

reacciones de enzimas

TABLE 15.5 Types of chemical reactions in metabolism

Type of reaction	Description
Oxidation-reduction	Electron transfer
Ligation requiring ATP cleavage	Formation of covalent bonds (i.e., carbon-carbon bonds)
Isomerization	Rearrangement of atoms to form isomers
Group transfer	Transfer of a functional group from one molecule to another
Hydrolytic	Cleavage of bonds by the addition of water
Carbon bond cleavage by means other than hydrolysis or oxidation	Two substrates yielding one product or vice versa. When H_2O or CO_2 are a product, a double bond is formed.

tipos de regulacion enzimatica

- 1) control alosterico
- 2) isoenzimas
- 3) modificacion covalente reversible
- 4) activacion proteolitica
- 5) cantidad de enzima presente

Vitaminas B

TABLE 15.3 The B vitamins

Vitamin	Coenzyme	Typical reaction type	Consequences of deficiency
Thiamine (B_1)	Thiamine pyrophosphate	Aldehyde transfer	Beriberi (weight loss, heart problems, neurological dysfunction)
Riboflavin (B_2)	Flavin adenine dinucleotide (FAD)	Oxidation-reduction	Chelosis and angular stomatitis (lesions of the mouth), dermatitis
Pyridoxine (B_6)	Pyridoxal phosphate	Group transfer to or from amino acids	Depression, confusion, convulsions
Nicotinic acid (niacin) (B_3)	Nicotinamide adenine dinucleotide (NAD^+)	Oxidation-reduction	Pellagra (dermatitis, depression, diarrhea)
Pantothenic acid (B_5)	Coenzyme A	Acyl-group transfer	Hypertension
Biotin (B_7)	Biotin-lysine adducts (biocytin)	ATP-dependent carboxylation and carboxyl-group transfer	Rash about the eyebrows, muscle pain, fatigue (rare)
Folic acid (B_9)	Tetrahydrofolate	Transfer of one-carbon components; thymine synthesis	Anemia, neural-tube defects in development
B_{12}	5'-Deoxyadenosyl cobalamin	Transfer of methyl groups; intramolecular rearrangements	Anemia, pernicious anemia, methylmalonic acidosis

Vitaminas

TABLE 15.4 Noncoenzyme vitamins

Vitamin	Function	Deficiency
A	Roles in vision, growth, reproduction	Night blindness, cornea damage, damage to respiratory and gastrointestinal tract
C (ascorbic acid)	Antioxidant	Scurvy (swollen and bleeding gums, subdermal hemorrhaging)
D	Regulation of calcium and phosphate metabolism	Rickets (children): skeletal deformities, impaired growth Osteomalacia (adults): soft, bending bones
E	Antioxidant	Lesions in muscles and nerves (rare)
K	Blood coagulation	Subdermal hemorrhaging

