put together by Alex Yartsev: Sorry if i used your images or data and forgot to reference you. Tell me who you are.
aleksei.igorevich@gmail.com

Failure To Thrive

Problem Summary: infant not feeding well; failing to thrive.
Cries during day and wakes at night → **FAILURE TO THRIVE**

DEFN OF FAILURE TO THRIVE

~~ SYMPTOM, NOT A DIAGNOSIS ~~

CURRENT GROWTH
→ weight < 3° percentile (NCHS growth chart)
→ weight for height <5° percentile
→ weight ≤ 20% ideal weight for height

GROWTH OVER TIME
→ slowing of weight gain
<20g/day 0-3m
<15g/day 3-6m
→ fall-off from prev estab growth curve
downward corssing ≥ 2 percentiles
→ documented weight loss

RELSHIP OF GROWTH PARAMETERS TO EACH OTHER

↓ WEIGHT	↓ WT & HT	↓ WT, HT, head
↓ intake ↑ loss hypermetabolic	struct dystrophy endocrine constit shortness	In utero insult Genetic abN

HISTORY *What is the cause??*

CONTEXT

→onset +/- Rx ***
→overall health

MEDICAL
→pregancy
→birth (preterm, growth)
→perinatal
→current illness/meds/Sx
→systems:
-vomiting / appetite
-stool patts
-distress/tiring c feeds

PSYCHOSOCIAL

→ 1° environment
→ support systems
→ finances
→ domestic sitn
→ bonding
→ child's routine

NUTRITIONAL

→intake
(schedule, amt, preparation)
breast-fed:
- milk control
- maternal diet, rest, meds
formula-fed:
- type
mixed:
- 3day diet Hx
→infant hunger/satiety cues
→meal Δe, feeder, position
→mech: N suck & swallow?
→feeding behaviour
→favourite / hated foods??
→weaning Hx
→family knowledge/ practices/beliefs

DEVELOPMENT

→ milestones
→ behaviour

INVESTIGATIONS

DETERMINED BY FINDINGS
→ **FBC** *anaemia, infxn,*
→ **serum electrolytes & creatinine** *kidney*
→ **LFT's**
→ **urinalysis (± culture)** *kidney damage, UTI, starving*

CATHETER FOR SAMPLE
→ **UEC** *kidney fn, solute load, dehydn*
→ **stool analysis** *fat, blood, WBCs*
→ **hormone assays**
→ **bone age**

PHYS EXAM

(1) **ORGANIC DISEASE or Δ** (2) **SEVERITY OF MALNUTRITION** (3) **ABUSE**
-COMPLETE PHYSICAL
-ABDO: liver, spleen, distension, pain, guarding →*peritonitis, IBD, GITI*
-CARDIO: murmurs →*congenital defect, cyanotic heart disease, failure*
-RESP: →*infxn, asthma, bronchiolitis*
-DEVELOPMENT:

OBSERVE FEEDING
→ feeding environment (home)
→ food: type & amt
→ feeding: pace & duration
→ parent-child interaction

↓ INTAKE

↓ appetite
-chronic disease
-psychosocial
ingestion probs
-neuro disorder
-craniofacial dfx
-dyspnoea
-muscle weakness
food unavailable
-poor feeding technique
-↓ volume
-inapprop food for age
-abuse

↓ ABSORPTION

Sml intestine
-Coeliac Δ
-food intolerance
-inflamm bowel Δ
-enzyme defics
Colon
-Hirschsprung Δ
Pancreas
-CF
Liver
-chronic cholestatic Δ
Drugs
Infections

EXCESS LOSS

Vomiting
-CNS path (↑ICP)
-GI obstruction
-metabolic probs
-gastroesoph. reflux Δ
-drugs/toxins
Diarrhoea
-inflamm bowel Δ
-infxn
-immunodeficiency
-CoeliacΔ / CF (fatty)
Renal loss
-diabetes
-tubular acidosis

ABNORMAL USE

Endocrine probs
-diabetes
-hypopituitarism
inborn errors of metab
-mitochondrial Δs
Chromosomal Δ
Prenatal insult
-foetal alcohol Δ

↑ NEED

Congenital heart Δ
Chronic respiratory Δ
Neoplasm
Chronic/recurrent infxn
Chronic anaemia
Hyperthyroidism

FAMILY
-poverty
-lack of support
-education / experience
-priorities
-social isolation

SOCIETAL
-poverty / chomage
-poor health services
-discrimination

MATERNAL
↓ bonding because
→irritability
→poor

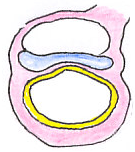
MANAGEMENT

GOAL	**OPTIONS**	***comment***
↑ WEIGHT & GROWTH -weight gain with ↑ intake confirms diagnosis, keep monitoring	(1) ↑ caloric intake (approp diet for catchup) (2) supplementation: ↑fats, *polyjoule* etc. (3) hospitalization - high risk abuse / severe neglect - medically unstable (severe malnutrition) - failed trialled outpatient mgt	Cost
TREAT UNDERLYING DISEASE		
PARENTAL SUPPORT	(1) Antenatal (preventive) (2) Education programs (+/-residential) → Tresilian / Karitane (3) Early childhood support services (4) Social network (mothers' groups) (5) Encourage paternal/family involvement	
EVALUATE MATERNAL DEPRESSION	(1) Edinburgh Post-natal depression scale (2) Anti-depressant meds / Counselling	
MONITOR GROWTH	Weekly visits: GP & Community Nurse Multidisciplinary team - 1° caregiver - nutritionist - social worker - child behaviour specialist - community social services Monitor ALL CHILDREN	
PROTECT CHILD	DOCS	
REFER TO PAEDIATRICIAN		

DEVELOPMENT OF THE GASTROINTESTINAL TRACT

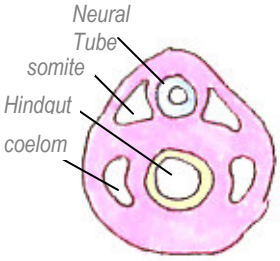
The GIT develops as an outpouching of the yolk sack.

WEEK 3:
the yolk sack is fully formed

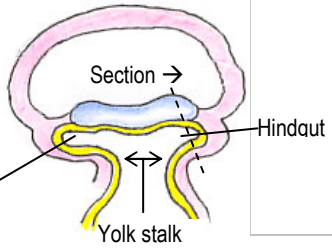


A SECTION THOUGH THE HINDGUT. Halfway through week3

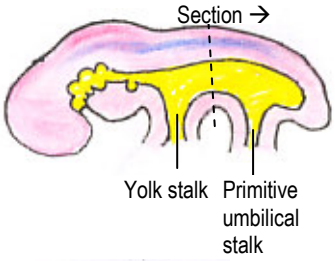
The gut here separates into 3 sections, the Hindgut, Foregut, and Midgut (which is presently occupied by the yolk stalk). These are very important; the arterial supply of the gut is faithful to these original divisons. Eg. the sup mesenteric artery supplies the structures which grew forth from the midgut.



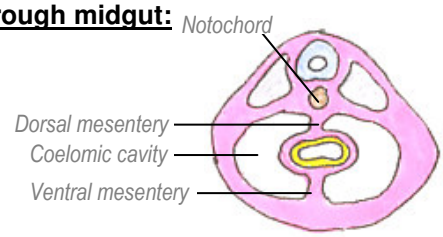
WEEK 3.5:
The yolk sack protrudes through into the embryo and forms two pockets which are the very first bits of the **foregut and hindgut.**
The Midgut is still part of the yolk stalk



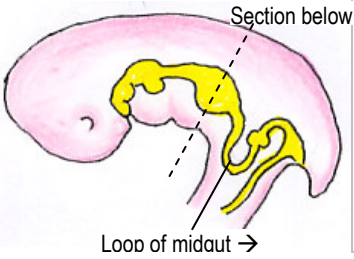
WEEK 4:
The embryo is now roughly a tube; the ventral cavity (coelom) contains the now-complete fore, mid and hind-guts. The yolk sack grows smaller.



Section through midgut:

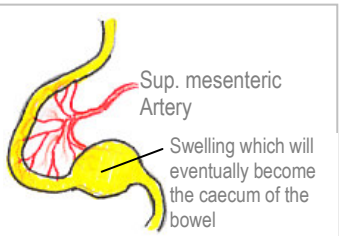


WEEK 5
A loop of the midgut begins to protrude through into the umbilical stalk, while the rest distends and prepares to separate into the stomach, spleen, pancreas and liver . The yolk sack has become one with the midgut.

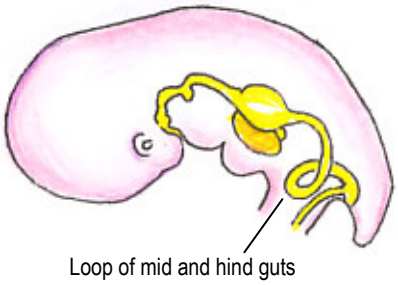


The Loop of Midgut

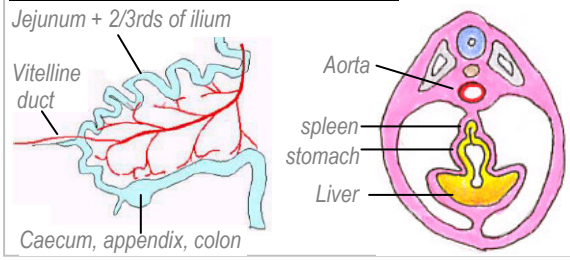
Anticlockwise rotation of this loop around the axis of the Superior Mesenteric Artery will position the ascending, transverse and descending colon in its normal adult position.



