NMDC221 Session 1
Session Overview

Introduction to Subject & outline of Assessments

Clinical decision making in Nutritional Medicine

Gastrointestinal & Alimentary Disease Part I

- Principles and considerations in nutritional medicine management of Gastro-oesophageal reflux disease (GORD)
  - Adult & Infant

- Nutritional management & consideration of drug-nutrient interactions

Class Activities
Introduction to Subject & outline of Assessments

This subject integrates and consolidates knowledge gained from previous health science and nutritional and dietetic medicine subjects through an exploration of the nutritional management of various health conditions and individual cases. Students learn treatment strategies that incorporate dietary modification and nutritional management and learn to critically evaluate these from the perspectives of traditional empirical knowledge and evidence-based research. Potential drug-herb-nutrient interactions are critically discussed and the fundamentals of case analysis are introduced.

Clinical Nutritional Medicine is an essential foundation for clinical practice.
# Introduction to Subject & outline of Assessments

<table>
<thead>
<tr>
<th>Assessment Tasks</th>
<th>Learning Outcomes Assessed</th>
<th>Session Content Delivered</th>
<th>Session Due</th>
<th>Weighting</th>
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</thead>
<tbody>
<tr>
<td><strong>Concept Maps/Schematics</strong> (3 x A4 single sided Each map worth 10%)</td>
<td>1,3,4</td>
<td>1-12</td>
<td>Sunday following Session 12</td>
<td>30%</td>
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<tr>
<td><strong>Case Study Analysis</strong> (1500 words)</td>
<td>1,2,3,4,5</td>
<td>14--21</td>
<td>Sunday following Session 24</td>
<td>35%</td>
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<tr>
<td><strong>Case Study Analysis</strong> (1500 words)</td>
<td>1,2,3,4,5</td>
<td>22-33</td>
<td>Sunday following Session 36</td>
<td>35%</td>
</tr>
</tbody>
</table>
Session Reading & resources

- **Recommended Reading**
  

- **Review**:
  
  - BIOH111 – Concept Map guidelines
  - NMDF211, NMDM121, BIOC211

**NOTE - Preparation for Session 3** - In Session 3 you will be introduced to **Outcome Measurement Tools** that you will also be using in clinic. Please print the **Gastrointestinal System Rating Scale questionnaire** and bring this to class.
Clinical Decision Making
Clinical Decision Making

- Additional to looking at the nutritional management and prescribing for various health conditions, this subject also seeks to introduce you to some of the tools and techniques used in case analysis.
- These concepts will be developed further in your pre-clinical skills subject – HMCL211, HMCL222, HMCL223
Clinical Decision Making

Clinical decision making in Nutritional Medicine includes:

- Holistic Case analysis/Schematic (concept map) work
- Identifying important case elements/system interactions, etc..
- Considering the individual and then;
  - Formulate Treatment Aims (*what* needs treatment) – including construction of a research strategy i.e. Create answerable clinical questions (PICO)
  - Formulate Treatment Plan, which includes Dietary & Lifestyle Health Goal, which are SMART. (*how* they are going to be treated)
  - Determine appropriate Nutritional supplementation/prescription

- Evaluate

(Leach, 2010)
Clinical Decision Making

Holistic Case analysis

In BIOH111 you studied the purpose and application of **Concept Maps** - a visual representation of a body of knowledge organised so that a number of points of interest (concepts) are linked to show their relationship with one another.

In this subject, we will be progressing your understanding of the *clinical* application of Concept Maps – aka **Schematic**, as a valuable case analysis tool.

This will also assist your ability to successfully complete Assessment One.
Clinical Decision Making

Concept Map/Schematic

- Similar to a Concept Map, a Schematic is a clinical tool that enables you to visually ‘map’ all the details of a case, both subjective and objective.
- This process allows you to ‘see’ holistically the most implicated body system/s and the key interrelationships leading to the Presenting Complaint/s (PC), which will in turn lead to an understanding of Treatment Aims (what needs treatment).
- A schematic process can also illuminate aspects of the case needing more investigation, information or diagnostics, which can all form part of your Treatment Plan.
Clinical Decision Making

Concept Map/Schematic

- All case information is ‘mapped’ via organ & body systems, around the central client details and PC
- Sometimes a Sign or Symptom may appear more than once – e.g., ↑ A/Biotics may be included in both GIT and Immune systems.
- Connecting arrows are then used to identify key holistic interrelationships
- These are then numbered in numerical order, creating an order of priority for your Treatment Aims
The understanding reached via the schematic process, can consolidate your holistic understanding of causative and sustaining factors present in a case.

