Basic Concepts in Nutrition Support for Critical Care Medicine

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Malnutrition is a common problem in hospitalized patients

- Up to 50% of hospital patients on admission can have clinical, hematological, biochemical or anthropometric evidence of protein energy malnutrition (PEM) caused by reduced intake of nutrient substrates. This worsens if untreated during stay in hospital and patients can continue to lose weight after discharge from hospital.
- >10% body weight loss is associated with increased morbidity, including:
  - Chest infection
  - Wound infection
  - Wound breakdown/delay healing
  - Development of pressure areas
  - Bacteremia/septicemia
  - Prolonged hospital stay
  - Increased incidence of readmission to hospital
  - Increased mortality

Malnutrition

- Kwashiorkor – Acute
  - Inadequate protein intake
  - Hypoalbuminemia
  - Fatty liver
- Marasmus – Chronic
  - Inadequate energy intake
  - Subcutaneous fat loss
  - Muscle wasting
- Marasmic-Kwashiorkor
  - Combination of Kwashiorkor and Marasmus

Patients requiring nutrition support

- Severely malnourished
  - Marked weight loss and muscle wasting
- Moderately malnourished
  - Reduced dietary intake in previous month
  - Nutritional parameters low/low-normal
- Normal/near-normal nutrition status
  - But at risk of developing PEM due to underlying disease or illness or trauma in absence of nutrition support

Nutritional care process

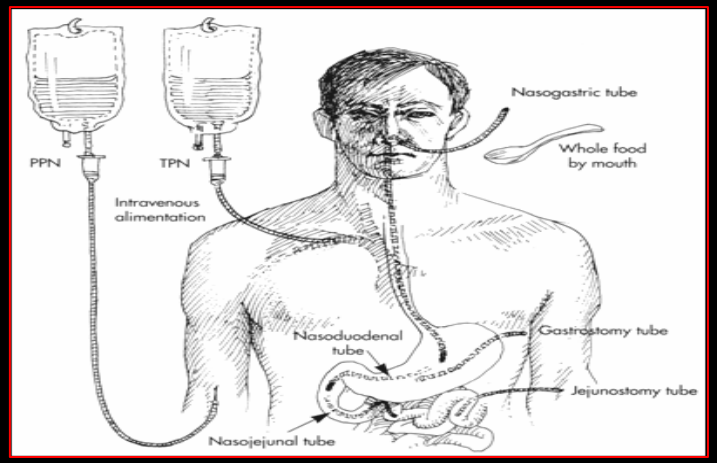
- The nutritional care process is a systemic approach to meeting the nutritional needs of a particular patient. It must incorporate the following actions:
  - Considering a patient's history, present state of nutrition, and disease status
  - Identifying individual nutritional needs
  - Planning a nutritional strategy that will meet both the general and particular needs of the patient
  - Providing food or more exotic nutritional components
  - Evaluating the results in order to provide corrective feedback
  - Facilitating discharge or transfer to another level of care

Assessment of energy requirement

- Energy requirement
  - Harris-Benedict (HB) equation
    - Men
      - EER (Kcal) = 66.5+13.75W+5.00H-6.77A
    - Women
      - EER (Kcal) = 655.1+9.56W+1.85H-4.67A
  - Activity factors
  - Stress factors
- Adjust body weight
  - ideal BW + 0.25 x (observed BW-ideal BW)
- Indirect calorimetry

Choices of route for nutritional support

- Enteral (EN)
- Parenteral (PN)
- PPN (both)

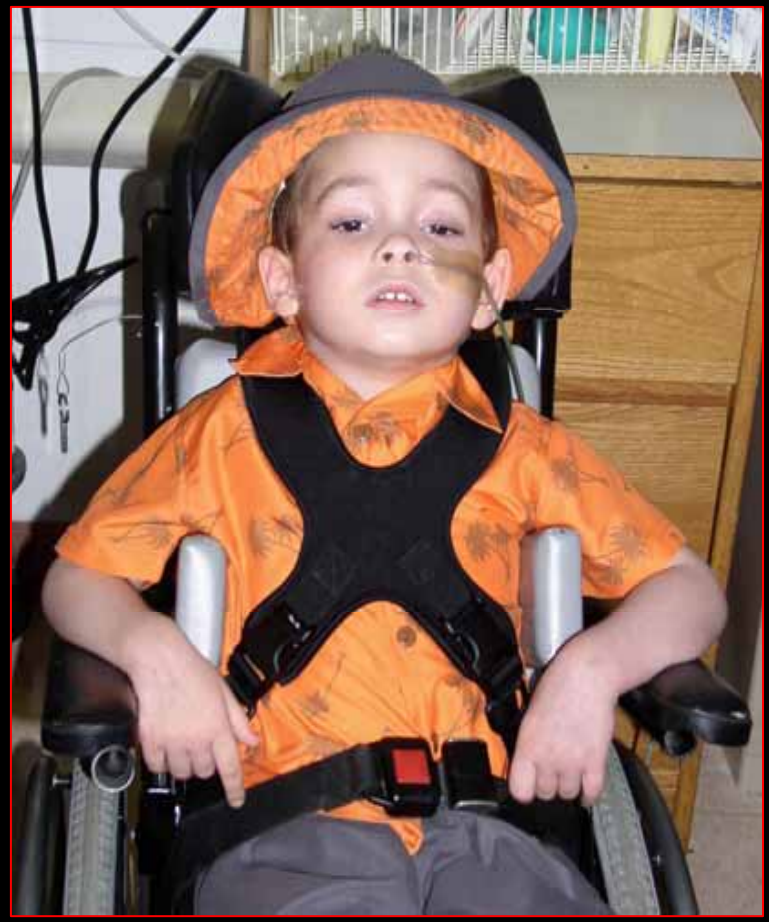


Nutritional screening criteria

- Diagnosis and past medical history associated with increased nutritional risk
- Diet information
- Physical assessment
- Anthropometric data
- Laboratory data

Factors to consider for EN

- **Access route**
- **Enteral formula**
- **Delivery technique**
- **Monitoring**
- **Complications**
- **Patient preference**

