Anorexia Nervosa

History of Presenting Illness

ASK: do you think you are thin?
Anorexics will amaze you with the poverty of their insight into their own condition.
ASK THE FAMILY: how are the other kids?
Often there are several eating disorders in the same family- perhaps stemming from the same risk factor

(diagnostic criteria from DSM IV)

- History of **weight loss** (or in children, lack of weight gain)
- Weight loss is **Self-induced through <u>avoidance</u>**
- Intrusive **dread of fatness**
- **Amenorrhea** (or in men, loss of sexual interest)
- Excessive **exercise**
- Use of **appetite suppressants**
- **History of eating disorders** in family
- ***BUT NOT BINGE/PURGE:***
  - NO RECURRENT EPISODES OF OVEREATING
  - NO "CRAVING" i.e. no compulsion to eat and then follow it with compensatory behaviour eg. vomiting

Differential Diagnoses (DDx)

- **Eating disorder (!)**
- **Stress-related autophagy**
- **Drugs**
- **Cancer**
- **Pregnancy**
- **Intestinal parasite**
- **Psychosocial ramifications of puberty**
- **Malabsorption disease (eg, coeliac)**
- **Hyperthyroidism**
- **Depression**
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Findings on History

OBESITY/THINNESS most strongly correlated with MOTHERS WEIGHT

- No necessary previous illness, but may have previous GIT disorder
- **History of eating disorder in family**
- Gradual decline of school/work performance, missing days etc.

Findings on Examination (Ex)

Look for signs of
- **ANAEMIA**
- **DEHYDRATION**
- **MALNUTRITION**
- **KETOACIDOSIS**

- Pale, thin, gaunt, sunken face/eyes (BMI below 17.5)
- Sullen/depressed
- Dark circles under eyes (~dehydration, hypovolumia)
- **Chapped lips**
- **Flaking skin**
- **Brittle hair**

SIGNS OF MALNUTRITION: protein loss…
…but: if there is a protein-loss enteropathy or some other GOOD reason for being emaciated, these signs will also be present.

- Halitosis (due to ketone bodies in blood stream)

Tests and Investigations

Blood Count: looking for metabolic abnormalities consistent with malnutrition
- Low haemoglobin (N = 1.15-1.6 g/L) due to iron deficiency
- Low WBC (N = 4 to $11x10^3$ per mm^3) due to malnutrition
- Low plasma glucose (N= 4 to 10 mmol/L; below 2.8 = coma) (or 7 - 11 mg/L)

Postural Hypotension: marked difference between standing and sitting/lying blood pressure; normal difference = 12

Urinalysis to eliminate pregnancy: **Expected <u>Negative</u>**

Stool Sample to eliminate intestinal infection/infestation **Expected <u>Negative</u>**

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Management

According to the 2004 review of the 1990 Mental health Act, anorexia does not fall into the NSW Mental Health Act definition of a mental illness unless the patient suffers a severe disturbance of mood with

By GP:

- referral to psychiatrist (specialist in eating disorders)
- does the pt require resuscitation, rehydration, nutrient replacement therapy?

By Specialist: DEFINITIVE TREATMENT:

- **Nutritional Rehabilitation:**
  - **Dietician** will work with pt. to devise a feeding regime to gain minimum healthy weight
    - **1st** take **detailed nutritional history** and ask about weight-loss behaviours
    - **INFORM** about dangers of over/under eating, excess exercise, starvation metabolism
    - **Then** when target weight is reached, a maintenance diet is prescribed

HOSPITALISATION may be needed if pt. is emaciated, or there is low compliance, or a family crisis supervenes.

- **Psychotherapy:** somatic focus must be combined with cognitive behavioural therapy and supportive psychotherapy. Aim is to:
  - understand the personal significance of weight loss;
  - help deal with weight gain;
  - to have her accept and become attuned to her body;
  - to improve her self esteem;
  - to assist her to reintegrate home, school and peer group.

