An introduction to human biochemistry

C. A. Pasternak

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C. A. Pasternak

Professor of Biochemistry, University of London
at St. George's Hospital Medical School

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Abbreviations

A	adenine
ACTH	adrenocorticotrophic hormone
ADP	adenosine diphosphate
Ala	alanine
AMP	adenosine monophosphate
Ara	arabinose
Arg	arginine
Asn	asparagine
Asp	aspartate
ATP	adenosine triphosphate
C	cytosine
cAMP	adenosine 3′,5′-cyclic monophosphate; cyclic AMP
CCK	cholecystokinin
CDP	cytidine diphosphate
CMP	cytidine monophosphate
CoA	coenzyme A
CoQ	coenzyme Q (ubiquinone)
CTP	cytidine triphosphate
Cys	cysteine
DNA	deoxyribonucleic acid
dRib	deoxyribose
ΔG	free energy change
ΔH	enthalpy change
ΔS	entropy change
FAD	oxidised flavine adenine dinucleotide
$FADH_2$	reduced flavine adenine dinucleotide
FFA	free fatty acid
FH_4	tetrahydrofolate
FMN	oxidised flavine mononucleotide
$FMNH_2$	reduced flavine nononucleotide
Fru	fructose
FSH	follicle stimulating hormone
Fuc	fucose
G	guanine
Gal	galactose
GalN	galactosamine
GDP	guanosine diphosphate
GH	growth hormone
Glc	glucose
GlcA	glucuronic acid
GlcN	glucosamine
Gln	glutamine
Glu	glutamate
Gly	glycine
GMP	guanosine monophosphate
GSH	reduced glutathione
GSSG	oxidized glutathione
GTP	guanosine triphosphate
Hb	haemoglobin
HbO_2	oxygenated haemoglobin
His	histidine
Hyp	hydroxyproline
IduA	iduronic acid
IgA	immunoglobulin A
IgD	immunoglobulin D
IgE	immunoglobulin E
IgG	immunoglobulin G
IgM	immunoglobulin M
Ile	isoleucine
kJ	kilojoule (= ~4.2 kilocalories)
Leu	leucine
LH	luteinizing hormone
Lys	lysine
Man	mannose
Met	methionine
mRNA	messenger RNA
MSH	melanophore stimulating hormone
NAD^+	oxidized nicotinamide adenine dinucleotide (DPN^+ in older literature)
NADH	reduced nicotinamide adenine dinucleotide (DPNH in older literature)
NANA	*N*-acetyl neuraminic acid
P_i	inorganic phosphate
Phe	phenylalanine
PP_i	inorganic pyrophosphate
Pro	proline
Rib	ribose
RNA	ribonucleic acid
rRNA	ribosomal RNA
s	sedimentation coefficient
Ser	serine
T	thymine
T_3	triiodothyronine
T_4	thyroxine
TDP	thymidine diphosphate
TG	triglyceride
Thr	threonine
TMP	thymidine monophosphate
tRNA	transfer RNA
Try	tryptophan
TSH	thyroid stimulating hormone
TTP	thymidine triphosphate
Tyr	tyrosine
U	uracil
UDP	uridine diphosphate
UMP	uridine monophosphate
UTP	uridine triphosphate
Val	valine
Xyl	xylose

Introduction

The aim of this book is to present in one volume the biochemistry and cell biology necessary for an understanding of the molecular basis of medicine.

The human body, like all other living organisms, is made up of cells. Cells, most of which are approximately the same size, contain within them distinctive structures such as nucleus, mitochondria, endoplasmic reticulum. The function of these structures is the same in all cells of the body and in the cells of all animals and plants. Part One of this book, which describes the workings of a living cell in terms of such organelles, is therefore as relevant to a mouse as to a human being. Indeed much of the information has been gained by studying mice rather than human beings. The realization that the chemical reactions that occur within cells are the same not only in animals and plants, but in microbial cells also, has led to most of the biochemical details of cellular activity being derived from simple and rapidly dividing bacteria such as *Escherichia coli*. Hence Part One is really an introduction to the function of cells in general. Defects in function that lead to disease are indicated in the appropriate places.

Practically every cell in the human body has the potentiality to synthesize all the proteins and other molecules that make up a human being. But only a *part* of that potentiality is ever expressed in any one cell. Clusters of cells, grouped together in tissues and organs, each express a discrete potentiality, superimposed on the basic biochemical pathways necessary to remain alive: muscle cells, for example, synthesize creatine and myosin, pancreatic cells synthesize digestive enzymes and insulin, lymphocytes synthesize immunoglobulins, and so forth. In Part Two this cellular specialization is described.

In so far as human biochemistry is studied in order to relate it to human disease, then, this book is an attempt to present in molecular terms the basis of that relationship. In order to create a book which is written within the economic as well as within the physical grasp of the student, only the main essentials of each topic have been presented. A short bibliography is appended for those who wish to pursue in greater depth aspects of the subject in which they are particularly interested.

