PROTEIN - CALORIE MALNUTRITION & STARVATION

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MALNUTRITION

WHO definition

"The cellular imbalance between the supply of nutrients & energy & the body's demand for them

to ensure growth, maintenance & specific functions."

PROTEIN-CALORIE MALNUTRITION

- Primary nutritional problem i
- First National Nutritional Disorder.

- The inadequate consumption of protein & energy as a result of primary dietary deficiency or conditioned deficiency may cause loss of body mass & adipose tissue.

- Frequent due to socio-economic factors limiting the quality & quantity of dietary intake.

- Prevalent in developing countries of Africa, Asia & South America.

- Markedly seen in infants & children.

ETIOLOGY

- Social & economic factors.
- Biological factors.
- Environmental factors.
- Role of free radicals.
- Age of host.

- Common causes among the **social, economic, biological & environmental** factors are :

- Lack of breast feeding & giving diluted formula.
- Improper complementary feeding.
- Over crowding in family.
- Ignorance.
- Illiteracy.
- Lack of health education.
- Poverty.
- Infection.

- **Role of free radicals** : Damage liver cell giving rise to Kwashiorkor.

- **Age of host** :

- Frequent in infants & young children whose rapid growth increases nutritional requirements.

- PEM in pregnant & lactating women can affect the growth, nutritional status & survival rates of their foetus, newborns & infants.

- Elderly can also suffer from PEM due to alteration of GI system.

CLINICAL FEATURES

Five forms of PEM are :

1. Kwashiorkor
2. Marasmus
3. Marasmic – Kwashiorkor
4. Nutritional dwarfing
5. Underweight child

KWASHIORKOR

- Ga language of Ghana - **" The sickness of the weaning".**
- Also called wet-protein energy malnutrition.
- Form of PEM characterized by protein deficiency.
- Refers to **an insufficient protein consumption** but with sufficient calorie intake.
- Usually appear in the age of 12 months when breast feeding is discontinued.
- May also develop at any time during a child formative years.

- It causes **fluid retention** (edema); dry ,peeling skin & hair discolouration.

- Can have long term impact on a child's physical & mental development & in severe cases may lead to death.

- Treated by adding food energy & protein to the diet.

SYMPTOMS

- ✓ Change in skin pigment.
- ✓ Diarrhea.
- ✓ Decreased muscle mass.
- ✓ Swelling(edema).
- ✓ Fatigue.
- ✓ Hair changes
- ✓ Lethargy.
- ✓ Increased & more severe infection due to damaged immune system.
- ✓ Failure to gain weight & grow.
- ✓ Shock(late stage).

Marasmus

- Greek word Marasmos- **"withering or wasting"**
- Severe protein calorie malnutrition characterized by **energy deficiency and emaciation**
- Causes stunted growth and wasting of muscles and tissue
- Develop between the age of 6 months & 1 years in children who weaned from breast feeding/who suffer from weakening conditions like chronic diarrhea.

SYMPTOMS

- ✓ Severe growth retardation.
- ✓ Loss of subcutaneous fat.
- ✓ Severe muscle wasting.
- ✓ Child looks thin and limbs appear as skin and bone.
- ✓ Wrinkled skin.
- ✓ Bony prominence.
- ✓ Frequent watery diarrhea & acid stools.
- ✓ Temperature is abnormal.
- ✓ Edema absent.

DIFFERENCE IN CLINICAL FEATURES BETWEEN MARASMUS AND KWASHIORKOR

CLINICAL FEATURES	MARASMUS	KWASHIORKOR
-MUSCLE WASTING	Obvious	Sometimes hidden by edema and fat
-FAT WASTING	Severe loss of subcutaneous fat	Fat often retained but not firm
-EDEMA	None	Present in lower legs, and usually in face and lower arms
-WEIGHT FOR HEIGHT	Very low	May be masked by edema
-MENTAL CHANGES	Sometimes quite and apathetic	Irritable, moaning, apathetic

CLINICAL FEATURES	MARASMUS	KWASHIORKOR
-APPETITE	Usually good	Poor
-DIARRHOEA	Often	Often
-SKIN CHANGES	Usually none	Diffuse pigmentation, sometimes 'flaky paint dermatitis'
-HAIR CHANGES	Seldom	Sparse, silky, easily pulled out
-HEPATIC ENLARGEMENT	None	Sometimes due to accumulation of fat

Protein-energy (calorie) malnutrition
(moderate energy and protein deficit)

Moderate energy deficit with severe protein deficit, especially in light of increased needs due to infections