Treatment must continue for a long period of time even after weight and eating patterns have normalised. Compulsory treatment may be necessary

Epidemiology

Mainly **Women (10 : 1) – TYPE A PERSONALITY is a risk factor**
Prevalent in cultures where food is plentiful
(worldwide prevalence = 0.5%; in America 2.3% in females)
Mortality ~ 10% chance every 10 years
OCDs in >20% of sufferers
Anxiety disorders in 65%
Depression in 68%

Prognosis`

The relapse rate is high (50% in the first year and 90% overall),
the death rate is 1% per year with 20% dead by 20 years,
the illness lasts around 5 years on average

Biochemistry of weight loss

energy intake of the body is balanced by its **energy output** ("energy balance equation"):
thus, increasing output or decreasing input will unbalance the equation and force autophagy
(where the body uses stores of energy to satisfy its basic metabolic needs)

Energy intake = food intake in kilojoules or calories

Energy output =

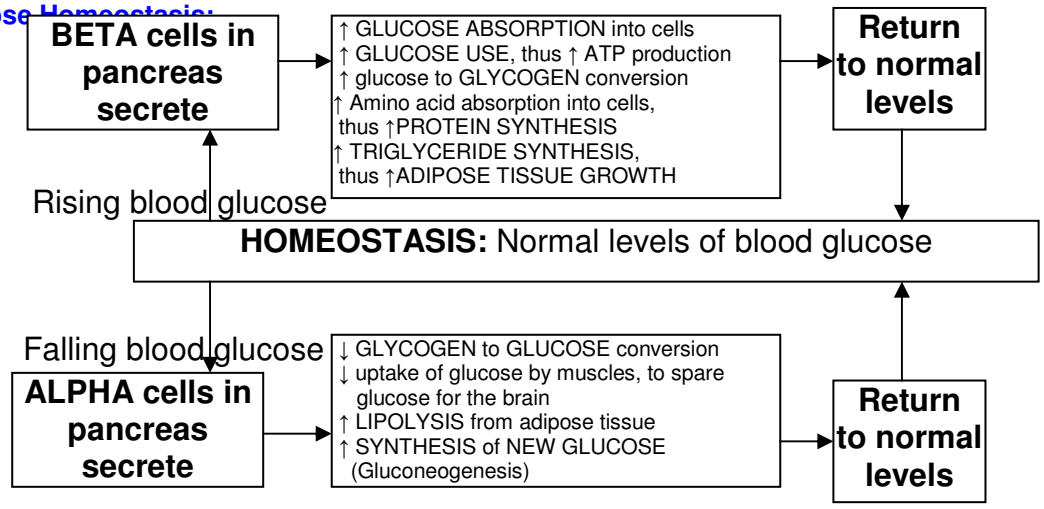
- resting metabolic rate (RMR),
- energy cost of arousal,
- the energy cost of work and activity,
- thermogenesis (heat production)
  - shivering,
  - non-shivering
  - diet-induced thermogenesis. On eating, there is a specific stimulation of the sympathetic nervous system which leads to thermogenesis.
    carbohydrate and protein eaten in excess may also stimulate thermogenesis.
    Fat does not elicit thermogenesis.

Biochemistry of starvation:

1st order of business: BRAIN NEEDS GLUCOSE; primary source is glycogen in the liver
OTHER ORGANS THAT CANT DO WITHOUT GLUCOSE: Testes, Kidney Medulla, Erythrocytes
Blood glucose falls by 2/3rds = COMA eg. in diabetes (all glucose gets bound in cells)