Acknowledgements

I am grateful to many colleagues, especially H. Baum, N. D. Carter, M. J. Clemens, J. A. Edwardson, D. A. Hems, M. R. Holloway, L. Hudson, J. S. Jenkins, P. Knox, T. Northfield, D. J. Perkins, H. P. Rang, and D. H. Williamson for helpful criticism of parts of this book. The careful typing of the manuscript by Helen Withers Green, and the production of the finished work by the staff of the Oxford University Press deserve especial mention. The facilities provided by Dr. S. Grisolia, Director of the Instituto de Investigaciones Citologicas, Valencia, made undisturbed work possible on a number of occasions. Chiefly I am indebted to my family for putting up with so many 'lost' week-ends, during which the weeds in our garden grew considerably faster than the pages of the book that follows.

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August, 1978.

Contents

Part One

Cellular potentiality

1

Cellular constituents and their function

1.1 Composition of cells

Cells, like all living matter, are made up largely of water. The remainder consists of proteins, fats, carbohydrates, nucleic acids, dissolved molecules, and inorganic ions. The approximate amounts that are present in the cells of liver are shown in Fig. 1.1. The distribution is approximately the same in other cells, with minor variations. Fat cells, for example, contain more fat, calcifying tissue, more inorganic ions, and so forth.

The liver is often taken as typical of animal cells, and much of the basic biochemistry of cells is derived from rat liver. However, it should be appreciated (i) that liver tissue contains a mixture of cells, such as hepatocytes, Kupffer cells, and cells of connective tissue, and (ii) that each of these is a specialized cell, secreting specific proteins and other substances. The liver cell is therefore no more typical of animal cells than a cell taken from the kidney or heart.

In fact, there is no such thing as a typical cell. But liver tissue is soft and easily homogenized, and this is an advantage for biochemical studies, which are carried out on solutions or suspensions of cell constituents rather than on the complicated structures of intact cells. Moreover liver does perform all the basic biochemical pathways described in Part One of this book, and hence is as suitable as any other cell. It is because the biochemistry of nucleus, mitochondria, or endoplasmic reticulum is the same in all cells, that it does not matter which type of cell one studies, *provided* that its specialization is not so intense as to mask the basic reactions. The red cell, which contains no nucleus, mitochondria, or endoplasmic reticulum, is an extreme example of the latter situation.

1.1.1 Proteins

1.1.1.1 Primary structure

Proteins are polymers of **amino acids**, linked by **peptide bonds**. The twenty amino acids commonly found in proteins, whether of animal, plant, or microbial origin, are shown in Fig. 1.2, together with a diagram of a peptide bond. Note that all amino acids except glycine have one or more asymmetric carbon atoms (that is, an atom that has each of its four valencies linked to a different substituent), and hence are optically active. All amino acids have the L(+) configuration about the α, or C-2, carbon atom (Fig. 1.2).

The breaking of a peptide bond by hydrolysis is a reaction in which energy is released (**exergonic reaction**); the formation of a peptide bond is the opposite (Fig. 1.3). That is to say, it is a reaction that 'requires' energy (**endergonic reaction**). Endergonic reactions do not take place unless (a) they are somehow coupled to an exergonic reaction, or (b) the concentration of the

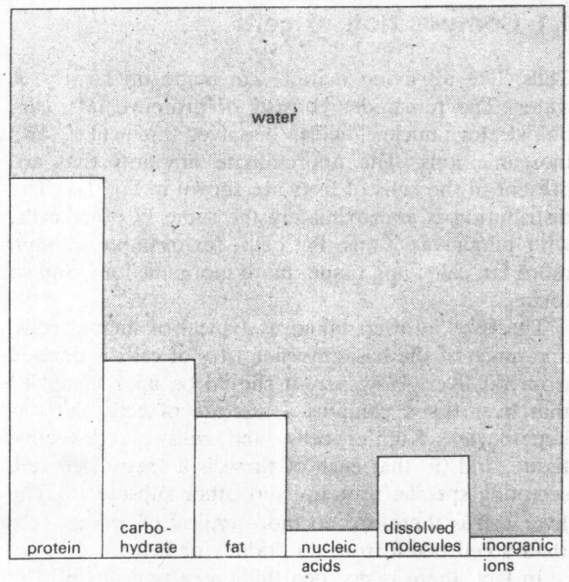


Fig. 1.1 Composition of cells

The approximate composition of liver cells is shown.

reactants is greatly increased. There is no way of 'supplying' energy as such; heat, for example (which is used to 'trigger' reactions – that is to lower the activation energy, see section 1.2.1) cannot be directly used to turn an endergonic reaction into an exergonic one. The use of the phrase 'energy-requiring' (or 'energy-supplying') is therefore not strictly accurate; it does however illustrate the nature of endergonic reactions so well that it is used throughout this book. The way in which endergonic reactions are made to occur in living cells, by (a) coupling to exergonic reactions or (b) increasing the concentration of reactants, is described in the chapters that follow.

Proteins are made up of one or more linear, unbranched chains of amino acids. This is known as the **primary structure** of a protein (Fig. 1.4). The chain is generally some hundreds of amino acids long, yielding proteins of molecular weight between 10 000 and 100 000.

