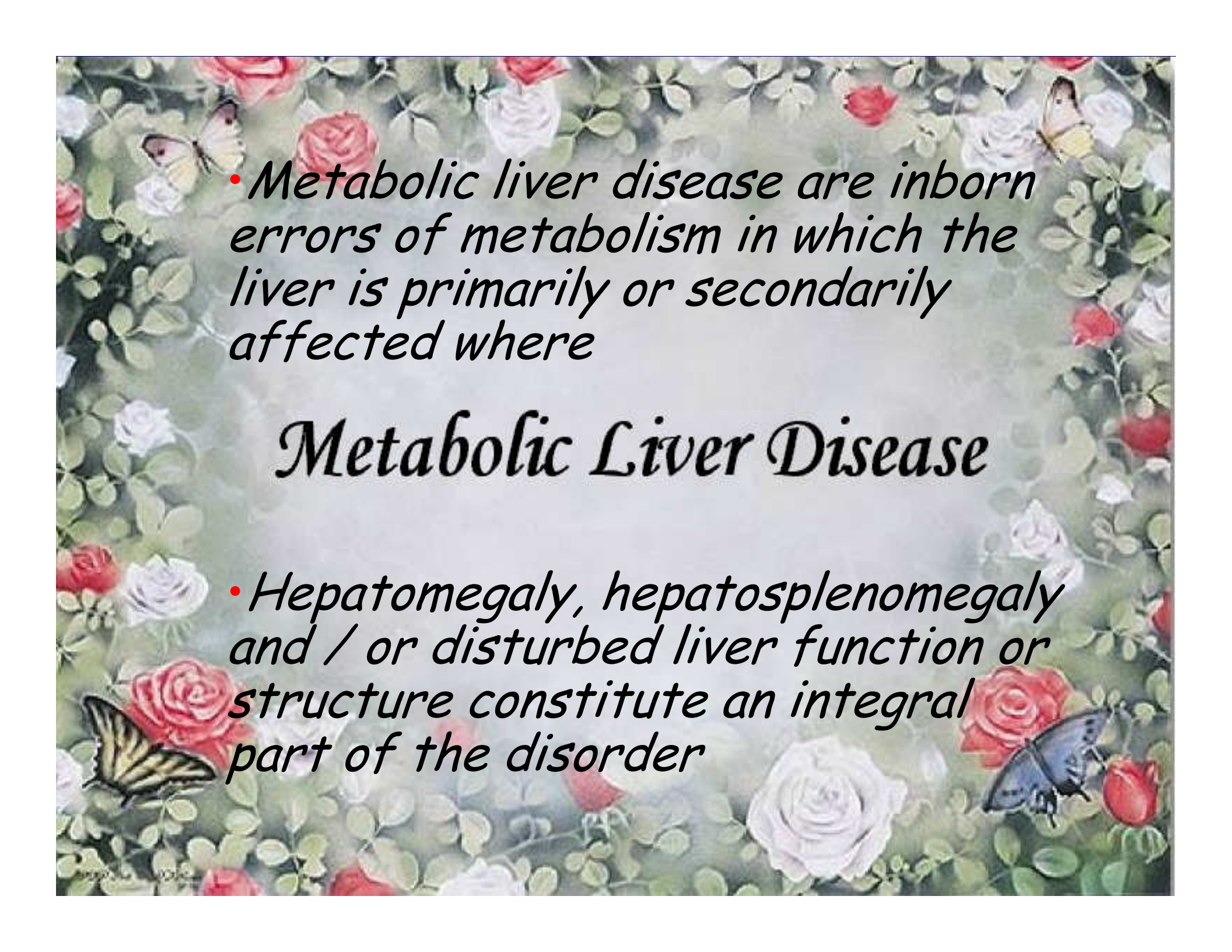




Nutritional  
management of  
metabolic liver disease

Malathi



- 
- *Metabolic liver disease are inborn errors of metabolism in which the liver is primarily or secondarily affected where*

## *Metabolic Liver Disease*

- *Hepatomegaly, hepatosplenomegaly and / or disturbed liver function or structure constitute an integral part of the disorder*

# Hepatic presentations of MLD



- Asymptomatic hepatomegaly.
- Chronic liver /cholestatic liver disease
- Acute hepatitis.
- Acute liver failure

