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 allosterico

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	Cheliosis 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

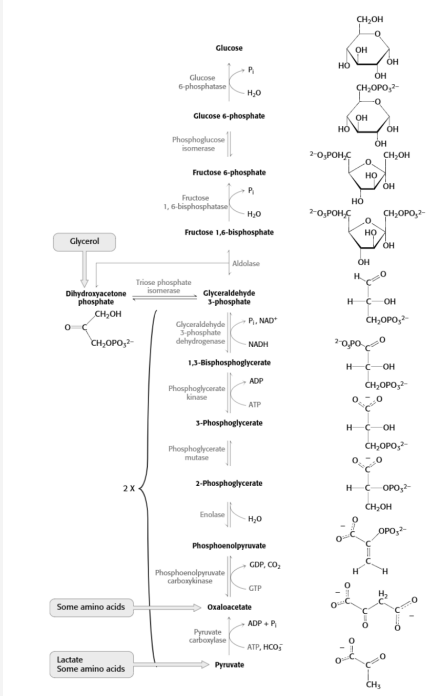
transportadores de electrones

TABLE 15.2 Some activated carriers in metabolism

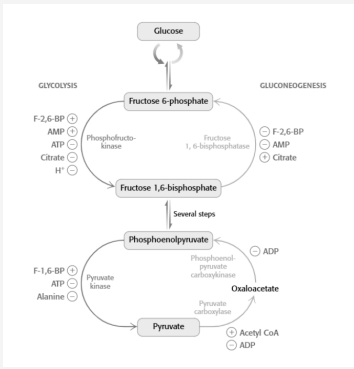
Carrier molecule in activated form	Group carried	Vitamin precursor
ATP	Phosphoryl	
NADH and NADPH	Electrons	Nicotinate (niacin) (vitamin B_3)
$FADH_2$	Electrons	Riboflavin (vitamin B_2)
$FMNH_2$	Electrons	Riboflavin (vitamin B_2)
Coenzyme A	Acyl	Pantothenate (vitamin B_5)
Lipoamide	Acyl	
Thiamine pyrophosphate	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 ⟶ oxaloacetate + ADP + P_i + $2H^+$
2	Oxaloacetate + GTP ⟺ phosphoenolpyruvate + GDP + CO_2
3	Phosphoenolpyruvate + H_2O ⟺ 2-phosphoglycerate
4	2-Phosphoglycerate ⟺ 3-phosphoglycerate
5	3-Phosphoglycerate + ATP ⟺ 1,3-bisphosphoglycerate + ADP
6	1,3-Bisphosphoglycerate + NADH + H^+ ⟺ glyceraldehyde 3-phosphate + NAD^+ + P_i
7	Glyceraldehyde 3-phosphate ⟺ dihydroxyacetone phosphate
8	Glyceraldehyde 3-phosphate + dihydroxyacetone phosphate ⟺ fructose 1,6-bisphosphate
9	Fructose 1,6-bisphosphate + H_2O ⟶ fructose 6-phosphate + P_i
10	Fructose 6-phosphate ⟺ glucose 6-phosphate
11	Glucose 6-phosphate + H_2O ⟶ + glucose + P_i

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oxidacion y reduccion

TABLE 18.1 Standard reduction potentials of some reactions

Oxidant	Reductant	n	E_0' (V)
Succinate + CO_2	α-Ketoglutarate	2	−0.67
Acetate	Acetaldehyde	2	−0.60
Ferredoxin (oxidized)	Ferredoxin (reduced)	1	−0.43
$2\,H^+$	H_2	2	−0.42
NAD^+	$NADH + H^+$	2	−0.32
$NADP^+$	$NADPH + H^+$	2	−0.32
Lipoate (oxidized)	Lipoate (reduced)	2	−0.29
Glutathione (oxidized)	Glutathione (reduced)	2	−0.23
FAD	$FADH_2$	2	−0.22
Acetaldehyde	Ethanol	2	−0.20
Pyruvate	Lactate	2	−0.19
$2\,H^+$	H_2	2	−0.00[1]
Fumarate	Succinate	2	+0.03
Cytochrome b (+3)	Cytochrome b (+2)	1	+0.07
Dehydroascorbate	Ascorbate	2	+0.08
Ubiquinone (oxidized)	Ubiquinone (reduced)	2	+0.10
Cytochrome c (+3)	Cytochrome c (+2)	1	+0.22
Fe (+3)	Fe (+2)	1	+0.77
$\frac{1}{2}O_2 + 2\,H^+$	H_2O	2	+0.82

Note: E_0' is the standard oxidation–reduction potential (pH 7, 25°C) and n is the number of electrons transferred. E_0' refers to the partial reaction written as Oxidant + e^- ⟶ reductant
[1]Standard oxidation – reduction potential at pH = 0.