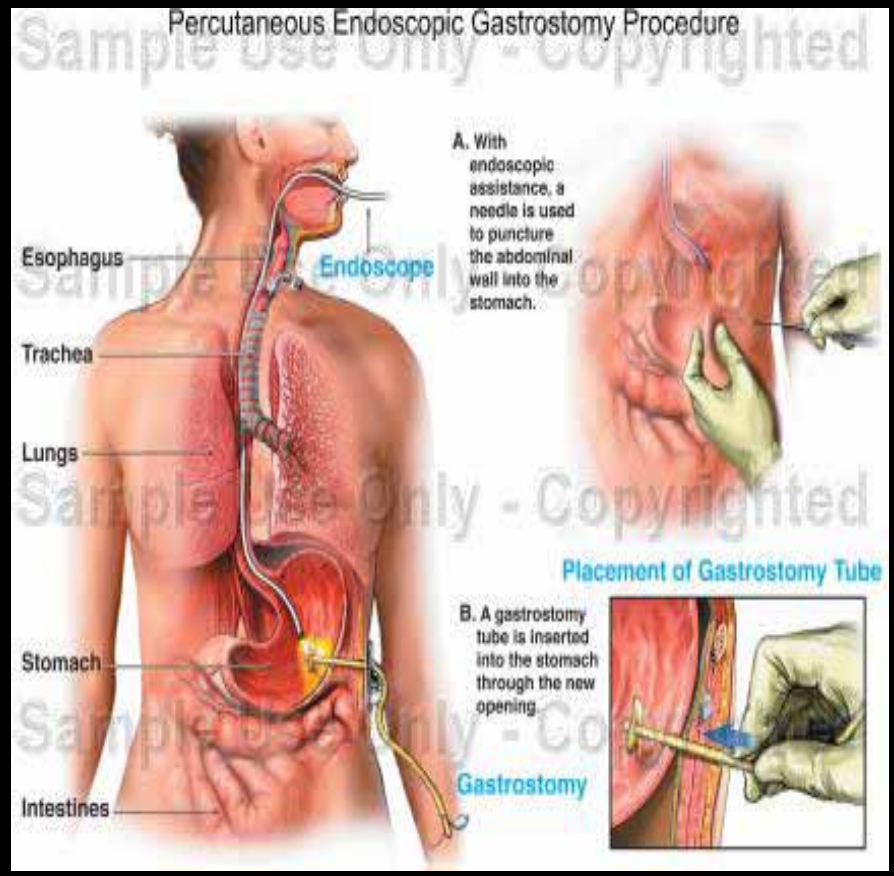


Access routes

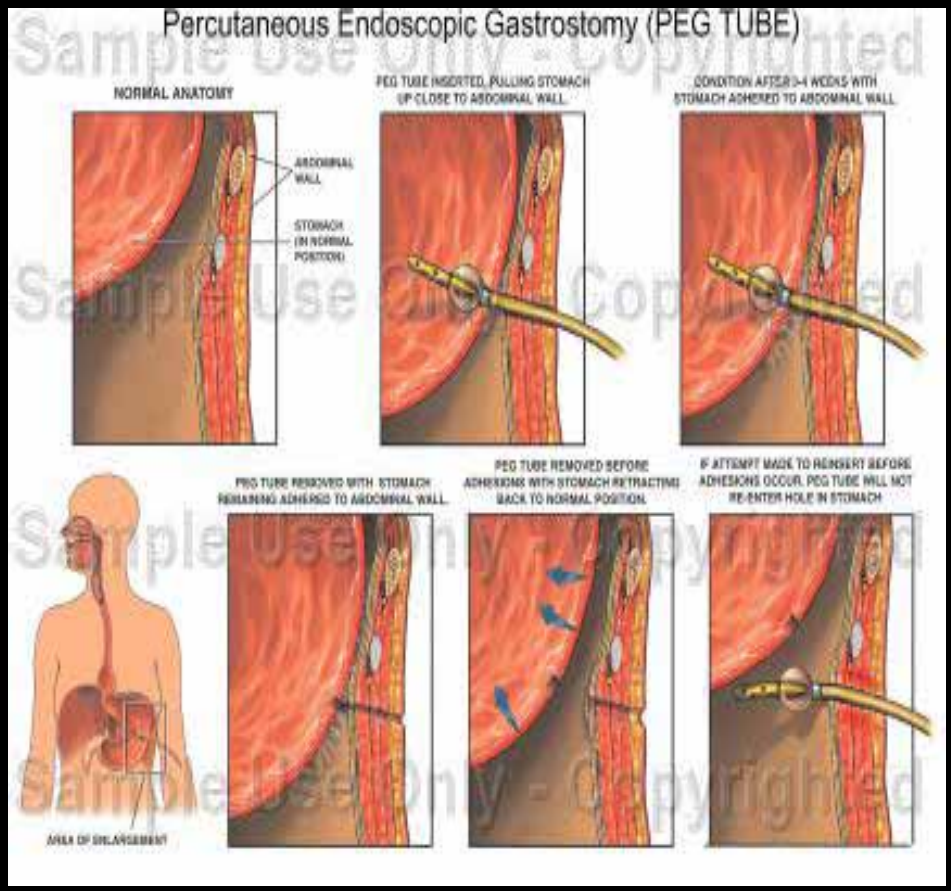
- Short-term feeding (<2 weeks)
  - Fine-bore nasoenteral tubes (FBT)
    - Nasogastric
    - Nasoduodenal
    - Nasojejunal
  - Dual function (gastric aspiration/jejunal feeding tubes)
- Long-term feeding (>2-4 weeks)
  - Gastrostomy
    - Surgical
    - Percutaneous endoscopic (PEG)
    - Fluoroscoic percutaneous
    - Laparoscopic
    - Button ostomies (for cosmesis)
  - Duodenostomy
    - Percutaneous endoscopic
  - Jejuonostomy
    - Surgical
    - Percutaneous endoscopic (PEJ)
    - Jejunal tubes through PEG
    - Needle catherter (NCJ)
    - Cuffed tube

Percutaneous gastrostomy - 1

- Surgically placed gastrostomies are rarely used now
  - unless concurrently at laparotomy
- Percutaneous placement is the technique of choice
  - Endoscoic/radiologic
  - Usually under local anesthesia
- Prior to placement ensure site is appropriae
- Site of placement should be away from skin folds
- Contraindiations include ascites and gross obesity



Percutaneous gastrostomy - 2



- Compliations include
  - Local wound infection
  - Granulating tissue
  - Necrotizing fascitis
  - Pneumoperitoneum
  - Intra-abdominal abscess
  - Tube displacement
  - Bleeding
  - Bowel perforation
- Complication rate should be no more than 1-2%
- If displaced or potentially misplaced
  - confirm placement radiologically