WEEK 6
The midgut loop elongates rapidly; its superior limb becomes the jejunum, and the inferior limb becomes the colon and appendix. The liver grows from the ventral mesentry; the spleen and pancreas grow from the dorsal mesentry

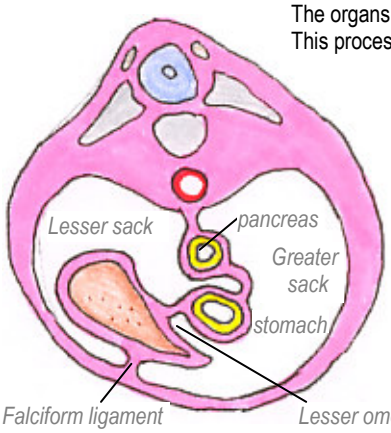


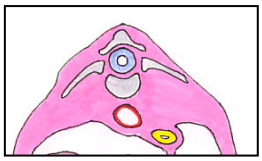
Section through midgut wk.5-6



Later in WEEK 6: section through midgut

The organs rotate 90 degrees to the right. This process begins in week 5

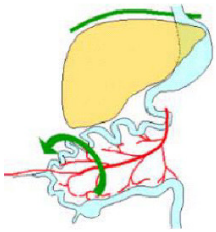




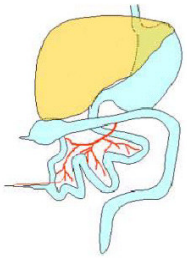
ABOVE: The pancreas fuses with the posterior abdominal wall

Rotation of the midgut loop

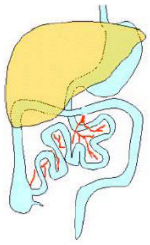
Thus the colon assumes its ascending-transverse-descending position



Caecum situated caudally 6 weeks



Caecum below right lobe of liver 10 weeks



Caecum in right iliac fossa 11 weeks

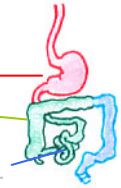
!! the ANUS and the MOUTH arise from different structures, are innervated separately from the gut, and receive a different blood supply.

RELATIONSHIP OF ADULT TO EMBRYO:

Foregut: *supplied by coeliac trunk*
- Oesophagus, stomach, proximal half of duodenum

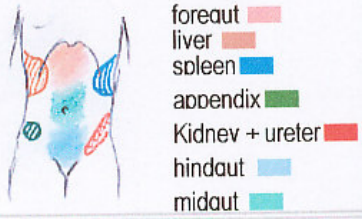
Midgut: *supplied by superior mesenteric artery*
- Distal duodenum, jejunum, ilium, caecum + appendix, ascending colon, 2/3rds of transverse colon until the splenic flexure

Hindgut: *supplied by inferior mesenteric artery*
- Left colic flexure, descending + sigmoid colon, rectum and upper anal canal.



Distribution of pain referral:

Refers via sympathetic afferents



POVERTY AND CHILD HEALTH

DEFINITIONS:

ABSOLUTE poverty

- Income insufficient to maintain basic subsistence; **cannot afford food or shelter** eg. Dickens
- 1995: one QUARTER of the total world population fits into this category: **less than 1 dollar per day**
- **this figure has DOUBLED SINCE 1975**

RELATIVE poverty

- income insufficient to pay for basic social roles, participate in relationships etc.
- the exact figure is determined by the experts, eg. the **"HENDERSON POVERTY LINE"**
- henceforth "poverty" in this summary will allude to this estimate

How to determine who's poor and who's not:

- "expert" estimate of how much it costs to subsist in Australia, at a given time
- a conservative estimate: NOBODY WILL ARGUE that those below this line are "very poor"
- lecturer quoted about $290 as being the poverty line for a single white female living alone

Distribution in Australia
12% live in poverty: 1.7 million people
13% of children live in poverty: 440,000 kids

Specific groups:

- of single parents 29% are in poverty
- of couples with 1 child 5% are in poverty; …of couples with 3 kids- 11%

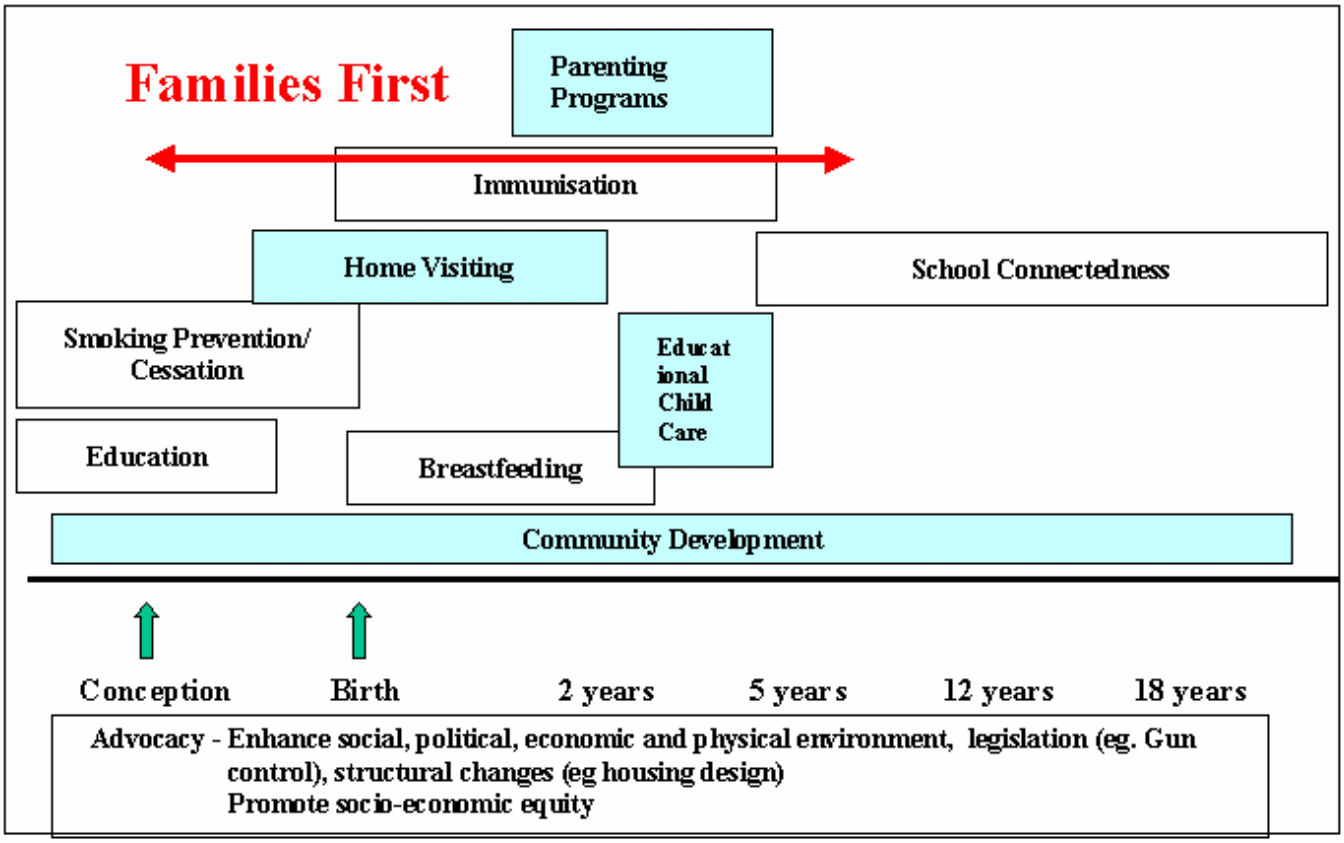
Why does this matter?

- There exists a direct relationship between income and life expectancy
- Chronic illness and hospital admissions are more frequent in unemployed population
- Chronic illness is more common among children of the unemployed
- Children of the poor are less likely to be breastfed

How has this changed?

- Rates of breastfeeding, child mortality (including indigenous population) and birth weight have improved
- Not much else has

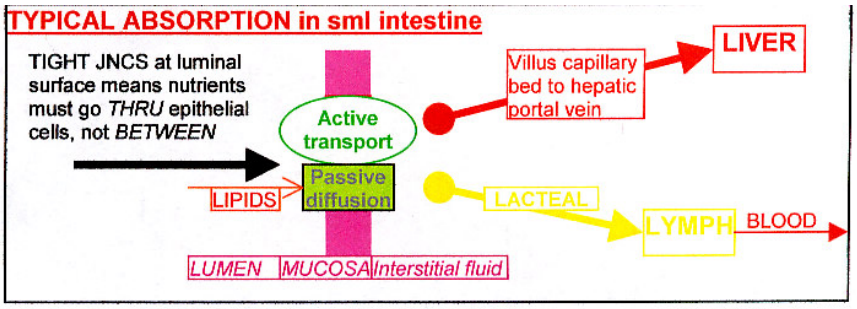
Policies and Programs directed at child health: a timeline for a hypothetical infant



DIGESTION & ABSORPTION OF NUTRIENTS

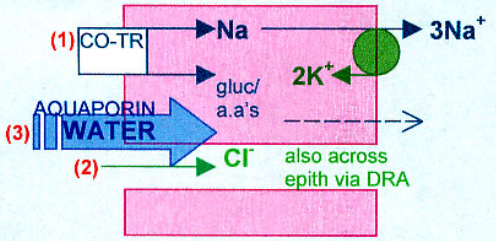
706_05

TYPICAL ABSORPTION in sml intestine



WATER & SALT – ABSORPTION @VILLUS

Water follows solutes: Na, Cl, HCO_3.
Solutes transported by ACTIVE TRANSPORT & LEAKY TIGHT JNCS
Main players: Na enters cell unwanted & pumped out by **SODIUM PUMP** (Na^+/K^+ ATPase) @ basolat membrane
(1) Na cotransported with glucose/aa OR
(2) Na exchanged for H+ etc. via NHES
→CREATES OSMOTIC GRADIENT FOR WATER TO FLOW DOWN



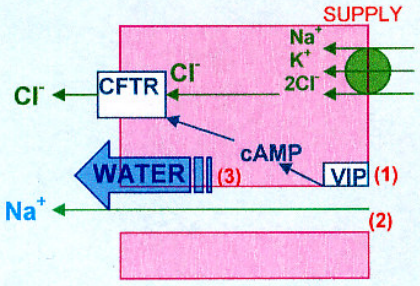
NHES mutn (no Na in), DRA mutn (no Cl in) → *DIARRHOEA*

