Concise Encyclopedia of Biochemistry

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de Gruyter

Concise Encyclopedia of Biochemistry

Walter de Gruyter
Berlin · New York 1983

Title of the Original, German Language Edition
Brockhaus ABC Biochemie
edited by
H.-D. Jakubke, Prof. Dr. rer. nat. habil.
H. Jeschkeit, Dr. rer. nat. habil.
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First printing, March 1983

Second printing with corrections
October 1983

CIP-Kurztitelaufnahme der Deutschen Bibliothek

Concise encyclopedia of biochemistry / [transl. into Engl., rev. and enl. by Thomas Scott; Mary Brewer]. – Berlin; New York: de Gruyter, 1983.
Einheitssacht.: Brockhaus -ABC Biochemie 'engl.'
ISBN 3-11-007860-0

NE: Scott, Thomas [Bearb.]; EST

Library of Congress Cataloging in Publication Data

Brockhaus ABC Biochemie. English.
Concise encyclopedia of biochemistry.

Translation of: Brockhaus ABC Biochemie.
1. Biological chemistry--Dictionaries. I. Scott, Thomas, 1935- II. Brewer, Mary. III. Title.
[DNLM: 1. Biochemistry--Dictionaries. QU 13 C744]
QD415.B713 1983 574.19'2'0321 82-22148
ISBN 3-11-007860-0

English Language Edition
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Typesetting: Dörlemann-Satz, Lemförde. – Printing: Karl Gerike, Berlin – Binding: Dieter Mikolai, Berlin. – Coverdesign: Rudolf Hübler, Berlin. **Printed in Germany.**

Verlag Walter de Gruyter & Co., Genthiner Str. 13, D-1000 Berlin 30, Tel.: (030) 26005-0, Telex 184027
Walter de Gruyter, Inc., 200 Saw Mill River Road, Hawthorne, N.Y. 10532, Tel.: (914) 747-0110, Telex 646677

Preface

The "Brockhaus ABC Biochemie" was first published in 1976 in Leipzig, the second edition followed in 1981. When we undertook to translate this book, based on the second German edition, it was clear that our work would also involve considerable updating of existing entries and the introduction of new material. Such a task can, of course, never be complete. It is a rare and fortunate author or editor in the life sciences, and particularly in biochemistry, whose material is still completely up to date at the time of publication; progress in this field is so rapid and shows no sign of abating. Therein, however, lies the excitement and challenge of this venture. Already we have started collecting, classifying and revising in preparation for a subsequent edition.

We have departed from the style of the German edition by quoting a few literature references. These have been included with some of the new material, and we hope they will be useful to readers who want more information than can be fitted into a work of this sort. Where possible, we have also given each enzyme its EC (Enzyme Commission) Number, according to the Recommendations (1978) of the Nomenclature Committee of the International Union of Biochemistry (published in "Enzyme Nomenclature" Academic Press, 1979).

We apologize to any biochemist whose pet compound, mechanism or pathway has been overlooked, and we should be grateful to receive suggestions for new entries. It is also recognized that a reference work should reach into the past, defining terms no longer used, but encountered when using the older literature. In this respect, suggestions from our more "senior" readers would be most welcome.

Finally, thanks are due to Dr. Rudolf Weber of de Gruyter Publishers for his guidance and encouragement in the preparation of the manuscript and the production of this book.

January 1983

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Using this book

Cross referencing is indicated by the word "see", and the subject of the cross reference starts with a high case letter, e.g. . . . in the Posttranslational modification of proteins (see), or . . . see Enzyme induction. Numbers, Greek letters and configurational letters at the beginning of names are ignored in the allocation of alphabetical order, e.g. β-Galactosidase is listed under G; L-Histidine under H; *N*-2-Hydroxyethylpiperazine . . . under H. The main entry title is printed in bold type, followed by synonyms in bold italics. The remaining text uses only two further types, normal and italics.

Abbreviations: (The standard biochemical abbreviations, e.g. ATP, NAD, etc. are found as entries in the appropriate alphabetical positions).

abb.	abbreviation
$[\alpha]$	specific optical rotation
b.p.	boiling point
c	concentration
°C	degrees Celsius
(d.)	with decomposition
ρ	density
IP	isoelectric point
M	molar
m.p.	melting point
M_r	relative molecular mass
n	refractive index
syn.	synonym

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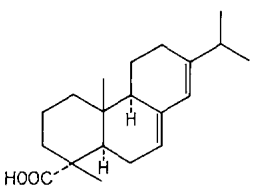
A: abb. for adenine; abb. for absorbance.

Å: Angstrom unit.

AAR: abb. for Antigen-antibody reaction (see).

A band: a transverse dark band, consisting of thick and thin filaments, seen in electron microscope preparations of myofibrils from striated muscle.

Abietic acid: a diterpene carboxylic acid, M_r 302.46, m.p. 173 to 175 °C, b. $p._{9.5}$ 248–250 °C,$[\alpha]_D$ – 106 °C (alcohol). A.a. and the isomeric neoabietic acid, m.p. 171 to 173 °C, $[\alpha]_D$ + 161° (alcohol) can easily be interconverted. These two resin acids are the main components of rosin (up to 90%), from which they are obtained by treatment with heat or acids, possibly as products of the rearrangement of other diterpene carboxylic acids. Amber contains derivatives of A.a.



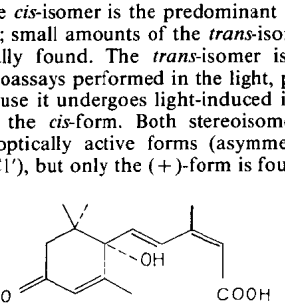
Abietic acid

Abrin: see Ricin.

Abscisic acid, abb. ***ABA, abscisin, dormin***: (S)-(+)-5-(1′-hydroxy-4′-oxo-2′,6′,6′-trimethyl-2-cyclohexen-1-yl)-3-methyl-*cis, trans*-2,4-pentadienoic acid, a widely occurring, sesquiterpene plant hormone. Its action is mainly inhibitory. M_r 264.3, m.p. 160 to 162 °C,$[\alpha]_D$ + 430°, λ_{max}260 nm. ABA appears to be ubiquitous in plants and acts as antagonist to the auxins, gibberellins and cytokinins. It inhibits growth and the germination of seeds. It induces dormancy in seeds and promotes the falling of leaves and fruits. It is thus present in relatively large quantities in fruits, dormant seeds, buds and wilting leaves. The β-D-glucose ester of ABA has been found in the yellow lupine (*Lupinus luteus*), rose (*Rosa*), beans (*Phaseolus*) and maple (*Acer pseudoplatanus*). It is assayed both spectroscopically and by biological tests based on its growth-inhibiting properties.

The biosynthesis of ABA is still unknown. A direct path from isopentenyl pyrophosphate via geranyl and farnesyl pyrophosphate, or formation from carotenoids by photochemical cleavage of violaxanthin via xanthoxin have both been proposed.

It was first isolated in 1963 from cotton bolls (9 mg/75 kg dry matter) by Addicot and Lyon and by Wareing from maple leaves (0.27 mg/27 kg dry matter). Its structure was determined in 1965. It exists in two stereoisomeric forms, depending on the *cis* or *trans* orientation of the $\Delta^{2,3}$ double bond. The *cis*-isomer is the predominant form in all plants; small amounts of the *trans*-isomer are occasionally found. The *trans*-isomer is active only in bioassays performed in the light, presumably because it undergoes light-induced isomerization to the *cis*-form. Both stereoisomers can exist in optically active forms (asymmetric C-atom at C1′), but only the (+)-form is found naturally.