A full clinical Schematic can be complex and act as an active case document, which can be referred back to and amended as your treatment/s with the client progress.
Systems Interaction

In future subjects and in your clinical practicum, you will be translating understandings reached via the schematic into holistic summaries – e.g.:

**Causative and sustaining factors**

- **1. Immune/GIT**: Frequent Abx and infection disrupts microbiome. Poor immunity (SIgA), decreased nutrient absorption, decreased intestinal repair. Dysbiosis.
- **2. NS/GIT**: Prolonged SNS response affects enteric and parasympathetic nervous systems. Decreased HCl, digestive enzymes and peristalsis. Stress response becomes adapted and eventually exhausted.
- **3. GIT/NS**: Reduced GIT synthesis of 5HT, reduced GABA, reduced cofactor absorption for Neurotransmitter synthesis and muscular innervation. Direct impact on mood, bowel motility, MS and eating habits.
- **4. GIT/Liver**: Dysbiosis and hyper-permeable GIT increase toxic/endotoxic loading on the liver.
- **5. Liver/GIT**: Suboptimal liver function influences Bile flow. Fat intolerance, decreased absorption of fat soluble nutrients.
- **6. GIT/Immune**: Poor GIT based immunity (SIgA), frequent Abx and caesarean birth suggests longstanding microbiome imbalance. Immune system dys-regulation.
Treatment Aims

- A ‘mapping’ process provides clarity about what needs ‘treatment’ in a case
- Treatment Aims are therefore a prioritised (and numbered) list of what needs ‘treatment, based on your case analysis/schematic work
- Treatment Aims are generally technical, utilise holistic and medical terminology and address/reflect the identified case priorities, causative and sustaining factors
- Treatment Aims in turn inform dietary and lifestyle Health Goals (SMART in nature) which represent how to achieve the identified Treatment Aim/s
Treatment Aim: Examples

- Useful words to consider in the construction of Treatment Aims are:

  - Regulate
  - Downregulate
  - Address
  - Increase
  - Improve
  - Restore
  - Support
  - Establish
  - Continue
  - Inhibit
  - Facilitate
  - Eradicate
  - Modulate
  - Optimise
  - Identify
  - Maximise
  - Manipulate
  - Rebuild
  - Stimulate
  - Strengthen
  - Prepare
Treatment Aim: Examples

- Treatment aims can be constructed as a general concept and then include relevant sub-branches. This is useful when the case is large and there are repetitive themes – e.g.:

  1. Improve digestive health, address microbiome dysbiosis and increase mucosal immunity
     - Increase SlgA
     - Decrease GIT permeability and reduce endotoxic load
     - Improve microbial diversity
     - Regulate GUT based neurotransmitter synthesis

  2. Reduce sympathetic nervous system dominance and regulate cortisol production
     - Support adrenal function/reserve
     - Increase HCl production
     - Regulate peristalsis
     - Regulate BSL
Treatment Aim: Examples

- Or be listed as a whole Aim without sub aims
  - E.g. Regulate digestive health and decrease permeability

- A Treatment Aim can also be a direction of assessment/testing - E.g. Confirm or rule-out potential liver based pathology (order LFT, assess enzymes, FLDP, etc.).
Schematic

Class Activity:

Using the provided template, map out the provided case details and then discuss the following:

- What are the key organ/s and body system interaction/s present in this case?
- Formulate two Treatment Aims which reflect the understanding reached – (refer to the language and TA structure on previous slides)
Clinical Decision Making

Integrated Treatment Approach
When taking a client's case (initial and repeat) it is also essential to:

- Ask about the use of all CAM, over-the-counter (OTC) and prescription medications: including practitioner prescribed and self-medicated
- Consider the age, sex and genetic predispositions of your patient.
- Concurrent disease states may have an effect on absorption, distribution, metabolism and excretion

(Braun & Cohen, 2010, p. 123)
Clinical Decision Making

Must also include consideration of pharmaceutical interventions/impact & potential for interactions:

- Reduced drug effect
- Added drug effect
  - Increased or decreased incidence of adverse effects
- Synergistic effect that is beneficial or negative
- Improvement in overall treatment of disease process
  - Restore tissues that may be adversely affected by drug
  - Reduced side effects associated with drug
  - Arrest nutrient deficiencies associated with drug
  - Reduce drug dose overtime

Drug mechanism of action & system involved (Schematic/concept 1 map 1)

(Braun & Cohen, 2010, p. 94)
Clinical Decision Making

Integrated Treatment Approach
Combined use of drug, herb &/or nutrient safety depends on:

- Predicted potential for an interaction
- Appropriate use of each intervention (dose, indication, route of administration, timing of administration etc.…)

(Braun & Cohen, 2010, p. 104)
Clinical Decision Making

Integrated Treatment Approach
Risk Rating - drugs most likely to have interactions are:
  o Drugs with a narrow therapeutic window
  o Drugs that are anti-coagulants, anti-hypertensives, anti-diabetic, anti-psychotic and anti-epileptic drugs

Patients at most risk of interactions are:
  o Elderly
  o Children
  o Patients with impaired renal or liver function

(Braun & Cohen, 2010, p. 105-106)
Clinical Decision Making

Integrated Treatment Approach

- Refer to drug information centres, peer-reviewed literature, reputable sources for information
- Document relevant information and recommendations on case notes
- Suspend the use of nutrients or herbs 5-7 days before surgery or childbirth, where appropriate

(Braun & Cohen, 2010, p. 123)
Gastro-Intestinal Tract

Please refer to relevant notes from the following subjects to review structure and function of this body system:
- BIOC211, BIOS222
Gastro-Intestinal Tract

GIT: Brief overview of Functions & Features:

- Ingestion
- Secretion (cells within the GIT secrete up to 9L per day of various acids, buffers or enzymes into the GIT)
- Mixing and propulsion - peristalsis
- Digestion: Both mechanical and chemical
- Absorption: of macronutrients and micronutrients.
- Defecation: removal of waste
Gastro-Intestinal Tract

GIT: Brief overview of Functions & Features:
- Major interface between our inner and outer worlds
- Total surface area is 100 times larger than our skin
- The number of living microbes inhabiting the digestive system equals the total number of cells in our body
- The digestive system is richly supplied with nerves
- Digestive health is essential to our wellbeing
- Digestive function deteriorates with age, especially gastric acid and pancreatic output.
- GALT (gut assisted lymphoid tissue/mucosa assisted lymphoid tissue)
- Consider how you will prescribe dependent upon their digestive function – dose, form (powder, liquid, tab, cap)
Gastro-Oesophageal Reflux (GORD)

Refer BIOC211 notes to review pathophysiology & diagnostic signs associated with GORD
Gastroesophageal Reflux Disease (GERD): Pathogenesis & Clinical Findings

Abbreviations:
LES: Lower esophageal sphincter

Primary GERD

- ↑ Intra abdominal pressure (obesity, pregnancy)
  - Hiatal Hernia: Portion of stomach protrudes above diaphragm
    - LES misaligned with level of diaphragmatic contraction
    - LES seal has ↓ structural support

- Foods
  - Coffee, alcohol, chocolate, mint, fatty meals, spicy, citrus

- Drugs
  - Beta-agonists, calcium channel blockers, anticholinergics

  Abnormal LES relaxation

Secondary GERD

- Scleroderma: (autoimmune disorder)
  - Collagen deposition around body, including lower 1/3 of esophagus
  - ↓ Peristalsis, ↓ LES tone

- Large meal, Delayed gastric emptying
  - Gas builds up in stomach, ↑ pressure on the cardia
  - Pressure receptors indirectly stimulate the vagus nerve

  Vagus nerve (CN-X) stimulates LES relaxation

Excessive or Prolonged Transient Lower Esophageal Sphincter Relaxation

HCl in stomach can be pushed up by ↑ intra-abdominal pressure (exercise, obesity, pregnancy) or positional changes (lying down)

Conditions with ↑ acid production (see ‘Peptic Ulcer Disease’)

- Repeated damage to esophagus
- Normal squamous epithelium replaced with columnar cells (metaplasia)
  - Barrett’s Esophagus: precancerous lesion
    - Over time, may progress to adenocarcinoma (1:1000)

- Scarring can form esophageal stricture
  - Stricture can bleed
  - Bright red hematemesis

- ‘Heartburn’ (Epi gastric/retrosternal burning sensation)
  - May persist despite treatment= ‘NERD’ (non-erosive reflux disease)

- Acid Regurgitation (water brash)
  - Aspiration of acid into larynx, lungs
  - Irritation of upper respiratory tract
  - Chronic cough (especially at night), asthma, hoarse voice

Legend: Pathophysiology  Mechanism  Sign/Symptom/Lab Finding  Complications

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Gastro-Oesophageal Reflux

- The primary protective factor from GORD is the LES (Richter J 2007)
  - Oesophageal mucosa is exposed to gastric contents for prolonged periods of time.
  - Symptoms include heartburn, regurgitation (worse for bending, straining or lying down) and ‘water brash’.