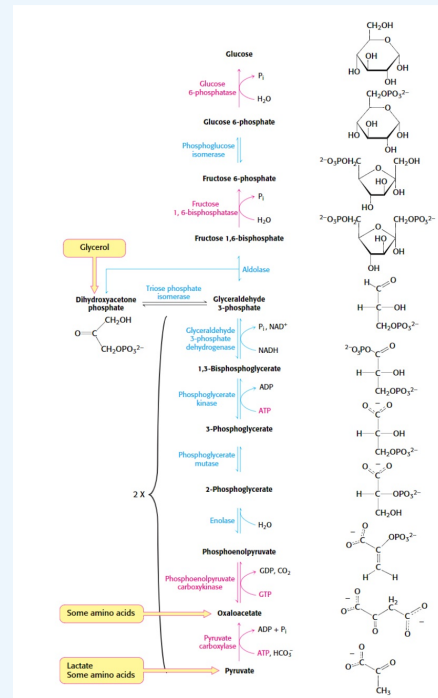
transportadores de electrones

TABLE 15.2 Some activated carriers in metabolism

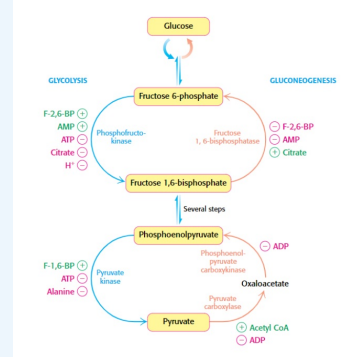
Carrier molecule in activated form	Group carried	Vitamin precursor
ATP	Phosphoryl	Nicotinate (niacin) (vitamin B_3)
NADH and NADPH	Electrons	Riboflavin (vitamin B_2)
FADH ₂	Electrons	Riboflavin (vitamin B_2)
FMN	Electrons	Riboflavin (vitamin B_2)
Coenzyme A	Acyl	Pantothenate (vitamin B_5)
Liposamide	Acyl	
Thiamine pyrophosphate	Aldehyde	Thiamine (vitamin B_1)
Biotin	CO_2	Biotin (vitamin B_7)
Tetrahydrofolate	One-carbon units	Folate (vitamin B_9)
S-Adenosylmethionine	Methyl	
Uridine diphosphate glucose	Glucose	
Cytidine diphosphate diacylglycerol	Phosphatidate	
Nucleoside triphosphates	Nucleotides	

Note: Many of the activated carriers are coenzymes that are derived from water-soluble vitamins.

Glicolisis



regulacion de glucosa



Gluconeogenesis

TABLE 16.6 Reactions of gluconeogenesis

Step	Reaction
1	Pyruvate + CO_2 + ATP + H_2O $\rightleftharpoons$ oxaloacetate + ADP + P_i + $2H^+$
2	Oxaloacetate + GTP $\rightleftharpoons$ phosphoenolpyruvate + GDP + CO_2
3	Phosphoenolpyruvate + H_2O $\rightleftharpoons$ 2-phosphoglycerate
4	2-phosphoglycerate $\rightleftharpoons$ 3-phosphoglycerate
5	3-phosphoglycerate + ATP $\rightleftharpoons$ 1,3-bisphosphoglycerate + ADP
6	1,3-bisphosphoglycerate + $NADH + H^+$ $\rightleftharpoons$ glyceraldehyde 3-phosphate + NAD^+ + P_i
7	Glyceraldehyde 3-phosphate $\rightleftharpoons$ dihydroxyacetone phosphate
8	Glyceraldehyde 3-phosphate + dihydroxyacetone phosphate $\rightleftharpoons$ fructose 1,6-bisphosphate
9	Fructose 1,6-bisphosphate + H_2O $\rightleftharpoons$ fructose 6-phosphate + P_i
10	Fructose 6-phosphate $\rightleftharpoons$ glucose 6-phosphate
11	Glucose 6-phosphate + H_2O $\rightleftharpoons$ glucose + P_i

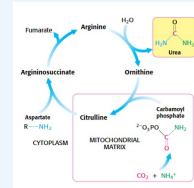
oxidacion y reduccion

Oxidant	Reductant	n	E° (V)
Acetate + CO ₂	α -Ketoglutarate	2	-0.67
Succinate	Ascorbic/Dehydro	2	-0.66
Ferredoxin (oxidized)	Ferredoxin (reduced)	1	-0.43
2H ⁺	H ₂	2	-0.82
NAD ⁺	NADH + H ⁺	2	-0.82
NADP ⁺	NADPH + H ⁺	2	-0.32
Lipote	Lipote (reduced)	2	-0.29
Gluconate (oxidized)	Gluconate (reduced)	2	-0.23
FMN	FADH ₂	2	-0.22
Ascorbic/Dehydro	Ethanol	2	-0.20
Pyruvate	Lactate	2	-0.19
2H ⁺	H ₂	2	-0.08
Formate	Succinate	2	+0.03
Cytochrome (b) (3)	Cytochrome (b) (2)	2	+0.03
Dihydroxyacetate	Acetate	2	+0.08
Ubiquinone (oxidized)	Ubiquinol (reduced)	2	+0.10
Cytochrome (c) (1)	Cytochrome (c) (2)	1	+0.22
Fe ³⁺ + 2H ⁺	Fe ²⁺ + 2H ⁺	1	+0.77
		2	+0.82

Note: E° is the standard reduction potential (pH 7, 25°C) and the number of electrons transferred. The values are for the partial reaction shown as 'Oxidant' $\rightarrow$ 'reductant'.