(S)-(+)-Abscisic acid

Absolute oils: see Essential oils.

Absorbance, *extinction, optical density*: a measure of the quantity of light obsorbed by a solution. It is equal to log I_0/I, where I_0 is the intensity of the incident light, and I is the intensity of the transmitted light.

Absorbance index: see Absorptivity.

Absorption coefficient: see Absorptivity.

Absorptivity: the proportionality constant ε, in Beer's law for light absorption: $A = \varepsilon lc$, where A is absorbance, l is the length of the light path, and c is the concentration. If concentration is expressed on a molar basis, ε becomes the *molar absorptivity, molar absorption coefficient*, or *molar extinction coefficient*, i.e. $\varepsilon = A/lc$, where l is the length of the light path in centimeters, and c is the molar concentration.

Acceptor RNA: see Transfer RNA.

Acceptor site: the ribosomal binding site for the aminoacyl-tRNA during protein biosynthesis.

Accumulation of metabolic intermediates: see Mutant technique.

Acetaldehyde, *ethanal*: CH_3-CHO, important intermediate in the degradation of carbohydrates. m.p. – 123 °C, b.p. 20.1 °C. In its activated form (see Thiamine pyrophosphate), it is involved in a number of reactions (see Alcoholic fermentation). Two molecules A. can undergo acyloin condensation to form Acetoin(see).

3′-Acetamido-3′-deoxyadenosine: see 3′-Amino-3′-deoxyadenosine.

Acetate kinase, *acetokinase* (EC 2.7.2.1): see Acetyl phosphate and Phosphoroclastic pyruvate cleavage.

Acetic acid, *ethanoic acid*: CH_3COOH, a very common monocarboxylic acid. m.p. 16.7 °C, b.p. 118 °C. A.a. occurs in the free form as the end

product of fermentation and oxidation reactions in some organisms. Acetate is formed metabolically by dehydrogenation of acetaldehyde, catalysed either by aldehyde oxidase (EC 1.2.3.1) or a $NAD(P)^+$-dependent aldehyde dehydrogenase (EC 1.2.1.3). The activated form of A.a., Acetylcoenzyme A (see) is a key substance in intermediary metabolism.

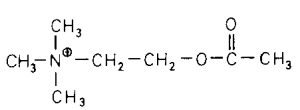
Acetogenins: see Polyketides.

Acetoin, *3-hydroxy-2-butanone, acetyl methyl carbinol*: CH_3-CO-CHOH-CH_3, a reduction product of diacetyl which arises under certain conditions as a side product of the pyruvate decarboxylase (EC 4.1.1.1) reaction. A. is also formed by decarboxylation of acetolactate by acetolactate decarboxylase (EC 4.1.1.5). It is oxidized in a reversible reaction to diacetyl by acetoin dehydrogenase (EC 1.1.1.5), and in some microorganisms it is converted to 2,3-butanediol by D(−)-butanediol dehydrogenase (EC 1.1.1.4).

Acetylcarnitine: see Carnitine.

Acetylcholine: a biogenic amine which is biologically highly active. M_r 163.2. Phylogenetically, A. is a very ancient hormone which appears even in protists. It could be a predecessor of the neurohormones.

A. acts as a cholinergic neurotransmitter in nerves and neuromuscular synapses; it induces a muscle contraction by changing the permeability of the sarcolemma. It is degraded by acetylcholinesterase (EC 3.1.1.7). Drugs which block the acetylcholine receptors (succinoylbischoline) cause muscles to relax (muscle relaxant for surgical operations). A. is found at the synapses in the central nervous system. It dilates blood vessels, causes a drop in blood pressure, and induces contractions in the smooth musculature of the bronchia and the gastrointestinal tract. It therefore promotes peristalsis in the latter.



Acetylcholine

Acetylcholinesterase (EC 3.1.1.7): "true cholinesterase", catalyses the hydrolysis of acetylcholine into choline and acetate. Due to the high turnover number of A. (0.5 to $3.0.10^6$ molecules substrate per molecule enzyme per min), the acetylcholine released at a synapse is hydrolysed within 0.1 ms. This enzyme is found in the central nervous system, particularly in the postsynaptic membranes of the striated muscles, the parasympathetic ganglia, the erythrocytes and the electric organs of fish. Crystalline A. (M_r 330000) has been isolated from the electric organ of the electric eel (*Electrophorus electricus*). It consists of 4 identical inactive subunits of M_r 82500; the half-molecules consisting of 2 covalently bound subunits (M_r 165000) are enzymatically active. Proteolytic attack on the subunits produces two fragments of M_r 60000 and 22500.

The active center of A. has two parts, the anionic binding site for the quaternary nitrogen, which is responsible for the alcohol specificity, and the esterase center, where a catalytic serine and histidine lyse the ester bond. The enzyme is inactivated by blockage of either the serine hydroxyl (by organic phosphate esters, such as diisopropylfluorophosphate or diethyl *p*-nitrophenylphosphate), or the anionic center by trimethylammonium derivatives. If the enzyme has been blocked by organophosphates, it can be reactivated by pralidoxime salts, which are therefore used as antidotes to organophosphate poisoning.

Acetyl-coenzyme A, *acetyl-CoA, active acetate*: $CH_3CO \sim SCoA$, a derivative of acetic acid in which the acetyl residue is bound by a high-energy bond to the free SH-group of coenzyme A. M_r 809.6, λ_{max} = 260 nm. The very reactive thioester has a high potential for transfer of the acetyl group, and is therefore a universal intermediate which provides the C_2 fragment for numerous syntheses. The free energy of the bond (34.3 kJ/mol = 8.2 kcal/mol), however, has no significance as a form of energy storage. In the transfer reactions mediated by acetyl-CoA, either the carboxyl group (electrophilic reaction) or the methyl group (nucleophilic reaction) can react.

By far the most important pathways for the synthesis of acetyl-CoA (Table) are 1) the oxidative decarboxylation of pyruvate, 2) the degradation of fatty acids and 3) the degradation of certain amino acids. The formation of acetyl-CoA involves either 1) the transfer of an acetyl residue from a suitable donor, such as pyruvate, and simultaneous reduction of NAD^+, or 2)the activation of free acetate in a one or two-step process which requires ATP and free coenzyme A.

Acetyl-CoA is the hub of carbohydrate metabolism and has a central position in overall metabolism. The products of carbohydrate, fat and protein metabolism are channeled via acetyl-CoA into oxidative degradation in the tricarboxylic acid cycle. The acetyl residue is used in the synthesis of esters and amides (e.g. acetylcholine, *N*-acetylglucosamine, *N*-acetylglutamate). Acetyl-CoA is also the starting point for isoprenoid synthesis via mevalonic acid and for fatty acid synthesis. The latter path is especially important in the transformation of carbohydrates into fat, and was elucidated in 1951 by Lynen and Lipman.

N*-Acetylglutamic acid, *N-acetylglutamate, abb. *Ac-Glu*: HOOC-CH($NHCOCH_3$)-CH_2-CH_2COOH, the acetylated form of glutamic acid, is the cofactor of carbamoyl phosphate synthetase (ammonia) (EC 6.3.4.16) and allosterically activates this enzyme. See Carbamoyl phosphate.