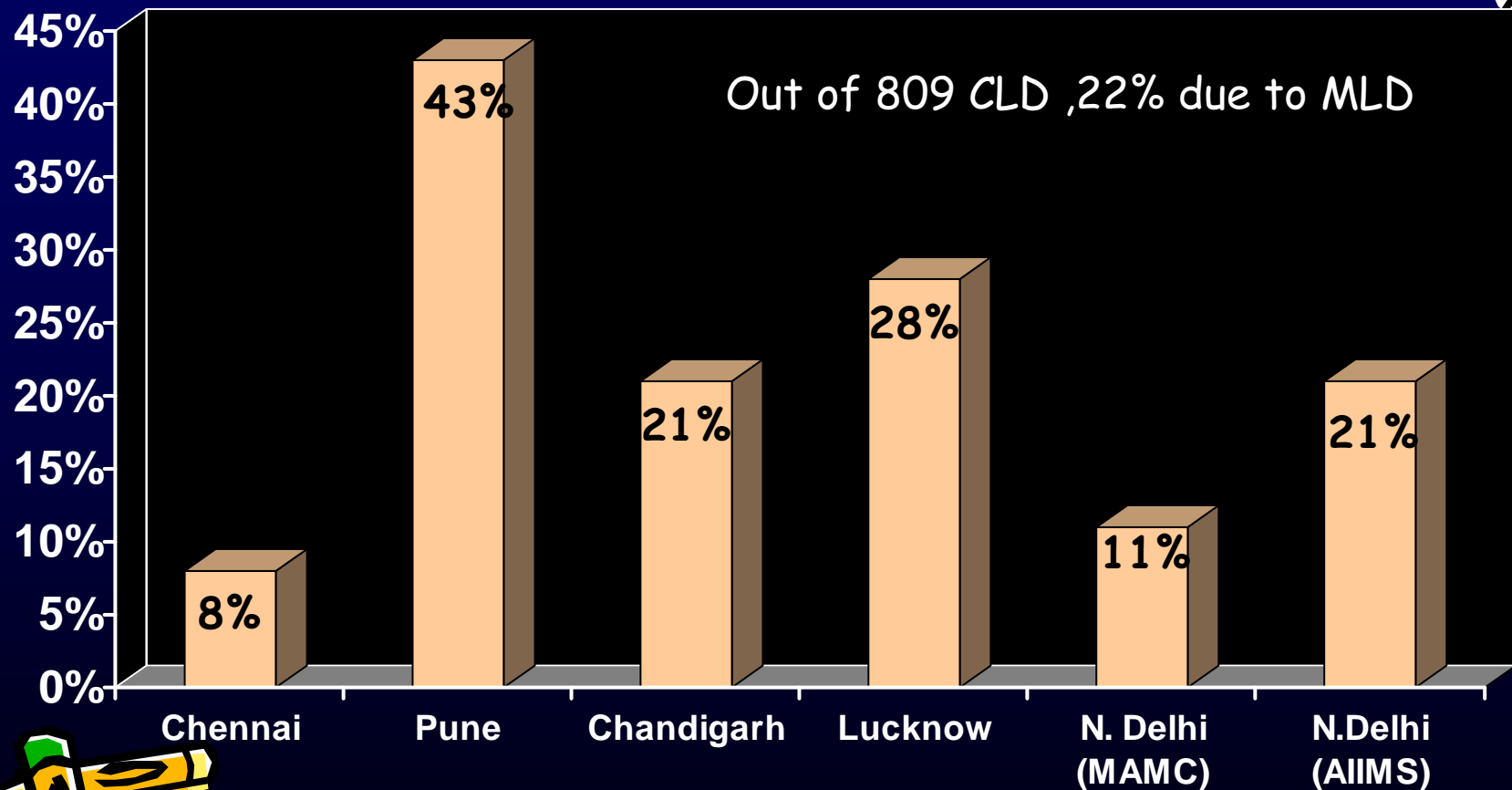


• End stage liver disease



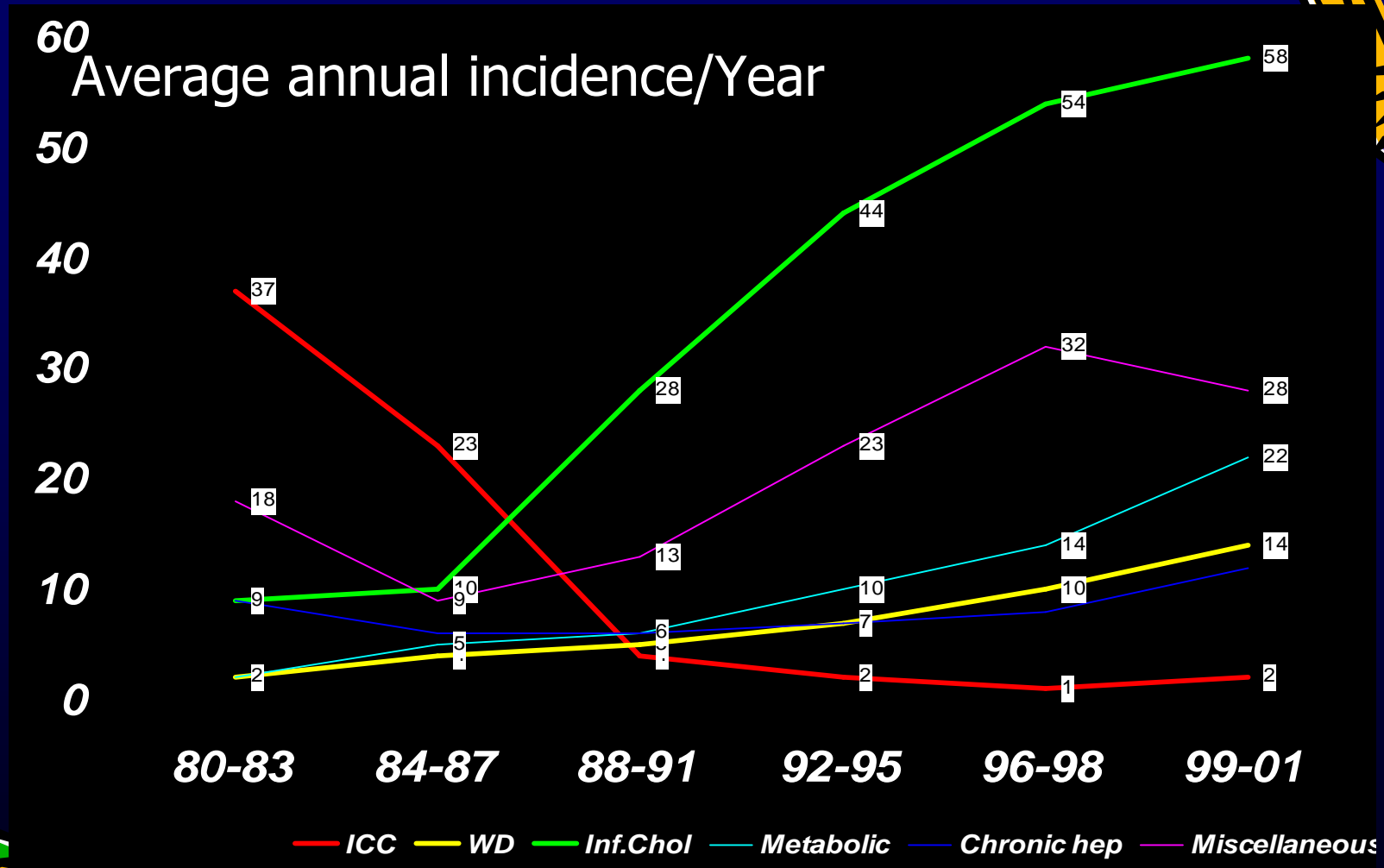
# MLD Presenting as CLD

Pediatric hepatology units



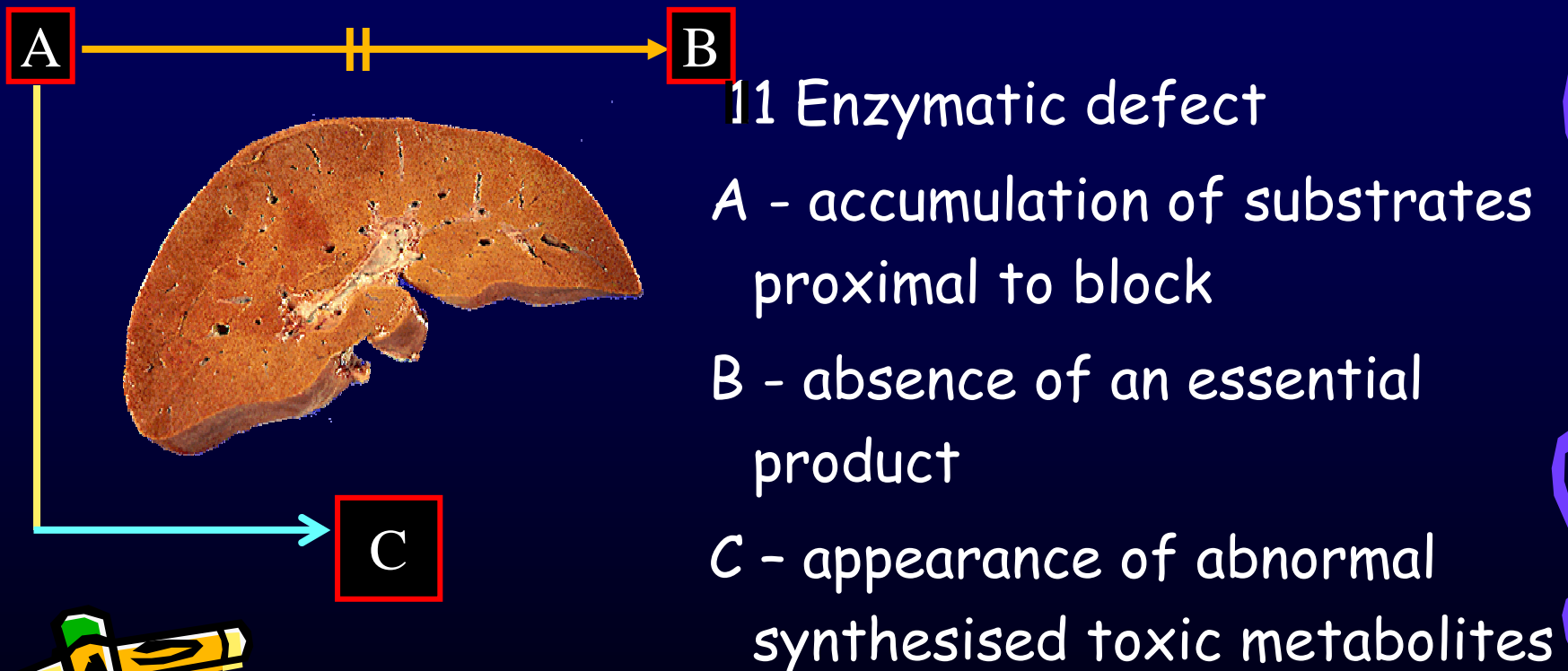
NK Arora Ind. JI ped 1999;66:597 - 103

# Liver Disease in KEM Pune 1980-2001



# Pathogenesis & Inheritance

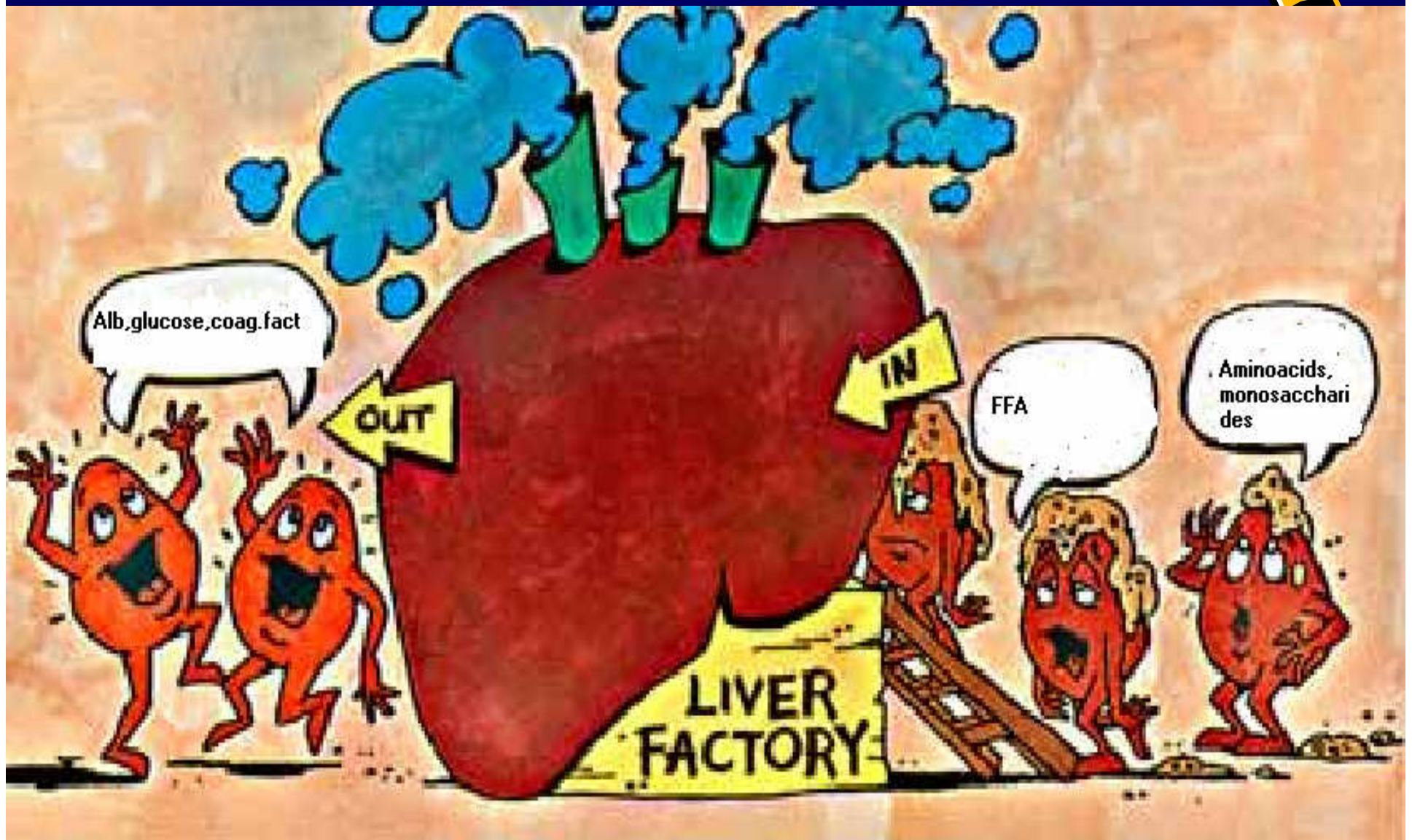
- Pathogenesis : Majority secondary to a well identified enzymatic defect and resultant metabolic block