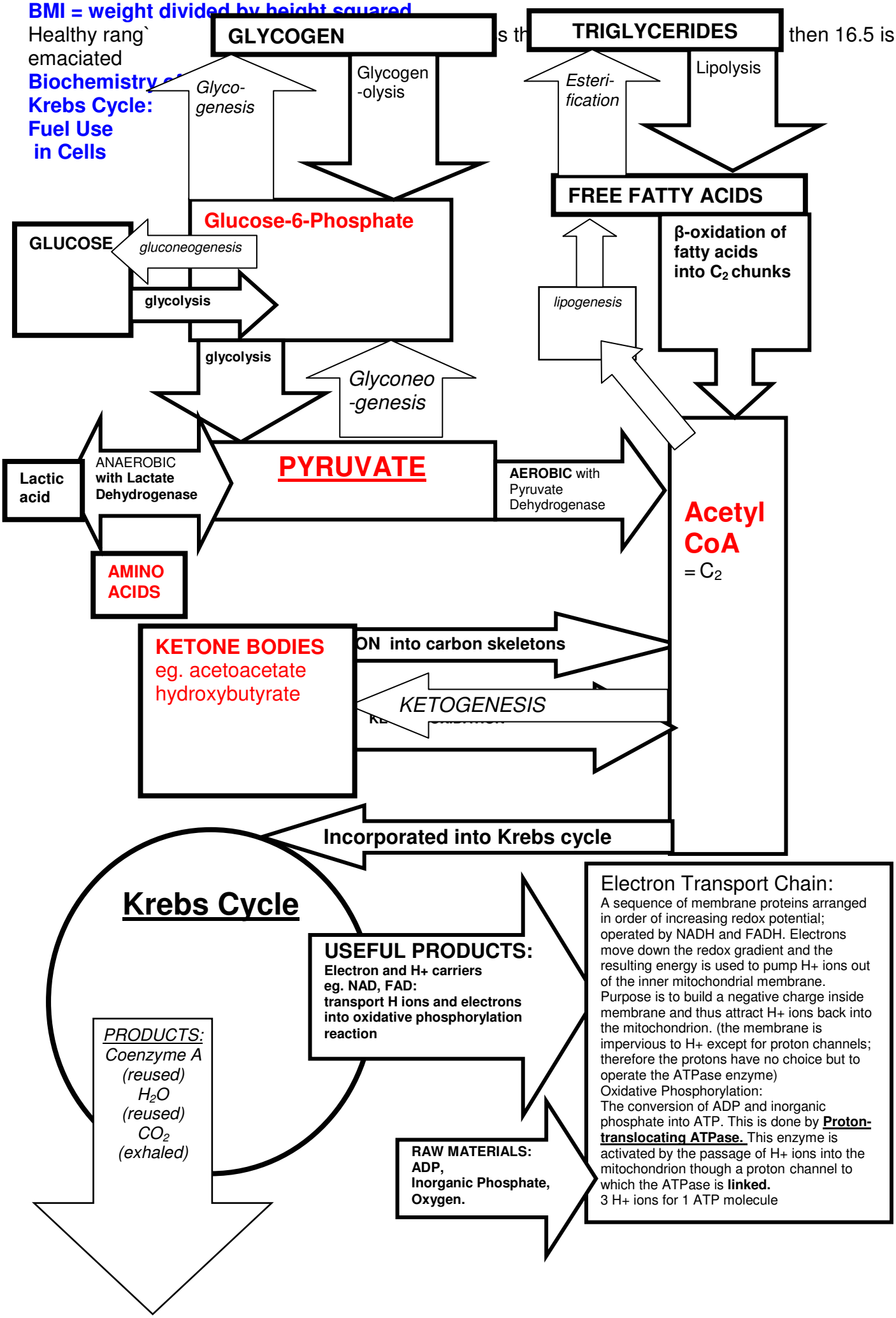
- STEP 1: **GLYCOLYSIS: GLYCOGEN** is catabolised to release a small amount of **glucose** for the brain
  LASTS 1 DAY-
  GLUCONEOGENESIS occurs: production of glucose out of raw materials eg glycerol
- STEP 2: **LIPOLYSIS** occurs: free fatty acids released into bloodstream,
  - to be used in β-oxidation: turn into **AcetylCoA molecules,** then get used in Krebs Cycle
  - KETONE BODIES are produced from AcetylCoA, which the brain can use instead of glucose
  - **FAT LASTS 2-3 MONTHS**: longer in fat people
- STEP 3: **LAST RESORT:**
  PROTEOLYSIS In MUSCLES occurs to release amino acids for the Kreb Cycle
  (get deaminated and turned into carbon chain skeletons, then slotted in wherever they fit along the cycle; ammonia is released as result) IF BRAIN IS STARVED permanent loss of frontal lobe matter occurs (!!)