^aStandard oxidation – reduction potential at pH = 0.

ciclo de urea



ciclo dea cido citrico

TABLE 21.2 Glycogen-storage diseases

Type	Inductive enzyme	Organ affected	Chaperon	Clinical features
V	Choline α -phosphatase	Liver and kidney	Increased amount in normal structure	Massive enlargement of the liver; failure to thrive, severe hypoproteinaemia, hypoglycaemia, hypocalcaemia, hypocalciuria
VI	Phosphatase	All organs	Massive increase in normal structure	Massive enlargement of the liver; hypoproteinaemia, hypocalcaemia, hypocalciuria
III	α -1,6-Glucosidase	Muscle and liver	Normal amount	Large liver, mild hypocalcaemia
IV	α -1,6-Glucosidase	Muscle and liver	Normal amount	Large liver, mild hypocalcaemia
IVb	Branching enzyme	Liver and spleen	Normal amount, very long incubation period	Progressive enlargement of the liver, hypoproteinaemia, hypocalcaemia, hypocalciuria
V	Phosphatase	All organs	Normal amount	Large liver, mild hypocalcaemia
Va	Phosphatase	Moderately increased amount	Normal structure	Large liver, mild hypocalcaemia
VI	Phosphatase	Liver	Increased amount	Large liver
VII	Phosphatidylcholine kinase	Muscle	Increased amount, normal structure	Large liver, mild hypocalcaemia
VIII	Phosphatidylcholine kinase	Liver	Increased amount, normal structure	Mild liver enlargement, mild hypocalcaemia

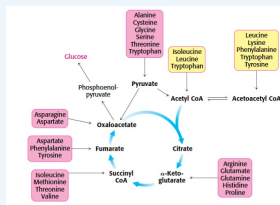
Note: Types I through VII are inherited as autosomal recessives. Type VIII is sex linked.

pentosas fosfato

TABLE 20.3 Pentose phosphate pathway

Reaction	Enzyme
Oxidative phase	
Glucose 5-phosphate + NADP ⁺ → 6-phosphogluconic acid lactone + NADPH + H ⁺	Glucose 6-phosphate dehydrogenase
6-phosphogluconic acid lactone + H ₂ O → 6-phosphogluconate + H ⁺	Lactonase
6-phosphogluconate + NADP ⁺ → ribulose 5-phosphate + CO ₂ + NADPH + H ⁺	6-phosphogluconate dehydratase
Nonoxidative phase	
Ribulose 5-phosphate → ribose 5-phosphate	Phosphopentose isomerase
Ribulose 5-phosphate → xylulose 5-phosphate	Phosphopentose epimerase
Ribulose 5-phosphate + xylulose 5-phosphate → sedoheptulose 7-phosphate + glyceraldehyde 3-phosphate	Transketolase
Sedoheptulose 7-phosphate + glyceraldehyde 3-phosphate → fructose 6-phosphate + erythrose 4-phosphate	Transaldolase
Xylulose 5-phosphate + erythrose 4-phosphate → fructose 6-phosphate + glyceraldehyde 3-phosphate	Transketolase

integracion de esqueletos de amino acidos



componentes etc

TABLE 18.2 Components of the mitochondrial electron-transport chain

Enzyme complex	Mass (kDa)	Subunits	Prosthetic group	Matrix side	Oxidant or reductant	Cytoplasmic side
NADH:Q oxidoreductase	>900	46	FMN Fe-S	NADH	Q	
Succinate:Q reductase	140	4	FAD Fe-S	Succinate	Q	
Q-cytochrome c oxidoreductase	250	11	Heme b _L Heme b _H Heme c ₁ Fe-S		Q	Cytochrome c
Cytochrome c oxidase	160	13	Heme a Heme a ₃ Cu ₂ and Cu ₁			Cytochrome c

Information from: J. W. DePierre and L. Ernster, *Ann. Rev. Biochem.* 46:215, 1977; Y. Hatfi, *Ann. Rev. Biochem.* 54:1015, 1985; and J. E. Walker, *Q. Rev. Biophys.* 25:253, 1992.

pasos en síntesis de Amino Ácidos

- 1) debe haber nitrogeno reactivo $\text{NH}_3 \rightarrow \text{NH}_4$
- 2) esqueletos carbonados (de glicolisis, ciclo de krebs, & pentosas fosfato)

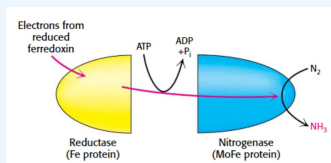
Principales formas de fijacion de nitrogeno

- 1) rhizobium 60%
- 2) Fritz Haber 15%
- 3) Rayos UV 25%

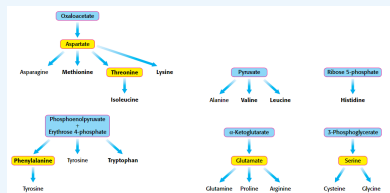
rhizobium

hacen fijacion de nitrogeno ($N_2 \rightarrow NH_3$).
son anaerobicos y simbioticos con plantas

complejo fijador de nitrógeno



familias biosintéticas de amino ácidos



20 amino acidos

ESENCIALEA	NO ESENCIALES
histidina	alanina
isoleucina	arginina
leucina	asparagina
lysina	aspartato
metionina	cisteina
fenilalanina	glutamato
treonina	glutamina
triptofano	glicina
valina	prolina
	serina
	tirosina