1.1.1.2 Secondary structure

The nitrogen and oxygen atoms of amino acids are in the normal valency states of 3 and 2 respectively, and therefore have one or two pairs of 'spare' unbonded electrons in their first octet. As a result they tend to attract any hydrogen atoms (themselves bonded to nitrogen or oxygen) that are available in the vicinity, to form a **hydrogen bond** (Fig. 1.5). In this bond, the 1S shell of the hydrogen atom becomes distorted in such a way that four (not two) electrons are able to associate with the hydrogen nucleus. The hydrogen atoms attracted are generally those of a water molecule, since water molecules predominate in biological fluids, but may also be part of another amino acid. Hydrogen bonds between amino acids and water contribute to the solubility of proteins in aqueous media. Hydrogen bonds between one amino acid and another amino acid four residues along the primary sequence give rise to a coiling of the primary structure (Fig. 1.6). The resulting **α-helix** is an example of what is termed the **secondary structure** of a protein. The hydrogen bond is much weaker than a covalent bond. Hence secondary structure is disrupted more easily than primary structure. For example, heating a protein in neutral solution at 60 °C, or adding a strong solution of urea or other compound that competes for hydrogen bonds, disrupts the α-helix. On the other hand, heating a protein in boiling 6N HCl for 24 hours is necessary to disrupt (that is, to hydrolyse, Fig. 1.3) a peptide bond.

The importance of the hydrogen bond to all forms of life cannot be overstressed. It contributes not only to the three-dimensional structure of proteins, as a result of which unique enzyme–substrate (section 1.2), or antibody–antigen (Section 11.2.1.1), interactions take place, but also to the pairing between complementary bases on two strands of nucleic acid, as a result of which the genotype (DNA) is faithfully reproduced at each cell division (Chapter 4) and as faithfully translated into protein via RNA in order to express its phenotypic potential (Chapter 5). In short, the hydrogen bond is crucial to the basic processes of living cells.

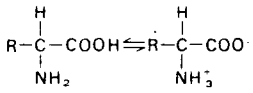
When the conformation of the amino acids in part of a protein chain is such as to make coiling into an α-helix difficult, that stretch of protein tends to exist in its straight, uncoiled form. Another manner of coiling through hydrogen bonds results in β-pleated sheets, as in silk fibroin. In proteins such as collagen that have a high content of proline and hydroxyproline (which lack a hydrogen atom attached to nitrogen, Fig. 1.2), hydrogen bond formation cannot occur. Instead a triple helix (called type 2 trans-helix), stabilized by proline–proline interactions, is formed (see section 10.3.2.1).

1.1.1.3 Tertiary structure

A strand of amino acids (whether in α-helix or not) as long as a globin chain (Fig. 1.4) – and globin is one of the smaller proteins known – tends to fold back on itself, producing **tertiary structure** (Fig. 1.7). The bonds involved in tertiary structure are (i) covalent bonds, (ii) hydrogen bonds, (iii) electrostatic bonds, and (iv) hydrophobic bonds.

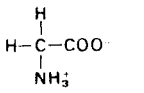
Covalent bonds: The only covalent bonds that contribute to tertiary structure are those between the sulphur of one cysteine residue and the sulphur of

General formula

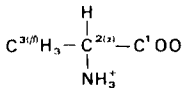


Aliphatic side-chains (R groups)

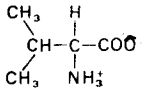
Unsubstituted



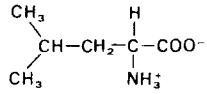
glycine (Gly)



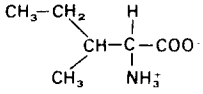
alanine (Ala)



valine (Val)

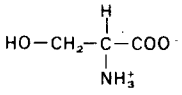


leucine (Leu)

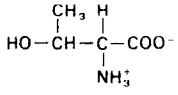


isoleucine (Ile)

OH-containing

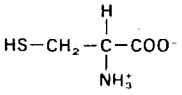


serine (Ser)

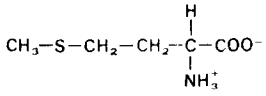


threonine (Thr)

S-containing

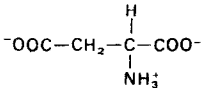


cysteine (Cys)
[see Fig. 1.8]

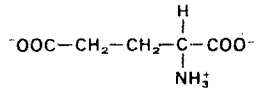


methionine (Met)

Negatively-charged

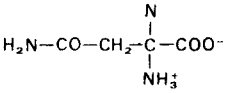


aspartate (Asp)

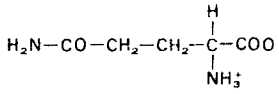


glutamate (Glu)

Asp and Glu are acidic, but their amides, Asn and Gln are neutral

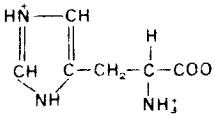


asparagine (Asn)

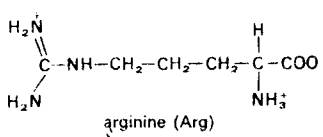


glutamine (Gln)

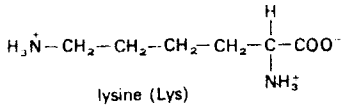
Positively-charged



histidine (His)