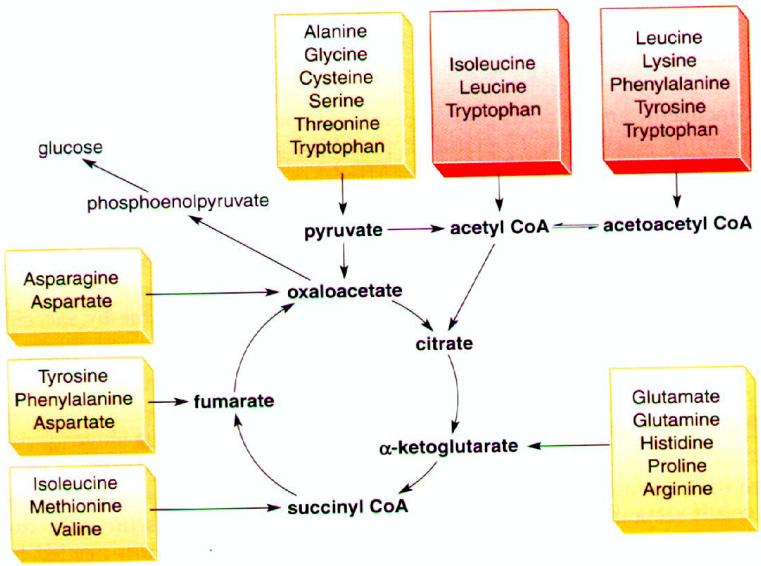
Glucose Homeostasis:



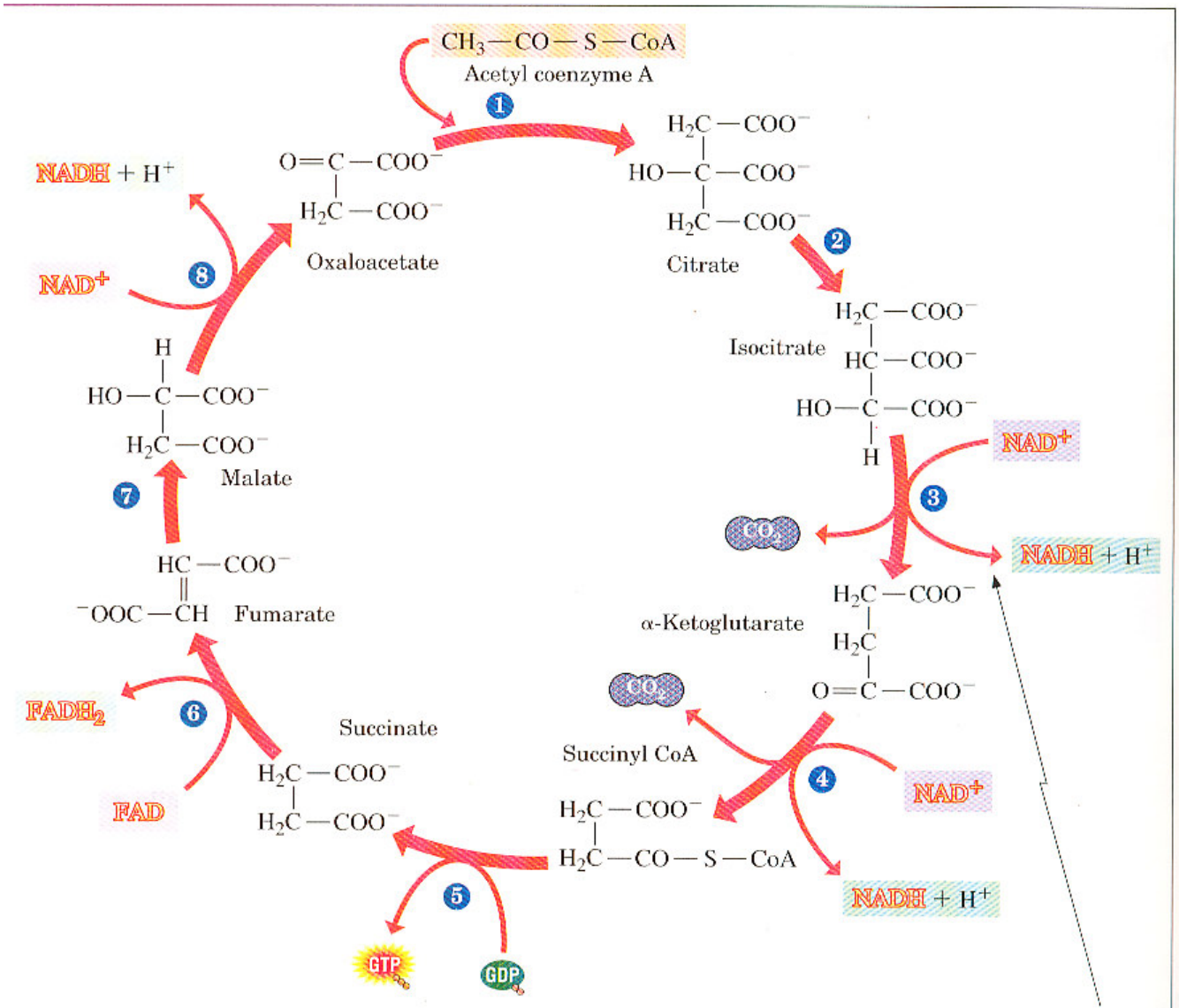
GLUCAGON converts ATP into Cyclic AMP; INSULIN re-converts it into AMP (deactivating it)
Cyclic AMP activates the protein kinases which activate glycogenolysis and deactivate glycogen synthesis

BMI = weight divided by height squared
Healthy rang` s th then 16.5 is
emaciated
Biochemistry
Krebs Cycle: Fuel Use in Cells





For the Biochemistry Psycho:
MITOCHONDRIAL METABOLISM



The curved arrows are a shorthand way of showing the reactants and products. For example, in step 3 the NAD^+ reacts with isocitrate to produce α-ketoglutarate, CO_2, NADH, and H^+. The last two then leave the site of the reaction.

FUNCTIONAL GIT ANATOMY
PBL 1

Secretion, gland: | **Location, action:** | **Absorption:** | **Relevant anatomy:**

Saliva (**Amylases**)
-From 3 pairs of salivary glands

Lingual **Lipase**
-from surface of tongue

MOUTH
-Mastication by teeth (food becomes a moistened compact *bolus*)
-Lubrication by saliva
-Carbohydrates broken down by **amylases**
-Triglycerides broken down by lingual **lipase**

Trace quantities of simple lipids + carbohydrates through the capillaries in the tongue and soft palate

OROPHARYNX
-Muscular swallowing action

LARYNGOPHARYNX
-Muscular swallowing action

OESOPHAGUS
-Muscular swallowing action

HCl
Intrinsic factor
-From parietal cells of gastric gland in the proximal stomach (the fundus)

Pepsinogen
-From "chief cells" in the base of gastric glands in the middle stomach (the "body")

Mucous to coat the walls of the stomach and protect them from acid/enzyme damage
-from Goblet cells in the pylorus (distal stomach)