Acetyl methyl carbinol: see Acetoin.

Acetyl phosphate: CH_3-$COOPO(OH)_2$, an energy-rich acyl phosphate. It is the product of acetate activation in some organisms: Acetate + ATP = A.p. + ADP; the reaction is catalysed by acetate kinase (EC 2.7.2.1). The back reaction can be used for ATP synthesis, for example in the phosphoroclastic cleavage of pyruvate.

Acid amides: see Carboxylic acids.

Acidic α_1-glycoprotein: see Orosomucoid.

Acid plants, *ammonium plants*: plants which ac-

Table. ***Reactions in which acetyl-coenzyme A is synthesized.***

Enzyme	Reaction	Occurrence/ Significance
Acetyl-CoA synthetase (EC 6.2.1.1)	$CH_3COO^- + ATP + CoA \rightleftharpoons CH_3CO\text{-}CoA + AMP + PP_i$	Yeasts, Animals, Higher plants
Acyl-CoA synthetase (GDP-forming) (EC 6.2.1.10)	$CH_3COO^- + GTP + CoA \rightleftharpoons CH_3CO\text{-}CoA + GDP + P_i$	Liver
Acetate kinase	$CH_3COO^- + ATP \rightleftharpoons CH_3CO\text{-}O\text{-}PO_3H_2 + ADP$ (Acetyl phosphate)	Microorganisms
Phosphate acetyltransferase (EC 2.3.1.8)	$CH_3CO\text{-}O\text{-}PO_3H_2 + CoA \rightleftharpoons CH_3CO\text{-}CoA + P_i$	Microorganisms
ATP citrate (*pro*-3*S*)-lyase (EC 4.1.3.8)	Citrate + ATP + CoA $\rightarrow CH_3CO\text{-}CoA$ + oxaloacetate + ADP + P_i	Outside the mitochondria
Pyruvate dehydrogenase complex (EC 1.2.4.1, 2.3.1.12 and 1.6.4.3)	$CH_3COCOO^- + NAD^+ + CoA$ (Pyruvate) $\xrightarrow[TPP,\ LipS_2]{}$ $CH_3\text{-}CO\text{-}CoA + CO_2 + NADH + H^+$	Mitochondrial particles
Acetyl-CoA transacetylase (EC 2.3.1.9)	$CH_3COCH_2CO\text{-}CoA + CoA$ (Acetoacetyl-CoA) $\rightleftharpoons 2CH_2CO\text{-}CoA$	Fatty acid degradation

Abb. TPP = thiamine pyrophosphate; $LipS_2$ = Lipoamide

cumulate organic acids in their leaf cells, which are neutralized by ammonium ions.

Aconitate hydratase, *aconitase*, (EC 4.2.1.3): a hydratase which catalyses one stage of the tricarboxylic acid cycle, the reversible interconversion of citrate and isocitrate. The reaction proceeds via the enzyme-bound intermediate, *cis*-aconitate. At equilibrium, the relative abundances are 90% citrate, 4% *cis*-aconitate and 6% isocitrate. Thus citrate is favored at equilibrium, but in respiring tissues the reaction proceeds from citrate to isocitrate, as isocitrate is oxidized by isocitrate dehydrogenase. The enzyme contains Fe(II) and requires a thiol such as cysteine or reduced glutathione. The Fe(II) ion forms a stable chelate with citric acid. X-ray analysis of Fe(II) complexes of tricarboxylic acids suggested the "ferrous wheel" hypothesis of aconitase action. According to this mechanism, three points on the *cis*-aconitate molecule are bound at separate sites on the enzyme surface; in addition the molecule is also complexed with the Fe(II) atom at the active center. The stereospecific *trans* addition of water to *cis*-aconitate to form either citrate or isocitrate is achieved by rotation of the ferrous wheel, which can add OH to either side of the molecule. Aconitase is inhibited by fluorocitrate. Two isoenzymes are present in animal tissues, one in the cytosol and one in the mitochondria.

Ref: Glusker, J. P., in Boyer, P. D. (ed.), *The Enzymes*, *5*, 434, Academic Press Inc., 1971.

Aconitic acid: an unsaturated tricarboxylic acid, usually occurring in the *cis* form, but sometimes in the *trans*. *cis*-A.a., m.p. 130 °C., *trans*-A.a., m.p. 194 to 195 °C. A.a. was discovered in free form in aconite, *Aconitum napellus*. The anionic form of *cis*-A.a. (propen-*cis*-1,2,3-trioic acid) is important as an intermediate in the isomerization of citrate to isocitrate in the Tricarboxylic acid cycle (see).

Aconitine: an Aconitum alkaloid (see Terpene alkaloids) from the roots of aconite (*Aconitum napellus*) and other *Aconitum* and *Delphinium* species. m.p. 197 to 198 °C, $[\alpha]_D^{20} -36°$ (benzene). A. is an esterified alkaloid. It is extremely poisonous and can cause death in adults at a dose of 1 to 2 mg by paralysing the heart and respiration. Its hydrolysis products are only slightly toxic. In spite of useful physiological properties, A. is rarely used in medicine, due to its toxicity. It is sometimes used internally as tincture for rheumatism and neuralgias and externally as a pain-killing salve.

In antiquity, aconitine preparations were used as arrow poisons by the Greeks and (East) Indians.

Aconitum alkaloids: a group of terpene alkaloids, some of them very poisonous, from various aconite (*Aconitum*) species. The best-known representative is aconitine.

ACP: abb. for acyl carrier protein.

ACTH: abb. for adrenocorticotropic hormone. See Corticotropin.

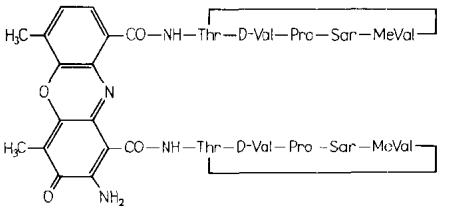
Actin: see Muscle proteins.

Actinidine: a widely occurring terpene alkaloid. See Valeriana alkaloids.

Actinomycins: a large group of peptide lactone antibiotics produced by various strains of *Streptomyces*. These highly toxic red compounds contain a chromophore, 2-amino-4,6-dimethyl-3-ketophenoxazine-1,9-dioic acid (actinocin), which is linked to two 5-membered peptide lactones by the amino groups of two threonine residues. The various A. differ only in the amino acid sequence of the lactone rings. In vivo, A. inhibit the DNA-dependent RNA synthesis at the level of transcription by interacting with the DNA. The concentration required for inhibition depends on the

base composition of the DNA; more is required for DNA with a low guanine content. A. are pharmacologically very important due to their bacteriostatic and cytostatic effect.

Actinomycin D (Fig.) is one of the most widespread A. Its spatial structure has been elucidated by NMR studies, and the specificity of its interaction with deoxyguanosine was demonstrated by X-ray analysis. Actinomycin D is used as a cytostatic, e.g. in the treatment of Hodgkin's disease.



Actinomycin D

Activated amino acids: see Aminoacyl adenylate.

Activated carbon dioxide: see Biotin enzymes.

Activated carboxylic acids: derivatives of carboxylic acids which are very reactive, and thus capable of reactions which the free acids do not undergo. The biochemically important A.c.a. are either anhydrides or thioesters.

Activated choline: see Cytidine diphosphocholine.