Inheritance : AR

AD, X linked, mitochondrial inheritance

# Internal chemical power house



# Metabolic liver Disease



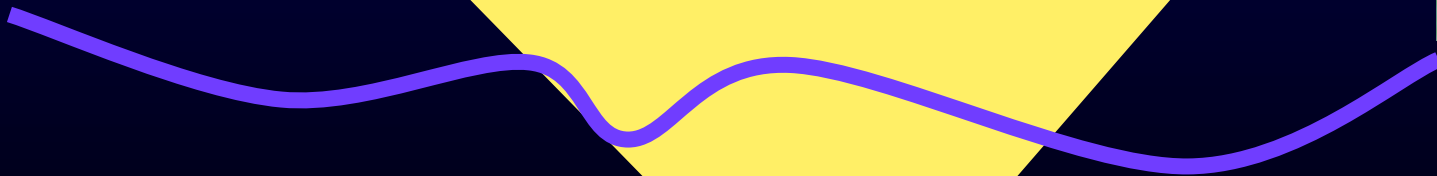
Carbohydrate	Galactosemia, GSD
Protein	A1AT, tyrosinemia
Lysosomes	Gaucher's, Niemann pick
Copper	Wilson's
Iron	Hemochromatosis
Bile Acid	PFIC, IBEBAM
Fatty Acid	FAO
Urea cycle	Urea cycle disorders
Peroxisomal	Zelleweger's
Miscellaneous	CF, NAFLD





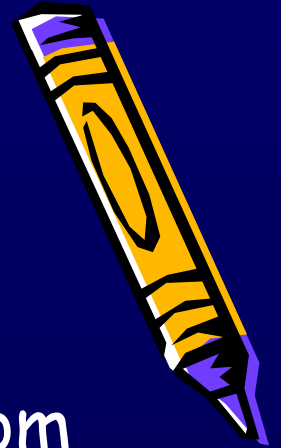
Is life worth living?

It depends on the liver!



# Common case scenarios

- **Mother:** doctor my child is suffering from liver prblem .Her appetite is very bad and she feels very weak.
- **Doctor:**Examines the child and asks what are you giving the child to eat?
- **Mother:**Nothing doc!I am very careful with her diet.No salt doctor,no oil ,nothing yellow,no non vegetarian.I am scared to give her the food we all eat.



# Nutrition in liver disease



NO



“Pathiyam or Pythiam”

# Facts

- The liver needs energy and protein for recovery and metabolic functions.



- In patients with liver disease if we do not provide adequate calories, protein and fat through exogenous source (food) the liver will steal from the endogenous bank (body stores) till it is available



# Nutritional management in MLD



- Role of liver in nutrition
- Effect of liver disease on nutrition
- Assessment of nutrition in liver disease
- Nutritional therapy in liver disease
- Nutritional therapy in specific metabolic liver disease



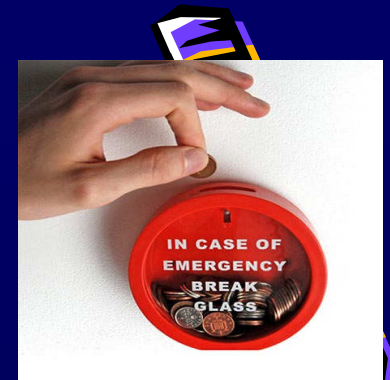
# Nutritional management in MLD



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# CHO metabolism



- Galactose and fructose → glucose
- Stores glucose → glycogen (glycogenesis)
- Glycogen → glucose (glycogenolysis)
- Lactic acid, AA, intermediates of TCA cycle → glucose (gluconeogenesis)
- Provides a regular and predictable supply of glucose to extrahepatic tissue



## ----- in Liver Disease

- Carbohydrate metabolism is disrupted
- Hypoglycemia occurs in acute on chronic liver disease or in acute liver failure
- Hyperglycemia may be + due to insulin resistance





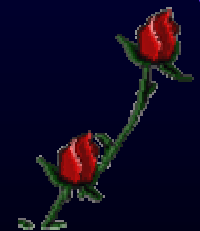
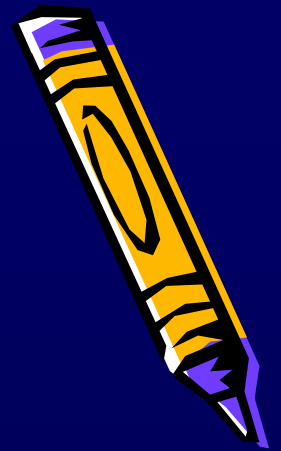
# Protein Metabolism

- Liver utilises amino acids for protein synthesis and gluconeogenesis.
- Regulates supply of amino acids to muscle.
- Converts excess of amino acids to urea.
- NE AA/EAA are catabolised in liver & muscle
- BCAA (valine, leucine and isoleucine) is utilised in protein synthesis, growth and + N<sub>2</sub> balance
- Synthesis of albumin and all coagulation factors (except VIII)



## ----- in CLD

- ↓ Protein synthesis → hypoalbuminemia
- (oedema, ascites)
- ↓ coagulation factors (coagulopathy)
- ↓ Prealbumin and retinol binding protein
- ↑ AAA (trypt, tyrosine, phenylalanine)
- ↓ BCAA (valine, leucine, isoleucine)



# Lipid Metabolism

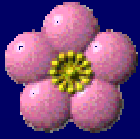
- Fat metabolism is co ordinated through liver,intestine,lymphatics and peri. tissue
- FFA released by adipose tissue is re esterified to TGL / oxidised to energy
- Synthesizes lipoproteins,cholesterol.
- Helps in absorption & storage of fat soluble vitamins



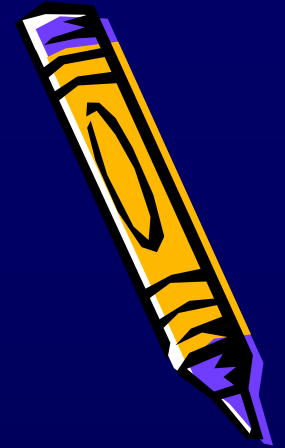
-----in CLD

- Lipoprotein levels are abnormal.
- Mild to moderate steatorrhea is present





# Vitamins and Minerals



- ↓ bile salts → malab of fat sol vitamins
- Vit A-night blindness,xerophthalmia,keratomalacia
- Vit D-rickets and osteomalacia.
- Vit E- areflexia,ataxia,peripheral neuropathy
- Vit K-(cofactor for II,V,IX,X) Coagulopathy
  
- Converts 1)Carotene to Vit A,
- 2)Folate to 5 MTHF acid and
- 3)Vit D to its active form.



# Vitamins and minerals



- Biochemical deficiency of water sol vitamins
- Iron deficiency due to GI bleed & ↓ intake
- Calcium is disturbed due to Vit D def.
- Zinc, Selenium and Chromium def can occur



# Topic

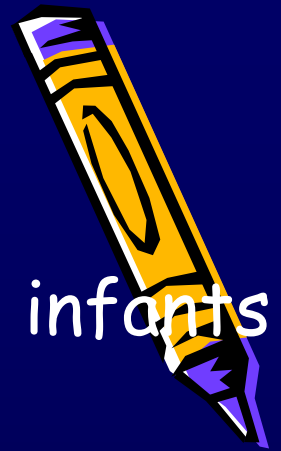


- Role of liver in nutrition
- Effect of liver disease on nutrition
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- Nutritional therapy in specific metabolic liver disease



# Malnutrition in LD

- Incidence of Severe Malnutrition in 60% of infants with liver disease (Beath 1993)
- PEM in 65-90% of advanced liver disease almost 100% in candidates for liver Tx (Di Cecco 1989)
- Malnutrition occurs irrespective of aetiology. Calorie depletion more in cholestatic disease whereas protein depletion in non cholestatic disease





# Malnutrition in LD

- Micronutrient def may be + without overt malnutrition.
- Fat soluble Vitamin and mineral def are common esp. with cholestasis.
- Malnutrition not a feature of acute liver injury but occurs with progression to liver failure.