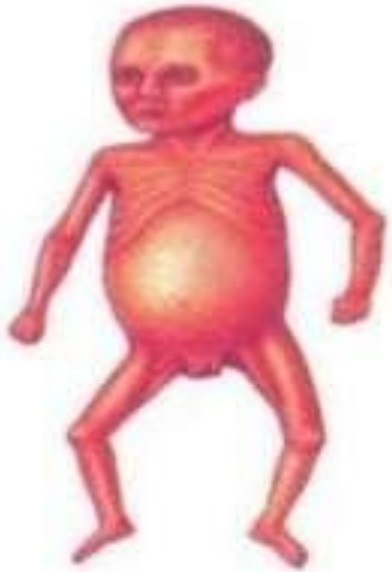
Growth, infections, and trauma put great nutritional demands on the body

Severe energy and protein deficit

Kwashiorkor
(edema with maintenance of some subcutaneous fat tissue)

Marasmus
(skin and bones appearance with little or no subcutaneous fat tissue)

Kwashiorkor



Marasmus

MARASMIC-KWASHIORKOR

Severely malnutritioned child with **features of both Marasmu & Kwashiorkor.**

- Features of Kwashiorkor : severe edema of feet &legs &alsc hands, lower arms, abdomen & face.

Pale skin &hair.

- Features of Marasmus:

Wasting of muscles of the upper arms, shoulders & chest.

NUTRITIONAL DWARFING OR STUNTING

- Some children adopt to prolonged insufficiency of food-energy & protein by a marked retardation of growth.

- Weight & height are reduced in same proportion ,so they appear superficially normal.

UNDERWEIGHT CHILD

- Children with subclinical PEM
  can be detected by their weight
  for height/age which are significantly low.

- Reduced plasma albumin.

- Risk for respiratory & gastric infections.

BIOCHEMICAL & METABOLIC CHANGES

- Significant changes in Kwashiorkor – hypoalbuminemia, hypoproteinemia & hypoglycemia.
- Plasma cortisol & growth hormone levels are high.
- Percentage of body water & extracellular water is increased.
- Electrolytes especially K & Mg are depleted.

- Levels of some enzymes are decreased.
- Circulating lipid are low.
- Ketonuria can occur & may cause decrease in urinary excretion of urea.
- In both Kwashiorkor & Marasmus , iron deficiency anemia & metabolic acidosis are present.

TREATMENT

Treatment strategy divided into 3 stages:

- Resolving life threatening conditions.
  - Hospital management
- Resolving nutritional status.
  -Dietary management
- Ensuring nutritional rehabilitation.

1. **Hospital Management:**

   Following conditions should be corrected – hypothermia, hypoglycemia, infections, dehydration, electrolyte imbalance, anemia & other vitamin & mineral deficiency.

2. **Dietary Management:**

   Diet from staple foods– inexpensive, easily digestible, evenly distributed & increased number of feedings.

3. **Rehabilitation:**

   Nutritional training for mothers- feeding their children back to health & use local food.

PREVENTION

- Promotion of breast feeding.
- Development of low cost weaning.
- Nutritional education.
- Family planning.
- Immunization.
- Early diagnosis & treatment.

STARVATION

STARVATION

- State of overall deprivation of nutrients.

  - Causes:

i. deliberate fasting-religious or political.

ii. famine conditions in a country or community.

iii. chronic wasting diseases(infection, inflammatory conditions, liver disease),cancer etc.

- Starved individuals has lax, dry skin, wasted muscles and atrophy of internal organs.

- Consequences of fasting:

  Plasma level of glucose, amino acid, triglycerides falls.

- Overview of fasting;

  Insulin secretion falls while glucagon is activated.

METABOLIC CHANGES IN STARVATION

Early, intermediate, advance stages of starvation.

- Early stage(2 days)
- Glycogenolysis & gluconeogenesis are important source of blood glucose.
- Energy from alternate source (beta oxdn FA, KB)

- Intermediate stage (24 days)
- Glycogen stores mostly depleted not serves as a source of blood glucose
- FA, KB supplied to heart, kidney, muscle.

- Advanced stage (>24 days)

• KB supplies to heart, kidney , muscle is decreased, limited to
brain only.

• Heart, kidney , muscle, depend on FA as main source.

• Gluconeogenesis will be enhanced due to increased activity
of enzymes pyruvate carboxylase , fructose-1,6-bis phosphatase.