WATER & SALT – SECRETION @CRYPT

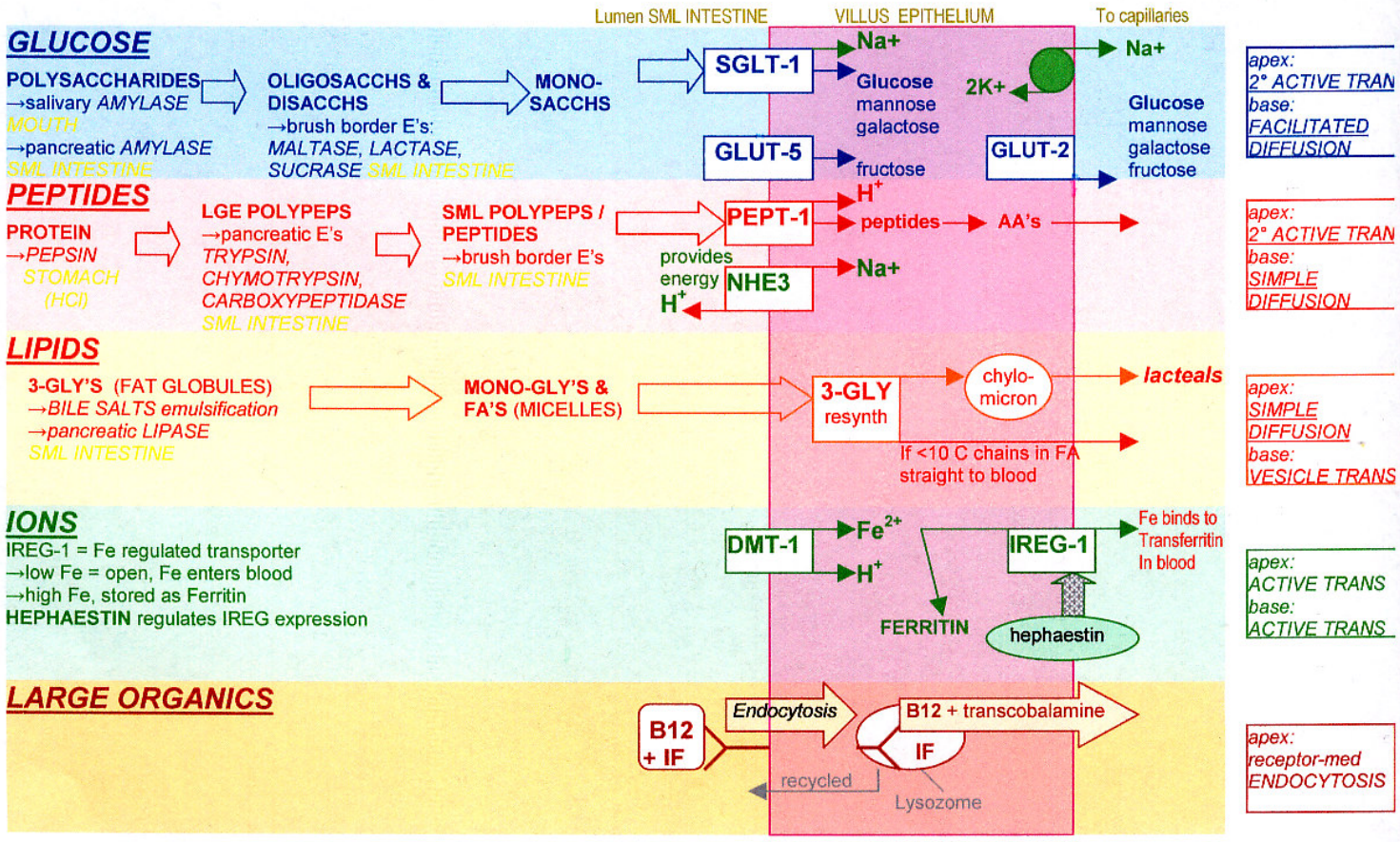
(1) osmotic gradient estab by food in lumen OR
(2) crypt secretes NaCl, water follows

CFTR = cAMP dependent Cl channel
Main players:
SUPPLY = Na,K-2 Cl cotransporter
ACTIVATOR = VIP
CHANNEL = CFTR

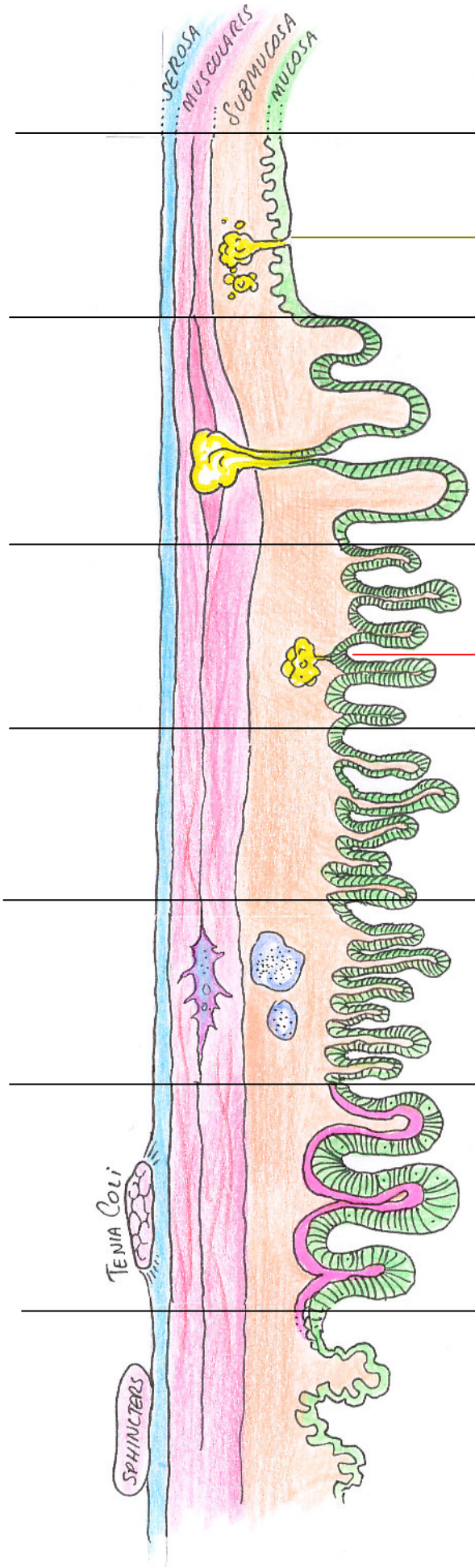
→CF=CFTR mutation malabsorption
→bacterial toxins (*cholera toxin)* (1)keep cAMP high, (2)blocks villus/crypt differentiation→ MASSIVE DIARRHOEA
stops at cell turnover (days)



Bicarbonate similarly secreted by GASTRO, DUO epithelium. **SUPPLY**=Na-bicarb contransporter , **CHANNEL**=CFTR



MICROSCOPY OF THE GUT:



OESOPHAGUS

2x layers of muscularis externa; upper 1/3 = striated muscle
lower 1/3 = smooth muscle
middle 1/3 = mixed histology
foamy-looking glands in the submucosa, secreting mucous for lubrication
submucosa mainly made of connective tissue
papillae are present to anchor the STRATIFIED SQUAMOUS EPITHELIUM
PLUS there is a properly visible Muscularis mucosae

STOMACH

3 layers of muscularis: oblique (inner), circular, longitudinal(outer)
muscularis also contains the glands at the bottom of the pits
GLANDS are filled with **PARIETAL (acid)** and **CHIEF CELLS (enzymes),** plus the neck has **MUCOUS CELLS** and there are **ENTEROENDOCRINE CELLS** throughout
MUCOSA is a simple columnar epithelium, "THE SURFACE MUCOUS CELL"
Cells migrate from the bottom of the pit (where the stem cells are) out to the top of the hill where they die and slough off; this is a common path for all the columnar cells in the GIT

DUODENUM

Is dominated by **VILLI** and the **CRYPTS** between them; V. absorb, C. secrete
VILLI are filled with loose connective tissue, strands of smooth muscle, lacteals and plasma cells
2 layers of muscularis, transverse and longitudinal (for peristalsis)
!! CHARACTERISTIC FEATURE !! are the Brunner's Glands, secrete mucus
there is also a lot of GOBLET CELLS throughout the small intestine

JEJUNUM

is unremarkable by anything except its lack of features which distinguish the DUODENUM and the ILIUM;
...but this is where most of your absorption happens
Villi are rather longer and more finger-like in the jejunum than in the duodenum

ILIUM

AYERBACH's PLEXUS: between the layers of muscularis, there are ganglia of the enteric nervous system; neurons project to the circular muscle, to other myenteric ganglia, to submucosal ganglia, or directly to the epithelium, and play an important role in regulating and patterning gut motility.
!! CHARACTERISTIC FEATURE !! PEYER'S PATCHES: pieces of MALT, (Mucosa-Associated Lymphoid Tissue) nodular, contain antigen-presenting cells and lymphoocytes.

COLON

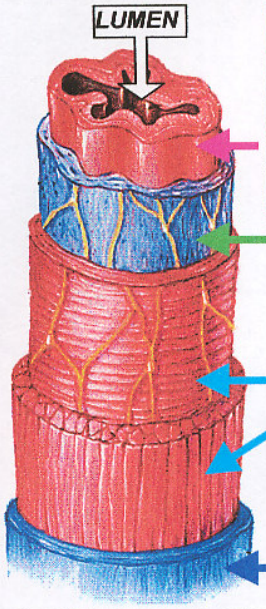
distinguished from the above by presence of longitudinal TENIA COLI muscle, and sack-like projections called HAUSTRA (plus there are fatty EPIPLOIC APPENDAGES which hang off the colon like christmas ornaments)
PLUS there is a properly visible Muscularis mucosae,
Straight tubular glands (crypts of Lieberkuehn) extend through the full thickness of the mucosa.
The appendix is characterised by large numbers of lymph nodules. In many adults, the normal structure of the appendix is lost and replaced by fibrous scar tissue.