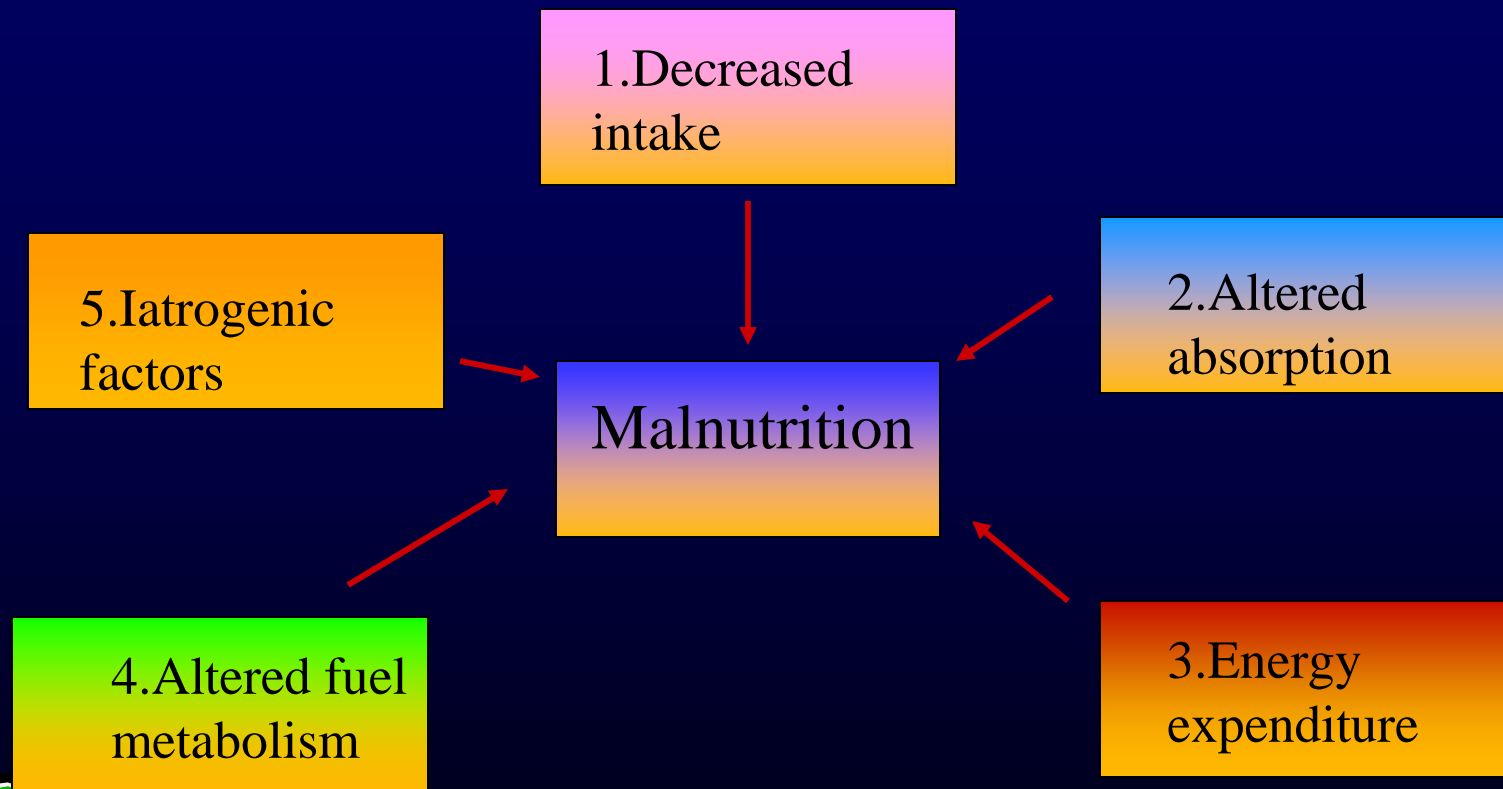


# Why this spectrum?

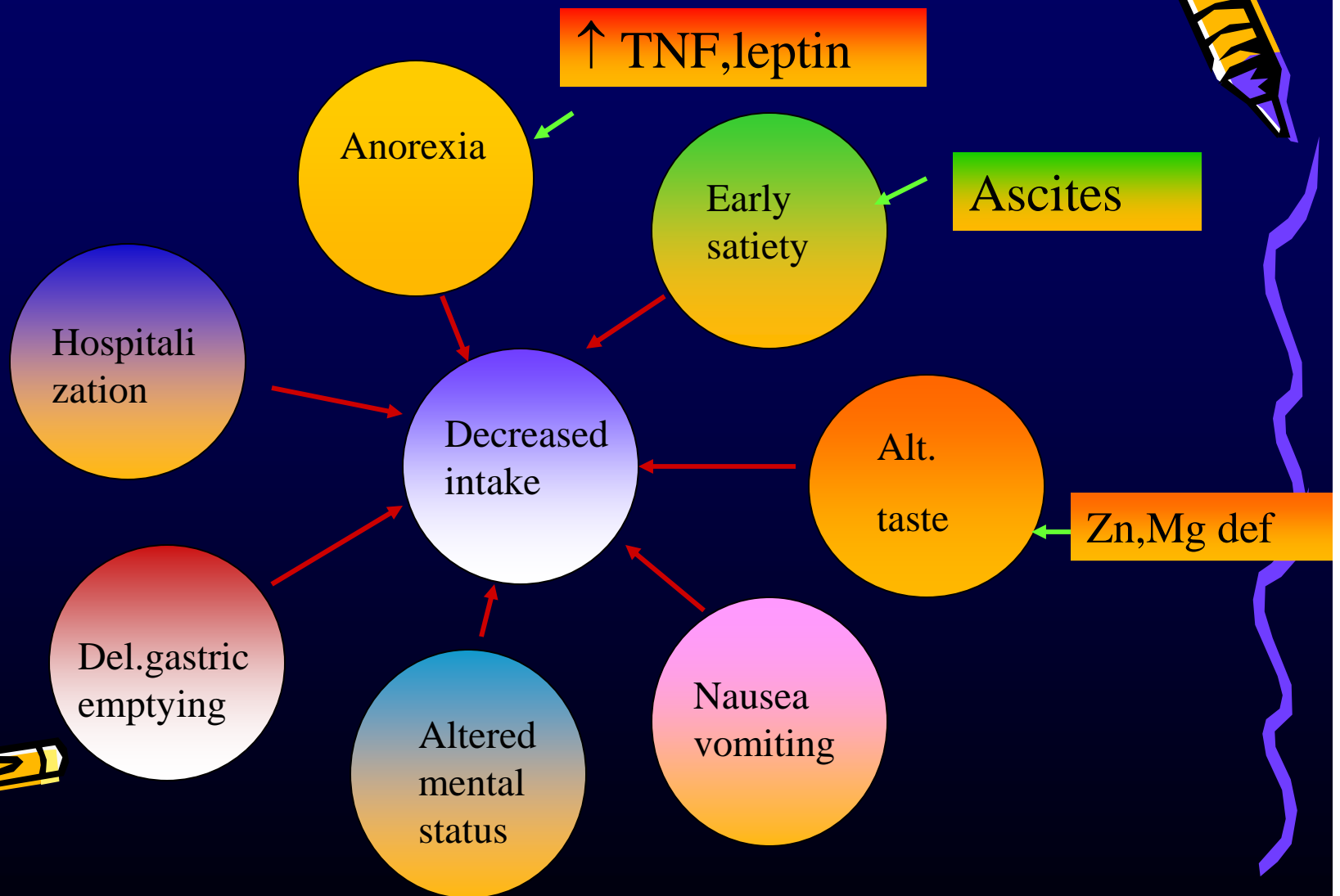
- Aetiology
- Severity and type of disease
- Methods used for assessment
- Setting where surveys are performed.



# Pathophysiology



# 1. Decreased Intake



## 2. Altered Absorption

- Fat malabsorption: ↓ bile secretion due to cholestasis impairs micelle formation essential for digestion of fat by pancreatic enzymes.
- Fat soluble vitamins also require micelles  
Vit A and D def seen in chr.cholestasis.( 30 % )
- Pancreatic insufficiency in PFIC,CF
- Bacterial overgrowth in cirrhosis (35-60%),  
mucosal edema in PHT.



# 3. Energy Expenditure



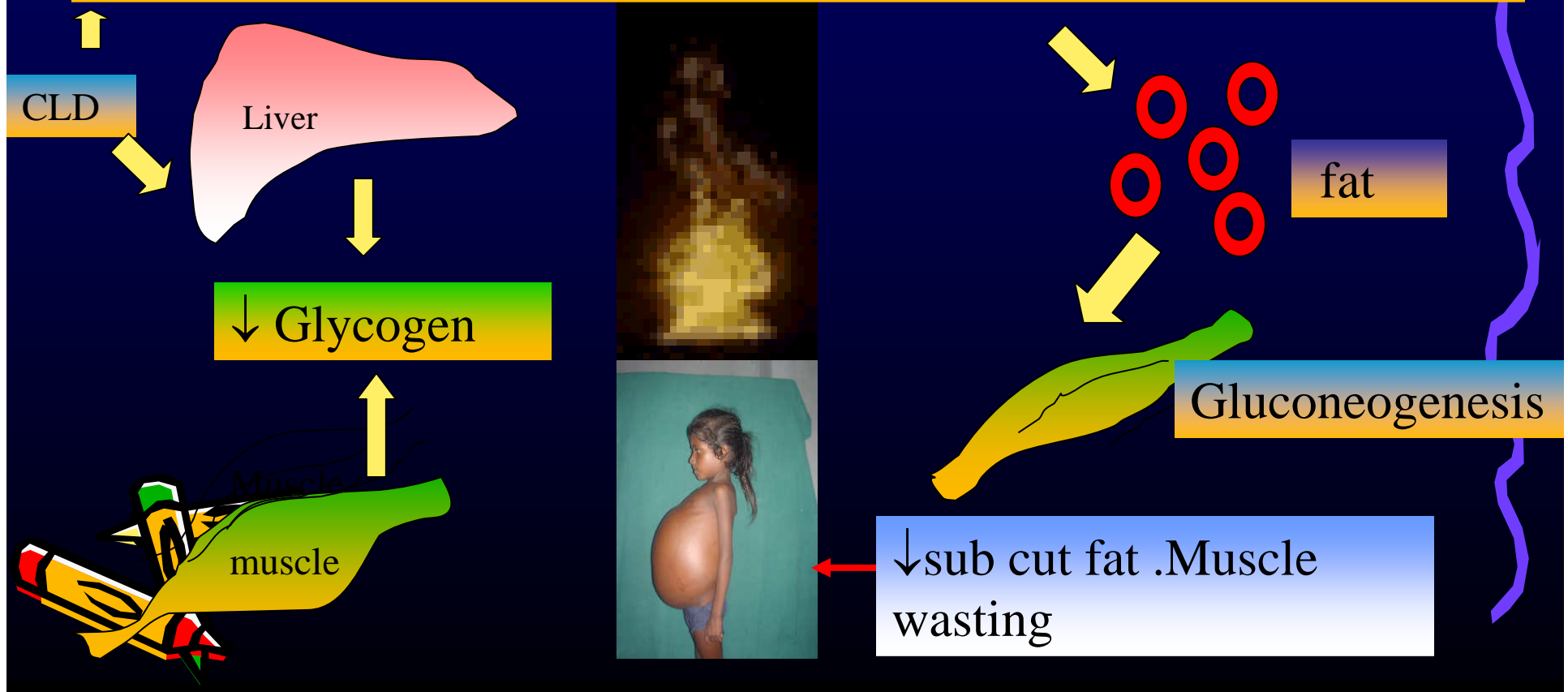
- Liver contributes 20% of Resting Energy Expenditure
- REE in CLD is variable. Biliary atresia ↑ ed by 30%. Adv liver disease -increased.
- Energy req. correlates with ↓ body mass & not with aetiology & duration of Liver Disease.
- ↑ nutritional requirement occurs acutely in ascites formation, SBP, variceal Hghe.