ORGANS INVOLVED

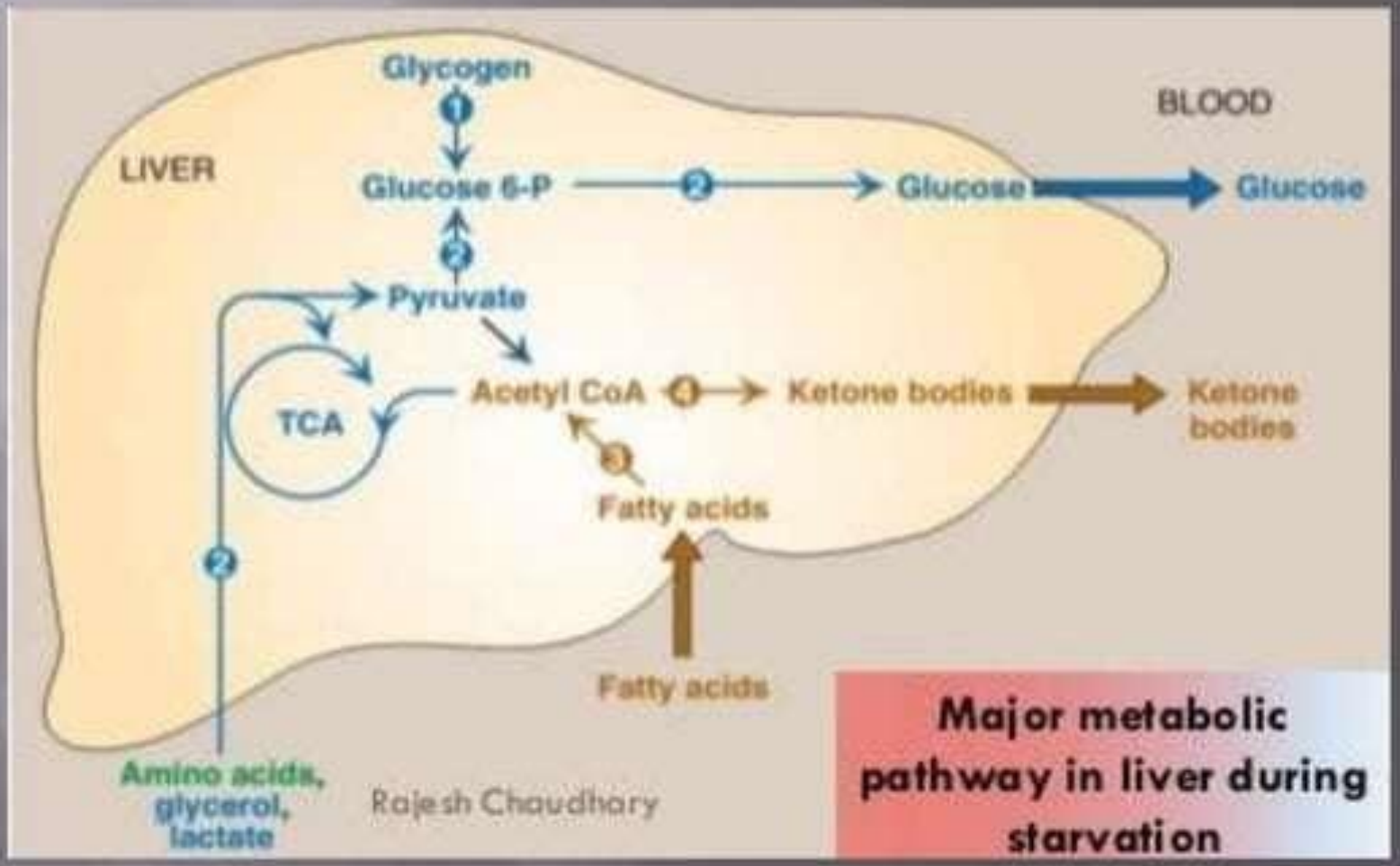
- Liver in starvation.
- Adipose tissue in starvation.
- Resting muscle in starvation.
- Brain in starvation.
- Kidney in long term starvation.