STOMACH
-Bulk storage of swallowed *bolus*
-Mechanical muscular churning of the *bolus* (peristalsis)
-HCl (pH 1.5-2.0) denaturates proteins, deactivates foreign enzymes, breaks down plant cell walls and animal connective tissue, activates **pepsin** from **pepsinogen**
-Pepsin breaks down proteins by attacking peptide bonds
-Intrinsic Factor facilitates absorption of vitamin B12 in the intestine
-Overall result is acidic viscous soup-like *chyme*

Nothing is specifically absorbed except some drugs (eg aspirin) and ethyl alcohol; this is due to the thick **mucous** coating of the stomach walls

Intestinal Juice to coat the walls of the small intestine and reduce the acidity of the chyme
-from submucosal glands all along the small intestine

Pancreatic alpha-amylase
Pancreatic lipase
Proteases
Peptidases
Nucleases
-from the pancreas

Bile
-from the liver; stored in gall bladder

DUODENUM
(About 25 cm of small intestine)
-Mixing of *chyme,* **intestinal juice** and digestive secretions of pancreas and liver
-Intestinal Juice coats the walls of the small intestine and reduce the acidity of the chyme
-Pancreatic alpha-amylase breaks down starches
-Proteases break down large protein complexes
-Peptidases break down proteins into amino acids
-**Nucleases** break down nucleic acids
-Bile emulsifies the lipids in the *chyme*
-Pancreatic lipase breaks down complex lipids into fatty acids

JEJUNUM
(About 250 cm of small intestine)
Absorption of nutrients

ILEUM
(About 350 cm of small intestine)
some absorption of nutrients

Absorption occurs mainly in the JEJUNUM:
-Peptides
-Amino acids
-Fructose
-Glucose
-Lipids
-Water minerals
-Vitamins

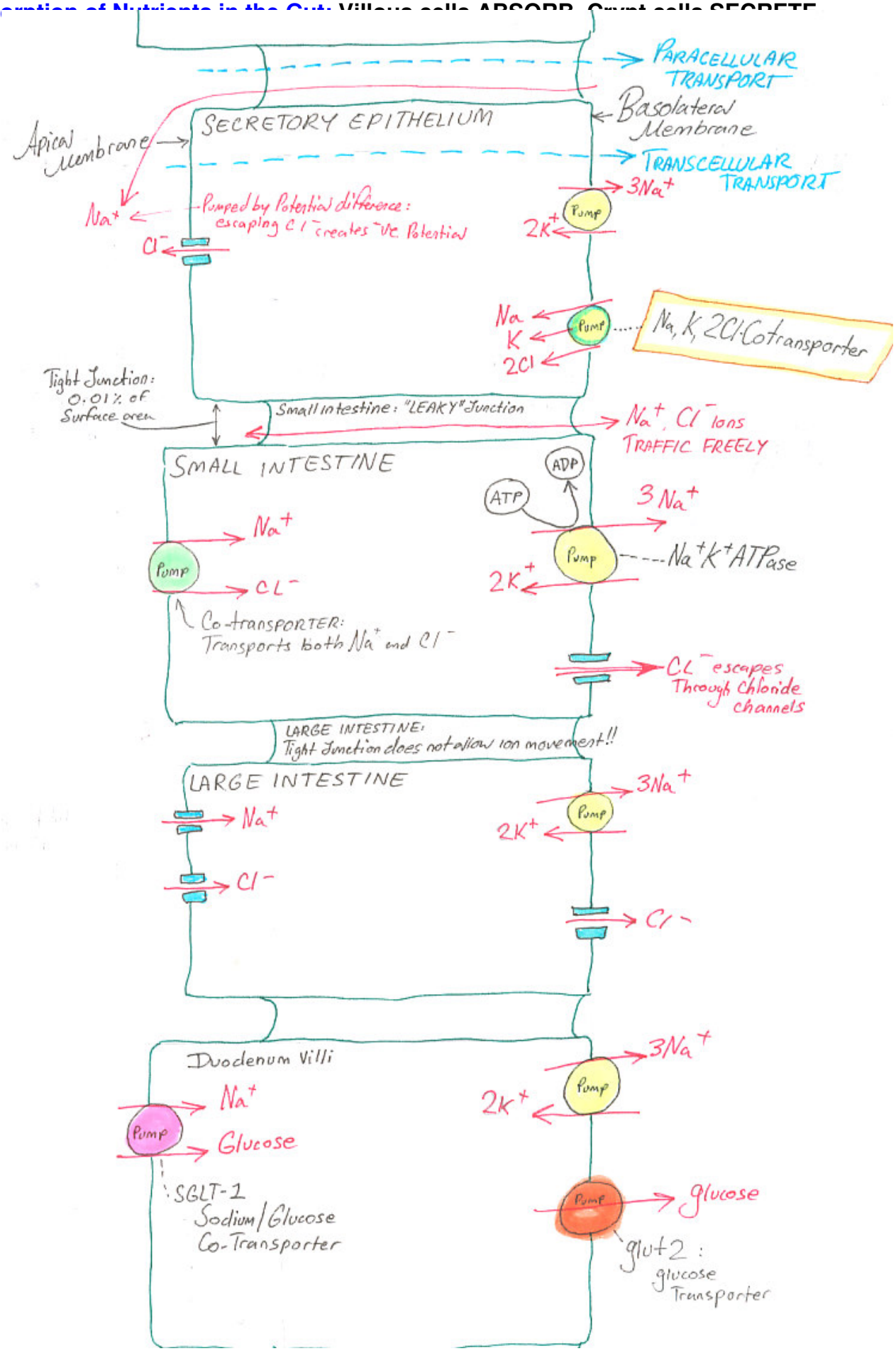
PROXIMAL COLON
(About 75 cm of total colon, comprising the ascending colon and transverse colon)
-Colonic bacteria generate **Vitamin K, Vitamin B5** and **Biotin**
-10% of all GIT absorption occurs in the proximal