# 4. Altered Fuel Metabolism



“Accelerated Starvation” with early recruitment of alternative fuel sources



# Factors affecting Fuel metabolism

- Increased or decreased metabolic rate
- Glucose intolerance/insulin resistance
- Rapid postprandial gluconeogenesis
- Reduced glycogen stores
- Elevated leptin
- Elevated TNF- $\alpha$
- Decreased insulin like growth factor-1





# 5. Iatrogenic



Overzealous diet restriction



Lactulose



Iatrogenic



Freq. paracentesis



Diuresis



# Cirrhosis



Poverty above



Richness below



# Topic



- Role of liver in nutrition
- Effect of liver disease on nutrition
- **Assessment of nutrition in liver disease**
- Nutritional therapy in liver disease
- Nutritional therapy in specific metabolic liver disease





# Assessment of nutritional status

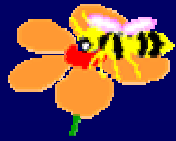
- A - Anthropometry
- B - Biochemistry
- C - Clinical
- D - Dietetic history/ drug/ disease diary



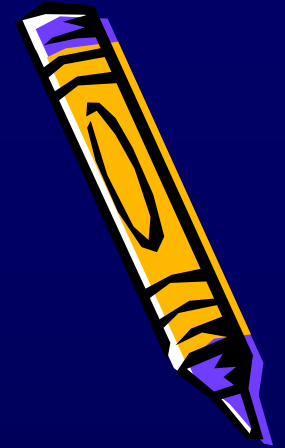
# Anthropometry

- Weight for age: underestimates nutritional deficits
- Organomegaly.
- Ascites
- Edema





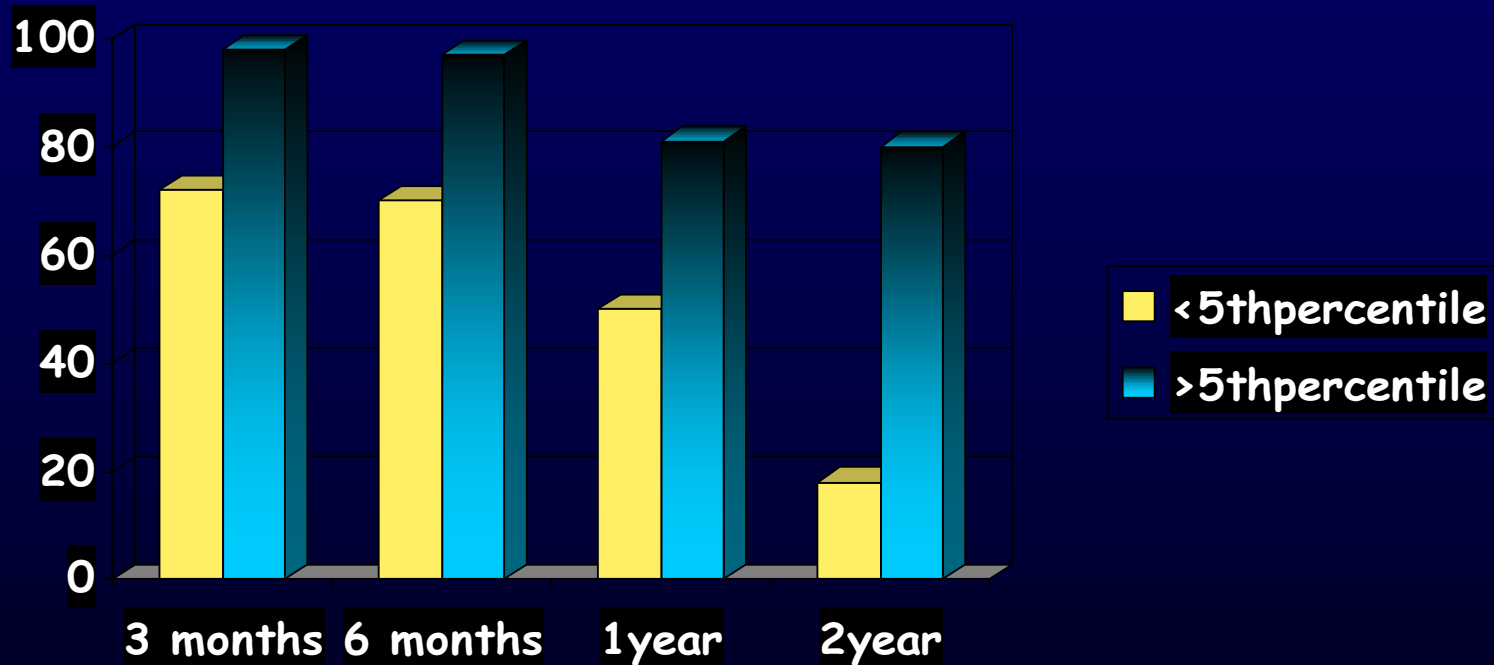
# Assessment of nutritional status



- Height for age
- BMI: calculation of obesity in NAFLD
- Head circumference: All children less than 3 y
- Triceps skin fold thickness.(TSF)
- Mid arm muscle circumference(MAMC)
- $MAMC = MAC - (3.14 \times TSF)$  *TSF not possible in infants <3m*
- Hand Grip.
- Subjective Global assessment (temporal muscle wasting, micronutrient def)



# Effect of Mid Arm Muscle Circumference on survival

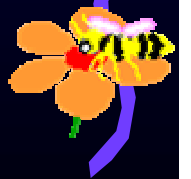


Survival of CLD patients with MAMC less than 5 th percentile compared to those with >5 th percentile

Caregaro L et al AmJClin Nutr 1996

# Assessment of nutritional status

- Prealbumin/Albumin X (↓ synthesis)
- Creat/Ht index.X (↓ synth or renal insuff)
- Body composition by bioelectric impedance X
- Skin testing for delayed hypersensitivity. X
- CT or DEXA X (↓ accuracy due to EC edema)
- Total Body water, Potassium X
- In Vivo Neutron activation analysis.(Highly accurate but expensive)






# Limitations of nutritional assessment



Body weight	Affected by organomegaly and body fluid changes
Anthropometric measurements	Influenced by fluid status
Serum protein	Affected by water retention and intercurrent infections
Urinary tests	Affected by many non-nutritional factors
Body composition by bioelectric impedance	Not yet validated



# Topic



- Role of liver in nutrition
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- Assessment of nutrition in liver disease
- **Nutritional therapy in liver disease**
- Nutritional therapy in specific metabolic liver disease





# Objectives of nutritional therapy

- Maintain, improve nutritional status or correct malnutrition.
- Prevent further liver cell injury and enhance regeneration.
- Prevent or alleviate HE or other metabolic disturbances amenable to nutritional therapy
- Maintain good nutrition while waiting for Tx.



# Nutritional Therapy

- provide adequate calories
- prevent protein from being catabolized
- promote anabolism.
- prevent hypoglycemia
- provide vitamins and micronutrients.



# CLD:Calories



- Infants: 120-150 cal/kg/day with 50% of cals from MCT
- Older children 1.2-1.5 x dietary ref value:  
eg 5yr old child 90 kcals/kg/day



# Calories



- Patients with ascites:
- Preferable to calculate the estimated "euvolemic" weight.
- Mild ascites: 3-5kg
- Moderate ascites: 7-9kg
- Severe ascites: 14-15kg



# Protein in CLD

- Infants 3-4 gm/kg/day.
- Older children :2.5-3 gm/kg/day





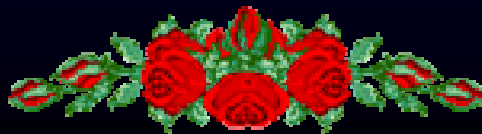
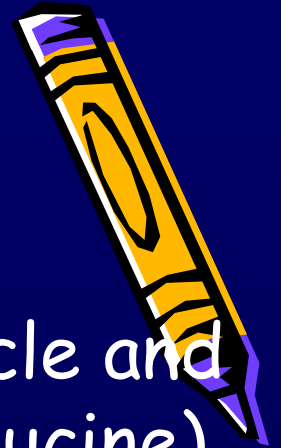
# Protein

- Compensated cirrhosis: 1-1.5 gm/kg/day
- Encephalopathy: restrict 0.6-0.8 gm/kg/day  
Slowly increase to 1 gm/kg/day.
- If protein sensitive and cannot tolerate even 0.8 gm/kg, veg protein / enteral feed enriched with BCAA and ↓ AAA is given
- Vegetable protein is low in methionine and ammoniagenic amino acids and rich in BCAA. Casein based are low in AAA and high in BCAA.



# BCAA

- BCAA is pro anabolic, promotes hepatic, muscle and plasma protein synthesis in CLD patients (leucine)
- Energy source for liver, muscle, brain and heart.
- Accelerate liver regeneration in animal models.
- BCAA helps in reducing and competes with AAA



# BCAA

- Fischer ratio: Serum BCAA to AAA, Correlates with liver function .
- Ratio  $<2$  is seen in children with persistent jaundice and likely to be a predictor of growth disturbance.
- Supplementation with BCAA improved Fischer ratio.
- Oral BCAA supplement 0.2-2 g/kg/day.
- ESLD: significant improvements in weight gain, liver function and QOL when supplemented over 1 year.
- More studies necessary to assess dose related response and the ideal dosage.