ANO-RECTAL JUNCTION

Rapidly does the simple columnar epithelium shapeshift into stratified columnar,
Then stratified cuboidal
Then stratified squamous

ALIMENTARY CANAL 4-basic layers

DIFFERENT TISSUE TYPES PREDOMINATE @ DIFFERENT AREAS → FUNCT DIFFS



MUCOSA (MUCOUS MEMBRANE) 3 SUBLAYERS
EPITHELIUM: SIMPLE COLUMNAR EPITHELIUM, ++ GOBLET CELLS
→ **mucus**: protects against autodigestion, lubricates passage
(@ stomach, sml intestine ENZYME & HORMONE-SECRETING CELLS)
LAMINA PROPRIA LOOSE AREOLAR CONNECTIVE TISS.
CAPILLARIES → **nourish & absorb nutrients**
LYMPH NODULES → **protection against gut pathogens**
MUSCULARIS MUCOSAE SMOOTH MUSCLE (thin)→**local movement**

SUBMUCOSA mod dense CONNECTIVE TISS.
(1)BLOOD & LYMPH VESSELS (2)LYMPHOID FOLLICLES
(3)NERVE FIBRES (4)++ELASTIC FIBRES → **mobility of mucosa**

MUSCULARIS (EXTERNA) SMOOTH MUSCLE: 2 LAYERS
→**peristalsis & segmentation**
INNER CIRCULAR → thicken to form **sphincters**
OUTER LONGITUDINAL

SEROSA = *VISCERAL PERITONEUM*
AREOLAR CONNECTIVE TISS covered with MESOTHELIUM (single layer of squamous epithelial cells) → **protection**
@ oesoph, replaced by **ADVENTITIA** FIBROUS CT → **binds oesoph to surrounding structs**
@ retroperitoneal organs: both serosa & adventitia

TONGUE

Ant 2/3 = oral cavity, post 1/3 = oropharynx
-NO SUBMUCOSA
SKELETAL MUSCLE runs in 3 perp planes
LINGUAL FRENULUM secures to floor
STRATIFIED SQUAMOUS EPITHELIUM (keratinized)
PAPILLAE (ant 2/3)
-filiform: lots, keratin → TOUGH, GRIP
-fungiform & circumvallate →TASTE BUDS
SEROUS GLANDS flush Δe for taste buds

SALIVARY GLANDS

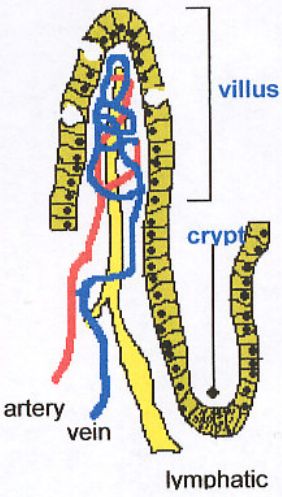
MAJOR: (OUTSIDE ORAL CAVITY)
PAROTID (serous), **SUBMANDIBULAR** (mixed), **SUBLINGUALS** (mucous mostly)
=*COMPOUND TUBULOALVEOLAR glands*
serous acini + mucous tubules→ducts
(continuous w oral epithelium distally)
MINOR: (ORAL CAVITY MUCOSA) continous secretion for moisture
****serous* (watery, enzymes, ions, ~mucin)
****mucous* (mucin)

(ORO) PHARYNX

STRATIFIED SQUAMOUS EPITH (no keratin)
MUCOSA: LP = DENSE, FIBROELASTIC
MUSCULARIS ext = SKELETAL (pharyngeal constrictors)
ADVENTITIA

OESOPHAGUS	*STOMACH*	*SML INTESTINE*	*COLON*	*RECTUM*
*STRONG ELASTIC (conduction & protection) *MUCUS (tubuloalveolar mucous glands)	CHEM DIGESTION (secretion) MECH (muscle churning)	CHEM DIGESTION (secretion) ABSORPTION 3 ways to max surface area!! Big folds –*plicae circulares* mucosa & submucosa Sml folds – *villi* epith & LP Cell folds – *microvilli* = **BRUSH BORDER*** plasma membrane, luminal surface	FLUID ABSORPTION MUCUS	MUCUS SPHINCTERS
STRATIFIED SQUAMOUS EPITH Non-keratinized, *THICK* **APC's (Langerhans)** LP **oesoph cardiac glands* (@ends) *ELASTIC network of LONGIT & CIRCULAR s.m.	SIMPLE COLUMNAR *RUGAE (folds)* → *expand* *SECRETORY SHEET* of columnar cells →*neutral MUCIN* -protect from acid pepsin -lubrication *GASTRIC PITS* tiny tubular infoldings of epith – glands at bottom of pits **cardiac:** *MUCOUS* **gastric:** (+++ thru-out) MUCOUS NECK CELLS -*acid mucin* PARIETAL CELLS – *HCl, IF* CHIEF CELLS – *pepsinogen* ENDOCRINE – *GHIH, glucagon, pancreatic peptide* **pyloric**: MUCOUS CELLS ENDOCRINE – *gastrin* **+++ Lcytes, ephils, mast cells**	SIMPLE COLUMNAR VILLI, CRYPTS, GLANDS *ENTEROCYTE* = main type of absorptive cell *LOTS!!!* →**digestive**: enzymes for disaccharides + polypeptides →**absorptive**: protein, aa's, carbo's (to capillaries in villi) lipids (to lymphatic channels) *GOBLET CELLS*: *less!* →*mucus* (lubricates, protects) (lifespan: 4-5d) *ENDOCRINE CELLS* *few* →*peptide hormones* (incl/ secretin, CKK, gastric inhib peptide, serotonin) *PANETH CELLS* **L-cytes** **++L-cytes** (migrate to lumen) **lymphatic nodules** (++ileum) grouped as **Peyer's patches** **M-cells** *LYMPHATIC VESSELS* – **FAT** muscle enters VILLI → pump action helps absorption	SIMPLE COLUMNAR CRYPTS ~ **NO VILLI** ~ GLANDS- bigger, closer *ENTEROCYTES* *GOBLET CELLS* ~~(*no enzymes secreted but digestion still occurs due to enzymes within faeces & bacterial flora here*)~~ →→→→→→→ →→→→→→→	SIMPLE COLUMNAR *RECTAL COLUMNS*: longit folds *ANAL VALVES* distally: transverse folds GLANDS *GOBLET CELLS* muscularis mucosae ← !! ALL END HERE!! → → STRATIFIED SQUAM EPITH (non-keratinized) → SSE (keratinized) @ *External anal sphincter* !! SKIN HERE !! *CIRCUMANAL GLANDS* → oily material
coarse COLLAGENOUS FIBRES, ELASTIC FIBRES LONGIT FOLDS: allow expansion *oesoph glands proper* (betw)	**+++Lcytes, ephils, mast cells**	*DUODENAL (BRUNNER'S) GLANDS* drain into overlying intestinal glands → *basic mucin* -nr pancreatic duct, protect from acid pepsin from stomach		*HAEMORRHOIDAL PLEXUS* +++veins (haemorrhoids=distension & protrusion into mucosa)
upper 1/3 = **SKELETAL** mid 1/3 = **MIXED** lower 1/3 = **SMOOTH:** autonomic ganglia in between *SPHINCTERS* formed by intra-luminal high P @ ends of oesoph	*3 LAYERS -outer LONGIT -mid CIRCULAR (thickens @ pylorus → *PYLORIC SPHINCTER* -inner OBLIQUE (fundus)	2 LAYERS	outer LONGIT forms 3 bands: *TAENIAE COLI* →gathers colon into *HAUSTRA* →folds in between are *PLICAE SEMILUNARES*	*INTERNAL ANAL SPHINCTER* inner CIRCULAR thickens **outer fades away** *EXTERNAL ANAL SPHINCTER* skeletal m surrounds dist anal canal
ADVENTITIA			ADVENTITIA @ ascending & descending (retroperit)	

GLANDS

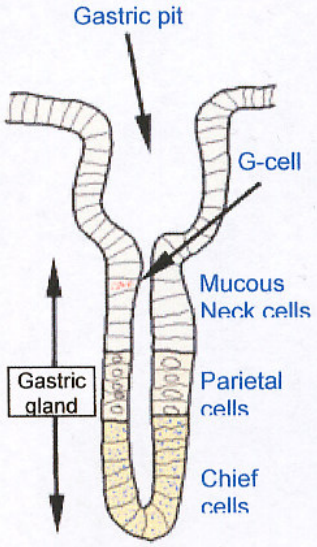


SML INTESTINE

VILLI = PROJECTIONS INTO LUMEN covered with ***MATURE ABSORPTIVE ENTEROCYTES*** also ***GOBLET CELLS***.
-short-lived, shed

CRYPTS = INVAGINATIONS OF EPITHELIUM AROUND VILLI lined with ***YOUNG SECRETORY EPITHELIAL CELLS***.
@ BASE = ***STEM CELLS*** constantly dividing to replace all epithelial cells on villi & crypts.