Liver in starvation

1. Carbohydrate metabolism
   - Increased glycogen degradation (glycogenolysis).
   - Increased gluconeogenesis.

2. Fat metabolism
   -Increased fatty acid oxidation
   -Increased synthesis of ketone bodies

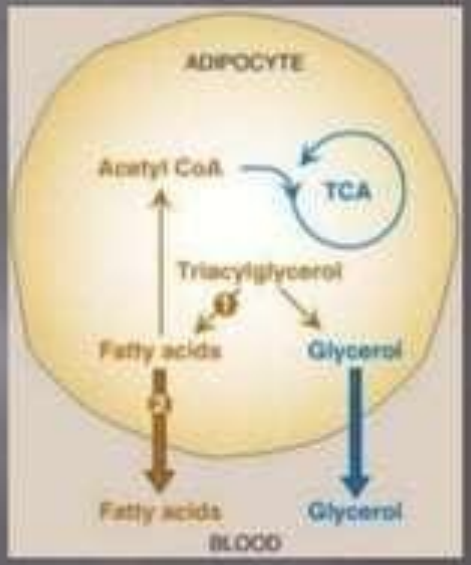
LIVER METABOLISM



ADIPOSE TISSUE IN STARVATION

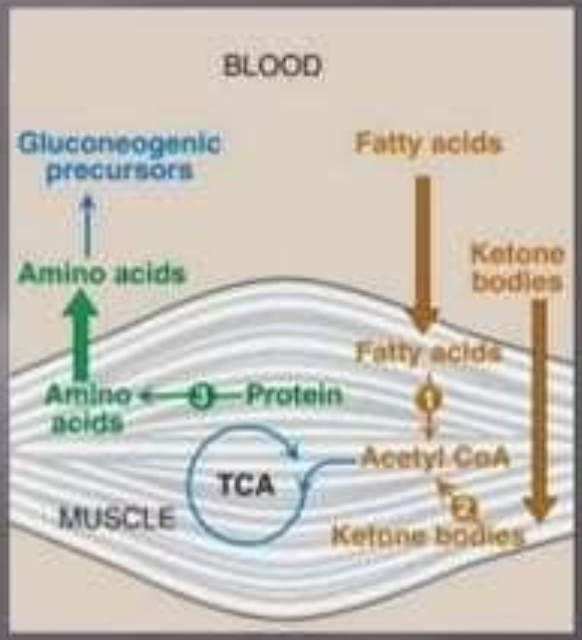
1. Carbohydrate metabolism
2. Fat metabolism

- Increased degradation of TAG (hormone sensitive lipase)
- Increased release of fatty acid
- Decreased uptake of fatty acid (lipoprotein , lipase of adipose tissue is low)



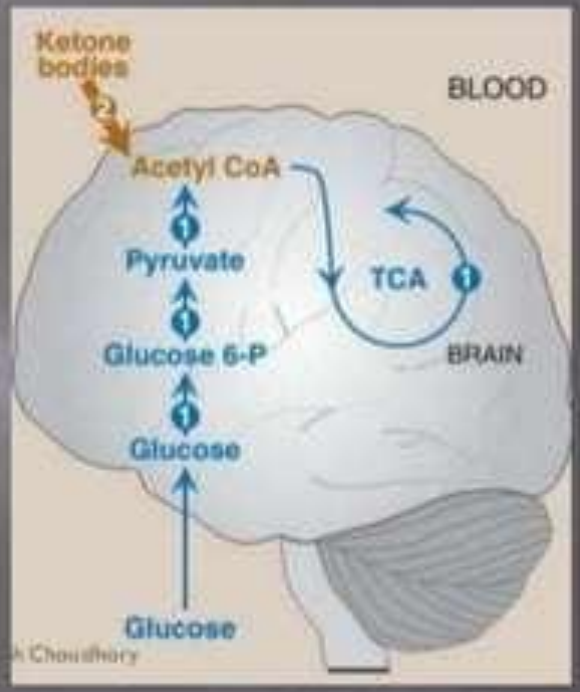
RESTING SKELETAL MUSCLE IN STARVATION

1. Carbohydrate metabolism
2. Lipid metabolism
   - During 1st two weeks : fatty acids from adipose tissue & ketone bodies from liver as fuel.
3. Protein metabolism
   - Alanine & glutamine are quantitatively the most important gluconeogenic amino acid .