# Carbohydrates



- CHO not restricted
- If patient has diabetes or insulin resistance provide upto 50-60% of kcals from complex CHO with a consistent CHO intake from day to day.
- A high CHO (50gms) snack at bedtime prevents breakdown of fat and protein during overnight fast.



# Fats

- Fats make food appetizing and delivers energy efficiently.
- Restrict only in fat mal absorption.
- If fat malabsorption is present use medium chain triglycerides.



# MCT

- MCT do not require micelle formation and are absorbed via the portal vein.
- ↓incidence of steatorrhea and promotes growth.
- Can be given in small doses through out the day mixed with food.(1-2ml/kg in 2-4 doses)
- Do not contain EFA and ∴ should be provided.
- High osmolality >600 mOsmo/kg and may cause cramping ,diarrhea. Rec.dose is 0.3 g/kg.
- Preferable not to cook in MCT oil,because of low temperature threshold , add after cooking.



# Sodium/Water



- Restrict Na only if there is moderate or massive ascites.
- Infants 1 mEq/kg/day and 2-3 mEq/kg/day in older children and adolescents
- Restrict fluid only in those with hyponatremia



# Vitamins and minerals

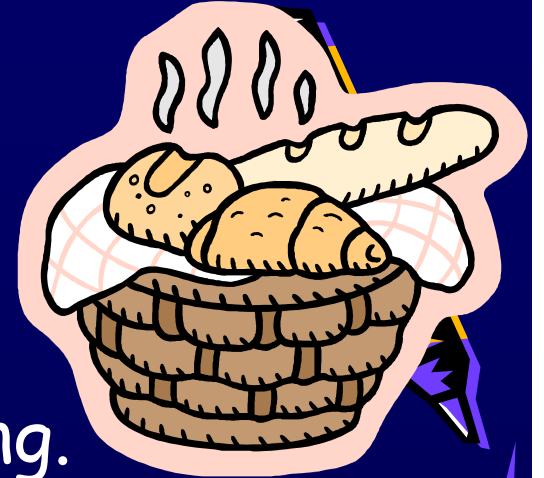
- Ensure adequate intake from diet or supplements.
- Vit B to be provided(B1,B6).
- Vit A,D,E in presence of cholestasis.
- Vit K if prothrombin time is prolonged.
- Calcium to be supplemented.
- Zinc (taste,dark adaptation) and Folic acid
- 5 mg/day beneficial.





# General Principles

- Avoid prolonged periods of fasting.
- Provide small meals and snacks / day.
- Encourage an evening snack to reduce duration of over night fasting.
- Oral liquid supplements help in increasing calorie and protein consumption.
- Avoid unnecessary diet restrictions.



# Recommendations in ALF



	CHO	PROTEIN	FAT
Age	8 to 9 mg/kg/min. (Max. 12.5 ) Tod. - 7, Adol. - 4	Infants 0.5- 1g/kg goal 1.5 to 1.9 g/kg Child > 1 yr - 0.8 to 1 g/kg	Child < 5 yrs - 40% Non Protein Energy Child > 5 yrs 30 to 35% 5-6% conc. > 1 yr. - 7%
Increments	1 g/kg/100 ml/day	0.25 to 0.5 g/kg	0.5 g/kg/100 ml
Type	Glucose polymers	Veg. proteins, BCAA	LC PUFA, MCT



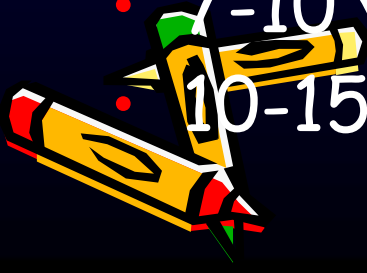
# ALF

- Energy expenditure
- If ventilated : Infants - 90 to 100 kcal/kg
- Non ventilated : 100 to 120 Kcal/kg
- Infants - Breast milk (0.67 kcal/ml)
- Vol restricted - BM + Carbohydrates + fat (1 kcal/ml)
- Children - 1 kcal/ml
- Volume restricted - 1.5 kcal/ml



# Fluid in ALF

- 60-75% of volume if cerebral edema is present.
- Premature - 180-200 ml/kg/day
- 0-1 yr - 150 ml/kg/day
- 1-3 yrs - 100 ml/kg/day
- 3-6 yrs - 90 ml/kg/day
- 7-10 yrs - 70 ml/kg/day
- 10-15 yrs - 60 ml/kg/day



# Nutrition in cholestasis

- Diet 200 Cal/kg: 125 % of RDA based on weight for height at 50 % percentile
- Glucose polymers: 24-27 cals /oz formula
- Protein 2 - 3 gm/Kg  
Dietary fat -MCT
- Milk with MCT skim milk + coconut oil 3ml + G.nut oil 1ml
- Vit A-5000 - 20,000 IU/day ,30,000IU /3m
- D - 30,000 - 60,000 IU IM once in 6m
- K- 1-5mg IM weekly
- E-50-400 IU oral/day
- B.complex & Vit C twice the RDA
- Ca, P, Zn, Mg,Se.Fe

# End Stage Liver Disease

- Fluid restrictions 60-80% maintenance
- Ascites (No added salt, 1 - 1.5 mmol./kg)
- Steatorrhea MCT, LCT
- Encephalopathy Temp. protein restrictions 0.5-1 g/kg

If NPO glucose 10-20%



# Enteral nutrition



- Those who are unable to meet nutrient needs by oral nutrition require tube feeds.
- Nutrient dense formula 1.5-2 cal/ml, lactose free, lactalbumin or casein based. Na <40mEq/L.
- If steatorrhoea +, lipids in the form of MCT.
- BCAA formula only if pts cannot tolerate the protein in standard formulae.
- Oesophageal varices are not a contraindication for placement of tubes



# Parenteral Nutrition



- Infections and metabolic complications more compared to enteral feeds.
- Reserved for those who are intolerant to enteral feed (G I bleed, ileus)
- Volume usually large. 25-40% fat energy
- Parenteral protein less chance of ppt HE
- Protein start at 0.5-0.7gm/kg  $\uparrow$  by 0.2gm/kg upto 1.5gm/kg. BCAA 35%.





# Topic



- Role of liver in nutrition
- Effect of liver disease on nutrition
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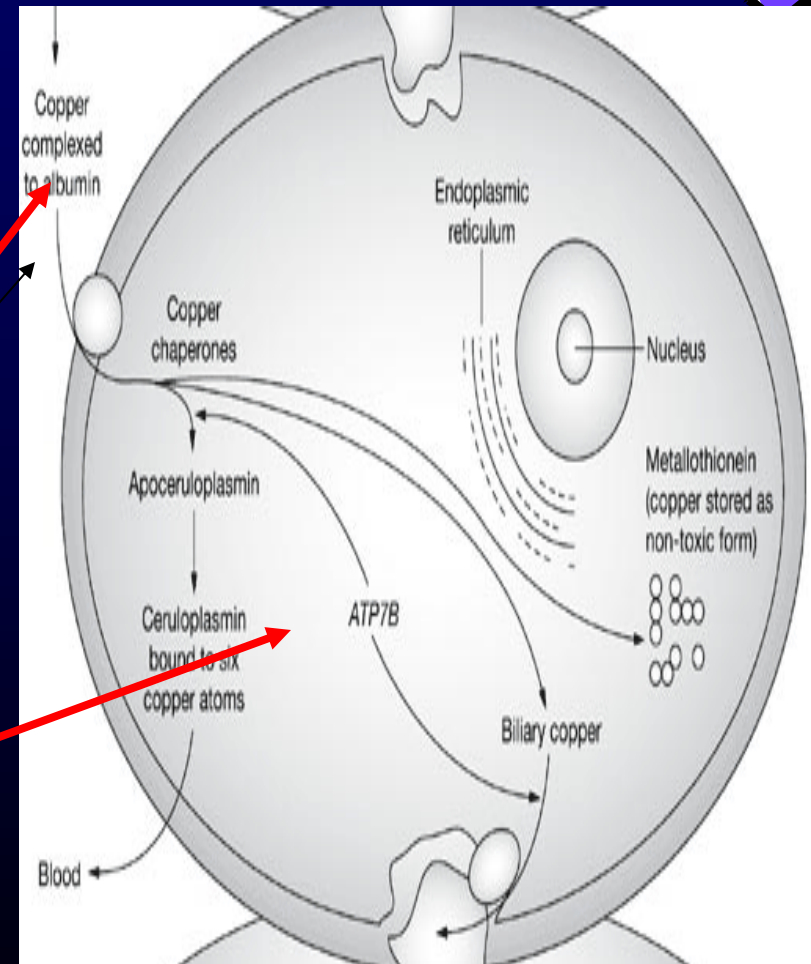
# 3 most common MLDs in India

- Wilson Disease.: Copper
- Glycogen storage disorder.
- Galactosemia



# Copper absorption & Metabolism

- Cu in adult diet is 2-4 mg /day.
- 55-75 % of copper is absorbed
- Daily req: adults is 1.3-1.7 mg.
- Absorbed and complexed with amino acids and albumin.
- Transported to hepatocytes.
- ATPB7 helps in incorporating copper into Cp and excretion in bile



# Wilson's disease



- **Defect:** AR disorder due to mutations in ATP7B gene located on chr.13. Defective transport of copper from the liver.
- **Causes:** Abnormal accumulation of copper in various tissues, liver kidney, brain, RBC
- **Effect:** Hepatic or Neuro manifestations or both.