Activated fatty acids: fatty acyl coenzyme A thioesters which, as high energy compounds, have a large potential for group transfer. They are formed during fatty acid biosynthesis, or by the activation of free fatty acids. Acyl CoA synthetases catalyse formation of the CoA derivatives according to the reaction:

$CH_3(CH_2)_nCOO^- + ATP + HS\text{-}CoA \rightarrow CH_3(CH_2)_nCO \sim SCoA + AMP + PP_i$

The reaction involves acyladenylate as an intermediate, which is cleaved by coenzyme A to form acyl-CoA and AMP. Several such enzymes are known, and they are named according to the length of carbon chain that shows optimal activity, e.g. acetyl CoA synthetase converts C_2 and C_3 fatty acids, octanoyl CoA synthetase (C_4 to C_{12}) and dodecanoyl CoA synthetase (C_{10} to C_{18}). Mitochondria also contain an acyl CoA synthetase that cleaves GTP to GDP and P_i. Acyl CoA derivatives of short chain fatty acids may also be formed in a transfer reaction involving succinyl-CoA, catalysed by thiophorases:

SuccinylSCoA + R-COOH → succinic acid + R-COSCoA

Activated fatty acids are in equilibrium with acylcarnitine in the organism. They are the starting point for fatty acid degradation.

Activated glucose: see Nucleoside diphosphate sugars.

Activated glycol aldehyde: 2-(1,2-dihydroxyethyl)-thiamine pyrophosphate, abb. ***DETPP***, glycol aldehyde bound to the C-2 atom of the thiazole ring of thiamine pyrophosphate. It is formed in carbohydrate metabolism by cleavage of a ketose and is transferred as C-2 group to an aldose in a transketolation reaction.

Activated amino acids: see Aminoacyl adenylate.

Activated fatty acids: derivatives of carboxylic acids which are very reactive, and thus capable of reactions that free acids do not undergo. Biochemically important A.f.a. are either anhydrides or thioesters; see, e.g. Acetyl-coenzyme A.

Activation hormone: see Insect hormones.

Activator protein: see Calmodulin.

Active acetaldehyde: α-hydroxyethylthiamine pyrophosphate, abb. ***HETPP***, the activated form of acetaldehyde formed by decarboxylation of active pyruvate. The aldehyde is bound to the C-2 atom of the thiazole ring of thiamine pyrophosphate. HETPP is an intermediate in alcoholic fermentation.

Active acetate: see Acetyl-coenzyme A.

Active aldehyde: see Thiamine pyrophosphate.

Active center: that part of an enzyme or other protein which binds the specific substrate and converts it to product (enzymes) or otherwise interacts with it (heme proteins, various carrier and receptor proteins). The A.c. of an enzyme thus consists of the actual catalytic center, which is relatively unspecific, and the substrate-binding site, which is responsible for the specificity of the enzyme. The A.c. may lie on the surface (in chymotrypsin, for example) or in a cleft (in lysozyme, papain, carboanhydrase or ribonuclease) in the enzyme molecule. It involves only a limited number of amino acid residues. The A.c. must be particularly flexible in order to bind its substrate and carry out catalysis. It therefore lacks regular structures, such as α-helix. The amino acids involved in catalysis may lie at a considerable distance from each other in the absence of a substrate; they are brought into play by conformational changes induced by the substrate when it binds (induced fit model suggested by Koshland) (see Chymotrypsin and Serine proteases). For example, the amino acids involved in the catalysis step in the serine proteases (including trypsin, chymotrypsin and elastase) are serine 195, histidine 57 and aspartate 102. The amino acids responsible for binding the substrate are serine 189 and glycine 216 in chymotrypsin, aspartate 189 and glycine 216 in trypsin, and serine 189 and valine 216 in elastase.

Information on the amino acids involved in the A.c. is obtained by specific marking with coenzyme, inhibitors or reagents specific for particular side chains. Some widely used irreversible inhibitors for the catalytic center of the serine proteases are tosyllysine chloromethyl ketone (TLCK), which selectively blocks the imidazole group of the histidine 57 in trypsin, diisopropylfluorophosphate (DFP), and phenylmethane sulfonyl fluoride (PMSF), which form stable esters with serine 195 of all serine proteases and many carboxyesterases.

Active CO_2: see Biotin enzymes.

Active formaldehyde: see Active one-carbon units; Thiamine pyrophosphate.

Active formate: see Active one-carbon units.

Active glucose: see Nucleoside diphosphate sugars.

Active glycolaldehyde: 2-(1,2-dihydroxyethyl)-thiamine pyrophosphate, abb ***DETPP***, glycolaldehyde bound to C-2 of the thiazole ring of thiamine pyrophosphate. It is formed in carbohydrate metabolism by cleavage of a ketose, and is transferred as a 2C group to an aldose in a transketolation reaction.

Active methionine: see *S*-Adenosyl-L-methionine.

Active methyl groups: see *S*-Adenosyl-L-methionine.

Active one-carbon units, abb. ***C_1 units:*** C_1 fragments which are activated by binding to tetrahydrofolic acid, or less commonly, to thiamine pyrophosphate. The active ethylenediamine group of tetrahydrofolic acid serves as a carrier for the metabolic transfer of a formyl or methyl group. Fig. 1 shows the active forms of tetrahydrofolic acid (THF). The various C_1 units can be interconverted while attached to THF (Fig. 2). The main source of C_1 units is the hydroxymethyl group of serine, which is transferred to THF by serine hydroxymethyltransferase (EC 2.1.2.1), forming hydroxymethyl-THF (activated formaldehyde). The formation of C_1 units in the course of histidine catabolism or the anaerobic degradation of purines is of particular importance. C_1 units are used in purine biosynthesis and as the donors of the 5-methyl group of thymine. The formation and uses of the C_1 units are given on the table. The most important are 1) active formaldehyde ($N^{5,10}$-methylene-THF, hydroxymethyl-TMF), 2) active formate (N^{10}-formyl-THF), 3) N^5-methyl-THF (see L-Methionine).

Table. ***Formation and uses of active one-carbon units.***

Type of C_1 unit	Biogenesis	Use
N^{10}-Formyl-THF	From formate + ATP by formyl-THF synthetase (EC 6.3.4.3); from $N^{5,10}$-methylene-THF	Purine synthesis after conversion to $N^{5,10}$-methylenyl-THF and $N^{5,10}$-methylene-THF
$N^{5,10}$-methylene-THF	From L-serine and THF by serine hydroxymethyltransferase (EC 2.1.2.1); from glycine and THF directly or via glyoxylate and formate	Purine synthesis; formation of the 5-methyl group of thymine and the methoxyl group of hydroxymethyl cytosine
N^5-Formimino-THF	By anaerobic purine degradation via formiminoglycine; by histidine degradation via formiminoglutamate	After conversion to $N^{5,10}$-methylenyl-THF and $N^{5,10}$-methylene-THF
$N^{5'}$-methyl-THF	From $N^{5,10}$-methylene-THF	Methionine synthesis.

Active pyruvate: α-lactyl-thiamine pyrophosphate. The lactyl is bound to the C-2 atom of the thiazole ring of the thiamine pyrophosphate. A.P. is an intermediate in the oxidative decarboxylation of pyruvate to acetyl-coenzyme A and in its decarboxylation to acetaldehyde in alcoholic fermentation.