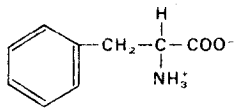
arginine (Arg)



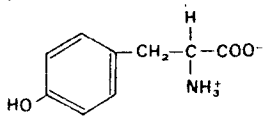
lysine (Lys)

Arg and Lys are basic; His is neutral [see Fig.1.10]

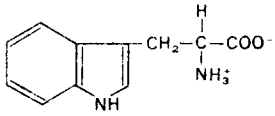
Aromatic side-chains (R groups)



phenylalanine (Phe)

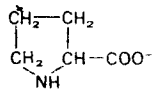


tyrosine (Tyr)

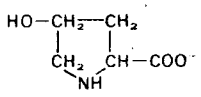


tryptophan (Try)

Imino acids

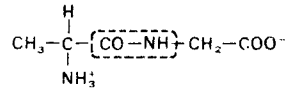


proline (Pro)



hydroxyproline (Hyp)

A peptide bond



alanyl glycine (Ala-Gly)

Fig. 1.2 Structures of common amino acids

The numbering of the carbon atoms is shown in the case of alanine.

another. The resulting **disulphide bond** is rather weak, in the sense that little energy is involved in the oxidation–reduction reaction (Fig. 1.8). Disulphide bonds are formed by bubbling oxygen through a solution of a protein, and are disrupted by addition of competing thiols such as mercaptoethanol (CH_3CH_2SH).

Hydrogen bonds: These bonds formed through the —OH of tyrosine, the free —COOH of aspartate and glutamate, the N of histidine and arginine, or the free NH_2 of lysine, contribute to tertiary structure.

Electrostatic bonds: These are formed between a positively and a negatively charged amino acid. The extent to which an amino acid is ionized depends on the tendency of its groups to ionize at the pH of the environment. The tendency is determined by the dissociation constant, and is expressed as **pK;** this is the pH at which the group is 50 per cent dissociated. It is

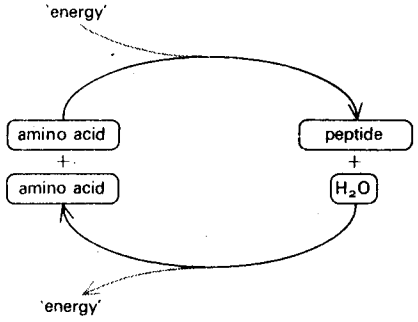


Fig. 1.3 Formation and rupture of peptide bonds

NH_2–Val His Leu Thr Pro Glu Glu Lys Ser Ala Val Thr
Gly Val Glu Asp Val Asn Val Lys Gly Trp Leu Ala
Gly Glu Ala Leu Gly Arg Leu Leu Val Val Tyr Pro
Leu Asp Gly Phe Ser Glu Phe Phe Arg Gln Thr Trp
Ser Thr Pro Asp Ala Val Met Gly Asn Pro Lys Val
Ser Phe Ala Gly Leu Val Lys Lys Gly His Ala Lys
Asp Gly Leu Ala His Leu Asp Asn Leu Lys Gly Thr
Leu Lys Asp Cys His Leu Glu Ser Leu Thr Ala Phe
His Val Asp Pro Glu Asn Phe Arg Leu Leu Gly Asn
Lys Gly Phe His His Ala Leu Val Cys Val Leu Val
Glu Phe Thr Pro Pro Val Gln Ala Ala Tyr Gln Lys
Lys His Ala Leu Ala Asp Ala Val Gly Ala Val Val
Tyr His COOH

Fig. 1.4 Primary structure of proteins

The sequence of the β-chain of haemoglobin is shown.

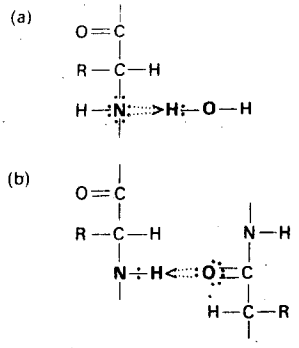


Fig. 1.5 The hydrogen bond

The electrons of the atoms that participate in H bond formation between (a) an amino acid and water and (b) an amino acid and another amino acid, are shown.

also the pH at which the group has maximal buffering capacity (Fig. 1.9). The pK values of the ionizable groups of amino acids and other biological molecules are given in Fig. 1.10.

Electrostatic bonds are relatively weak and can be disrupted by solutions of strongly ionized salts such as NaCl, or by high concentrations of H^+ (low pH) or OH^- (high pH) (Fig. 1.11).

Hydrophobic bonds: The term **hydrophobic bond** (literally 'water-loathing') is apt, since the interaction is the result of a mutual avoidance of the aqueous milieu

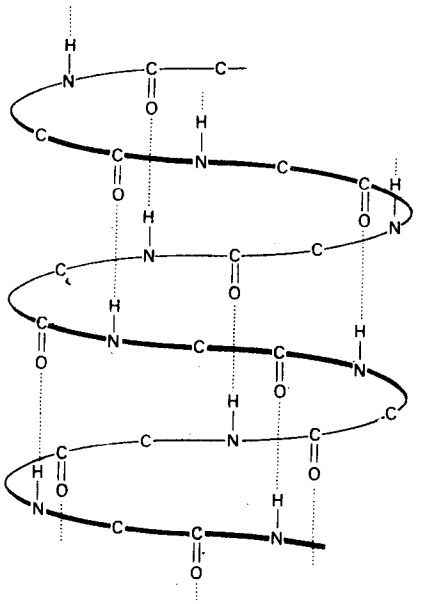


Fig. 1.6 Secondary structure of proteins: the α-helix

The α-carbon atoms are shown unbonded for clarity; and the H bonds appear longer than they actually are.