enfermedades de almacenamiento de glucosa

TABLE 21.2 Glycogen-storage diseases

Type	Defective enzyme	Organ affected	Glycogen in the affected organ	Clinical features
I Von Gierke	Glucose 6-phosphatase or transport system	Liver and kidney	Increased amount; normal structure.	Massive enlargement of the liver; failure to thrive; severe hypoglycemia, ketosis, hyperuricemia, hyperlipemia.
II Pompe	α-1,4-Glucosidase (lysosomal)	All organs	Massive increase in amount; normal structure.	Cardiorespiratory failure causes death, usually before age 2.
III Cori	α-1,6-Glucosidase (debranching enzyme)	Muscle and liver	Increased amount; short outer branches.	Like type I, but milder course.
IV Andersen	Branching enzyme (α-1,4⟶α-1,6)	Liver and spleen	Normal amount; very long outer branches.	Progressive cirrhosis of the liver; liver failure causes death, usually before age 2.
V McArdle	Phosphorylase	Muscle	Moderately increased amount; normal structure.	Limited ability to perform strenuous exercise because of painful muscle cramps; otherwise patient is normal and well developed.
VI Hers	Phosphorylase	Liver	Increased amount.	Like type I, but milder course.
VII	Phosphofructokinase	Muscle	Increased amount; normal structure.	Like type V.
VIII	Phosphorylase kinase	Liver	Increased amount; normal structure.	Mild liver enlargement; mild hypoglycemia.

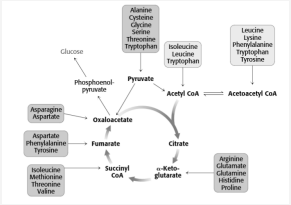
Note: Types I through VII are inherited as autosomal recessives. Type VIII is sex linked.

pentosas fosfato

TABLE 20.3 Pentose phosphate pathway

Reaction	Enzyme
Oxidative phase	
Glucose 6-phosphate + $NADP^+$ ⟶ 6-phosphoglucono-δ-lactone + NADPH + H^+	Glucose 6-phosphate dehydrogenase
6-Phosphogluconо-δ-lactone + H_2O ⟶ 6-phosphogluconate + H^+	Lactonase
6-Phosphogluconate + $NADP^+$ ⟶ ribulose 5-phosphate + CO_2 + NADPH + H^+	6-Phosphogluconate dehydrogenase
Nonoxidative Phase	
Ribulose 5-phosphate ⟺ ribose 5-phosphate	Phosphopentose isomerase
Ribulose 5-phosphate ⟺ xylulose 5-phosphate	Phosphopentose epimerase
Xylulose 5-phosphate + ribose 5-phosphate ⟺ sedoheptulose 2-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



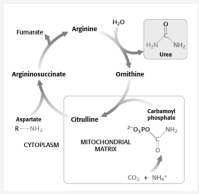
componentes etc

TABLE 18.2 Components of the mitochondrial electron-transport chain

Enzyme complex	Mass (kDa)	Subunits	Prosthetic group	Oxidant or reductant: Matrix side	Oxidant or reductant: Membrane core	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_H Heme b_L Heme c_1 Fe-S		Q	Cytochrome c
Cytochrome c oxidase	160	13	Heme a Heme a_3 Cu_A and Cu_B			Cytochrome c

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

ciclo de urea



ciclo dea cido citrico

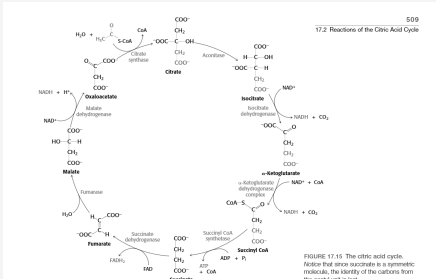


FIGURE 17.15 The citric acid cycle. Notice that since succinate is a symmetric molecule, the identity of the carbons from the acetyl unit is lost.