Delivery techniques

- Constant infusion
  - Should be used whenever possible with a pump
- Intermittent infusions
- Bolus feeding
  - 100-400 ml over 10-30 minutes several times daily

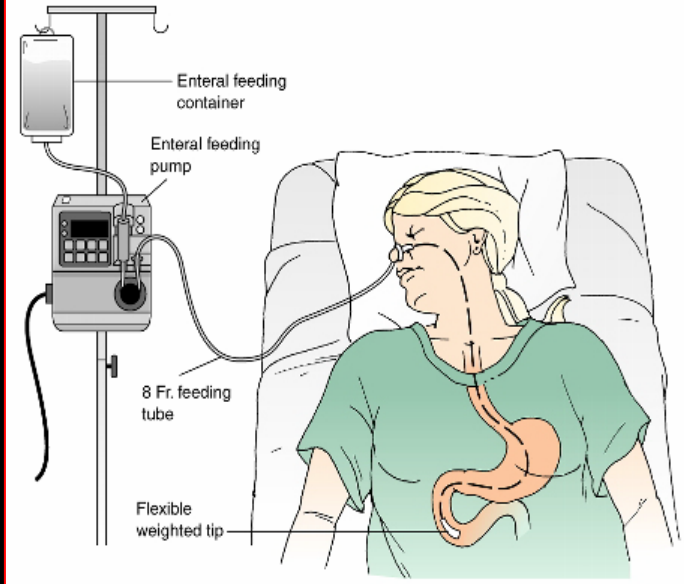


Figure 36-6 Nasoenteric tube feeding by continuous controlled pump.

Choices of EN formula

- Blenderized
- Polymeric
- Monomeric
- Disease-specific

Monitoring

- Starter regimens
  - Full strength if possible
- Recumbency/Semi-recumbency
  - Prevent regurgitation/aspiration
- Gastric residual volumes (GRV)
  - Reduce infusion rate if GRV > 200 ml (4 hours after last feeding)
- Prevent contamination
  - Closed sterile diet containers
  - Diet reservoirs

Nutritional assessment

- General history
- Dietary assessment
  - Weight dietary record (actual)
  - Dietary history (recall)
  - 24-hour recall
- Examination
- Investigations

Contraindications to enteral nutrition

- Main contraindications of EN
  - An inability to meet nutrient needs via the enteral route alone
  - Complex fluid balance problems
  - Intestinal obstruction
  - Paralytic ileus
- If there is partial gastrointestinal function, PN should be used and supplemented with EN

Influences of NPO

- Inhibition of saliva and digestive tract secretions
- Inhibition of GI motility and splanchnic circulation
- Increased virulence of potentially pathogenic microorganisms in subflora
- Reduction and inhibition of the protective flora
- Atrophy of the mucosa of the small intestine and colon
- Increased microbial translocation

Indications for Parenteral Nutrition (PN)

- All nutrition requirements can be given solely via the parenteral route
- Aucte/chronic, temporary/permanent intestinal failure or whose nutrition needs cannot be met by enteral route
- Common patients group include
  - Short bowel syndrome
  - Radiation enteritis
  - Acute pancreatitis
  - Prolonged ileus
  - High intestinal fistulae
  - Severe mucositis
  - Partial gastrointestinal function

Peripheral PN

- PPN should be considered for any patient with a non-functioning or partial functioning gastrointestinal tract requiring feeding for <10-14 days.
- Peripheral vein thrombophlebitis (PVT) is minimized by avoiding excess glucose levels, use of lipid emulsions, use of All-in-One (AIO) admixture.
- IV cannulas must be removed at first sign of PVT
  - Local erythema
  - Swelling
  - Hardness
  - Pain
  - extravasation

Central venous access

- Use if PN is anticipated for > 10-14 days
- Allow rapid dilution of the hyperosmolar nutrient admixtures
- Access with CVCs by open procedures or blind percutaneous puncture
- CVC tip should lie in the superior vena cava (SVC) or the right atrium
- Routes include
  - Subclavian vein
  - External/Internal jugular vein
  - Cephalic vein (PICC, peripheral inserted central catheter)
  - Femoral vein (PICC)

Infusion techniques

- Use volumetric infusion pumps to deliver accurate volumes and avoid potential complications such as accidental rapid administration of large volumes of PN solutions
- 24 hour continuous infusion
  - Most widely used in hospital practice
  - Simple to administer
  - Permit safe delivery of large volumes of PN solutions
- Cyclic (Intermittent) PN
  - Practiced by most home PN patients to increase the “infusion-free” part of the day
  - Can be used in hospital for mobile patients
  - Mimics the physiological profile of the normal “meal-eater”
  - Permits disconnection during the day allowing normal activities
  - May reduce fat and glycogen deposition

TPN related complications

- Insertion related
  - Air embolism
  - Arterial puncture
  - Arrythmias
  - Chylothorax
  - Hemothorax
  - Pneumothorax
  - Hemopericardium
  - Nerve injury
  - Catheter malposition
- Catheter related
- Metabolic
- Hepatobiliary dysfunction

TPN related complications

- Insertion related
- Catheter related
  - Displacement of catheter
  - CVC luminal occlusion
  - CVC fracture
  - Erosion of CVC tip through vessel wall
  - CVC thrombosis
  - CVC related infection/sepsis
- Metabolic
- Hepatobiliary dysfunction

TPN related complications

- Insertion related
- Catheter related
- Metabolic
  - Hyper/hypoglycemia
  - Hypophosphatemia
  - Hypercalcemia
  - Hyper/hyponatremia
  - Hyper/hypokalemia
  - Hyperosmolar diuresis
- Hepatobiliary dysfunction

TPN related complications

- Insertion related
- Catheter related
- Metabolic
- Hepatobiliary dysfunction
  - Elevated of hepatic enzymes (AST, ALT)
  - Jaundice
  - Biliary sludging
  - Fatty liver
  - Gallstones
  - Hepatic failure

Monitoring Nutrition Support

- Goal
  - Be aware of, anticipate and minimize feeding/infusion-related complications
  - Confirm adequacy of nutrition support and response to support
  - Baseline measurement recorded to see changes on treatment progression

