicated in tissue repair mechanisms or in immune defense
• Effect on the composition of the intestinal microflora and modulation of the intestinal mucosal immune response

• Clinical response to EN is associated with –
  ✅ Correction of the imbalance between proinflammatory and anti-inflammatory cytokines (reduced IL6, increased TGFβ)
  ✅ Reduction in lymphokine-secreting cells in the intestinal mucosa

EEN and Crohn’s disease

- Disease remission (70-80%) in new CD cases
- Improved quality of life

- Improvement of weight and height parameters (in 10 weeks to 6 months)
- Improved PCDAI scores

- Improvement in inflammatory markers
- Mucosal healing at endoscopy (74% vs 33% with steroids at 10 weeks, \( p<0.05 \))
EEN and Crohn’s disease

Factors determining EEN use

• Physician belief (62% European vs 4% American Ped gastroenterologist)
• Patient and parent consent and compliance
• Cost, palatability and invasiveness of NG use
• Growth and nutritional status
• Situations precluding use of steroids
Meta-analysis (children)

- n=147, 5 trials, pooled RR 0.95 (95% CI 0.67-1.34)
- N=144, 4 trials, pooled RR 0.97 (0.7-1.4)*

Limited data, good studies required

Equally effective as steroids in inducing remission

Aliment Pharmacol Ther 2007; 26, 795–806*, JPGN 2000;31:8-
EEN and Crohn’s disease

Children
- **EEN equally effective as steroids**
- Growth issues are vital
- Growth failure ~50%
- Underweight ~ 90%
- **Better compliance to EEN- parental control, support by dieticians and physicians, evident benefit on weight and height growth**

Adults
- **Less effective than steroids** (6 trials, pooled OR of 0.33 favoring steroids 95% CI 0.21 to 0.53)
- Growth not important
- **Poor compliance ~21% in meta-analysis** *

<table>
<thead>
<tr>
<th>EEN</th>
<th>Steroids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improves nutrition</td>
<td>Easy to administer</td>
</tr>
<tr>
<td>No side effects</td>
<td>Cheap</td>
</tr>
<tr>
<td>Mucosal healing better</td>
<td>No extra counselling</td>
</tr>
<tr>
<td>Motivated, compliant patient</td>
<td>Side effects: growth, bone density</td>
</tr>
<tr>
<td>Cost, palatability</td>
<td>Poorer mucosal healing</td>
</tr>
<tr>
<td>Polymeric preferred  (better acceptance and taste lower cost, no NG feeds)</td>
<td></td>
</tr>
</tbody>
</table>
EEN and remission

• Duration of EEN variable 3-12 weeks, majority 6-8 weeks

• Mean time for obtaining remission 11-18 days*

• Recommendation: 3-4 week trial for observation for efficacy and total duration of min 8 wks, may be increased to 12 weeks

• No difference in the efficacy of elemental versus non-elemental formulas (10 trials, n-334, OR 1.10; 95% CI 0.69 to 1.75).

• No difference in efficacy based on fat content (7 trials, n-209, low fat vs high fat < 20 g vs > 20 g/1000 kCal, OR 1.13; 95% CI 0.63 to 2.01).

EEN and site of disease

- Initial SB > colon (remission rate, isolated colonic 50%, ileocolonic 82%, ileal 97.1%)*
- Equal in isolated colon vs isolated SB (15/19 vs 10/13)
- Cochrane review - insufficient data to favour one disease site over another, use in all

Mode of administration

Both oral EEN and continuous NG feeding for 8 weeks are equally effective to induce remission [oral (75%) vs (85%) NG]^
Enteral nutrition for maintenance of remission

- **Advantage:** minimizing use of steroids/ immunosuppressive drugs and maintaining good nutrition
- **Supplemental EN** (any type, along with normal food, duration of EN - 1 year or more)
- **Significantly higher rate of clinical remission in those on EN vs without EN**
- **Higher amounts of enteral formula associated with higher remission rates:**
  - ≥30 vs. <30 kcal/kg ideal body weight/day or half the requirement as EN
- **Problems:**
  - Patient selection (better compliance given EN)
  - Several patients on concomitant medications (5-ASA or azathioprine)
  - Available evidence is inadequate, large RCT are necessary
- **Enteral nutritional supplementation could be considered as an alternative or as an adjunct to maintenance drug therapy in Crohn’s disease**

Eur J Gastroenterology & Hepatology 2010, 22:1–8
Cochrane Database of Syst Rev 2007, Issue 3. CD005984
EEN and remission