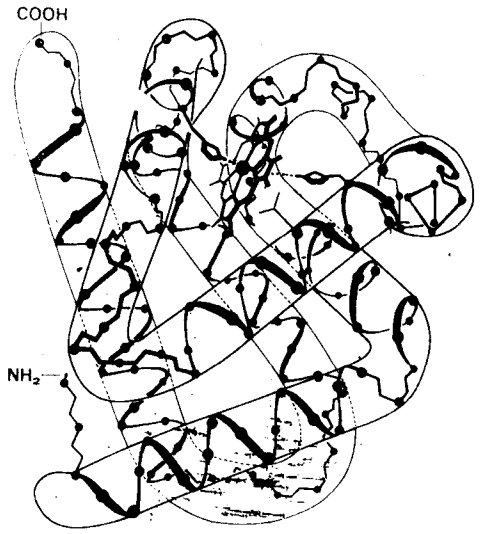


Fig. 1.7 Tertiary structure of proteins

One of the β-chains of haemoglobin is shown. Each amino acid is indicated by a dot. The large dot surrounded by a planar ring in the upper right part of the molecule is the Fe atom and the surrounding porphyrin, known as haem. The 'sausage-like regions indicate the folding of the chain due to tertiary interactions. Within each region, sequences of α-helix can be seen. The structure of the α-chains is similar. Reproduced from a model constructed by Dr. M. F. Perutz, with permission.

by two adjacent groupings. Aromatic groups (of phenylalanine, tyrosine, and tryptophan) and long chains of CH_2 groups (as in leucine, isoleucine, valine, etc.) are the main contributing species. The bonds are

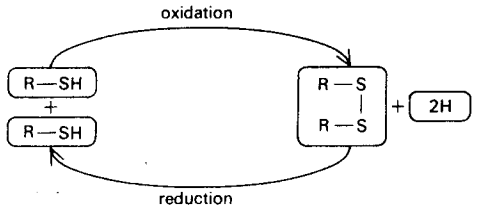


Fig. 1.8 Formation and rupture of disulphide bonds

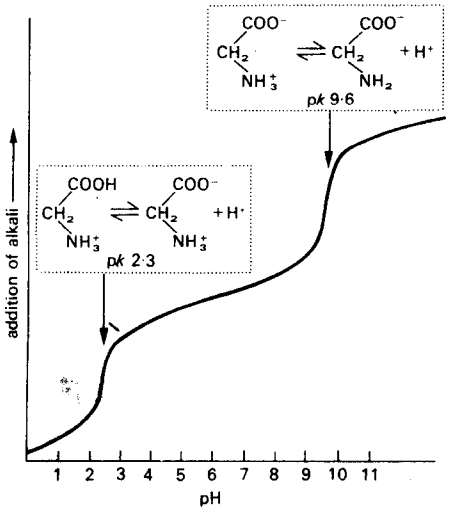


Fig. 1.9 Determination of p*K*

A break in the titration curve (shown for glycine) gives the p*K*.

Amino acids

	α-COOH	α-NH_2	R group	
glycine	2·3	9·6		
aspartate	2·1	9·8	3·9	(-COOH)
glutamate	2·2	9·7	4·2	(-COOH)
histidine	1·8	9·2	6·0	
cysteine	1·7	10·8	8·3	(-SH)
tyrosine	2·2	9·1	10·1	
lysine	2·2	9·0	10·5	(-NH_2)
arginine	2·2	9·0	12·5	

Phosphorylated compounds

inorganic phosphate	2·1, 7·2, 12·4
AMP	3·7, 6·3
glucose 6-P	1·0, 6·1

Fig. 1.10 p*K* values of some ionizable groupings in biological molecules

weak and are disrupted by heat or by competition for another hydrophobic compound such as chloroform.

Of these four types of bond, hydrophobic bonds are probably the most important for the determination of tertiary structure. Electrostatic bonds are the least important, since most of the charged amino acids 'face outwards'; that is, they interact with the water molecules of the environment, rather than with each other.