DYNAMICS
Stem cells commit to 1 of 4 lines:
(1)*ENDOCRINE* (2)*GOBLET* (3)*PANETH* (4)*ENTEROCYTE*:
these differentiate as they ascend
SECRETORY → ABSORPTIVE
ABSORPTION
→ capillary bed → systemic circulation
→(fats) lymphatic vessel: *CENTRAL LACTEAL* → thoracic duct → circulation



STOMACH

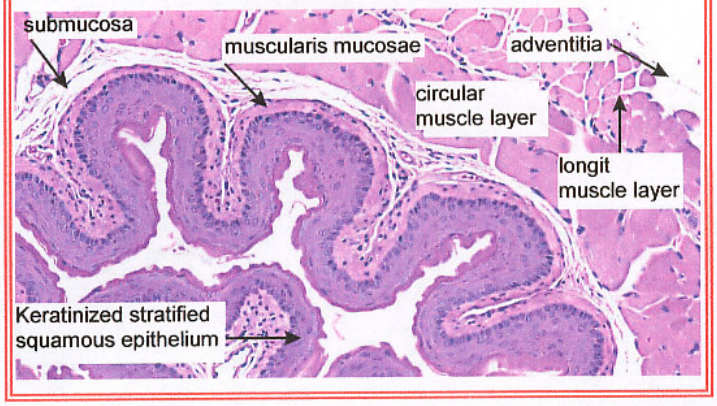
GASTRIC PITS = tubular infoldings of epithelium, replaced every 4-5d by MIGRATING CELLS

~~~THESE OVERLAY~~~

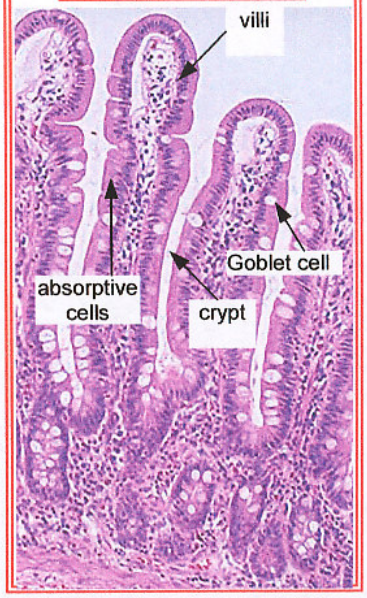
GASTRIC GLANDS: the most abundant glands in stomach. More than one may open into each pit.
****Endocrine cells (apart from G-cells) secrete → blood / intercell space (not lumen)*

PLURIPOTENT STEM CELLS at the bottom of the pit
→ most migrate to lumen to become **gastric lining epithelial cells** (exfoliation)
→ some go downwards
-MUCOUS NECK differentiate into CHIEF CELLS
-ENDOCRINE
-PARIETAL go both ways
(if necrotic→shed, apoptosis→eaten)

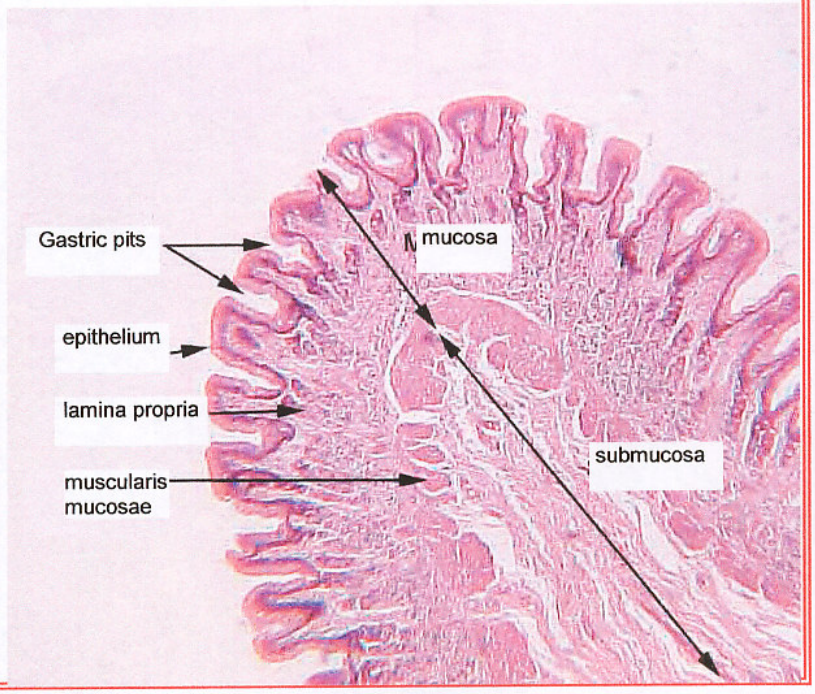
OESOPHAGUS



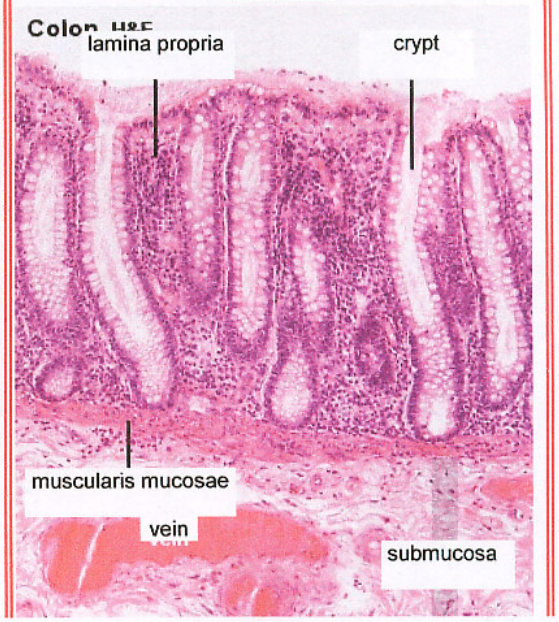
SML INTESTINE



STOMACH



COLON



: LACTATION and BREAST MILK BIOCHEMISTRY

Breast development: starts @ embryo, incomplete until 1st pregnancy

Embryology: two lines of buds parallel to midline; only one pair normally develops.

@ 12 weeks of foetus: 20 primitive ducts

@18 weeks: canalised;

deep portions = double layer of epithelium, later = alveolar ducts

+ myoepithelium which will push milk → nipple

@ PUBERTY: oestrogen, prolactin, insulin, GH and corticosteroids influence the following:

- Fat deposition
- Duct division + branching + enlargement
- Increased vascularity

@ first pregnancy:

placental oestrogen + progesterone + human placental lactogen + prolactin + maternal insulin + corticosteroid,

→ alveolar cells become secretory by 20th week of gestation

BUT: progesterone inhibits lactation; so as soon as the progesterone-secreting placenta is out of there, the lactation BEGINS!

COLOSTRUM: the 1st thing out of the breast; for 3-5 days

- yellow liquid high in protein (particularly immunoglobulin)
- only moderate amounts of sugar and fat.
- THEN → gradual conversion to milk secretion.

BREAST MILK:

- **proteins (lactalbumin, lactoglobulin) lactose, water and fat.**
- Milk is isotonic with plasma with lactose accounting for ½ the osmotic pressure.
- Milk **contains all vitamins except Vitamin K,**
- has a low salt content,
- is alkaline,
- is sterile
- contains immunoglobulins,
- contains growth factors important for gut development.

Human milk contains all the nutrients which are required for a healthy term baby of a well nourished mother for the first four to six months. No supplements are necessary other than vitamin K given at birth to prevent haemorrhagic disease of the newborn, a condition almost exclusively seen in breast-fed infants.

BF reduces the risk or severity of pyloric stenosis, respiratory illness, gastrointestinal tract disease, inflammatory bowel , some childhood cancers, otitis media, urinary tract infections, Sudden Infant Death Syndrome (SIDS) and juvenile onset diabetes mellitus.

Breastfeeding helps THE MOTHER: by protecting against premenopausal breast and ovarian cancer and osteoporosis. It also helps the mother to regain her pre-pregnancy body weight and provides a contraceptive effect.

COMPOSITION OF BREASTMILK favours the colonisation of the infant colon with the non-pathogenic lactobacillus bifidus and discourages colonisation with pathogenic bacteria.

NO FORMULA WILL EVER APPROACH THE PERFECTION OF MOTHERS' MILK

Hard to copy: Human milk varies with the gestational and chronological age of the baby, the timing of sample collection both during the feed and during the day, with the diet, nutritional status, menstrual cycle and genetic make-up of the mother, and with the content and time elapsed from the mother's last meal.

bioactive components:

live cells

immunoglobulins related to infections the mother has been exposed to,

large variety of oligosaccharides (about130)

varying growth modulators which include hormones and enzymes.(eg. insulin, growth factors (IGF1), and lipases)

PLUS long chain polyunsaturated fatty acids and proteins such as human lactoferrin

Compared with other mammalian milks, human milk is

- low in protein
- high in substances necessary for brain maturation, eg.
  - lactose
  - cholesterol
  - long chain polyunsaturated fatty acids
  - readily absorbed iron.

With the exception of infants with congenital lactase deficiency, an extremely rare disorder inherited as an autosomal recessive condition**, virtually all infants are capable of digesting lactose in mammalian milks**.

In most mammals, lactase activity declines after weaning; in contrast, in man lactase activity persists into adult life in approximately 30% of the world's population

The major macronutrients in rank order are

- water
- lactose
- fats
- oligosaccharides.

Fat provides nearly 50% of the energy from milk,

DO NOT feed them whole cows milk!

HAVE TO USE INFANT FORMULA for the 1st year …until they can drink from a cup

then you can start on full cream cows milk.

SUCKLING:

The baby places the whole areolar in the mouth with the nipple next to the back of the tongue. The baby's tongue presses against the roof of the mouth, and starting at the anterior (gum) margin rolls back against the palate. This action allows the stored milk to be squeezed into the mouth (oropharynx).

Suckling induces release of prolactin that induces milk secretion,
and oxytocin causes the let-down response (causing the myoepithelium to contract and eject the milk)
The baby swallows about 2 ml at a time.

!! BEWARE !! HIV and Hep C are transmissible through milk!!

LACTATION HAS A CONTRACEPIVE EFFECT:

The high prolactin levels associated with suckling inhibit ovulation

NORMAL NUTRITION IN THE FIRST 6 MONTHS:

SO, you're an infant. Your gut and kidneys are immature. You cannot tolerate solids and high renal solute loads **THUS you have to suckle, it's the best thing ever.**

Breastfeeding practices in Australia

Not every mother chooses to breastfeed. In
From the 1940s to 1960s, breastfeeding prevalence rates fell in many westernised countries as breastfeeding became less popular in line with the drive to modernity.
The 1970s saw a return in popularity of breastfeeding, at first among educated women.
Breastfeeding initiation rates rose from then around 70% to now 90%, and rates at one year from less than 10% to now 20%.

The most common reason given for early cessation of breastfeeding is an "inadequate supply of breastmilk", probably reflecting a lack of support in the early stages.