(Kumar & Clark, 2005, pp. 275-276)

(Tortora & Grabowski, 2003, p.861)
Signs and symptoms infants -

- Excessive crying is the most prevalent reason for an infant visiting the doctor in the early stages of life affecting approx. 30% babies (Douglas, 2005).
- And approx. 50% of infants in the first years of life present with GORD, most resolving in the first year of life (Mahan & Escott-Stump, 2008, p.656).
- Excessive crying can predispose infant to GORD after approx. 3-4 months of age (Douglas, 2005). Therefore look at feeding practices (overfeeding, possible cows milk allergy, referral to lactation consultant); sensory nourishment (baby wearing); parental responsiveness (reading baby cues); sleep management (co-sleeping, adequate rest) all important considerations.
Gastro-Oesophageal Reflux- Infants Cont.

Excessive crying and GOR in infants.

(Douglas P 2005)

EXCLUDE DISEASE eg urinary tract infection, otitis media, intussusception, incarcerated hernia, torsion of testis, fractured clavicle, neurological impairment, congenital heart disease

If > 3-4 months and persistently vomiting

FEEDING MANAGEMENT
Consider:
• frequent feeds

If breast fed
If bottle fed

PARENTAL RESPONSIVENESS
Consider:
• prompt parental response to cues
• maintaining exchange of sensory data: infant in same room as caregiver

SENSORY NOURISHMENT
Consider:
• sling/backpack
• outdoor walks
• increased social contact
• massage
• comfort eg tight nappy? irritating garment? overdressing?

SLEEP MANAGEMENT
Consider:
• nocturnal co-sleeping
• sensory nourishment instead of daytime sleep struggle

TRIAL PROTON PUMP INHIBITOR in addition to consideration of feeding management, parental responsiveness, sensory nourishment and sleep management

If crying continues

Referral to paediatric gastroenterologist (endoscopy)

If symptoms of cow’s milk allergy

Prompt referral to Lactation Consultant
Trial elimination of dairy products from maternal diet

Referral to paediatrician
(hydrolysed protein or amino acid formula)
? Referral to Lactation Consultant (relactation)
GORD

Differential Diagnosis:-

- Hiatus Hernia
- Helibacter pylori
- Peptic ulcer
- Acute and chronic gastritis
- Oesophageal cancer
- Gallstones
- SIBO
Risk Factors

- Weight (BMI >25) was associated with a 2.5-3 fold increase in symptoms (Corley & Kubo, 2006); erosive esophagitis and oesophageal adenocarcinoma, gastric cardia adenocarcinoma (Nilsson et al. 2003; Hampel et al. 2005); risk of these disorders increased with increasing weight.

- Helibacter pylori. There’s a negative association between h. pylori and GORD as seen in Asian countries which have a lower incidence of GORD and western countries, a higher incidence (Richter, 2007).

- Transient lower oesophageal sphincter (LES) relaxation and spatial separation of the LES diaphragm (hiatus hernia) (Richter, 2007).

- Smoking – reduction of LES pressure (Mahon, Escot-Stump & Raymond, 2012).

- Genetics.