Monitoring Nutrition Support

- Methods
  - Record baseline data and changes in hematological, biochemical, and anthropometric (MAC, MAMC, TSF) parameters.
  - In patients on long-term EN or HPN serum levels of trace elements and vitamins will additionally be required as appears clinically appropriate
  - In the first week measure daily
    - Urea and electrolytes
    - Phosphate
    - Glucose
    - Fluid I/O
    - Weight
    - Nitrogen balance

Monitoring Nutrition Support

- Once stable patients may be assessed twice weekly
  - Full blood count
  - Coagulation screen
  - Urea and electrolytes
  - Calcium
- The following may then be measured weekly
  - Weight
  - Liver function
  - Nitrogen balance
  - MAC, MAMC, TSF

Monitoring Nutrition Support

Serum protein	Normal value	Half-life	Clinical significance
Albumin	3.5-5.0 g/dL < 2.1, severe	21 days	Useful in long-term assessment Reliable indicator of morbidity/mortality
Transferrin	200-400 mg/dL <100, ssevere	8-10 days	Influenced by iron status Increased level with pregnancy, iron therapy
Prealbumin	10-40 mg/dL <5, severe	2-3 days	Short term nutritional index Better index of visceral protein
Retino-binding protein (RBP)	2.7-7.6 mg/dL	12 hours	Reflects actual changes in protein malnutrition Increased levels in chronic renal failure

Dietary requirement

- Protein (20-25%)
  - 0.75 g/kg/day (in stress, 1.5-2.0 g/kg/day)
  - Provide 4 kcal/g
- Sugar (45-50%)
  - Minimal requirement is 100 g/day (380 kcal) to meet the obligate glucose-using tissues
  - Maximal rate of glucose oxidation is 4-5 mg/kg/min (7.2 g/kg/day)
  - Provide 3.4 kcal/kg (hydrated glucose); 4 kcal/kg (oral form)
- Fat (30%)
  - 1 g/kg/day (<2.5g/kg/day) lipid emulsion
  - provide 9.1 kcal/g (enteral form); 10 kcal/g (emulsion, fat+glycerol+emulsifiers)

Carbohydrate

- Essential fuels used by glycolytic tissues; normally the major or sole energy sources for the CNS and peripheral nerves, RBCs, and some phagocytes.
- During prolonged starvation, the glucose requirement of the brain decreases as adaptation to ketone oxidation occurs.
- Maintain hepatic glycogen stores, which may protect hepatocytes during hypoxia or exposure to toxins.

Lipid

- The most concentrated forms of energy.
- Stabilize, support, and protect vital structures.
- Complex with fat-soluble molecules like some vitamins are used as structural components in biological membranes.

Protein

- Major structural component of the body.
- Some are essential (histidine, isoleucine, leucine, valine, methionine, cysteine, phenylalanine, tyrosine, threonine, tryptophan, and lysine) because they cannot be synthesized by the body.
- Act as peptide hormones, enzymes, and antibodies.
- May join with carbohydrates to form glycoproteins, to serve as plasma proteins and immune globulins, and components of connective tissue cells membranes and mucous secretions.

Nutrition in renal failure

- Provide protein in the range of 0.6-0.8 g/kg/day (before dialysis), and 1.2-1.5 g/kg/day (during dialysis)
  - 1 g/hour is removed by PD or HD
- Use essential amino acids, preferably
- Use lipid emulsion to reduce fluid intake
- Provide water soluble vitamins (B, C), and avoid large amount of fat soluble vitamins (A, D, E)

Nutrition in hepatic failure

- Decrease protein intake in hepatic failure and encephalopathy (0.5-0.7 g/kg/day)
- These patients have altered plasma amino acids profiles with increased concentrations of aromatic acids (phenylalanine, tyrosine, and tryptophan) and methionine, and decreased branched-chain amino acids (valine, leucine, and isoleucine)
- Decrease sodium intake (excrete nearly sodium-free urine), avoid excess trace elements (copper and magnesium are excreted by biliary system)

Nutrition in pulmonary failure

- Prevent excess CO_2 production
  - prevent overfeed
  - increase ratio of fat calories to carbohydrate calories
- Avoid refeeding syndrome
- Use enteral route if at all possible