Lack of response (~20-30% of patients)

• Partial EN (significantly poorer response 42% vs 15%)*
• Poor compliance (meta-analysis- 21% of adults * vs 9-15% children, parental supervision)
• Inadequate energy intake
• Intolerance of the feed
• Resistant disease- severe disease, stricture

Role of diet as an etiological agent

- No particular diets seem to have any particular triggers or help in remission of Crohn's disease.
- Any dietary intervention will have to be done under supervision.
- Linear growth and puberty will have to be the main focus.
- “Listen to your belly”
Diet in IBD

- Lack of evidence
- Most of advice is anecdotal
- Various diets have been advised to be avoided
- High fiber diet
- Caffeine, alcohol, sorbitol, carbonated drinks
- Fat containing diet
- hot & spicy food
- No routine use of TPN
Lactose elimination in Crohn's Disease

- There are inflammatory bowel disease (IBD) patients avoid lacteal products without evidence of lactose malabsorption, probably because of incorrect patient perceptions and arbitrary advice from physicians and diet books.
- Spanish Study 2004
  - 7/24 in IBD (CD&UC) and 5/25 (control)
  - No difference
- 2002-von tirpitz et al, Germany in their study mention that milk intolerance is a problem in relapse due to decrease lactase levels but not predominant cause in CD.
- The key points are-Lactase enzyme activity, SIBO, Small intestine transit time.
Fish Oil-Omega-3 in Crohn's disease

- Cochrane review in 2009
- Randomized controlled trials with placebo
- 6 studies
- 3 reported a significant reduction in 1 year follow up
- But two large studies did not find any differences.
Probiotics in EEN

- *Lactobacilli GG*, *Escherichia coli* strain Nissle 1917, VSL#3, *Saccharomyces boulardii*

- All trials had small numbers
- No statistical difference was seen
- No evidence to suggest that probiotics are beneficial for the maintenance of remission in CD.
"Fermentable, Oligo-, Di-, Mono-saccharides And Polyols".

- No evidence it is of benefit in IBD
Cochrane review on EEN-2007&2009

- Paediatric trials and meta analysis
- Showed feeds to be equally effective as corticosteroids.
- Intolerance to formula and inadequate volume are the main pitfalls in all studies.
- Remission rates are three times more with EEN when compared to PEN
Enteral nutrition: Inducing remission and maintaining remission---Is it elemental, semi elemental, polymeric?

- Liquid formula: Elemental (single amino acids), semi-elemental (small peptides of 4/5 amino acids), polymeric (whole protein)
- Calorie density of most feeds is between 0.7 and 1.5 kcal/mL
- Oral, NG, gastrostomy tube
# Pediatric Crohn's Disease Activity Index Calculator

**Cincinnati Children's**

## History (Recall; 1 week)

**Abdominal pain:**
- None
- Mild -- Brief, does not interfere with activities
- Mod/severe - daily, longer lasting affects activities, nocturnal

**Stools (per day):**
- Formed stools or up to 1 liquid stool, no blood
- Up to 2 semi-formed with small blood, or 2-5 liquid with or without small blood
- Any gross bleeding, or ≥ 6 liquid, or nocturnal diarrhea

## Patient Functioning -- General Well-Being

- No Limitation of activities, well
- Occasional difficulty in maintaining appropriate activities, below par
- Frequent limitation of activity, very poor

## Laboratory (values obtained within the past week)

- **Sex:** Male
- **Age (whole years):** 4
- **Hematocrit %:** 36
- **ESR (mm/hr):** 0
- **Albumin (g/dl):** 4.5

## Examination

**Weight**
Success of EEN

• No side effects.

• Compliant patients took it all orally-No NGT or PEG Feeds or hospital admissions.

• Team work.

• Support for parents.

• Co-operation of child and compliance.

• Increasing number of patients opting for EEN.

• From start of EEN none of them have had steroid therapy.
Limitations of EEN

• Lack of availability or alternatives other than peptide feeds in India.

• Cost in non-affordable patients-total cost is around 20k for 8 weeks.

• Co-operation of child and family is paramount.

• Review after completion of feeds only through telemedicine.
Conclusion

- EEN equally effective as steroids in inducing remission (70-80%) in CD children, less effective in adults
- Polymeric formula preferred over elemental (oral or NG) due to lower cost, better taste and equal efficacy
- Minimum 8 weeks duration recommended
- No definite effect of site of disease, fat composition or added glutamine to formula
- Requires commitment of physician, dietician, patient and family
- Better designed studies are required