Infant formulas

If Breast milk is unavailable, a standard infant formula should be substituted.

Cow's milk, sweetened condensed milk, or powdered cow's milk, are inappropriate

Amount of formula

A normal growth rate is the best sign of adequate nutritional intake.

A formula fed infant generally needs 100-120 kcal/kg/day,
..which is provided by **150-180 ml of formula/kg/day.**
As babies grow older they feed less frequently but have larger volumes at each feed.
At the age of 6 months, an infant would usually be having 4-5 feeds per day of 180-240 ml per feed.

Water and other fluids :

IF BREASTFED : NO NEED FOR FLUID SUPPLEMENT

If you water down beast milk, the infant will fail to thrive and go jaundiced

Introduction of solid foods @ 6 months

From 6-7 months through to 11-12 months = **progress from sucking to chewing and biting**.
MUST TRY EVERYTHING!! learn different tastes and textures, and the mouth-feel of different foods.
If babies miss out on this experience, they then tend to **resist eating lumpy food,** which may lead to significant feeding difficulties in the second year.

BUT: NOT TOO SALTY OR TOO SWEET! Dont want your baby habituated to these flavours

Early introduction to solids = world of harm;

- undernutrition (decreased nutrient intake through immature gut)
- increased morbity (diarrhoea, allergies)
- unnecessary stress on kidneys
- infant becomes used to high levels of sugar and salt

Late introduction to solids = not so good either

- growth-faltering
- difficulties in introducing new foods into the diet.

NORMAL BIRTH WEIGHT: 3.4kg
… should be
!! DOUBLED AT 5 months !!
…
!!! TRIPLED !!!
at 1st birthday

NUTRITION and DEVELOPING COGNITIVE FACULTIES

HARD TO STUDY:
usually poor nutrition goes together with poor everything else
eg. poor parents, low education level, no intellectual stimulation

HUNGRY BABY = STUPID BABY
Preterm infants = most at risk

Mothers who elect to breast feed in developed countries are of a **higher social and education status** than those who do not.
But.. Differences observed betwenh breast and bottle feeding may be due to the differences in mother-child contact and attachment (bonding) rather than human milk constituents.

BEST RESULTS: good nutrition AND mental stimulus
Although either one of these alone is still pretty good
BREAST MILK CONTAINS NERVE GROWTH FACTOR AND IGF-1, + iron, zinc etc

CONTROVERSIAL QUESTIONS: poly-unsaturated fatty acids. Apparently good for brain, but → conflicting results; SO nobody is allowed to add these PUFAs to their formula.

WORLD SCALE: IRON and IODINE = most scarce micro nutrients. **IODINE ESPECIALLY IMPORTANT!**
→ 20 million preventable cretins swarm the globe !! all for lack of Iodine

IRON DEFICIENCY = commonest deficiency worldwide, **40-45% of ALL children are anaemic (10% in developed countries!)**
= poor attention span, lower intelligence scores and perception.

Poor performance by iron deficient adolescent girls in developed countries is improved with iron supplementation.

…But… Should making a child smarter be promoted as another reason for breast feeding?

ENERGY BALANCE

ENERGY IN = ENERGY OUT.

1 kcal = 4.184 kJ
1 kJ = 0.239 kcal
I joule = the energy used when 1 kilogram is moved 1 metre by a force of 1 newton.
The newton is the unit of force that accelerates 1 kg by 1m/sec

4 components to total energy expenditure over 24 hours:

Basal metabolic rate

= **largest single component**

influenced by:

body composition
(lean tissue has a higher metabolic rate than adipose tissue)

SEX
(largely through differences in body composition, with males generally having more lean tissue for a given weight than females)

AGE
(decreases with advancing age eg ~210-220 kJ/m ^{2} /h at age 5 years; 180-190 kJ/m ^{2} /h at age 15 years; 140-150 kJ/m ^{2} /h at age 80 years - note that BMR is here adjusted for surface area which is easier to measure than lean body mass)

PHYSOLOGICAL STATE
eg increased in pregnancy

PSYCHOLOGICAL STATE
eg increased with acute anxiety

MEDICATIONS
eg increased with caffeine, nicotine; decreased with beta-blockers

DISEASE
eg increased with fever (~13% over basal for every degree Celsius), infections, malignancy, hyperthyroidism, burns (40 - 100% increase)

Energy cost of growth

…it costs energy to make breast milk, and its very expensive to grow flesh.

Gain of 1 g lean body mass =
= 5.4 - 7.1 kJ
Gain of 1 g fat mass =
= 50 kJ

Overall, each gram of weight gain requires ~21 kJ of energy.

Note: 1 metabolic equivalent (MET) is equivalent to BMR. Thus, an activity that is twice the energy cost of BMR is 2 METs.

The composition of weight gain is not the same at all times during growth.
For example, during the first 4 months about 40-45% of weight gain is body fat,

whereas by age 2 years it is much lower (less than 10%)

Therefore,
]**the energy cost of weight gain is much higher in early infancy than by age 2 years.**
This is a major reason why a decrease in energy intake in very young children can have a more significant impact on

Thermogenesis

consists of
cold thermogenesis
(shivering and non-shivering)
and
post-prandial thermogenesis.
One is to maintain the body tenpereature at the homeostatic set point; the other is a result of energy being sued to digest and absorb food.

. **Over the course of a day, it is usually about 6% of basal metabolic rate**.
The exact amount depends upon a range of factors including macronutrient intake eg protein digestion and metabolism leads to increased postprandial thermogenesis when compared with the digestion and metabolism of carbohydrates or fats.

Energy cost of activity

Physical activity accounts for 20-40% of total daily energy expenditure in most individuals,
→ more for athletes;
→ less for sloths

The amount of energy expended depends upon the intensity and duration of the different activities carried out in a day

Physical activity incorporates both sports-like exercise as well as **non-exercise activity thermogenesis (NEAT),** which includes such things as fidgeting and incidental movement.

In adults, energy requirements can be estimated by

1) first calculating basal metabolic rate using published equations (eg Harris-Benedict equation)

2) and then multiplying by a factor to account for the level of physical activity eg 1.3 for sedentary activity, 1.5 for moderate physical activity and 2 for strenuous activity.

BELOW:
Estimates of energy costs of selected activities, expressed as a multiple of BMR, or metabolic equivalents [METs]

Activity	Energy cost (multiple of BMR)
Rest	1.