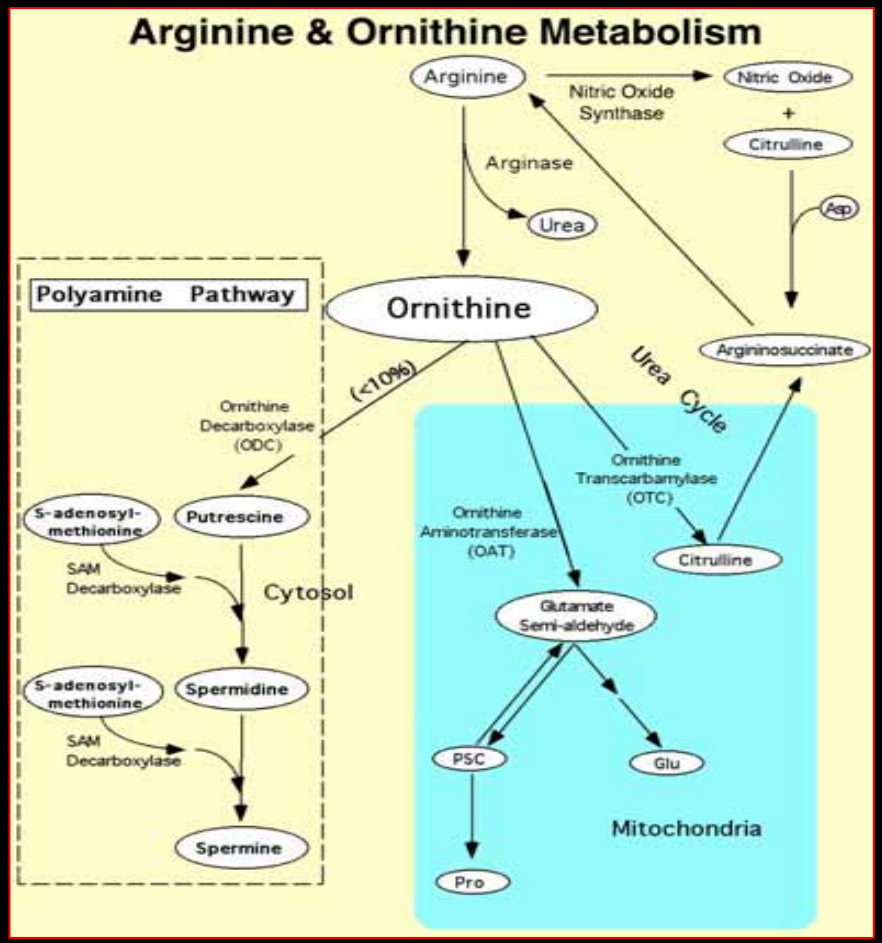
Re-feeding syndrome

- Metabolic and physiologic consequences associated with the depletion, repletion, and compartmental shifts of phosphorus, potassium, and magnesium as well as alterations in glucose metabolism and fluid status.
- Accompanying clincal swquellae can be significant if the intracellular shifts of electrolytes are severe; indeed, death may result from overzealous refeeding.
- Effects
  - Hypophosphatemia
  - Hypokalemia
  - Hypomagnesemia
  - Glucose and Fluid intolerance

Special nutrients

- Arginine
  - Promote proliferating T cells after mitogen or cytokines stimulation
- Glutamine
  - Becomes “conditionally” essential amino acid during sepsis or stress
- Omega-3 fatty acid
  - The end products are neither proinflammatory nor immunosuppressive
- Branched-chain amino acids (BCAA)
  - Primary energy source for muscle protein

Arginine - overview



- Arginine (or L-Arginine) is an amino acid involved in numerous areas of human biochemistry, including ammonia detoxification, hormone secretion, and the immune system. Arginine is also well known as a precursor to nitric oxide, a key component of endothelial-derived relaxing factor. The endothelium is the lining inside blood vessels and arginine supplements help make more nitric oxide. Therefore, arginine, by making more nitric oxide helps to relax and dilate blood vessels.
- L-arginine augments T-cell response and may be helpful in hastening surgical recovery, as well as in cancer and AIDS.