Hydrogen, electrostatic, and hydrophobic bonds are weak in comparison to covalent bonds. What makes the tertiary structure of a protein stable is the *number* of bonds that are involved, rather than the strength of any one bond. Disruption of tertiary structure is caused by the same agents (mild heat, exposure to urea) that disrupt secondary structure, as well as by extremes of pH, by mild oxidation, and so forth. The unfolded protein that results is said to be denatured. Provided that denaturation is carried out in dilute solution and gently enough, so that precipitation, for example, is avoided, the process is reversible, and the protein can be renatured by restoration of the original conditions. In other words, the native (undenatured) conformation of a protein tends to be the most stable. Because no bond involved in secondary or tertiary structure is strong, little free energy is released when a protein is denatured. On the other hand there is a big difference in **entropy** (section 3.3.1) between a highly coiled and folded structure (low entropy) and an unfolded, randomly orientated one (high entropy). In other words, when a protein is denatured the heat (enthalpy) change, ΔH (section 3.3.1), is approximately equal to the entropy change ($T\Delta S$), with only a small **free energy change** (ΔG); when a denatured protein is hydrolysed, there is in addition a big free energy change (ΔG). The concepts of entropy and free energy are more fully discussed in section 3.3.1.

1.1.1.4 Quaternary structure

Several proteins are made up of a number of polypeptide chains or subunits, which may be identical or different. Insulin, for example, consists of different A- and B-chains (Fig. 9.17), chymotrypsin consists of different A, B, and C chains (Fig. 1.29); haemoglobin consists of two α- and two β-chains, each with its own haem residue (Fig. 1.12), lactate dehydrogenase consists of four chains, each of which is either α or β (Fig. 2.10) while immunoglobins consist of two light and two heavy chains (Fig. 11.4). This combination of subunits – by bonds similar to those involved in tertiary structure – is known as **quaternary structure**.

1.1.2 Fats

Fats (also called **lipids**) are either neutral or charged. The neutral fats comprise **triglyceride**, **cholesterol**,

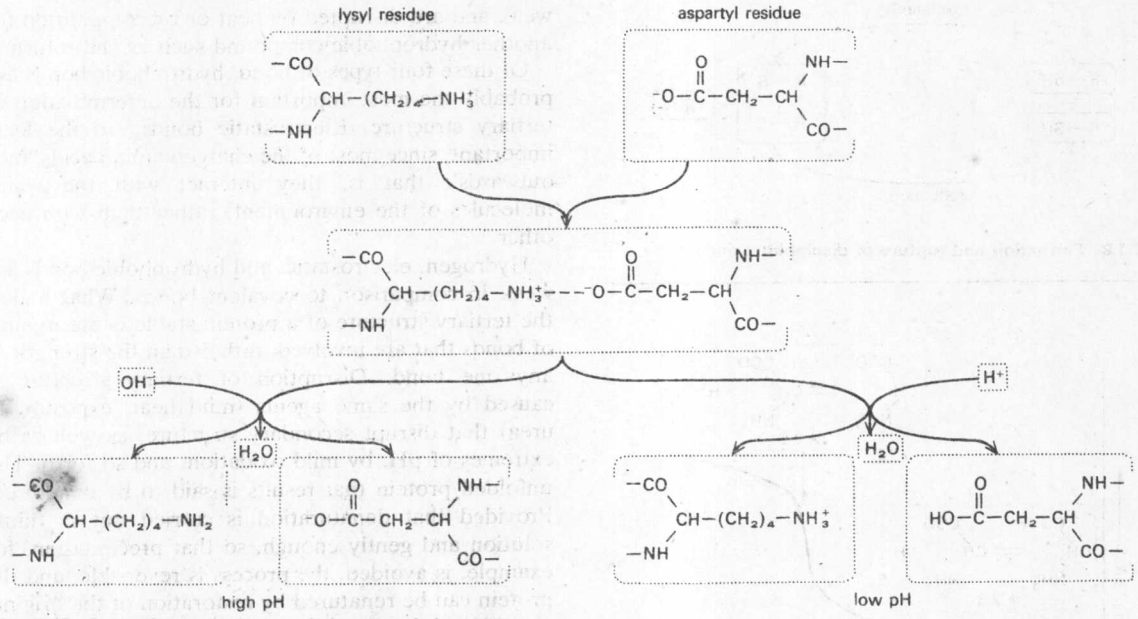


Fig. 1.11 Formation and rupture of electrostatic bonds

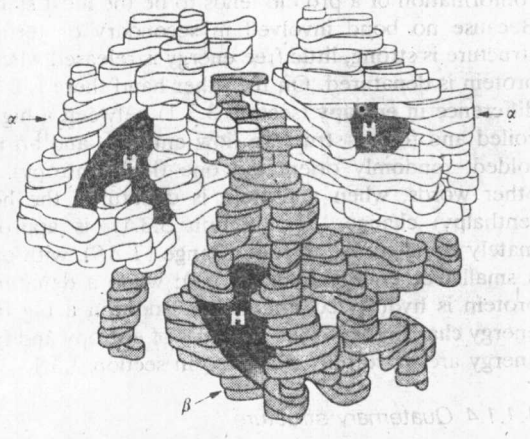


Fig. 1.12 Quaternary structure of proteins

The quaternary structure of haemoglobin consists of two α-chains and two β-chains. In this Figure, two of the α-chains, and one of the β-chains, are seen; the other β-chain is hidden. The three haem groups associated with the chains that are visible, are shown as flat discs labelled H. This illustration, derived from an X-ray crystallographic reconstruction, does not show the individual atoms (as in Fig. 1.7), but rather the 'electron densities'. Reproduced from a model constructed by Dr. M. F. Perutz, with permission.

cholesteryl esters, and certain **glycolipids**. The charged fats comprise **phospholipids** and acidic glycolipids. The structures of these compounds are given in Fig. 1.13. In so far as fats are 'small' molecules (molecular weight less than 1000), they do not have the secondary or tertiary structure of proteins. Their shape is determined, like that of individual amino acid residues, by the nature of the chemical bonds involved. Cholesterol, for example, has the 'puckered' shape typical of other cyclic, non-aromatic, hydrocarbons; long-chain fatty acids have a flexible shape, with regions of rigidity where double bonds occur.