**Diet :** Avoid copper containing food



SAMUEL ALEXANDER KINNIER WILSON

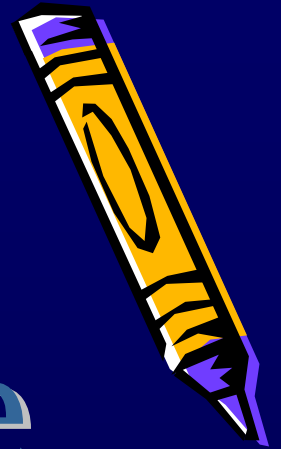
(1878-1937)

# Wilson disease

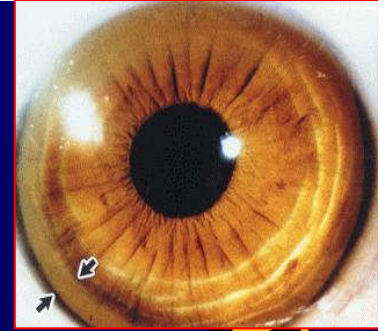
- World wide prevalence is 1 in 50,000.
- More common in India and is the leading cause of MLD in India
- Treatable CLD if detected early



2010

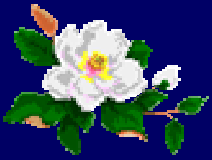


# Wilson's disease

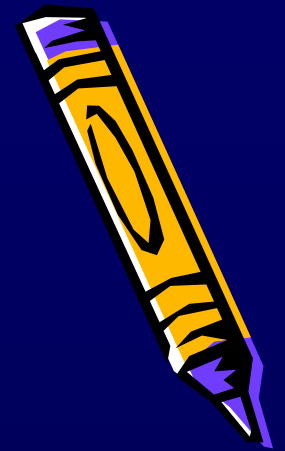


- Age usually more than 3 years
- Varied clinical presentation depends on the mutation
- High index of suspicion is important.
- Serum ceruloplasmin, KF ring and 24 hr urinary copper are 3 important investigations





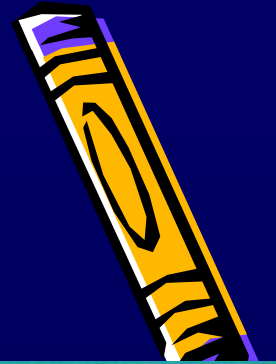
# Diet



- Water content of  $Cu < 1$  ppm.
- Avoid Cu vessels for storing or drinking water
- Strict diet during the initial treatment(1 yr) Avoid liver, shellfish, nuts, beans, chocolates, mushrooms.
- Lacto vegetarian diet
- How long? May be relaxed a little ,once stabilised.



# Liver Transplant is a “cure”



Diet is only an adjunct therapy



Life long medication is difficult







Chubby Child

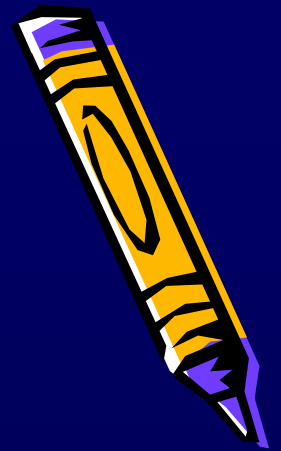
Yet has

Chronic liver Disease !!



# Glycogen Storage Disorder

- Important MLD in India
- Group of AR inherited disorder due to defect in metab. of glycogen
- Increased accumulation of normal or abnormal glycogen in various tissues
- Liver most severely affected. Skeletal muscle heart, kidney, bones, brain may be involved

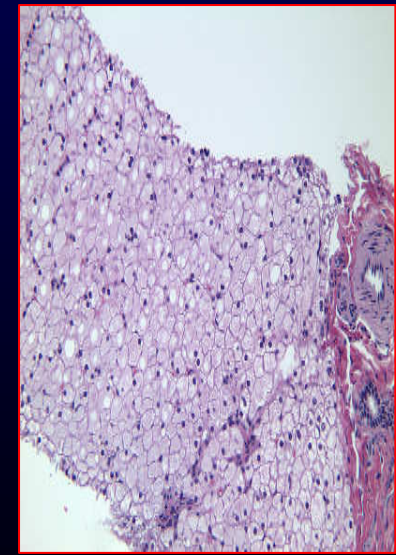


# Glycogen Storage Disorder



- More than 13 types of GSD. I, III are common (III & IV → cirrhosis)
  - Hepatomegaly
  - Hypoglycemia + seizures
  - Voracious appetite

Short stature





# GSD I: Liver not a good bank.



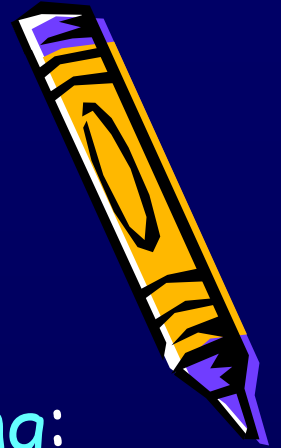
- Liver is able to convert the glucose in food to glycogen. All savings account converted to fixed deposit.
- During fasting - unable to convert the glycogen stored in the liver back to glucose. When in want cannot release cash from fixed deposit.
- Children with GSD cannot fast
- They are prone for hypoglycemic seizures
- ~~Aims~~ Aims to prevent hypoglycemia



# Night feeding important !

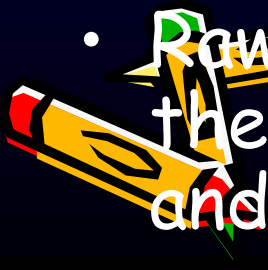
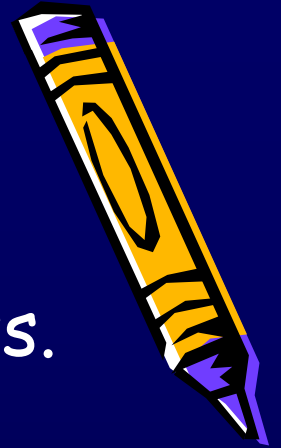
- I. Continuous nocturnal intragastric feeding:
- Infusion of elemental diet with dextrans is used. Glucose or dextrose infusion may be administered to lower cost.
- Drip to be controlled with electric infusion pump.
- <6 yrs: 7-9 mg/kg/min
- Older children: 5-6 mg/kg/min x 10hrs.
- Adults: 3-4 mg/kg/min x 8-10 hrs

II. Uncooked Corn starch : at 10pm, 2am, 6am

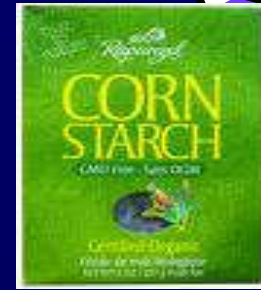


# Corn starch therapy

- Good alternative in children also for adults.
- Starch is a mixture of amylose 20-30% and amylopectin.
- Digestibility depends on the amylose content.
- *Cooked CS behaves as a rapid CHO.*
- *Raw CS is a slow CHO, releases glucose under the hydrolytic activity of pancreatic amylase and supplies glucose for many hours.*



# Corn starch therapy



- **Dose:** Optimal dosage of corn starch for children with GSD type I is 1.75mg/kg -2.5 mg/kg of ideal weight every 6 hrs
- Provides glucose 5.3-7.6 mg/kg/min.
- **Mix with water ,skim milk** at room temperature ratio 2:1.
- **Age:** children > 1 year since pancreatic activity is low in small children(some studies report that it can be used)





# Oral Feeding

- Foods rich in starch at intervals of 3-4 hrs.
- First morning feed should be given immediately or 30 minutes after nocturnal enteral feeding is discontinued or 4 hr after night dose of corn starch.
- According to age 5-6 feedings during the day
- CHO 60-70%, fat 25-30% as fat, 10-15 % proteins,
- Avoid or limit fruits ,milk ,sugar because they are converted to glycogen & lactate & contribute to lactic acidosis.
- Permit alternatives: rice bran,puffed rice,potato