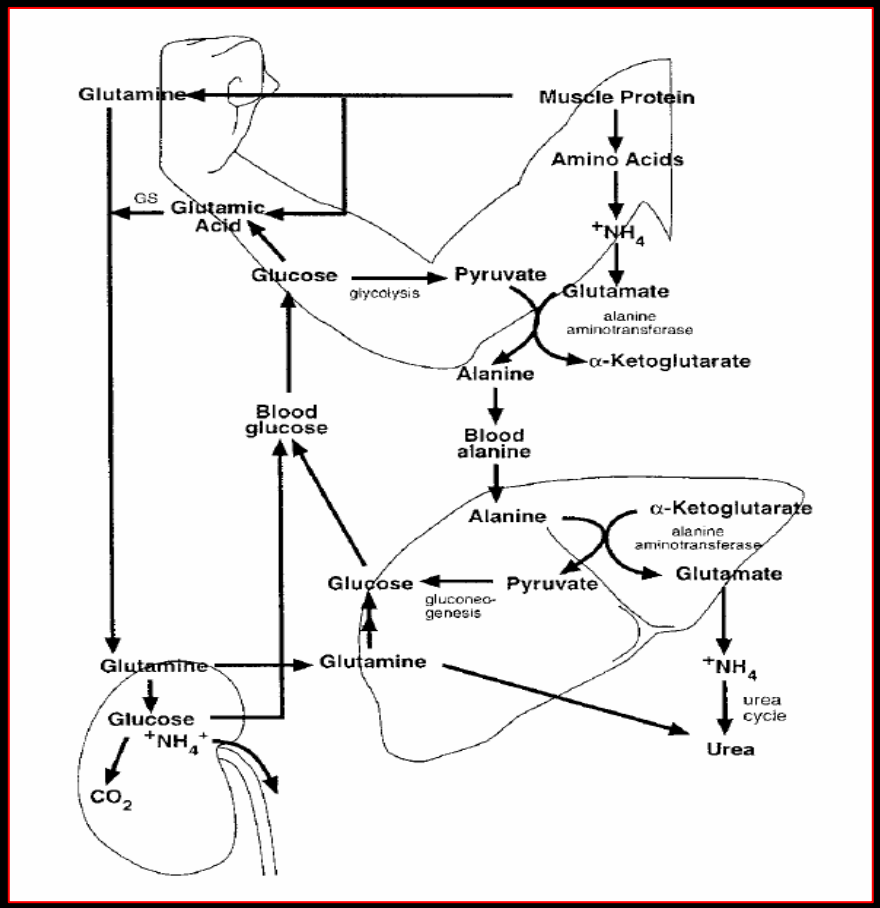
Glutamine - overview

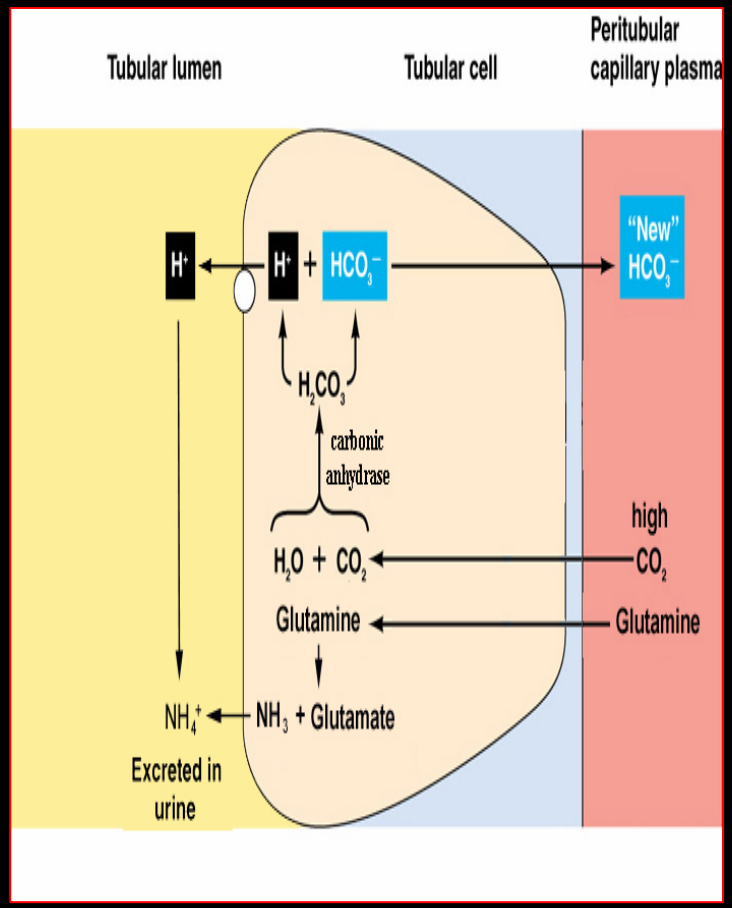
Functions of glutamine

	Substrate of protein synthesis (codons: CAA, CAG)
	Anabolic/trophic substance for muscle; intestine ("competence factor")
*	Controls acid-base balance (renal ammoniagenesis)
*	Substrate for hepatic ureagenesis
*	Substrate for hepatic/renal gluconeogenesis
*	Fuel for intestinal enterocytes
	Fuel and nucleic acid precursor and important for generation of cytotoxic products in immunocompetent cells
	Ammonia scavenger
	Substrate for citrulline and arginine synthesis
	Nitrogen donor (nucleotides, amino sugars, coenzymes)
	Nitrogen transport (one third circulating N) (muscle; lung)
	Precursor of δ-aminobutyric acid (via glutamate)
	Shuttle for glutamate (central nervous system)
	Preferential substrate for glutathione production?
	Osmotic signaling mechanism in regulation of protein synthesis?
	Stimulates glycogen synthesis
	L-Arginine-NO-metabolism

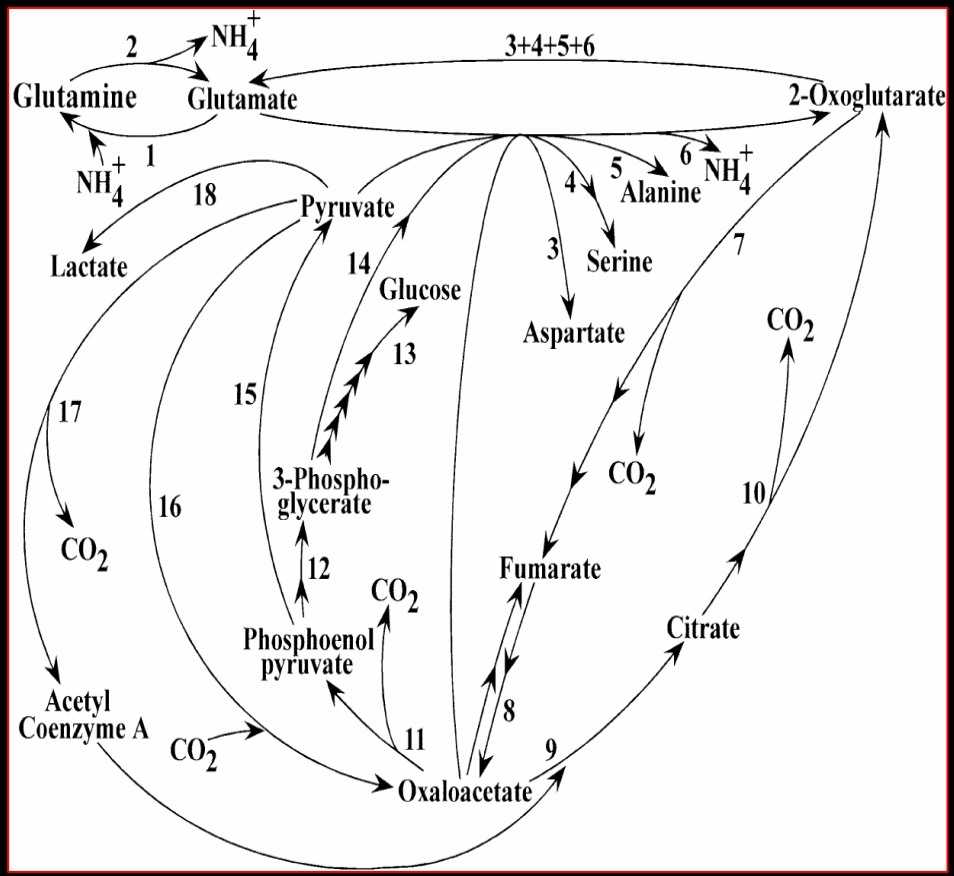
- L-glutamine is the most prevalent amino acid in the bloodstream and because human cells readily synthesize it, is usually considered a non-essential amino acid. It is found in high concentration in skeletal muscle, lung, liver, brain, and stomach tissue. Skeletal muscle contains the greatest intracellular concentration of glutamine, comprising up to 60% of total body glutamine stores, and is considered the primary storage depot and exporter of glutamine to other tissues .
- Under certain pathological circumstances, including injury, sepsis, prolonged stress, starvation, the body's tissues need more glutamine than the amount supplied by diet and biosynthesis.

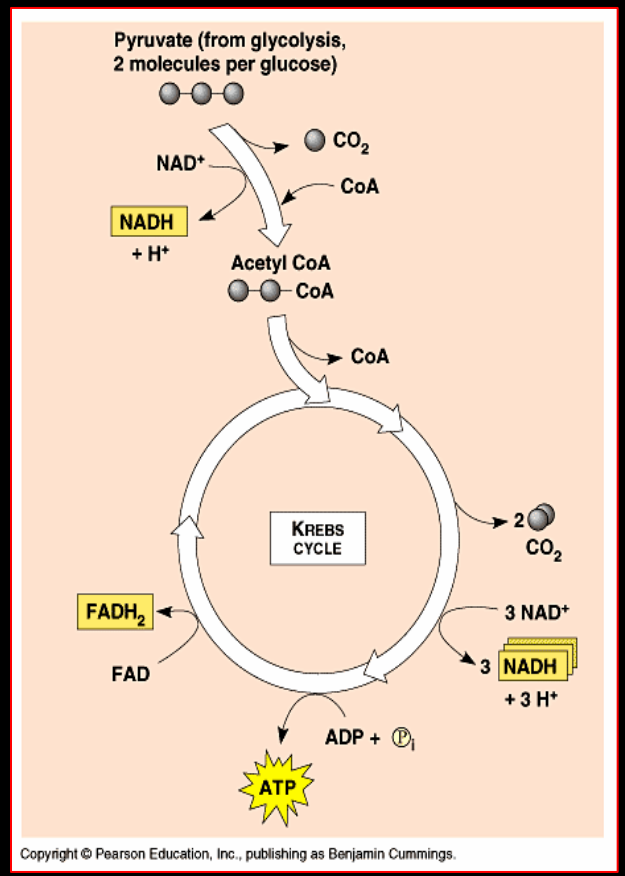
Glutamine – Gluconeogenesis and Ammonium Ion