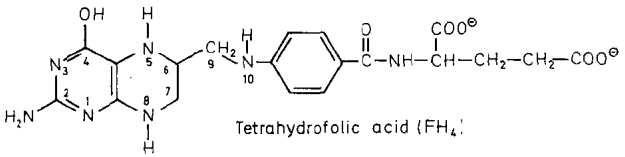
Active succinate: the high-energy thioester succinyl-coenzyme A. It is important as an intermediate in the tricarboxylic acid cycle.

Active sulfate: see Phosphoadenosine phosphosulfate.

Active transport: a process in which solute molecules or ions move across a biomembrane from lower to higher concentration, i.e. against the concentration gradient. Since thermodynamic work is involved, A.t. must be coupled to an exergonic reaction. In primary A.t., the coupling is direct. The transport of Na^+ and K^+ ions across a cell membrane by the Na^+,K^+-ATPase system, for example, requires the simultaneous hydrolysis of ATP. Secondary A.t. utilizes the energy of an electrochemical gradient established for a second solute to transport the first. One form of secondary A.t. is *cotransport*, in which the transport of one solute drives that of the other. An example is the Na^+-dependent transport of certain sugars and amino acids in animal cells: the concentration of Na^+ in the cell is maintained at a level far below the intercellular concentration by the Na^+,K^+ pump. A specific transport protein (carrier) binds both glucose and Na^+ outside the cell and releases them on the inside. The process is energetically favorable because the Na^+ is moving from a region of higher concentration to lower concentration. In other cases, the membrane potential generated by electron flow along the respiratory chain drives the active transport of sugars or amino acids. A third form of A.t. is called *group translocation* because the solute is changed in the course of transport. An example is the *phosphotransferase system* in some bacteria, in which sugars are phosphorylated in the course of transport. An interesting feature of this system is that phospho*enol*pyruvate rather than ATP is the phosphate donor.

A.t. processes are highly specific, and they are saturable. This implies that enzyme-like proteins, or *carriers*, mediate the transport. (The term "carriers" also applies to the mechanism of Facilitated diffusion [see]).

The bacterial transport systems called *permeases*



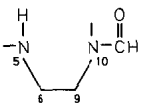
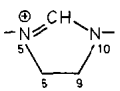
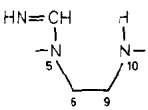
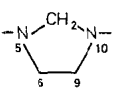
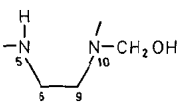
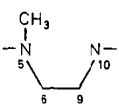
Active form	Reactive part of FH_4	Group transferred
N^{10} –Formyl–FH_4		–CH (Formyl–)
$N^{5,10}$ –Methenyl–		
N^{5} –Formimino–		
$N^{5,10}$ –Methylene–		$\rangle CH_2$ (Formaldehyde–)
N^{10} –Hydroxymethyl–		$\rangle CH_2$ (Formaldehyde–)
N^{5} –Methyl–		$-CH_3$ (Methyl–)

Fig. 1. ***Structure of tetrahydrofolic acid and active one carbon units***

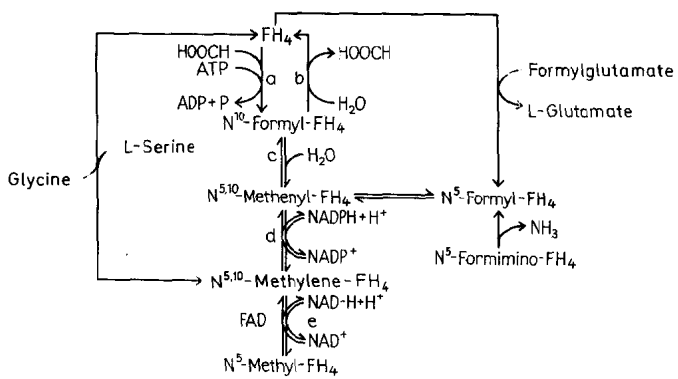


Fig. 2. ***Interconversions of active one carbon units.***
a Formyl-FH_4 synthetase (EC 6.3.4.3)
b Formyl-FH_4 deformylase (EC 3.5.1.10)
c 5,10-Methylene-FH_4 synthetase (EC 6.3.3.2)
d Methylene-FH_4 dehydrogenase ($NADP^+$) (EC 1.5.1.5)
e 5,10-Methylene-FH_4 reductase ($FADH_2$) (EC 1.1.99.15)

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have been extensively studied by genetic and other means. The protein product of the lactose permease (y) gene has been isolated, as have a number of membrane proteins which bind the substrates of other permeases. These proteins are probably components of the respective permeases.

Actomyosin: see Muscle proteins.

Acylcarnitine: see Carnitine.

Acyl carrier protein, abb. ***ACP:*** a small, acidic, heat-stable globular protein which is part of the fatty-acid synthesizing complex in *Escherichia coli* and other bacteria, yeasts and plants. It is the carrier of the fatty acid chain during its biosynthesis. The primary structure of the ACP from *E. coli* has been determined: it contains 77 amino acids and has M_r 8847. The protein itself contains no sulfur; but it carries a molecule of phosphopantetheine (which possesses-SH) linked via a phosphate ester to the hydroxyl of serine 36. All acyl residues formed during fatty acid biosynthesis are bound as thioesters to the SH-group of this prosthetic group. The M_r of the ACP isolated so far lie between 8600 *(Clostridium butyricum)* and 16000 (yeast).

Synthetic apo-ACP protein, a polypeptide representing amino acids 2 to 74 of the *E. coli* protein, functions as substrate for the holo-acyl-carrier-protein synthase (EC 2.7.8.7); the product is biologically as active as natural holo-ACP.

Acylmercaptan: see Thioester.

Adair-Koshland-Nemethy-Filmer model: see Cooperativity model

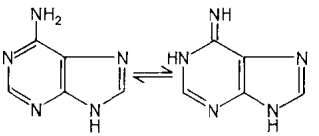
Adaptive enzymes: see Regulation of enzyme synthesis.

Adaptor hypothesis: a suggestion made by Crick to explain the translation of the genetic code. He proposed that there must be an adaptor between the information-carrying nucleic acid and the protein being synthesized which was able to "recognize" both kinds of molecules. The discovery of tRNA and the corresponding amino acyl-tRNA synthetases confirmed his hypothesis.

Addictive drugs, ***psychotropic drugs:*** drugs which create a sense of euphoria, and which have a strong potential for addiction. Addiction leads to physical dependence and is usually accompanied by increased tolerance, so that increasing quantities of the drug must be administered to achieve the desired effect, and doses can be survived that would otherwise be fatal. Painful withdrawal symptoms occur in the absence of the drug. Most Narcotics (see) are A.d., e.g. morphine, cocaine, barbiturates, alcohol. Specific Opiate receptors (see) have been demonstrated in the central nervous system, which bind morphine and structurally related addictive narcotics like heroin.

Addison's disease: see Adrenal corticosteroids.

Adenine, abb. ***A*** or ***Ade***: 6-aminopurine, one of the common nucleic acid bases. M_r 135.13, m.p. 365 °C (d.),sublimes above 220 °C. A. is also part of the adenosine phosphates and other physiologically active substances, including various nucleoside antibiotics. A. is found in free form in various plants, especially in yeasts. It is synthesized de novo from adenosine monophosphate, or is formed by degradation of nucleic acids. Adenine deaminase (EC 3.5.4.2) removes the 6-amino group to give hypoxanthine.