DISTAL COLON
(About 75 cm of total colon, comprising the descending colon and sigmoid colon)
-Storage of wastes and reabsorption of water

RECTUM
-Peristaltic expulsion of wastes

The Proximal colon absorbs:
-Water
-Vitamin K
-Biotin
-Vitamin B5
-Some bile salts
-Urobilinogen (product of bacterial metabolism of bilirubin from bile)
-Toxins (ammonium ions, indole, scatole, and hydrogen sulfide

Absorption of Nutrients in the Gut: Villous cells ABSORB, Crypt cells SECRETE



Absorption of:

- **Water:**
  - **driven by solute;** lipid bi-layer readily admits water (20% of total)
  - **Most water (80%) gets transported by transport proteins AQUAPORINS (passively)**
- **Gases:**
  - Completely passive (by diffusion)

Protein transport is both SATURABLE and INHIBITABLE:
SATURABLE transport: eg. glucose: when there is an end-point for absorption, and then no more**.**
INHIBITABLE transport can be interrupted by specific blockers
Protein transport usually requires sodium to pump

Behavioural science:

Taking a meaningful nutritional history:
RECORD: time consuming but accurate log of all consumed foods/drinks; depends on compliance.
Most useful if run over longer periods
24 hr RECALL: quick, provides a snapshot of intake- how good is the patients memory?
Diet History: for long-term accustomed food intake, eg. *on average, what do you eat in an average day?*
- may be useless if the pt has poor memory or the diet is highly variable
Food Frequency Questionnaire- accurate but depends on pt motivation, patience, memory and intelligence.
WHICH METHOD TO CHOSE? Depends:

- **want accurate measurements or descriptive assessment?**
- **Short or long-term?**
- **Can the pt be relied on to provide an accurate assessment?**

Genetics

Obesity and thinness are most closely related to the normal weight of the biological mother

Pharmacology

most commonly non-specific **antidepressants,** either for depressive illness or for obsessive compulsive symptoms which may impede recovery
ALSO perhaps a Sustagen ™ type protein+carbohydrate re-feeding schemata