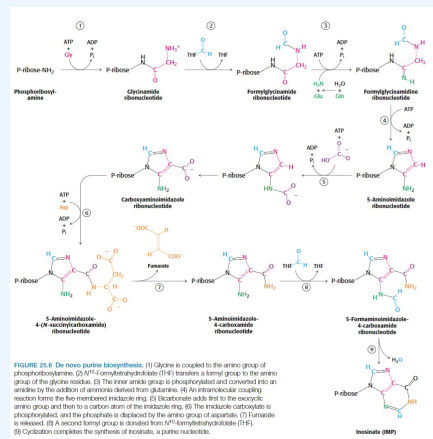
enfermedades de metabolismo de amino acidos

Disease	Enzyme deficiency	Symptoms
Citrullinemia	Argininosuccinate lyase	Lethargy, seizures, reduced muscle tension
Tyrosinemia	Various enzymes of tyrosine degradation	Weakness, liver damage, mental retardation
Albinism	Tyrosinase	Absence of pigmentation
Homocystinuria	Cystathionine β -synthase	Scoliosis, muscle weakness, mental retardation, this blood fair
Hyperphenylsermia	α -Aminoadipic semialdehyde dehydrogenase	Seizures, mental retardation, lack of muscle tone, ataxia

enzimas en síntesis de novo

Step	Enzyme
1	Glycinamide ribonucleotide (GAR) synthetase
2	GAR transformylase
3	Formylglycinamide synthase
4	Aminimidase ribonucleotide synthetase
5	Carbamoylaminimidase ribonucleotide synthetase
6	Succinylaminimidase carbamoylase ribonucleotide synthetase
7	Adenylosuccinate lyase
8	Aminimidase carbamoylase ribonucleotide transformylase
9	Isoicase monophosphate cyclohydrolase

biosíntesis de purimidinas



síntesis de ácidos grasos

Step	Reaction	Enzyme
1	$\text{Acetyl CoA} + \text{HCO}_3^- + \text{ATP} \rightarrow \text{malonyl CoA} + \text{ADP} + \text{P}_i + \text{H}^+$	Acetyl CoA carboxylase
2	$\text{Acetyl CoA} + \text{ACP} \rightleftharpoons \text{acetyl ACP} + \text{CoA}$	Acetyl transacylase
3	$\text{Malonyl CoA} + \text{ACP} \rightleftharpoons \text{malonyl ACP} + \text{CoA}$	Malonyl transacylase
4	$\text{Acetyl ACP} + \text{malonyl ACP} \rightarrow \text{acetoacetyl ACP} + \text{ACP} + \text{CO}_2$	β -Ketoacyl synthase
5	$\text{Acetoacetyl ACP} + \text{NADPH} + \text{H}^+ \rightarrow \text{hydroxyacyl ACP} + \text{NADP}^+$	β -Ketoacyl reductase
6	$3\text{-hydroxyacyl ACP} \rightleftharpoons \text{crotonyl ACP} + \text{H}_2\text{O}$	Hydroxyacyl dehydratase
7	$\text{Crotonyl ACP} + \text{NADPH} + \text{H}^+ \rightarrow \text{butyryl ACP} + \text{NADP}^+$	Enoyl reductase

oxidacion acidos grasos

Step	Reaction	Enzyme
1	Fatty acid + CoA + ATP $\rightleftharpoons$ acyl CoA + AMP + PP _i	Acyl CoA synthetase (also called fatty acyl-CoA ligase)*
2	Camelate + acyl CoA $\rightleftharpoons$ acyl camelate + CoA	Camelate acyltransferase (also called camelate transferase)
3	Acyl CoA + FAD $\rightarrow$ trans- Δ^2 -enoyl CoA + FADH ₂	Acyl CoA dehydrogenase (several isozymes have different chain-length specificity)
4	trans- Δ^2 -Enoyl CoA + H ₂ O $\rightleftharpoons$ 3-hydroxyenoyl CoA	Enoyl CoA hydratase (also called enoylase or 3-hydroxyenoyl CoA hydratase)
5	3-hydroxyenoyl CoA + NAD ⁺ $\rightleftharpoons$ 3-ketoacyl CoA + NADH + H ⁺	3-hydroxyenoyl CoA dehydrogenase
6	3-ketoacyl CoA + CoA $\rightleftharpoons$ acyl CoA + acyl CoA (shortened by 2 C)	β -ketoacyl CoA (also called thiolase)