Amino form Imino form

Tautomeric forms of adenine

Adenine arabinoside: see Arabinosides.

Adenine deaminase, ***adenase*** (EC 3.5.4.2): see Purine degradation.

Adenine xyloside: see Xylosylnucleosides.

Adenosine, abb. ***Ado:*** 9-β-D-ribofuranosyladenine, M_r 267.24, m.p. 229 to 231 °C, $[\alpha]_D^{20}$ −61.7° (c= 0.7, water). Phosphorylated derivatives of Ado are metabolically important. See Nucleosides.

Adenosine deaminase (EC 3.5.4.4): see Taka amylase.

Adenosine 3′-phosphate 5′-phosphosulfate: see Phosphoadenosine-phosphosulfate.

Adenosine phosphates, ***adenine ribonucleotides:*** important as components of nucleic acids and as the major form in which chemical free energy is stored and transferred. They are also important metabolic regulators, for example in glycolysis and the tricarboxylic acid cycle. The biologically significant derivatives, including cyclic adenosine 3′,5′-monophosphate, carry the phosphate ester on the C-5 of the ribose.

1. ***Adenosine 5′-monophosphate***, abb. ***AMP***, M_r 347.22, m.p. 196 to 200 °C (d.), $[\alpha]_D^{20}$ −26 ° (c =1.0, 10% HCl), −47.5 ° (c =2.0, 2% NaOH), is synthesized de novo from inosinic acid (see Purine biosynthesis) and also arises by cleavage of pyrophosphate from adenosine triphosphate.

2. ***Adenosine 5′-diphosphate***, abb. ***ADP***, M_r 427.22 $[\alpha]_D^{25}$ −25.7 °, is formed either by adding a second phosphate to AMP (see Adenylate kinase), or by removal of a phosphate from ATP; the latter conversion may be catalysed by one of the adenosine triphosphatases (EC 3.6.1.3), or by an enzyme which transfers the phosphate to another organic molecule (kinase). The energy stored in the anhydride bond of ADP can be made available by the enzyme adenylate kinase, which catalyses the reaction 2 ADP → ATP + AMP. ADP is the phosphate acceptor in substrate and oxidative phosphorylation and photophosphorylation, in which it is converted into ATP.

3. ***Adenosine 5′-triphosphate***, abb. ***ATP***, M_r 507.19, $[\alpha]_D^{22}$ −26.7 ° (c = 3.095), was discovered in 1929 by Lohmann. It is extremely important as the universal energy "currency" of every living cell. The energy is stored in the two high-energy phosphate bonds.

Biosynthesis of ATP. ATP is the immediate pro-

duct of all processes in the cell leading to the chemical storage of energy. It is biosynthesized by phosphorylation of ADP in the course of substrate phosphorylation, oxidative phosphorylation and cyclic and non-cyclic photophosphorylation in plants. Another source of energy for ATP formation is other high-energy phosphates and the reaction catalysed by adenylate kinase.

Cleavage of ATP. ATP has a high potential for group transfer. Several groups can be removed (Fig. 1, Table 1).

Table 1. ***Free energy of hydrolysis of ATP in kJ/mol (kcal/mol)***

Removal of orthophosphate:	
$ATP \rightarrow ADP + P_i$	29.4 (7.0)
Removal of pyrophosphate:	
$ATP \rightarrow AMP + PP_i$	36.12 (8.6)
$PP_i \rightarrow P_i + P_i$	28.14 (6.7)

pyrophosphate may be hydrolysed by inorganic pyrophosphatase (EC 3.6.1.1), which makes the transfer reaction essentially irreversible.

d) Transfer of the adenosyl residue and release of both orthophosphate and pyrophosphate, for example in the synthesis of *S*-adenosyl-L-methionine.

Uses of ATP. The chemical energy stored in ATP is used in chemical reactions, including the synthesis of specific macromolecules from the corresponding monomeric components and the synthesis of activated compounds. Often an endergonic reaction is driven forward by enzymatic coupling to the hydrolysis of ATP. Many metabolic pathways, including the biosynthesis and β-oxidation of fatty acids, degradation of glucose, urea synthesis, nucleotide synthesis and the transformations of sugars require ATP.

ATP provides energy for the contraction of muscles and the motion of cilia and flagella. In some

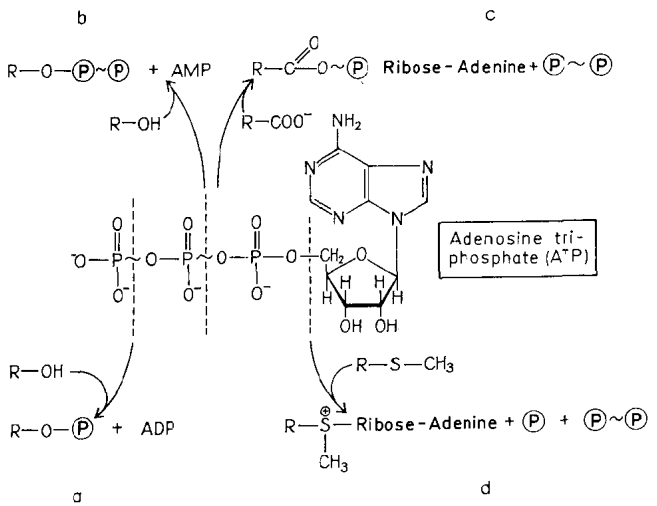


Fig. 1. ***Possible cleavages of adenosine 5′-triphosphate.***

a) Transfer of orthophosphate to alcoholic hydroxyl groups, acid groups or amide groups and release of ADP. The enzymes which catalyse these reactions are the kinases, which can also catalyse the synthesis of ATP from ADP.

b) Transfer of the pyrophosphate residue and release of AMP, for example in the synthesis of 5-phosphoribosyl-1-pyrophosphate from ribose 5-phosphate in the course of purine biosynthesis.

c) Transfer of the AMP residue and release of pyrophosphate. The receiving group is given a higher group-transfer potential in this process, which occurs in the activation of fatty acids and amino acids in synthetic pathways. The released

organisms, ATP can provide the energy for bioluminescence. Electric fish can generate electric current by hydrolysing ATP. Active transport of many substances across membranes depends on a source of ATP.

Other nucleoside triphosphates, which are energetically equivalent to ATP, are important in some metabolic reactions: cytidine triphosphate in phosphatide biosynthesis, guanosine triphosphate in protein synthesis and oxidative decarboxylation of 2-oxoacids (see Tricarboxylic acid cycle), inosine triphosphate in some carboxylations, uridine triphosphate in polysaccharide synthesis.

In the living organism, the adenosine phosphates are in equilibrium and are regarded collectively as the adenylic acid system. The physiological

concentrations for ADP and ATP are around 10^{-3} mol/l. The ratio of the forms is called the energy charge and is given by the equation $EC = \frac{[ATP] + 0.5[ADP]}{[ATP] + [ADP] + [AMP]}$. The square brackets indicate molar concentrations. If all A. is in the form of ATP, the energy charge is 1; otherwise it is smaller than 1.