Gastro-Oesophageal Reflux

Treatment Aims will include:

- Manage issues with reduced swallowing capacity:
  - Neurological disorders (e.g. stroke)
  - Decreased saliva production (e.g. elderly, anticholinergics)

- Manage any issues with delayed gastric emptying:
  - Fatty meals
  - Lying down within 45 minutes of eating
  - Diabetes

(Mahan & Escott-Stump, 2008, p. 657)
Gastro-Oesophageal Reflux

Therapeutic Actions

- Minimize agents that increase acid production:
  - Smoking – prolongs acid clearance. Higher rate of GERD in smokers versus nonsmokers.
    (Watanabe et al. 2003)
  - Drugs (e.g. NSAIDs, aspirin)
  - Foods and beverages e.g. coffee, tea, carbonated drinks, chocolate, citrus, spicy foods

- Minimize irritant effects on the oesophagus:
  - Drugs (e.g. tetracycline's, iron salts, aspirin, NSAIDs)
  - Foods (e.g. citrus foods, tomatoes, coffee)
    (Mahan & Escott-Stump, 2008, p. 657)
Gastro-Oesophageal Reflux

Clinical and dietary interventions (Adults) can include:-

- Minimise or manage foods / agents that reduce sphincter tone. These include –
  - Chocolate, fatty meals, carminatives (spearmint, peppermint), smoking, alcohol.
  - Drugs (calcium channel blockers, diazepam, theophylline)
  - Hormones (oestrogen, progesterone)
  - Physiologic factors (e.g. prostaglandin, glucagon, vasoactive intestinal peptide) (Mahan & Escott-Stump, 2008, p. 657)

- Include - antioxidant rich foods, legumes and whole grains, chamomile, cinnamon, ginger. These aid in regulating GIT motility, act as anti-inflammatory and anti-microbial agents and may reduce nausea and excessive salivation. Cabbage and cabbage juice aid the healing of irritated tissue from acid regurgitation (Sarris & Wardle, 2010, pp. 78-79).
Gastro-Oesophageal Reflux

Clinical and dietary interventions (Infants) can include:-

- Consider feeding practices; check sleep quality and quantity; baby cues; mothers diet in b’fed infant (high in dairy or chocolate); feeding position


- Maternal or infant dairy-free diet may be beneficial. Supplement with soy, goat milk alternatives or extensively hydrolysed whey formula

- Carob bean gum (0.33mg/100ml formula) powder has been found to decrease the severity and frequency of GORD. Contains galactomannans which protect from acid (Georgieva et al 2016).

(Sarris & Wardle, 2010, p 78-9)
Key nutrients to consider

- Slippery Elm
- Glutamine
- Betaine hydrochloride
- Aloe Vera (inner leaf juice)
- Vitamin C
- Probiotics (*L. rhamnosus*; *B. infantis*)
- Cabbage juice
## Slippery Elm in Gastro-Oesophageal Reflux

### Mechanism of action
- Contains high amounts of mucilage → traps water to form a gel-like substance.
- Gel-like substance adheres to the mucous membranes and forms a barrier against stomach acid → preventing pain, ulceration and cellular changes (Braun, 2006).

### Evidence of clinical application
- First used by Native Americans as a healing salve for wounds, ulcers and inflammation (UMMC, 2014)
- There have been no scientific studies done on the use of Slippery elm bark and GORD, however there is a significant amount of traditional and empirical evidence supporting its use.

### Dosage
- **Acute:** 1500 - 2000mg per day, for 4 – 8 weeks. An additional 400 – 500mg can be taken if acute pain is being experienced. (UMMC, 2014)
- **Chronic/Maintenance:** 800 – 1500mg per day.

### Other considerations
- Contraindicated in pregnancy.
- Can be made as a tea or taken as a powder. Also prescribed in capsules.
- There is a theoretical concern that because it lines the digestive tract, it might affect the absorption of drugs. Separate the dosage by 2 hours. (Braun, 2006)
## Glutamine in Gastro-Oesophageal Reflux

| Mechanism of action | • A key nutrient for rapidly proliferating cells  
|                     | • Reduces oxidative damage  
|                     | • The preferred respiratory fuel for enterocytes – vital in maintaining a healthy intestinal lining  
|                     | • Maintain secretory IgA production → Antimicrobial (especially if GORD is being driven by *H. pylori* overgrowth) (Braun & Cohen, 2010) |
| Evidence of clinical application | •Although commonly prescribed for GORD, there are no large scale or specific studies on glutamine supplementation and GORD. Much of the clinical application has come from extrapolating data and actions identified in studies done on other gastrointestinal conditions.  
|                     | •Despite this, glutamine has been shown to be effective in a clinical setting and based on the mechanism of action, is often prescribed by naturopaths and holistic nutritionists. |
| Dosage | Best taken as a powder formulation. 4 – 6 grams per day, in divided doses. (i.e. 2 – 3g, twice a day) (Braun & Cohen, 2010) |
| Other considerations | None |
Betaine Hydrochloride in Gastro-Oesophageal Reflux