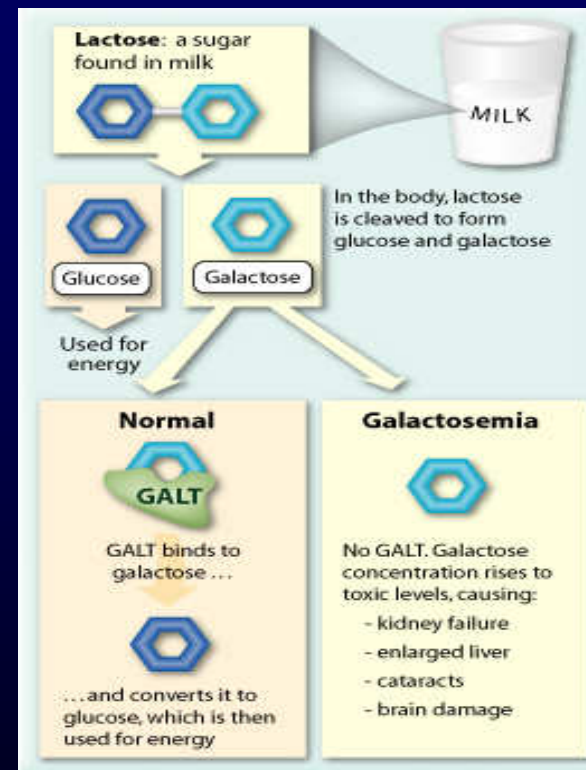






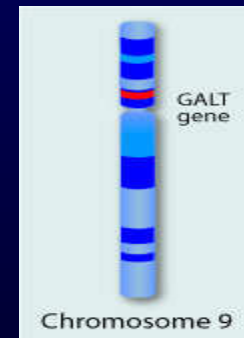
# Galactose metabolism

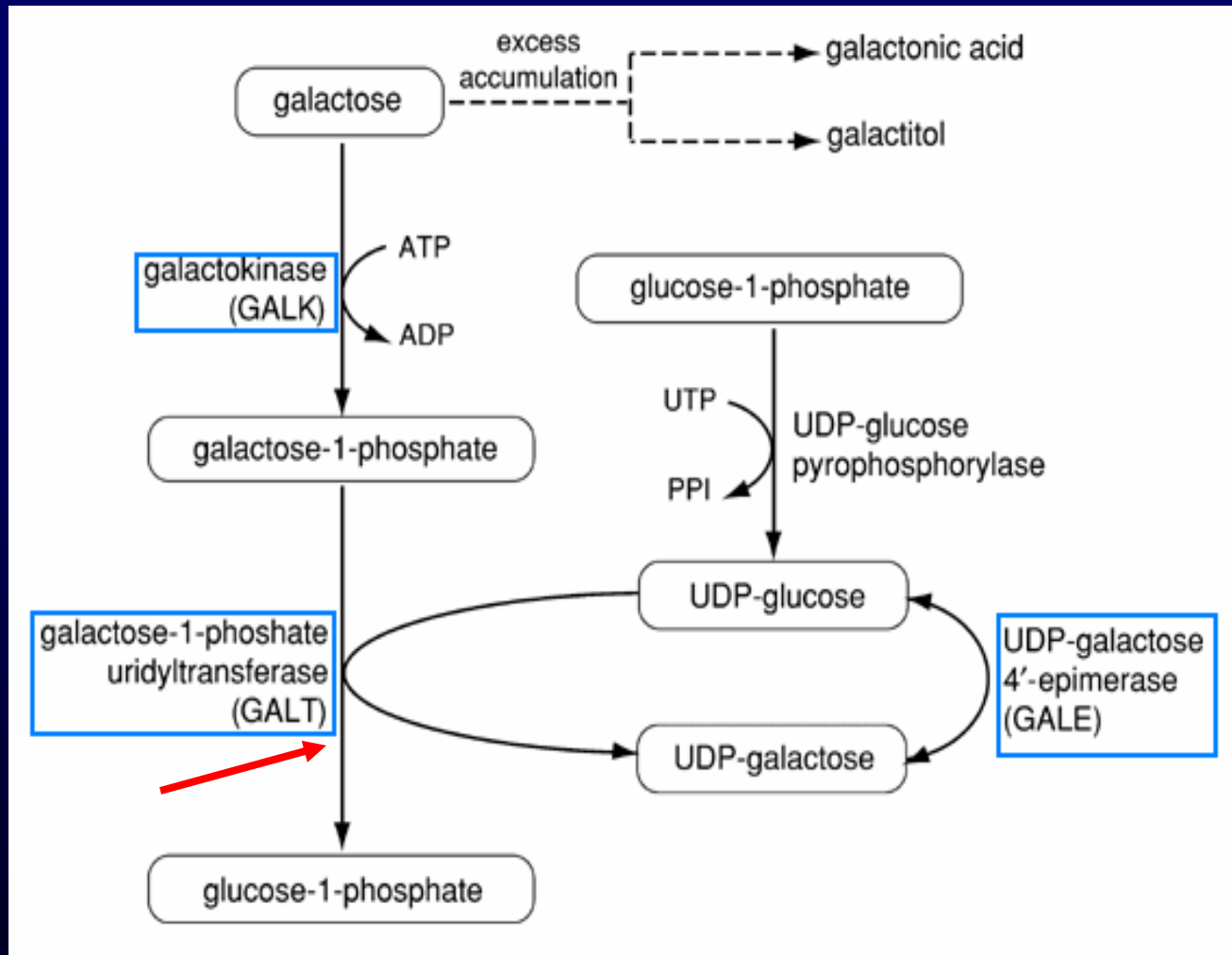
- Galactose is a monosaccharide derived from hydrolysis of lactose .
- Lactose is hydrolysed to glucose and galactose by the disaccharide lactase in the brush border of the intestine.
- Galactose is transported across the brush border membrane of the enterocyte and metabolised to glucose through a series of reactions.



# Galactosemia

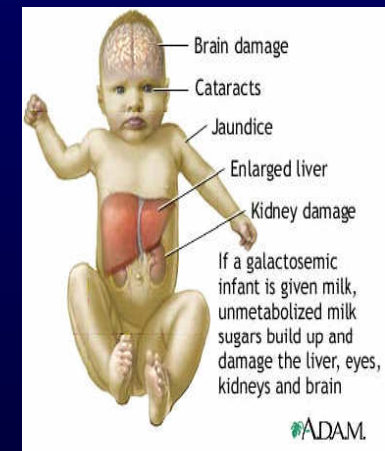
- Classical Galactosemia is an inborn error of metabolism resulting from a deficiency of the human galactose-1-phosphate uridyl transferase enzyme. Mutation in ch 9
- AR.1 in 50,000 live births
- Transferase deficiency results in more severe symptoms compared with other defects in galactokinase and epimerase deficiency.





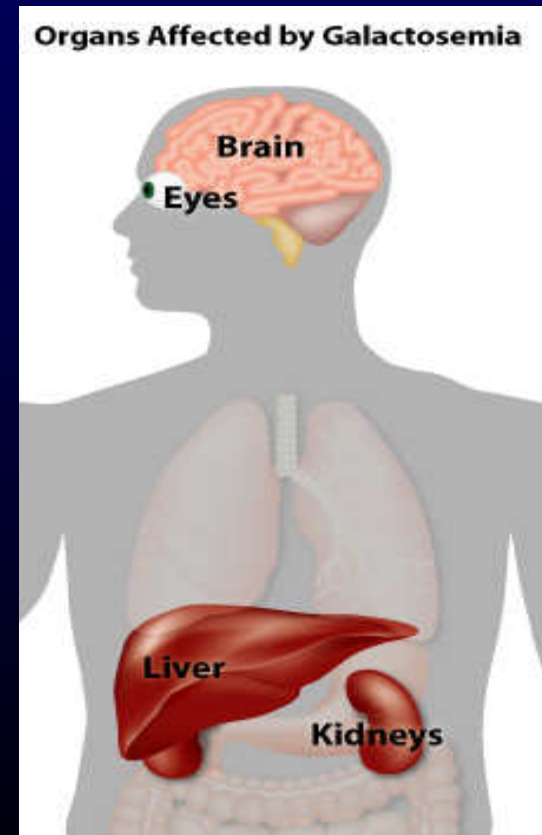
# Galactosemia

- At birth: Vomiting, diarrhea, Jaundice after milk consumption
- Hepatomegaly
- Hypoglycemia
- Failure to thrive.
- Cataracts
- Ascites - fluid accumulation in the abdomen
- High Galactose concentrations in urine



# Galactose toxicity

- Defective galactosylation of complex molecules may play a role.
- Accumulation of 2 products
- galactose 1 phosphate.
- Galactitol





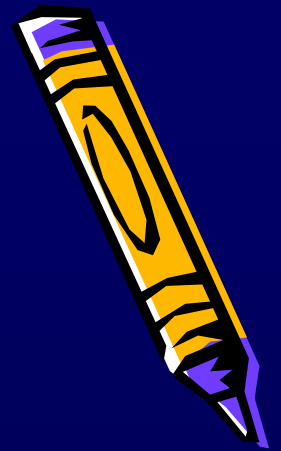
# Galactosemia

- Vomiting
- Hepatomegaly
- Lethargy
- Diarrhea
- Increased susceptibility to bacterial infections
- Hemolytic anemia
- Cataracts
- Renal Dysfunction
- Premature Ovarian Failure
- Poor growth
- Delayed speech development
- Mental retardation
- Death



# Diagnosis

- Neonatal screening available.
- The presence of reducing substances in the infant's urine with normal or low blood sugar while the infant is being fed breast milk or a formula containing lactose.
- Measurement of enzyme activity in the red blood cells (fluorometric assay and Beutler assay)
- Prenatal diagnosis by direct measurement of the enzyme galactose-1-phosphate uridyl transferase



# Galactosemia



**The treatment for galactosemia is restriction of galactose and lactose for life.**

# Dietary management

- Complete elimination is the desired goal but difficult to accomplish.
- Some have advocated galactose to be restricted to less than 125 mg /day.
- Within 72 hrs all acute symptoms show marked improvement.
- Hepatic dysfunction normalise within 1 week.
- Asymptomatic heterozygote mothers should avoid milk and milk products



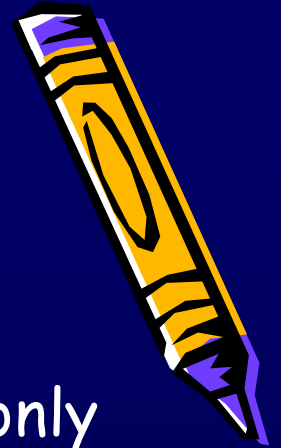
# Dietary management

- In spite of Elimination of milk and milk product:
- Ongoing galactose toxicity occurs :
- I) may be from grains, fruits and vegetables..
- Persimmon, Papaya, tomato 35, 28, 23 mg/100g
- Banana 9, apple 8, Carrot 6, potato 1.2, beetroot 0.8.
- II) Endogenous production from UDP galactose .
- Endogenous production far exceeds exogenous galactose intake which typically amounts to 20-40 mg/d on an appropriately restricted diet.



# Dietary management

- Elimination of dietary galactose is currently the only available approach to transferase enzyme deficiency
- Early diagnosis and nutritional intervention results in survival, reversal of acute symptoms and biochemical manifestations.
- Normal growth and complete normalisation of liver functions in the majority of patients.
- MR, neurologic disorders, ovarian failure and growth inhibition continue to persist in survivors.



# Conclusion

- Liver plays an important role in nutrition
- Nutrition is significantly affected in liver disease.
- Nutritional therapy is important in management of liver disease.
- Specific diets may be therapeutic or adjuncts in the management of MLD

Thank You