TABLE 17.2 Citric acid cycle

Step	Reaction	Enzyme	Prosthetic group	Type*	$\Delta G'^\circ$: kJ mol^{-1}	$\Delta G'^\circ$: kcal mol^{-1}
1	Acetyl CoA + oxaloacetate + H_2O ⟶ citrate + CoA + H^+	Citrate synthase		a	−31.4	−7.5
2a	Citrate ⟺ cis-aconitate + H_2O	Aconitase	Fe-S	b	+8.4	+2.0
2b	cis-Aconitate + H_2O ⟺ isocitrate	Aconitase	Fe-S	c	−2.1	−0.5
3	Isocitrate + NAD^+ ⟺ α-ketoglutarate + CO_2 + NADH	Isocitrate dehydrogenase		d + e	−8.4	−2.0
4	α-Ketoglutarate + NAD^+ + CoA ⟺ succinyl CoA + CO_2 + NADH	α-Ketoglutarate dehydrogenase complex	Lipoic acid, FAD, TPP	d + e	−30.1	−7.2
5	Succinyl CoA + P_i + ADP ⟺ succinate + ATP + CoA	Succinyl CoA synthetase		f	−3.3	−0.8
6	Succinate + FAD (enzyme-bound) ⟺ fumarate + $FADH_2$ (enzyme-bound)	Succinate dehydrogenase	FAD, Fe-S	e	0	0
7	Fumarate + H_2O ⟺ L-malate	Fumarase		c	−3.8	−0.9
8	L-Malate + NAD^+ ⟺ oxaloacetate + NADH + H^+	Malate dehydrogenase		e	+29.7	+7.1

*Reaction type: (a) condensation; (b) dehydration; (c) hydration; (d) decarboxylation; (e) oxidation; (f) substrate-level phosphorylation.

pasos en sintesis de Amino Acidos

1) debe haber nitrogeno reactivo NH3->NH4

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 (N2->NH3).
son anaerobicos y simbioticos con plantas

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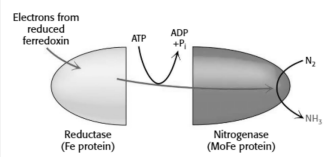
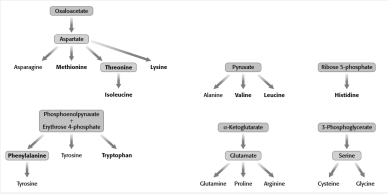
complejo fijador de nitrogeno



familias biositnteticas de amino acidos



20 amino acidos

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

enefermedades de metabolismo de amino acidos

TABLE 23.4 Inborn errors of amino acid metabolism

Disease	Enzyme deficiency	Symptoms
Citrullinemia	Arginosuccinate 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, thin blood hair
Hyperlysinemia	α-Aminoadipic semialdehyde dehydrogenase	Seizures, mental retardation, lack of muscle tone, ataxia

enzimas en sintesis de novo

TABLE 25.2 The enzymes of de novo purine synthesis

Step	Enzyme
1	Glycinamide ribonucleotide (GAR) synthetase
2	GAR transformylase
3	Formylglycinamidine synthase
4	Aminoimidazole ribonucleotide synthetase
5	Carboxyaminoimidazole ribonucleotide synthetase
6	Succinylaminoimidazole carboxamide ribonucleotide synthetase
7	Adenylosuccinate lyase
8	Aminoimidazole carboxamide ribonucleotide transformylase
9	Inosine monophosphate cyclohydrolyase

biosintesis de purimidinas

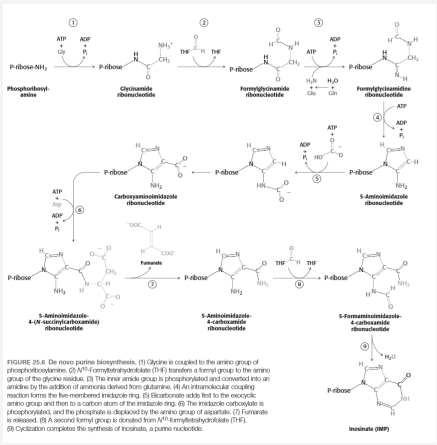


FIGURE 25.6 De novo purine biosynthesis. (1) Glycine is coupled to the amino group of phosphoribosylamine. (2) N^{10}-Formyltetrahydrofolate (THF) transfers a formyl group to the amino group of the glycine residue. (3) The inner amide group is phosphorylated and converted into an amidine by the addition of ammonia derived from glutamine. (4) An intramolecular coupling reaction forms the five-membered imidazole ring. (5) Bicarbonate adds first to the exocyclic amino group and then to a carbon atom of the imidazole ring. (6) The imidazole carboxylate is phosphorylated, and the phosphate is displaced by the amino group of aspartate. (7) Fumarate is released. (8) A second formyl group is donated from N^{10}-formyltetrahydrofolate (THF). (9) Cyclization completes the synthesis of inosinate, a purine nucleotide.