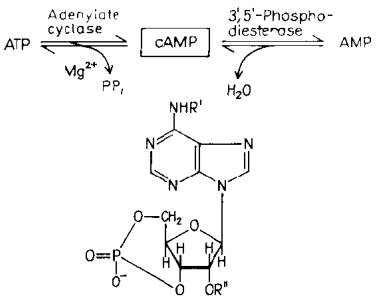


Fig. 2. ***Synthesis of cyclic adenosine 3',5'-monophosphate; and the structure of cyclic N^6, $O^{2'}$-dibutyryladenosine 3',5'-monophosphate.***

4. ***Cyclic adenosine 3',5'-monophosphate***, abb. ***3',5'-AMP, cyclo-AMP, cAMP,*** M_r 329.2. cAMP was discovered by Sutherland in 1956 as a heat-stable factor in the liver. It is generated from adenosine triphosphate (Fig. 2) by adenylate cyclase (EC 4.6.1.1), and is degraded to AMP by 3':5' cyclic-nucleotide phosphodiesterase (EC 3.1.4.17), which is specific for cyclic nucleotides. The activities of these two enzymes determine the intracellular level of cAMP. Physiological amounts of various substances, for example pyridoxal phosphate in *Escherichia coli*, can reduce the activity of the adenylate cyclase. The phosphodiesterase of mammals is inhibited by nucleoside triphosphates, pyrophosphate, citrate and methylated xanthines (especially theophylline) and stimulated by nicotinic acid. The adenylate cyclase is activated by a number of hormones, which leads to increased formation of cAMP, but only in the target cells of those hormones. In this way cAMP serves as a "second messenger" for a number of different hormones. In addition, it affects the initiation of production of hormones and their release, e.g. acetylcholine, glucagon, insulin, melanotropin, parathyrin, vasopressin and corticotropin. cAMP has a central function in the hormonal regulation of animals and microorganisms, and possibly also of higher plants.

The variety of effects of cAMP (Tables 2 and 3) implies a similarity in the basic molecular mechanisms of these effects. It might lie, for example, in cAMP-sensitive enzymes, the specific protein kinases. cAMP also affects the equilibrium among various metabolic pathways, e.g. glycogen metabolism.

In many cases, the physiological effects of cAMP are only seen in the presence of calcium ions.

The exogenous "artificial" control of the intracellular cAMP level is becoming medically important. Substances which raise this level have been successfully used in treatment; for example, treatment of psoriasis with the alkaloid papaverine, which inhibits the cyclic nucleotide phosphodiesterase; and with the tissue hormone dopamine, which stimulates the formation of cAMP in the epidermis. cAMP also inhibits the growth of certain tumors.

Because it is very polar, cAMP penetrates the cell membrane only in very small quantities. Its synthetic derivatives have better permeability because they have been made more lipophilic by substitution with organic acids. The most commonly used is $N^6,O^{2'}$ dibutyroyladenosine 3',5'-monophosphate, abb. DBcAMP. A number of other cyclic 3',5'-nucleotides with special functions have been found to occur naturally.

Table 2. ***Occurrence and effect of cyclic adenosine 3',5'-monophosphate (according to Hardeland)***

Organisms	Effect
1) Protozoa *Paramecium*	Activation of the protein kinase
2) Bacteria *Escherichia coli*	Release of glucose inhibition of enzyme induction (see Catabolite repression) Initiation of messenger RNA synthesis mediated by a specific cAMP receptor protein (catabolite gene activator protein) Inhibition of the degradation of messenger RNA bound to ribosomes. Stimulation of the synthesis of many enzymes.
Serratioa marcescens *Salmonella typhimurium* *Proteus inconstans* *Aerobacter aerogenes* *Brevibacterium liquefaciens*	Release of catabolite repression and stimulation of the synthesis of β-galactosidase (*Brevibacterium liquefaciens* excretes cAMP into the medium)
Photobacterium fischerei	Release of catabolite repression and production of bioluminescence.

Table 2 (continued on page 10)

Table 2. ***Occurrence and effect of cyclic adenosine 3′,5′-monophosphate (according to Hardeland)*** (cont.)

Organisms	Effect
3) Fungi Slime molds *Dictyostelium discoideum*	Extracellular signal transmitter. Cell aggregation as response to chemotactical stimuli.
Polysphondylium pallidium	Does not respond to cAMP, has a different akrasin
Yeasts *Saccharomyces cereviseae*	Affects the oscillation and the redox equilibrium in the course of glycolysis. Affects sporulation
4) Invertebrates e.g. annelids *(Golgingia, Nereis)* starfish	Activation of protein kinases
Liver fluke *(Fasciola)* Blowfly *(Calliphora)*	Transmission of the effect of serotonin
5) Vertebrates Frog, toad, turkey, pigeon, rat, mouse, guinea pig, rabbit, human	"Second messenger" in the transmission of hormone stimuli
6) Higher plants Barley (endosperm)	Enzyme induction during germination Stimulation of the synthesis of amylase
Peas, lettuce Weeds	Effects on germination

Table 3. ***Metabolic processes regulated by cyclic adenosine 3′,5′-monophosphate (after Nelboek)***

Process	Tissue/Organism	Stimulation + Inhibition −
Effect on enzyme activity:		
Fructose-bisphosphatase (EC 3.1.3.11)	Kidney	+
Glycogen synthase (EC 2.4.1.11)	Muscle, liver	−
Phosphorylase kinase (EC 2.7.1.38)	Muscle	+
Phosphorylase phosphatase (EC 3.1.3.17)	Adrenals	−
Protein kinase (EC 2.7.1.70)	Various	+
Enzyme induction:		
β-Galactosidase (EC 3.2.1.23)	*Escherichia coli*	+
Lactose permease		+
Galactokinase (EC 2.7.1.6)		+
Glycerol kinase (EC 2.7.1.30)		+
Glycerophosphate permease		+
L-Arabinose permease		+
Fructokinase (EC 2.7.1.4)		+
Tryptophanase (EC 4.1.99.1)		+
D-Serine dehydratase (EC 4.2.1.14)		+
Thymidine phosphorylase (EC 2.4.2.4)		+
Glucose-6-phosphatase (EC 3.1.3.9)		+

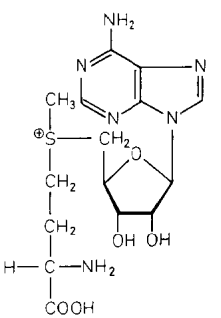
Table 3 (continued on page 11)

Table 3. ***Metabolic processes regulated by cyclic adenosine 3',5'-monophosphate (after Nelboek)*** (cont.)

Process	Tissue/Organism	Stimulation + Inhibition −
Phospho*enol*pyruvate carboxykinase (ATP) (EC 4.1.1.49)	Liver	+
L-Serine dehydratase (EC 4.2.1.13)		+
Tyrosine aminotransferase (EC 2.6.1.5)		+
Physiological effects:		
Amylase secretion	Salivary glands, pancreas	+
Calcium resorption	Bones	+
DNA synthesis	Thymocytes	+
Fat synthesis	Liver	−
Gluconeogenesis	Liver	+
Glycogenolysis	Liver	+
Urea synthesis	Liver	+
Secretion of hormones	Various glands	+
Ketogenesis	Liver	+
Contractility	Heart muscle	+
Fat degradation	Fat tissue	+
Permeability	Kidneys	+
Protein synthesis	Liver	−
HCl secretion	Stomach epithelium	+
Cell aggregation	Thrombocytes	+
Cell aggregation	Slime mold	−

S-Adenosyl-L-homocysteine: see *S*-Adenosyl-L-methionine.