| Mechanism of action | “Betaine hydrochloride supports the pH decline in the stomach thereby, potentially improving nutrient digestibility” Betaine has shown an anti-apoptotic effects and promotes cell proliferation.  
• Betaine accumulates in cell organelles and replaces inorganic ions, allowing protective enzymes and the cell membranes to remain viable.  
• The chloride component is directly used to increase stomach acid production. (Eklund et al. 2005) |
| Evidence of clinical application | “If low stomach acid is the cause of reflux symptoms, betaine hydrochloride may be useful.” (Hechtman, 2012)  
• There are no studies on betaine hydrochloride supplementation and the effects on Gastro-oesophageal reflux. Clinical application has developed from assessing the underlying drives of GORD and the biochemical pathways of betaine hydrochloride. |
| Dosage | There is no typical dosage for betaine hydrochloride. Practitioners should follow manufacturers guidelines. |
| Other considerations | “Betaine hydrochloride should only be taken with protein containing meals and the dose should be built up slowly”. (Hechtman, 2012)  
• Patients may feel a warming sensation in their abdomen. This should be relieved by food. |
# Aloe Vera in Gastro-Oesophageal Reflux

## Mechanism of action

- Contains a glycoprotein fraction that increases the expression of receptors and cell proliferation to enhance wound healing (in vivo).
- “Aloe contains 2 dihydroisocoumarins that demonstrate free radical scavenging properties. This is also supported through the inclusion of flavonoids and polysaccharides which supports healing and antioxidant activity” (Braun & Cohen, 2010)

## Evidence of clinical application

- “The gel reduces oxidation of arachidonic acid and as such, prostaglandin formation → anti-inflammatory.”
- “A study in rats found that aloe gel reduced vascularity and swelling by 50% in inflamed synovial pouches”. (Braun & Cohen, 2010)

## Dosage

- Best administered as a liquid gel.
- Start with 25 – 50mls, twice per day to test for tolerance. Can go up to 100mls, twice per day in more severe cases (Langmead et al. 2004)

## Other considerations

- Generally well tolerated, although some preparations contain the latex, which has a laxative effect.
## Vitamin C in Gastro-Oesophageal Reflux

### Mechanism of action
- Vitamin C is an electron donor, accounting for most of its biological functions → anti-oxidant.
- Adequate vitamin C status prevents degranulation of tissue and allows for adequate wound healing.
- Assists with the proper formation of collagen, elastin and fibronectin, thereby helping to improve sphincter tone. (Braun & Cohen, 2010)

### Evidence of clinical application
- There is evidence to suggest that a vitamin C deficiency exists in gastric diseases. Further to this, the overgrowth of *H. pylori* has been shown to contribute to creating this deficiency (Aditi & Graham, 2012).
- Very limited studies of vitamin C administration for GORD are available.

### Dosage
- 500mg twice per day (Hechtman, 2012)

### Other considerations
"The degree of the absorption depends on the dose ingested and decreases as the dose increases. At a low concentration most vitamin C is absorbed by the sodium vitamin C co-transporter 1 in the small intestine and reabsorbed in the renal tubule. At high concentrations, these transporters become saturated, limiting the amount that can be absorbed." (Braun & Cohen, 2010)
## Other nutrient considerations

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magnesium</td>
<td>400-800mg</td>
<td>Sphincter tone</td>
</tr>
<tr>
<td>Digestive enzymes</td>
<td>Enzymes with food (dose varies on product)</td>
<td>Improves food breakdown, increases nutrient absorption</td>
</tr>
<tr>
<td>Selenium</td>
<td>25-250mcg</td>
<td>Deficiency has been linked to the progression of Barrett’s oesophagus</td>
</tr>
<tr>
<td>Zinc</td>
<td>10-100mg/day</td>
<td>Deficiency is linked to Barrett’s oesophagus and the risk of oesophageal adenocarcinoma. Increase would healing</td>
</tr>
<tr>
<td>Omega 3 Fish Oil</td>
<td>1-6gm /day</td>
<td>Anti-inflammatory</td>
</tr>
</tbody>
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(Mahan & Escott-Stump, 2008, p. 658; Sarris & Wardle, 2010, p. 77)
## Gastro-Oesophageal Reflux