sintesis de acidos grasos

TABLE 22.2 Principal reactions in fatty acid synthesis in bacteria

Step	Reaction	Enzyme
1	Acetyl CoA + HCO_3^- + ATP ⟶ malonyl CoA + ADP + P_i + H^+	Acetyl CoA carboxylase
2	Acetyl-CoA + ACP⟺ acetyl ACP + CoA	Acetyl transacylase
3	Malonyl CoA + ACP⟺ malonyl ACP + CoA	Malonyl transacylase
4	Acetyl ACP + malonyl ACP ⟶ acetoacetyl ACP + ACP + CO_2	β-Ketoacyl synthase
5	Acetoacetyl ACP + NADPH + H^+ ⟺ D-3-hydroxybutyryl ACP + $NADP^+$	β-Ketoacyl reductase
6	D-3-Hydroxybutyryl ACP⟺ crotonyl ACP + H_2O	3-Hydroxyacyl dehydratase
7	Crotonyl ACP + NADPH + H^+ ⟶ butyryl ACP + $NADP^+$	Enoyl reductase

oxidacion acidos grasos

TABLE 22.1 Principal reactions in fatty acid oxidation

Step	Reaction	Enzyme
1	Fatty acid + CoA + ATP⟺ acyl CoA + AMP + PP_i	Acyl CoA synthetase (also called fatty acid thiokinase and fatty acid CoA ligase)*
2	Carnitine + acyl CoA⟺ acyl carnitine + CoA	Carnitine acyltransferase (also called carnitine palmitoyl transferase)
3	Acyl CoA + E-FAD ⟶ trans-Δ^2-enoyl CoA + E-$FADH_2$	Acyl CoA dehydrogenases (several isozymes having different chain-length specificity)
4	trans-Δ^2-Enoyl CoA + H_2O⟺ L-3-hydroxyacyl CoA	Enoyl CoA hydratase (also called crotonase or 3-hydroxyacyl CoA hydrolyase)
5	L-3-Hydroxyacyl CoA + NAD^+ ⟺ 3-ketoacyl CoA + NADH + H^+	L-3-Hydroxyacyl CoA dehydrogenase
6	3-Ketoacyl CoA + CoA⟺ acetyl CoA + acyl CoA (shortened by C_2)	β-Ketothiolase (also called thiolase)

*An AMP-forming ligase.

lipoproteinas del plasma

TABLE 26.1 Properties of plasma lipoproteins

Plasma lipoproteins	Density (g ml^{-1})	Diameter (nm)	Apolipoprotein	Physiological role	Composition (%) TAG	CE	C	PL	P
Chylomicron	<0.95	75–1200	B-48, C, E	Dietary fat transport	86	3	1	8	2
Very low-density lipoprotein	0.95–1.006	30–80	B-100, C, E	Endogenous fat transport	52	14	7	18	9
Intermediate-density lipoprotein	1.006–1.019	15–35	B-100, E	LDL precursor	38	30	8	23	11
Low-density lipoprotein	1.019–1.063	18–25	B-100	Cholesterol transport	10	38	8	22	21
High-density lipoprotein	1.063–1.21	7.5–20	A	Reverse cholesterol transport	5–10	14–21	3–7	19–29	33–57

Abbreviations: TAG, triacylglycerol; CE, cholesteryl ester; C, free cholesterol; PL, phospholipid; P, protein.

regulacion de apetito

TABLE 27.2 Gastrointestinal peptides that regulate food intake

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

Information from: M. H. Stipanuk, ed., *Biochemical, Physiological, Molecular Aspects of Human Nutrition*, 2d ed. (Saunders/Elsevier, 2006), p. 627, Box 22-1.

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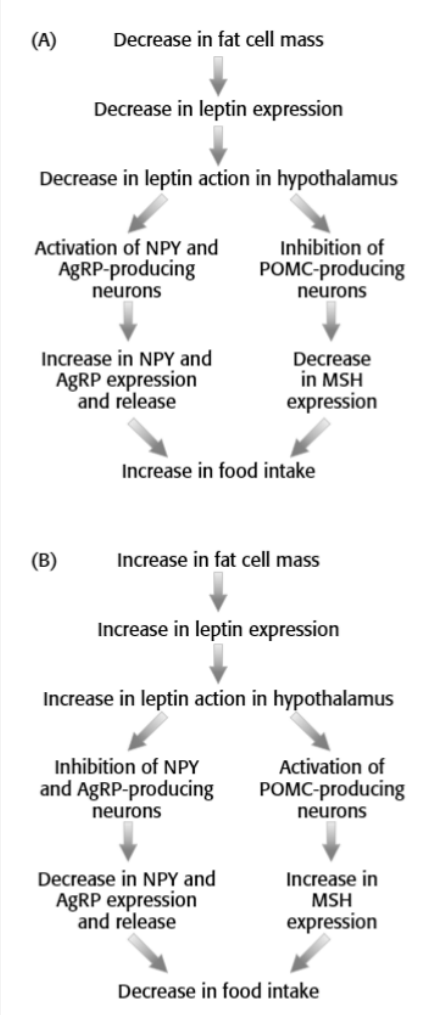
efecto de leptina



consumo de metabolitos

TABLE 27.5 Fuel metabolism in starvation

Fuel exchanges and consumption	Amount formed or consumed in 24 hours (grams) 3d day	40th day
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.

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