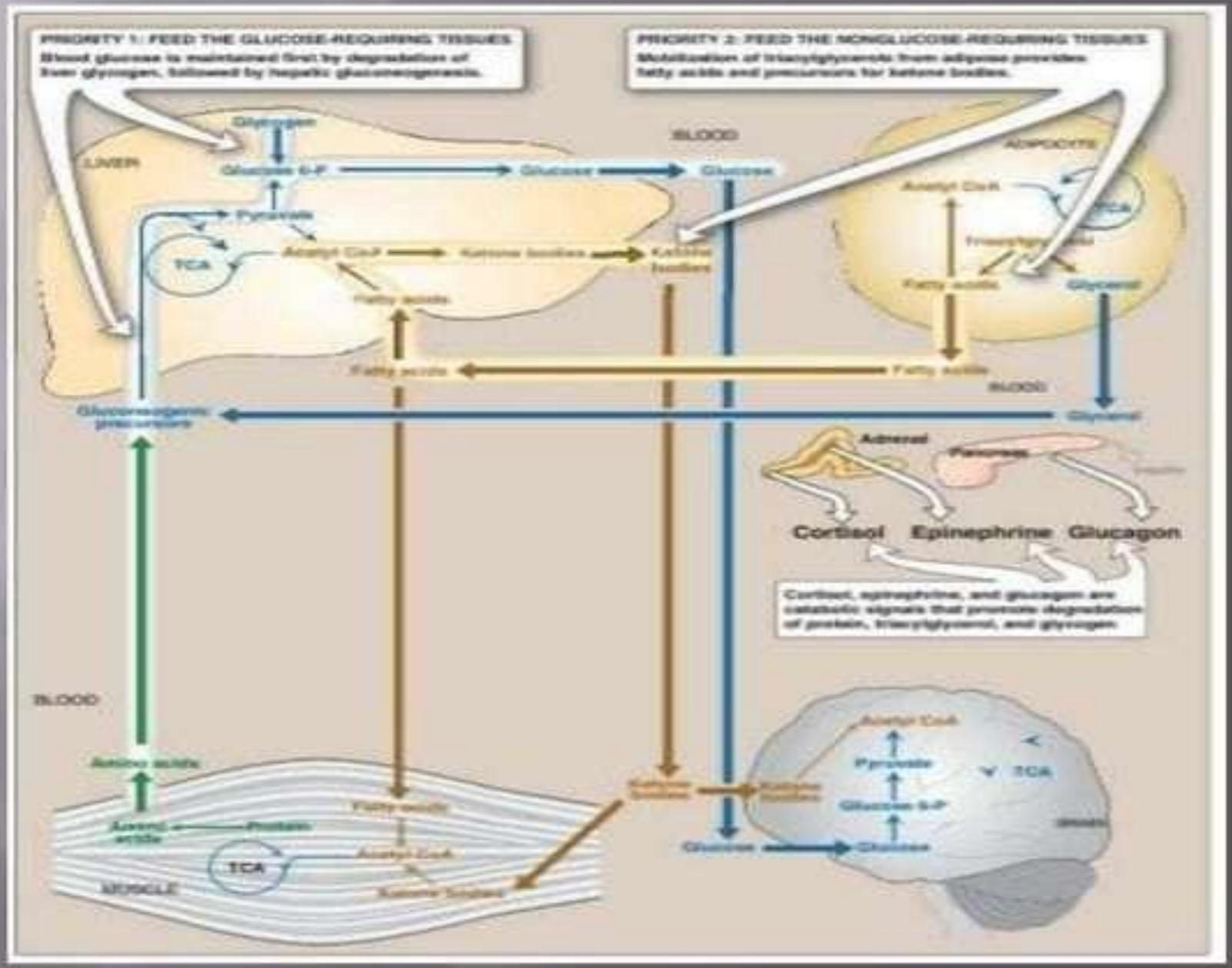


BRAIN IN STARVATION

- During starvation, insulin
  independent tissue, brain
  continue to utilize glucose.



- Brain also utilizes ketone bodies as energy source.


PRIORITY 1: FEED THE GLUCOSE-REQUIRING TISSUES
Blood glucose is maintained first by degradation of liver glycogen, followed by hepatic gluconeogenesis.
PRIORITY 2: FEED THE NONGLUCOSE-REQUIRING TISSUES
Mobilization of triacylglycerols from adipose provides fatty acids and precursors for ketone bodies.
Glycogen
LIVER
Glucose 6-P
Glucose
BLOOD
Pyruvate
TCA
Acetyl CoA
Ketone bodies
Fatty acids
ADIPOCYTE
Glycerol
Gluconeogenic precursors
Adrenal
Pancreas
Cortisol
Epinephrine
Glucagon
Cortisol, epinephrine, and glucagon are catabolic signals that promote degradation of protein, triacylglycerol, and glycogen.
Amino acids
Protein
MUSCLE
BRAIN

KIDNEY IN LONG TERM STARVATION

- Kidney expresses the enzymes of gluconeogenesis , including glucose-6- phosphatase.
- In late fasting about 50% of gluconeogenesis occurs.
- Provides compensation for the acidosis that accompanies the increased production of ketone bodies.

COMPLICATION

REFERENCE

- Textbook of pathology-6th edition-Harsh Mohan.
- http://www.bettermediciene.com/article.

THANK YOU