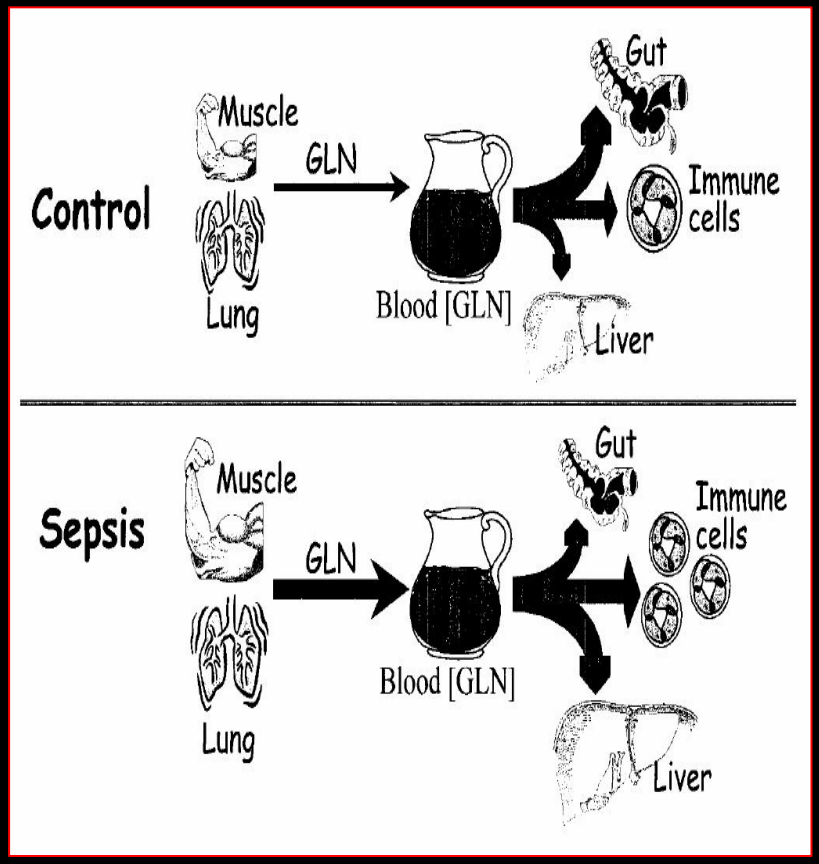


Glutamine and Krebs Cycle





Glutamine - immunomodulation in sepsis



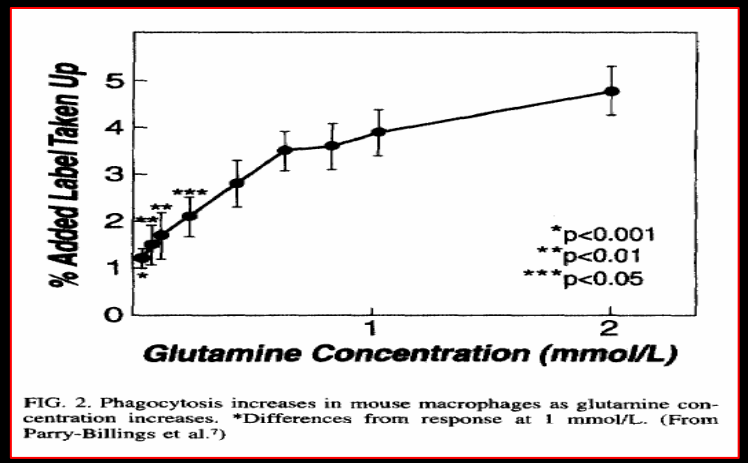


FIG. 2. Phagocytosis increases in mouse macrophages as glutamine concentration increases. *Differences from response at 1 mmol/L. (From Parry-Billings et al.[7])

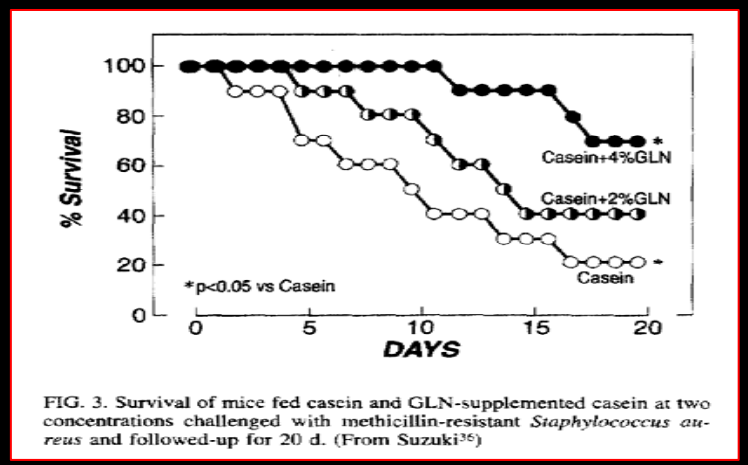
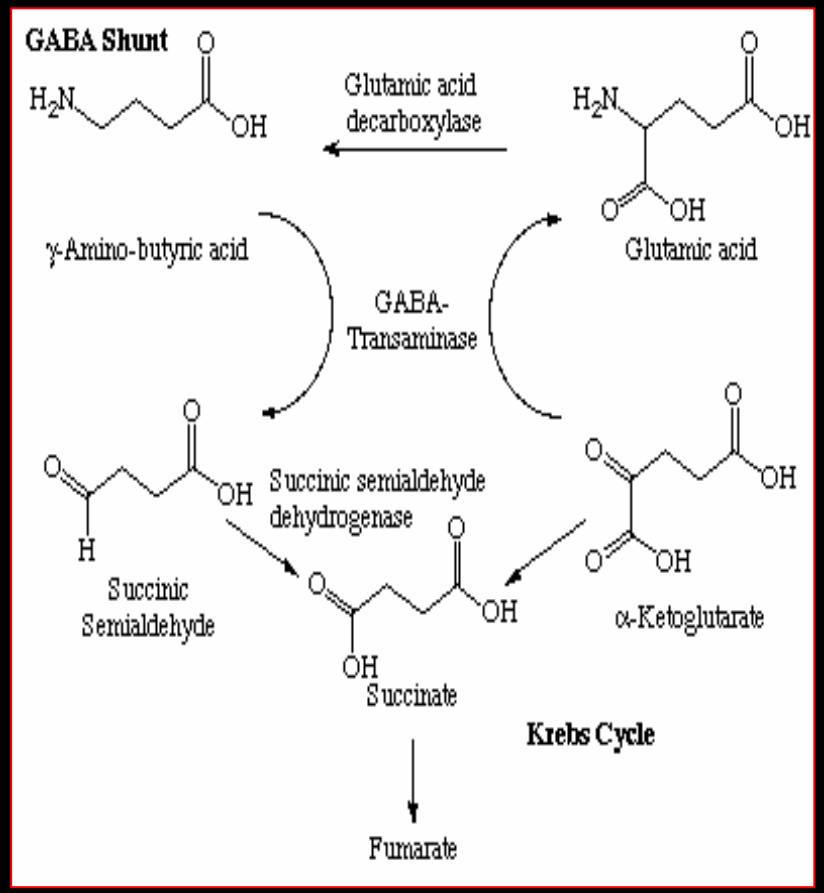