All organic molecules fall roughly into one of two categories. They are either **polar** (**hydrophilic**) or they are **apolar** (**hydrophobic**). Polar molecules tend to form hydrogen bonds with water, which makes them soluble in aqueous media. Apolar molecules are incapable of forming hydrogen bonds with water, and are therefore insoluble in aqueous media; instead, they tend to form hydrophobic bonds with each other. Neutral fats fall into the latter category. They exist either as fat droplets in intracellular or extracellular fluid (for example chylomicrons), or are embedded within the apolar regions of biological membranes (Fig. 1.14).

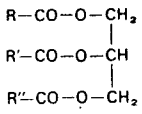
Charged fats and neutral glycolipids are molecules having two distinct regions: a polar and an apolar one. As a result they are termed amphipathic molecules. The polar region is in contact with water or other polar molecules; the apolar region is in contact with apolar molecules or with the apolar region of another amphipathic molecule (Fig. 1.14). No covalent bonds are involved in linking lipids to each other or to other molecules. The importance of membranes to living cells is discussed below (section 1.3).

1.1.3 Carbohydrates

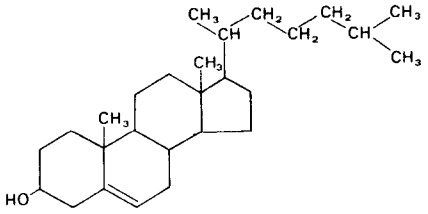
The term carbohydrate is used to denote **monosaccharides** such as glucose or fructose, **disaccharides** such as sucrose or lactose, and **polysaccharides** such as

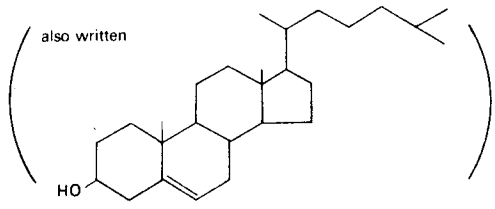
NEUTRAL

triglyceride

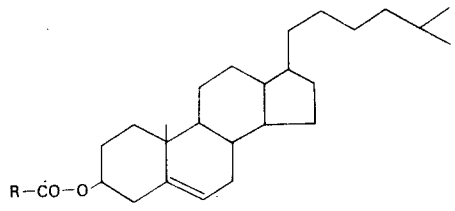


cholesterol

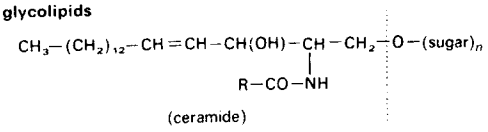




cholesteryl ester



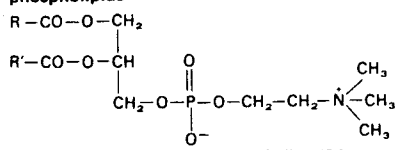
glycolipids

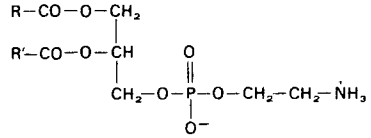


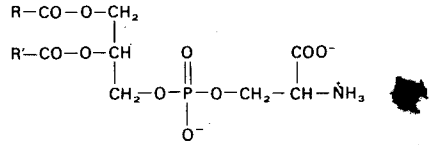
n = 1, 2, 3, or 4 residues; the sugars are generally glucose, galactose, *N*-acetyl glucosamine, or *N*-acetyl galactosamine

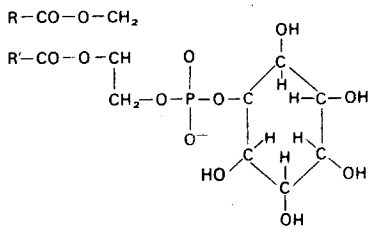
CHARGED

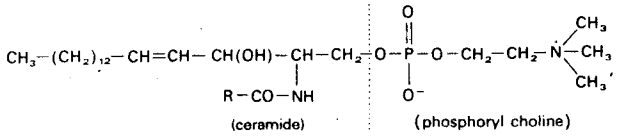
phospholipids

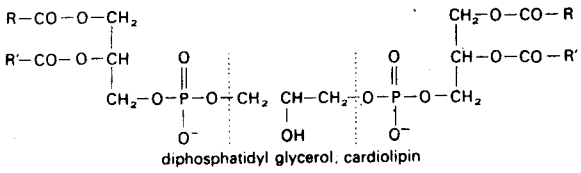




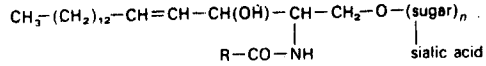




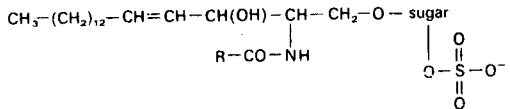




acidic glycolipids



Sialic acid-containing glycolipids are known as gangliosides; generally there are 1–3 sialic acid residues