***S*-Adenosyl-L-methionine, *S*-(5'-deoxyadenosine-5')-methionine, active methionine, active methyl,** abb. ***S*-Ado-Met, SAM:** a reactive sulfonium compound which is the most important methylating agent in cellular metabolism (see transmethylation.) M_r of the free cation 398.4. The natural form is the L-(+)-isomer. $[\alpha]_D^{24}$ of SAM $^+Cl^-$ = +48.5 (c = 1.8 in 5N HCl). Due to the asymmetry of the sulfonium group, there are 4 stereoisomers. SAM is unstable at room temperature, both as the solid, and in aqueous solution. It is formed by activation of L-methionine with ATP: Met + ATP → SAM + PP_i + P_i. The adenosine residue of the ATP is transferred to the methionine.



***S*-Adenosyl-L-methionine**

The transmethylation reaction produces, in addition to the methylated product, *S*-adenosyl-L-homocysteine: systematic name *S*-(5'-deoxyadenosine-5')-homocysteine. It may be reconverted to SAM after cleavage into adenosine and L-homocysteine. L-Homocysteine is the substrate of dimethylthetin-homocysteine methyltransferase (EC 2.1.1.3). See Methionine.

Adenylate cyclase (EC 4.6.1.1): see Adenosine phosphates.

Adenylate kinase, *myokinase* (EC 2.7.4.3): a trimeric enzyme found in the mitochondria of muscles and other tissues. It is resistant to heat and acid. M_r 68 000, subunit M_r 23 000. It catalyses the conversion of two molecules of ADP into ATP + AMP, thus making available the energy of the ADP. At equilibrium, the concentrations of the three adenosine phosphates are nearly equal. In many energy-requiring reactions ATP is converted into pyrophosphate and AMP (see Adenosine phosphates). A.k. is important because it catalyses the first stage (AMP to ADP) in the conversion of this AMP into ATP.

Adenylic acid: see Adenosine phosphates.

Adenylosuccinate, *N*-succinyladenylate, abb. ***sAMP:*** 5-aminoimidazole-4-*N*-succinocarboxamide ribonucleotide, an intermediate in purine biosynthesis. M_r 463.31.

Adenylylsulfate reductases: enzymes of sulfur metabolism which reduce either phosphoadenylylsulfate (APS reductase) or adenylylsulfate. Adenylylsulfate reductase (EC 1.8.99.2) is identical with one component of the sulfate reductase in sulfate assimilation, since adenylylsulfate is the donor of the sulfate group. The table shows the properties of some of these reductases. The re-

ductase is in every case a complex of three components, an adenylylsulfate transferase (see sulfate assimilation, Fig. 1), a low-molecular-weight carrier and the actual adenylylsulfate reductase. Phosphoadenylylsulfate reductase from *Saccharomyces cerevisiae* requires NADPH and has been partly purified and fractionated.

Table. ***Properties of adenylylsulfate reductases from various organisms***

Organism	pH optimum	M_r	Comments
Desulfovibrio[1]	7.4	220000	Contains 1 molecule FAD and 6 to 8 atoms nonheme iron
Thiobacillus thioparus[1]	7.4	170000	Contains 1 molecule FAD and 8 to 10 atoms nonheme iron
Thiocapsa roseopersicina[1]	8.0	180000	Contains 1 molecule FAD, 4 atoms non-heme iron and 2 atoms heme iron 60 to 80 fold enrichment to a homogeneous preparation in the ultracentrifuge
Chlorella pyrenoidosa[2]		330000	Partly purified enzyme

1. With $Fe(CN)_6^{3-}$; 2. a thiol as electron donor; the enzyme from *Chlorella* is active with phosphoadenylylsulfate only in the presence of 3′-nucleotidase.

Adermine: vitamin B_6. See Vitamins.

ADH: abb. for Antidiuretic hormone. See Vasopressin.

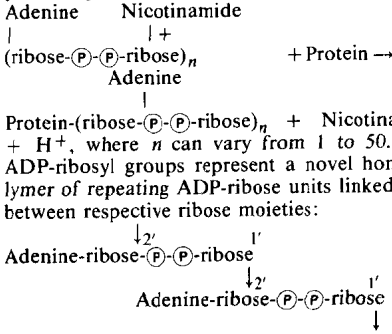
Adiuretin: see Vasopressin.

Adjuvant: a mixture of oils, emulsifiers, killed bacteria and other components which serves to intensify unspecifically the immune response. The A., which is not (supposed to be) itself antigenic, is injected several times, intramuscularly or subcutaneously, together with an antigen into an animal to produce the maximal amount of antibodies. In experimental immunology, Freund's incomplete A., an emulsion of paraffin oils which protects the antigen from too rapid degradation, and Freund's complete A., which contains in addition killed mycobacteria or tuberculosis bacteria, are most commonly used. A used in the production of vaccines are aluminum hydroxide and calcium phosphate gels. They are thought to activate the phospholipase A of the macrophages, so that more lecithin is converted to lysolecithin. The latter can itself act as an A., and this may be the reason that it sometimes leads to oversensitivity (allergy).

Ado: abb. for Adenosine.

ADP: abb. for Adenosine 5′-diphosphate.

ADP-ribosylation of proteins: attachment of monomeric or polymeric ADP-ribosyl groups to a protein by transfer from NAD^+:



, where n can vary from 1 to 50. Poly ADP-ribosyl groups represent a novel homopolymer of repeating ADP-ribose units linked 1′–2′ between respective ribose moieties:

The free energy of hydrolysis of the β-N-glycosidic linkage of NAD^+ is −34.35 kJoules (−8.2 kcal)/mole at pH 7 and 25 °C; it is therefore a so-called high energy bond, and NAD^+ can act as an ADP-ribosyl transferring agent. The transfer of one ADP-ribosyl group (n= 1 in above equation) is catalysed by ADP-ribosyl transferase. Formation and concomitant transfer of poly ADP-ribose to an acceptor is catalysed by poly(ADP-ribose) synthetase (n is greater than one in the above equation).

Diphtheria toxin, produced by strains of *Corynebacterium diphtheriae* that carry β-phage, inhibits protein synthesis in eukaryotic cells by catalysing the transfer of an ADP-ribose moiety from NAD^+ to elongation factor 2. *Pseudomonas* toxin catalyses a similar reaction. T4 phage catalyses the monomeric ADP-ribosylation of RNA polymerase and other proteins in *Escherichia coli*. Choleragen activates adenylate cyclase by catalysing transfer of ADP-ribose from NAD^+ to the enzyme.

Poly ADP-ribose groups are found in eukaryotic chromosomal proteins, mitochondrial protein and histones.

The biological function of the ADP-ribosylation of proteins in eukaryotic cells is not known, but the occurrence of poly ADP-ribosyl groups in nuclear proteins, particularly in association with chromatin, suggests a regulatory role in nuclear function.

The nature of the linkage to protein is not known, but it appears to involve attachment to basic amino acids. In the choleragen-activated ADP-ribosylation of adenylate cyclase, an arginine residue appears to be the chief receptor for ADP-ribose.

Ref: Hayaishi, O. and Ueda, K. *Ann. Rev. Biochem.* (1977) ***46***, 95–116. "ADP-ribosylation of nuclear proteins" by Purnell, M.R., Stone, P.R. and Whish, W.J.D., *Biochemical Society Transactions* (1980) ***8***, 215–227.

Adrenal corticosteroids, *adrenocorticoids, corticosteroids, corticoids, cortins:* an important

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