Desk work	1.5
Driving	1.6
Standing	2.0
Walking slowly on level ground	2.5
Walking quickly	4
Cycling slowly	4
Running slowly	6
Tennis	6-8
Swimming, light-moderate effort	8
Elite athlete (maximum aerobic capacity)	**24**

Table 2: Estimated average energy requirements of children 1-10 years

Age (y)	Boys (kJ/kg/d)	Girls (kJ/kg/d)
1-2	435	450
2-3	410	427
5-6	385	368
9-10	301	260

FAILURE TO THRIVE

A BABY SHOULD GAIN ABOUT 150g per week

= a description, not a diagnosis.
= describes a toddler or infant whose growth rate is abnormally low
= causes include all kinds of illness, malnutrition and lack of love (?)
= LOOK AT PERCENTILE CHART: but in first few months of life, the average weekly weight gain is better
!! Nutritional influence on growth takes place on a background of the baby's genetic potential for growth.

Table 1 shows the minimum average acceptable weight gain
(grams/week) between 6 and 12 weeks according to the weight percentile at 6 weeks.

Wt percentile at 6 wks	Boys	Girls
p3	170	145
p10	160	137
p75	133	112
p97	130	103

The lower limits for weight gain for boys and girls together for small babies is about 150 g per week, and for bigger babies 120 g per week. Overall the lower limit for acceptable weight gain is greater for the smaller babies.

WHO recommended anthropometric indices:

weight-for-height,

- . Low weight-for-height , also known as wasting or thinness (<~3rd centile of weight for height), indicates in most cases a recent and severe process of weight loss, which may be associated with severe disease or acute starvation

height-for-age

- Low height-for-age, or stunted growth (height <~3rd centile of height for age), reflects a process of failure to reach linear growth potential as a result of suboptimal health and/or nutritional conditions. For children in the age group below 2-3 years, low height-for-age probably reflects a continuing process of "failing to grow" or "stunting"; for older children, it reflects a state of "having failed to grow" or "being stunted".

weight-for-age

- . Low weight-for-age , or underweight (weight <~3rd centile of weight for age), can reflect both a chronic process (ie long-term poor nutrition) as well as an acute process.

SYMPTOMS AND SIGNS:

Mild:

- child is irritable and unhappy,
- hair is lack lustre, coarse or sparse,
- the skin is dry.

Moderate/severe:

- wasting which is best seen by observing the child from side on,
- = reduction in muscle mass in the buttock
- loose skin evident on the medial aspects of the thighs and buttock.
- Generalised muscle wasting can be associated with oedema, reflecting hypoproteinaemia.

chronic undernutrition in children may present as stunting only,

history, physical examination and longitudinal growth data will show whether the child is thriving or not.

COMPARE TO PARENTS: not abnormal for little tiny people to have little tiny children

Pre-term children will appear short and wasted if plotted according to actual age;

THUS: plot their graph according to EXPECTED age (i.,e when they were due) rather than actual age

Correcting the age to the expected, rather than actual, birthdate should be made for weight until 24 months, and for height until 40 months.

Age (mo)	Weight gain (g/day)	Length gain (mm/day)
0-3	25-30	0.9
3-6	20	0.7
6-9	15	0.5
9-12	10	0.4
12-18	8	0.4
18-24	6	0.3

Conceptual Overview of Gastrointestinal Anatomy — 7.06 Lecture 1

ARTERIAL SUPPLY

- **abdominal AORTA**
  - **T12: Coeliac trunk**
    - Left gastric → stomach + oesophagus
    - splenic → spleen + stomach + pancreas
    - Common hepatic → Liver, gallbladder, duodenum, pancreas
  - **L1: Sup. mesenteric**
    - Inf. pancreatico-duodenal → Pancreas, duodenum
    - ieiunal → jejunum
    - ileal → ilium
    - ileocolic → ilium, caecum, appendix
    - RIGHT COLIC
    - MIDDLE COLIC
    - LEFT COLIC
    - → Anastomose → MARGINAL (all along the colon)
  - **L3: Inf. mesenteric**
    - Sigmoida → sigmoid colon
    - Superior rectal → Rectum above pectinate line

VENOUS DRAINAGE

FOREGUT → Splenic vein
HINDGUT → Inf mesenteric
MIDGUT → Sup mesenteric

→ **PORTAL VEIN** → **Sinusoids of the liver** → **Hepatic veins** → **INFERIOR VENA CAVA**

ANASTOMOSIS between the portal and caval venous systems:
!! site of rupture !! if the portal pressure is too high!
- **Oesophageal wall**: bleeding into stomach, hematemesis, black stool
- **Anal canal:** hemorrhoids
- **Abdo wall around umbilicus:** "Caput Medusae" radiating veins
- **Posterior Abdominal Wall**

LYMPHATIC DRAINAGE:
FOREGUT → coeliac nodes
MIDGUT → sup mesenteric nodes
HINDGUT → inf mesenteric nodes

→ **Via INTESTINAL TRUNKS: → to cysterna chili; → to thoracic duct**

INNERVATION:
FOREGUT + MIDGUT → Vagus (parasympathetic), T5-T12 thoracic splanchnic (sympathetic)
HINDGUT → S2-S4 pelvic splanchnic (parasympathetic); L1-L2 Lumbar Splanchnic (sympathetic)

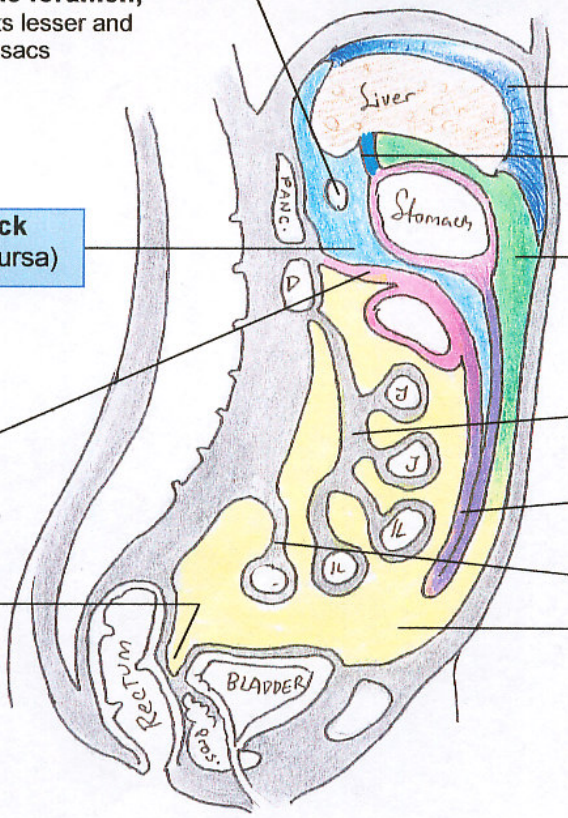
The CAVITIES

Epiploic foramen, connects lesser and greater sacs

Lesser Sack (omental bursa)

Transverse mesocolon

Retrovesicular pouch:
"behind the bladder":
In an upright position, when there is pus in the abdomina cavity, it will collect here→ and this is very convenient as it is right next to the rectum, thus one can palpate the pouch (pain and irritation are abundant when exudate collects there)



Falciform ligament
Lesser omentum
Supracolic space
mesentery
Greater omentum
Sigmoid mesocolon
Infracolic space

SALIVATION CONTROL

INTRINSICS continuously secrete for moisture
EXTRINSICS activated by **FOOD!!** (ie. ΔpH, pressure, also lower GI irritation)
TO BRAIN: Food → chemoreceptors → **salivatory nuclei** @ brainstem
Pressoreceptors
FROM BRAIN: CN VII, IX → ↑ **serous** output **PARASYMPATHETIC CONTROL**
→ ↑ **mucous** output under **SYMPATHETIC CONTROL**

PROPULSION: (1) muscle contraction
(2) mucus secretion to lubricate

PHARYNX

SWALLOWING CONTROL
TO BRAIN: food stimulates **tactile receptors** here
(invol reflex activity follows)
FROM BRAIN:
swallowing centre @ medulla, Pons → CN X →
mm of pharynx & oesophagus → *peristalsis*

MOUTH *INGESTION*

TEETH: *MECH DIGESTION (chew)*
TONGUE: *CHEM DIGESTION (starch)*
SALIVA: *initiation of PROPULSION*
(1) **Lubricant & food softener**(mucin)
(2) **Protects teeth**
-Ca^{2+}, PO_3 = integrity
-bicarb = neutralizes bact acid attack
-AMYLASE degrades sugars before bact can use
-anti-bact agents: incl LYSOZOME, DEFENSINS
(3) **digestion**: AMYLASE (polysaccharide breakdown)
LIPASE (only works when no
gastric acid to inhibit → neonates)
(4) **growth factors:** oral mucosa health
↓↓ saliva = probs TALKING, SWALLOWING, EATING