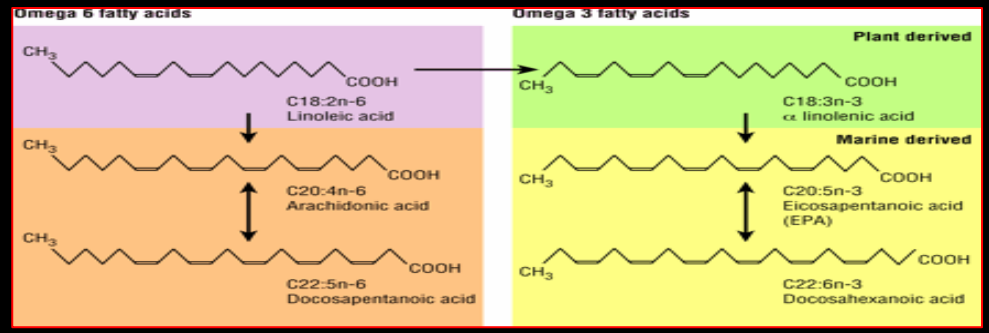
FIG. 3. Survival of mice fed casein and GLN-supplemented casein at two concentrations challenged with methicillin-resistant *Staphylococcus aureus* and followed-up for 20 d. (From Suzuki[35])

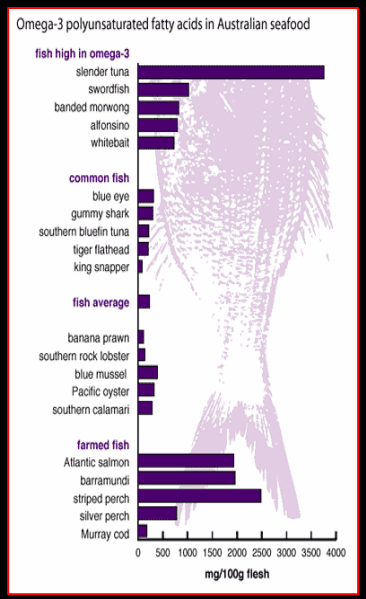
Glutamine in Cancer Treatment

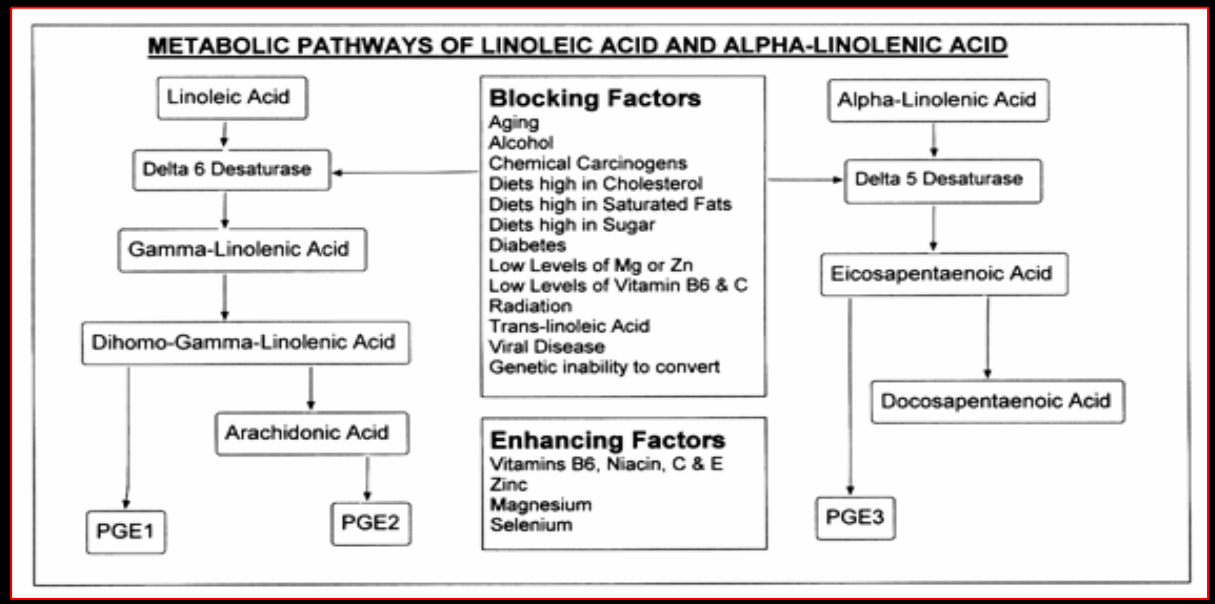
- The role of glutamine as an adjuvant oncology treatment is very promising. The simple process of glutamine supplementation may help patients experience fewer side effects from therapy, including GI damage, mucositis, stomatitis, and neuromuscular pain.
- Glutamine passes freely across the blood-brain barrier. Once in the brain, it's converted to glutamic acid and increases the concentration of GABA (gamma-aminobutyric acid). Both glutamic acid and GABA are essential for proper mental function.



ω-3 fatty acids *vs.* ω-6 fatty acids








Thanks for your attention!