* Acyl CoA synthetase is also called acyl-CoA synthetase.

lipoproteínas del plasma

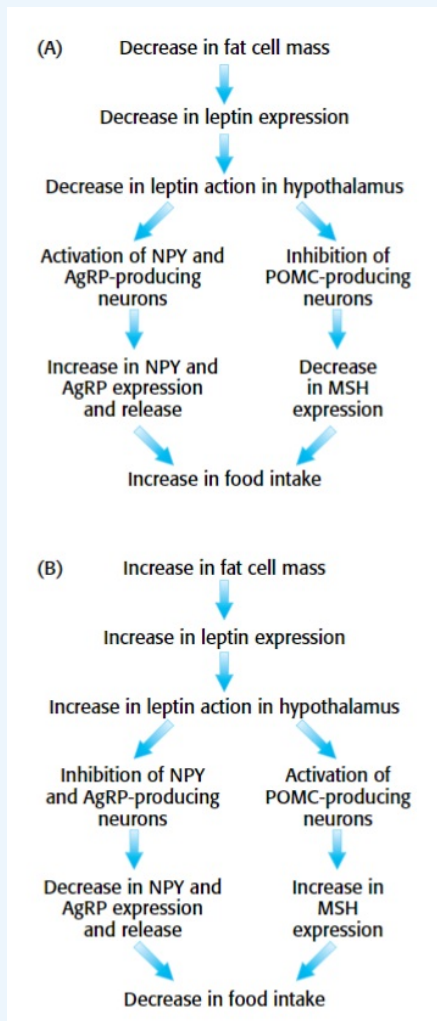
Plasma lipoproteins	Density (g ml ⁻¹)	Diameter (nm)	Apolipoprotein	Physiological role	Composition (%)				
					TAG	CE	C	PL	P
Chylomicrons	0.915–0.96	75–120	B-4, C-1, E	Dietary lipid transport	86	3	1	8	
Very low density lipoproteins	0.95–1.006	30–80	B-100, C-1, E	Endogenous lipid transport	52	14	7	18	
Intermediate-density lipoproteins	1.06–1.019	15–35	B-100, E	LDL precursor	38	30	8	23	1
Low-density lipoproteins	1.019–1.063	18–25	B-100	Cholesterol transport	10	38	8	22	1
High-density lipoproteins	1.063–1.21	7.5–20	A	Reverse cholesterol transport	5–10	14–21	3–7	19–29	35–50

regulacion de apetito

Appetite-suppressing signals
Cholecystokinin
Ghrelin-like peptide 1
Ghrelin-like peptide 2
Amylin
Semaglutin
Bombesin
Enterostatin
Apolipoprotein A IV
Gastric inhibitory peptide
Appetite-enhancing peptides
Ghrelin

Information from M. H. Sipman, ed., *Biochemical, Physiological, Molecular Aspects of Human Nutrition*, 2nd ed. Saunders/Elsevier, 2001, p. 877, Table 27.2.

efecto de leptina



consumo de metabolitos

TABLE 27.5 Fuel metabolism in starvation

	Amount formed or consumed in 24 hours (grams)	
	3d day	40th day
Fuel exchanges and consumption		
Fuel use by the brain		
Glucose	100	40
Ketone bodies	50	100
All other use of glucose	50	40
Fuel mobilization		
Adipose-tissue lipolysis	180	180
Muscle-protein degradation	75	20
Fuel output of the liver		
Glucose	150	80
Ketone bodies	150	150

reservas de metabolitos

TABLE 27.4 Fuel reserves in a typical 70-kg man

Organ	Available energy in kilojoules (kcal)		
	Glucose or glycogen	Triacylglycerols	Mobilizable proteins
Blood	250 (60)	20 (45)	0 (0)
Liver	1700 (400)	2000 (450)	1700 (400)
Brain	30 (8)	0 (0)	0 (0)
Muscle	5000 (1200)	2000 (450)	100,000 (24,000)
Adipose tissue	330 (80)	560,000 (135,000)	170 (40)

Data from G. F. Cahill, Jr., *Clin. Endocrinol. Metab.* 5:398, 1976.