CLEFT PALATE = *food to nose*

OESOPHAGUS

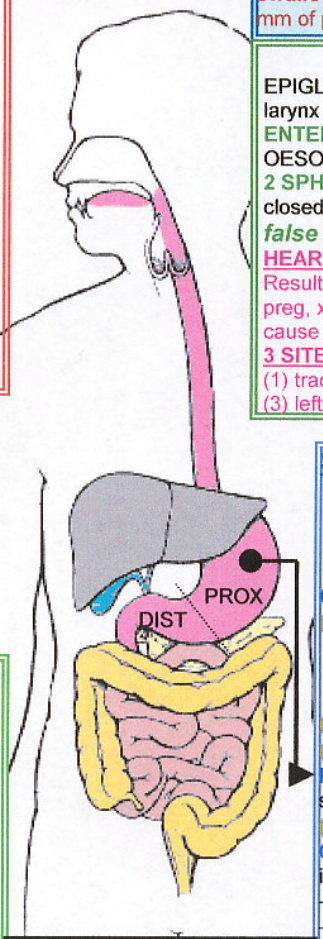
EPIGLOTTIS guides bolus here by blocking entry to larynx
ENTERS ABDO by piercing Diaphragm @ OESOPHAGEAL HIATUS
2 SPHINCTERS: upper & lower oesophageal closed when not swallowing, collapsed between
false PERISTALSIS: intrinsic control (no CNS)
HEARTBURN: gastric acid regurgitates into oesoph
Result of abdo contents pushed upwrds: obesity, preg, xs consumption, running, hiatus hernia. Can cause **oesophagitis, oesophageal ulcers**
3 SITES OF COMPRESSION
(1) trachea bifurcation (2) crossing of aortic arch
(3) left ventricle = **potential hazards for swallowing**

SECRETIN → bile produxn → **LIVER** ↓ bile → **GALLBLADDER** (stored) sml gallstones
CCK bile release

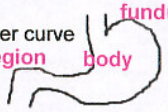
PANCREAS
Pancreatic juice
- bicarb
- pro-enzymes
Main Pancreatic duct

Hepato-pancreatic sphincter

fatty CHYME — MAJOR DUODENAL PAPILLA



STOMACH



CARDIAC SPHINCTER = in point
PYLORIC SPHINCTER = outpoint (gatekeeper)
Fundus: HCL, enzymes
Body: mucus
Pyloric region: mucus, enzymes
(1) STORAGE for duo
(2) MECH DISRUPTION
PACEMAKER sends waves of contraxns, food swept along, crashes into pylorus → pulverised
(3) CHEM DEGRADATION: secretion →
GASTRIC ACID @ distal 2/3 by PARIETAL CELLS in glands **HCl (pH ~ 1)**
→ ~ PROTEIN DEGRAD
→ ANTI-BACTERIAL bact overgrowth if ↓ secretn
→ activates PEPSIN
PEPSIN = acid sensitive (needs pH<3)
Produced as **PEPSINOGEN** @ CHIEF CELLS
→ PROTEIN DEGRAD to peptide frags needed in GI regulation (also anti-bact)
INTRINSIC FACTOR
→ VIT B12 ABSORPTION: secreted by PARIETAL CELLS, complexes with B12, complex absorbed @ Terminal Ileum. Autoimmune Δ, surgery, parietal cell damage → Pernicious anaemia
MUCUS: secreted from SURFACE EPITH.
→ MUCOSAL PROTECTION BARRIER (from acid, pepsin, sharp contents) = thick layer
→ **diffusing agent**: neutralizes bicarb output from surface epith & acid from lumen Helicobacter pylori destroys mucosal layer → **ulcers**

SML INTESTINE DIGESTION, ABSORPTION

Pyloric sphincter = inpoint **~20ft (relaxed)**
Ileocaecal valve = endpoint
DUODENUM (retroperitoneal) = SHORTEST, parts 1-4, cradles head of PANCREAS ***!REACTION VESSEL!***
JEJUNUM (infraperitoneal) ***NUTRIENT ABSORBER***
rich arterial supply & lymphatic capillaries (lipid exit)
ILEUM (infraperitoneal) } malabsorption
(1) CHEM DIGESTION: hotpot of
→ **bicarb** from PANCREATIC DUCT, xtra-hepatic BILE DUCTS, DUODENAL MUCOSA
Serves to neutralize **chyme** & provide proper Δe for pH-sensitive enzymes
→ **digestive enzymes** mostly pancreatic → Ltd digestion
brush-border enzymes @ villi → **reduce to simple units**
→ **bile: FAT** digestion & absorption!!
!!!ALL FOOD CLASSES FULLY DIGESTED!!!
(2) MECH DIGESTION & PROPULSION
Sm muscle segments intestine → continual mixing of contents → **SLOW** mvt → suffic time to digest & absorb
If rapid pumping of chyme by pylorus, osmotic water loss from blood into intest lumen would be dangerously ↑↑
(3) ABSORPTION
Of breakdown products of CARBOHYDRATE, PROTEIN, FAT, NUCEIC ACIDS, VITAMINES, E-LYTES, WATER

& DIGESTION IN THE GIT

706_03, 04

LARGE INTESTINE

CAECUM starting point, ILEOCAECAL VALVE
APPENDIX twisted structure = perfect hideout for bacteria → APPENDICITIS
COLON (retroperitoneal)
ASCENDING→R colic flexure→TRANSVERSE→ L colic→ DESCENDING→enters pelvis→SIGMOID
TAENIAE COLI: "bands" to reduce colon length
CHEMICAL DIGESTION
Bacterial Flora digest undigested carbos and synthesize B VITAMINS & VIT K
(& STORAGE)
ABSORPTION
Remaining WATER, E-LYTES (esp NaCl), VITAMINS produced by bact
PROPULSION = main function
haustral contraxns (food→distend→contract→propel) SLOW (30mins), Mixing effect aids water absorption
= mvts over **sml areas**
Mass peristalsis (3-4x/day) POWERFUL
= mvts over **large areas**

RECTUM

3 CURVES (R→L→R)
= **RECTAL VALVES** → allows escape for flatus while trapping faeces

ANAL CANAL

2 SPHINCTERS
→ **internal anal sphincter** (invol, sm muscle)
→ **external anal sphincter** (voluntary, striated m): covered HAIRLESS SKIN
[INFANTS CANNOT CONTROL]

DEFAECATION REFLEX
(1) faeces stretch Rectum →depolarizes sensory fibres →sp cord
(2) parasym efferents cause: CONTRACTION rectal walls, RELAXATION internal sphincter
(3) voluntary signals to relax/close external sphincter

DEFAECATION: rectal mm contract to expel faeces
Valsava's maneuver helps: close epiglottis, contract diaphragm & abdo mm →↑↑ intrabdo P
DIARRHOEA: results from rushed passage thru lge intestine → not enough time for water absorption [irritation of colon by bacteria] **If prolonged → dehydration, e-lyte imbalance**
CONSTIPATION: stool TOO LONG in colon → XS water absorption [causes: ↓ fibre, ↓exercise, poor bowel habits, laxative abuse, emotional upset]

WHO OWNS THE BREAST? Breastfeeding CDT

breast feeding = the number one Dietary Guideline for Children in Australia.
= GOLD STANDARD; keep going as long as you can, at least 6 months

According to the World Health Organisation:

- Of 64% of the world's infants, 35% are breast fed for 4 months.
- **only 32 per cent of Australian babies were being breastfed exclusively at six months. :(**
- Still not good enough; some mothers only breastfeed while in hospital
- **The recommended duration is 6 months → THEN INTRODUCE SOLIDS, eg. CEREALS**
  (By the age of six months, iron and zinc stores are falling in infants who have been exclusively milk-fed (either breast or formula).
- At this time also, the development of feeding behaviour has progressed from sucking to biting (and then to chewing by 7 - 9 months).
- **Delayed introduction of solids may result in growth-faltering** and difficulties in being able to introduce a variety of foods into the diet.

LIST OF FOODS in order of appropriateness:

Rice cereal from 6 months; iron fortified (50 mg per 100g, but not heme form)
Mashed banana from 6 months
Minced chicken from 6-9 months
Finely chopped chicken from 12 months, if already chewing
Cows milk from 12months; approximately 4.0 g fat per 100 g

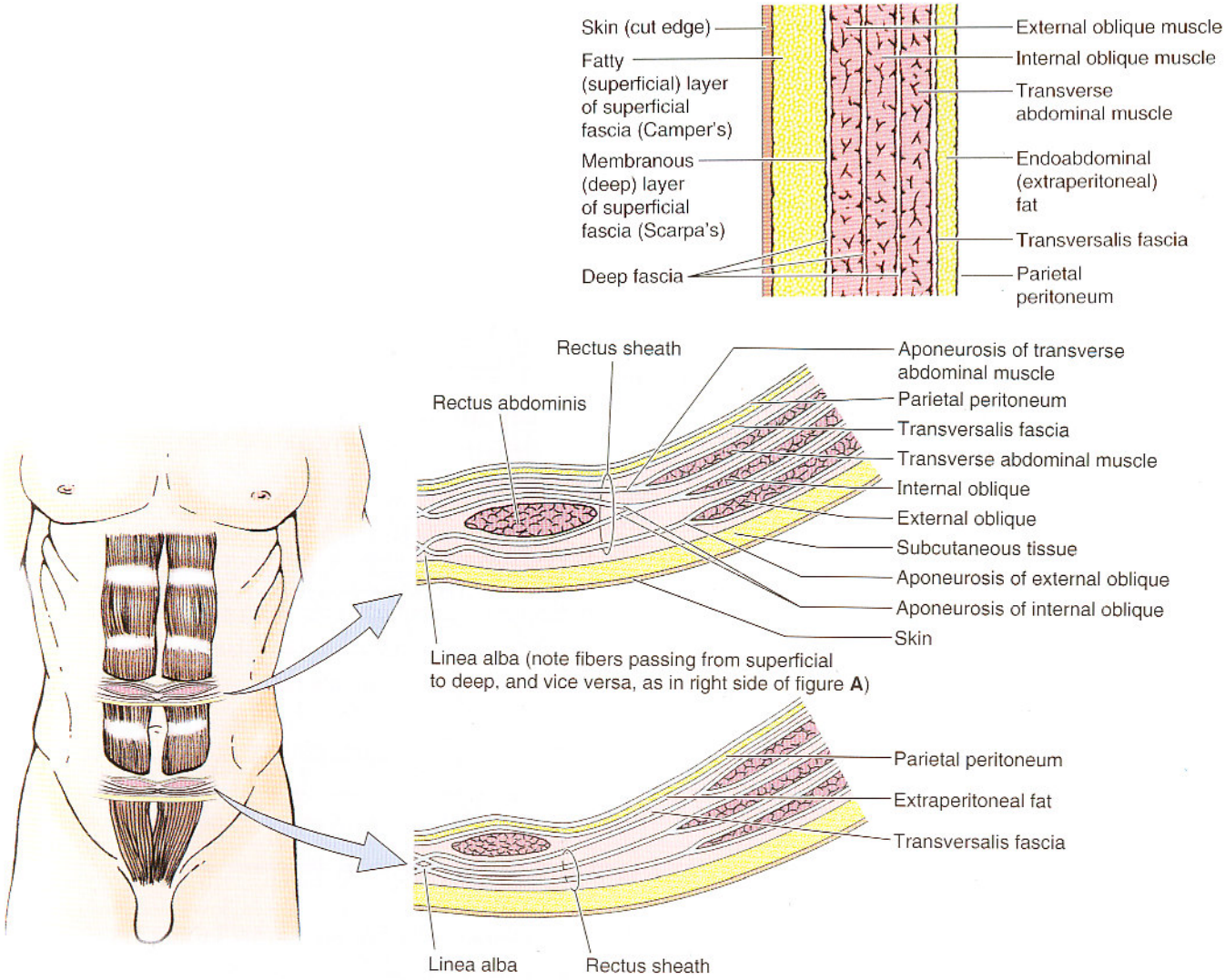
FACTOIDS:

- The energy needs per kg of body weight are greater in infancy than during adolescence.
- The greatest energy needs (adjusted for body weight) are during the first few months of life

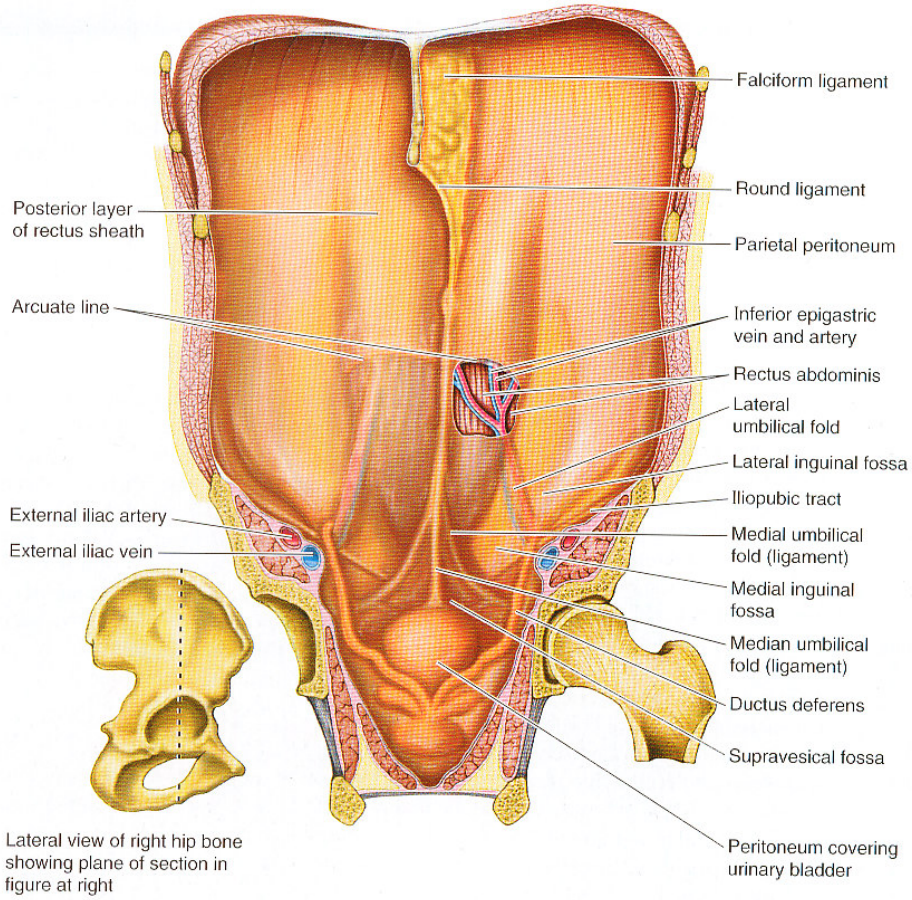
Mature human milk has slightly more fat content than cow's milk: human milk 4.2 g/100g and cow's milk 3.9 g fat/100g.

Breast milk energy content ranges from 271 - 285 kJ per 100g. (same as formula)

ANTERIOR ABDOMINAL WALL



Below: viewed posteriorly (male)



Superficial Anatomy of the Abdomen: Localisation