<table>
<thead>
<tr>
<th>Drug</th>
<th>Drug Action</th>
<th>Side Effects</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>OTC Antacids</td>
<td>Neutralizes acid by increasing bicarbonate and mucous. Inactivates pepsin. Binds bile salts</td>
<td>Diarrhoea (Mg), constipation (Al), hypophosphataemia (with low phosphate intake), hypercalcaemia</td>
<td><strong>Vitamin C</strong>: increases aluminium absorption. Separate doses by 2 hrs.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Folate &amp; Iron</strong>: reduces absorption of both. Separate doses by 2 hours.</td>
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(Braun & Cohen, 2010, p1096; Bryant & Knights, 2011, p. 538)
# Gastro-Oesophageal Reflux

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<tr>
<td>‘Raft’ Antacids:</td>
<td>Mixes with stomach acid forms a slimy jelly ‘raft’ reducing acid, pepsin &amp;</td>
<td>Low incidence of side effects (less than 5%).</td>
<td>Vitamin E &amp; Calcium : reduces absorption of both</td>
</tr>
<tr>
<td>Alginic Acid</td>
<td>bile acids on mucosa</td>
<td>Constipation</td>
<td></td>
</tr>
<tr>
<td>Raft’ Antacids:</td>
<td>Increases bicarbonate and mucus production, localized PG release = repair</td>
<td>Sucralfate : nausea, headache, rash, dizziness &amp;</td>
<td>Vitamin E &amp; Calcium : reduces absorption of both</td>
</tr>
<tr>
<td>Sucralfate</td>
<td>of gastric mucosa</td>
<td>indigestion.</td>
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(Braun & Cohen, 2010, p1096; Bryant & Knights, 2011, p. 538)
# Gastro-Oesophageal Reflux

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<tr>
<td><strong>H2-Antagonist</strong></td>
<td>Blocks H2 receptors on parietal cells. Reduce amount, and dissolution of gastric acid raises pH and reduces the activity of pepsin</td>
<td>Diarrhoea, nausea, constipation, headache, dizziness, skin rash</td>
<td><strong>Folate, B12 &amp; Iron</strong> : reduces absorption of both. Separate doses by 2 hrs.</td>
</tr>
<tr>
<td><strong>Proton Pump Inhibitors (PPI’s)</strong></td>
<td>Reduces gastric output via non-competitive &amp; irreversible bonds with H+/K+ ATPase on parietal cells. Action lasts for days after stopping the drug.</td>
<td>Diarrhoea, nausea, abdominal pain, headache. Sustained reduction of HCl causes increase in gastrin.</td>
<td><strong>Folate B12 &amp; Iron</strong> : reduces absorption of both. Separate doses by 2 hrs.</td>
</tr>
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(Braun & Cohn, 2010, p. 1097; Bryant & Knights, 2011, pp. 541-543)
## Gastro-Oesophageal Reflux

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<tr>
<td><strong>Dopamine 2 Antagonists:</strong></td>
<td>Inhibition effect on dopamine = reduced gut motility. Increases gastric emptying. Increases lower oesophageal sphincter tone</td>
<td><strong>Metaclopramide:</strong> drowsiness, fatigue, nervousness, anxiety, diarrhoea, insomnia.</td>
<td>None found</td>
</tr>
<tr>
<td>Metaclopramide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Domperidone</td>
<td>As above</td>
<td><strong>Domperidone:</strong> less side effects (minimal BBB crossover)</td>
<td>None found</td>
</tr>
</tbody>
</table>

(Bryant & Knights, 2011, p. 541)
Pharmacology Considerations
Phases Affecting Drug Activity

Administration of Drug

Available portion of the drug is absorbed

PHARMACEUTICAL PHASE
Drug is dissolved

PHARMACOKINETIC PHASE
Drug moves into the blood stream

Drug is available for distribution

Drug distributed to organs & tissues

Drug metabolized & / or excreted

PHARMACODYNAMIC PHASE
Drug-molecular target interaction

Drug Effect

(Bryant, Knight & Salerno, 2007, p 112)

Refer to Pharmacology handout
TUTORIAL  _  Watch the following video TED talk.

How our microbes make us who we are

- TED Talk (17.24 minutes) *How our microbes make us who we are*
- [http://www.ted.com/talks/rob_knight_how_our_microbes_make_us_who_we_are](http://www.ted.com/talks/rob_knight_how_our_microbes_make_us_who_we_are)
References


References


References


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