Sulphate-containing glycolipids are known as sulphatides (cerebroside sulphates); generally the sugar is galactose or glucose

The R groups shown in the figure are fatty acyl résidues. The commonest fatty acids are:

C_{16}: $CH_3(CH_2)_{14}COOH$ palmitic acid

C_{18}: $CH_3(CH_2)_{16}COOH$ stearic acid

$C_{18:1}$: $CH_3(CH_2)_7CH=CH(CH_2)_7COOH$ oleic acid

Also important are the polyunsaturated (essential) fatty acids:

$C_{18:2}$: $CH_3(CH_2)_4CH=CH—CH_2—CH=CH—(CH_2)_7COOH$ linoléic acid

$C_{18:3}$: $CH_3—CH_2—CH=CH—CH_2—CH=CH—CH_2—CH=CH—(CH_2)_7COOH$ linolenic acid

$C_{20:4}$: $CH_3(CH_2)_4[CH=CH—CH_2]_3CH=CH(CH_2)_3COOH$ arachidonic acid

(see Figs 1.13 and 1.14 for structures of sugars and sialic acid).

Fig. 1.13 Structures of common lipids

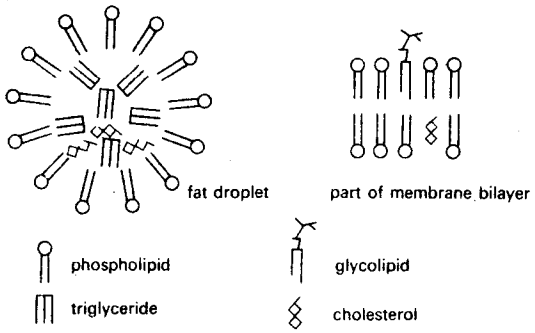
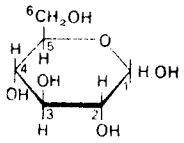


Fig. 1.14 Arrangement of lipids in aqueous environments

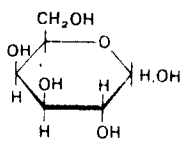
In each case (fat droplet or membrane bilayer), polar groups are in contact with the environment.

Monosaccharides

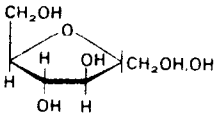
Hexoses



D-glucose (Glc)

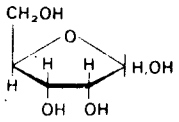


D-galactose (Gal)

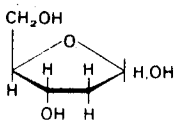


D-fructose (Fru)

Pentoses

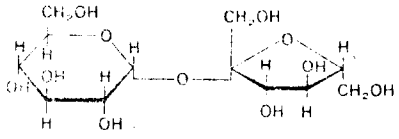


D-ribose (Rib)

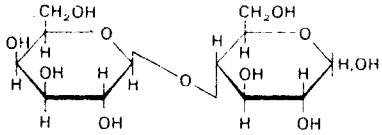


2-deoxy D-ribose (dRib)

Disaccharides

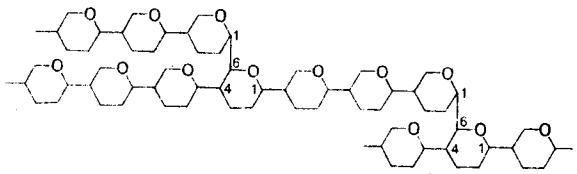


sucrose [D-glucosyl-(1→2) D-fructoside]



lactose [D-galactosyl-(1→4) D-glucose]

Polysaccharides



glycogen (poly D-glucosyl $\left\{\begin{matrix}(1\rightarrow4)\\(1\rightarrow6)\end{matrix}\right\}$ glucose)

Fig. 1.15 Structures of common carbohydrates

The numbering of the carbon atoms is shown in the case of glucose.

glycogen or starch (Fig. 1.15). In addition, carbohydrate residues linked covalently to lipid or protein are found in most cells of the body. The glycolipids, having the characteristics of fats, have already been described (Fig. 1.13). **Glycoproteins**, which have the characteristics of proteins, contain some carbohydrate. Glycolipids and glycoproteins are important components of the plasma membrane (section 6.2.1). Protein–carbohydrate complexes that are predominantly carbohydrate, known as **proteoglycans**, occur largely in the spaces outside cells (section 10.3). They are heteropolymers containing amino sugars and uronic acids (Fig. 1.16) in addition to simple sugars.

The sugar residues of carbohydrates do not interact with each other in the way that the amino acid residues of proteins do. Instead they have a tendency, because of their many oxygen atoms, to form hydrogen bonds with water. As a result the sugar residues are spread out in random manner and the molecules become rather voluminous. The high viscosity and