

美国医师执照考试

High-Yield™ *Biochemistry*

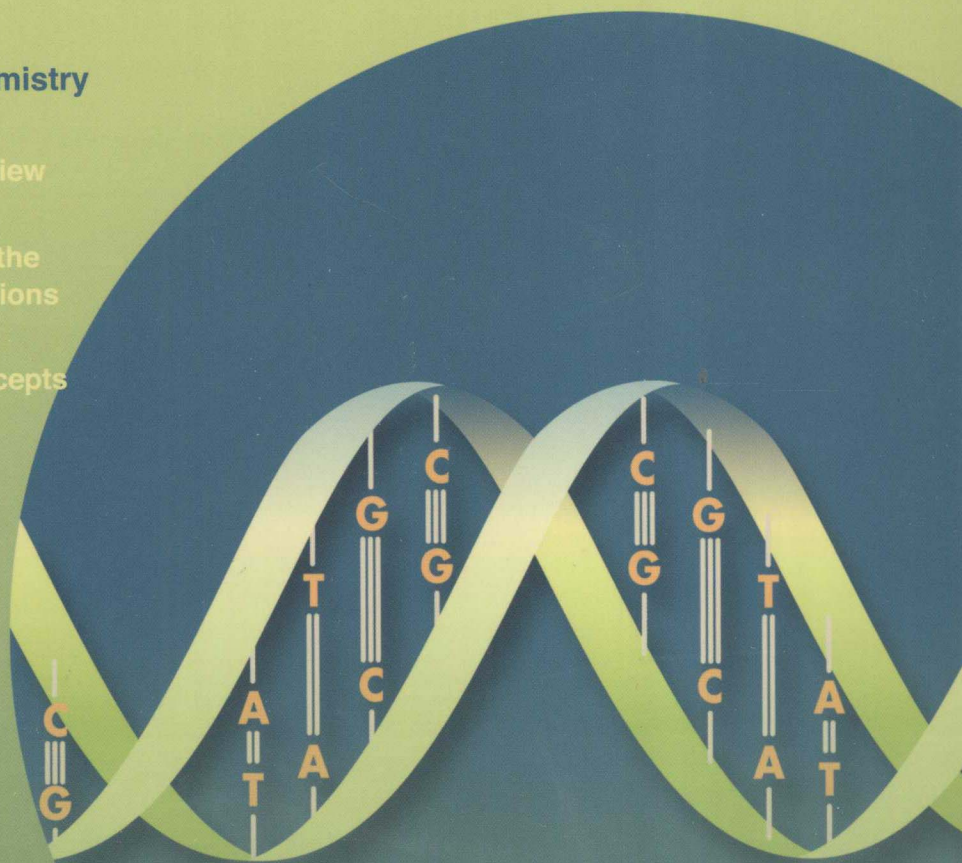
生物化学

(第3版)

R. BRUCE WILCOX

High-Yield™ Biochemistry
is designed to:

- Provide a quick review of biochemistry
- Help equip you for the biochemistry questions on Step 1
- Clarify difficult concepts



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美国医师执照考试

High-YieldTM 生物化学

Biochemistry

(第3版)

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北京大学医学出版社

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High-Yield™ 系列丛书是针对美国医师执照考试 (United States Medical Licensing Examination, USMLE) 的知名品牌图书, 受到世界各地读者的欢迎。该系列丛书具有以下特色:

1. 内容高度概括, 重点突出, 有利于读者快速掌握学科的核心知识。
2. 编排新颖, 既有基础知识要点的介绍, 又有以疾病为核心的综合归纳, 并体现了相关学科的横向联系。
3. 语言规范、地道, 既有利于读者快速掌握专业词汇, 又有利于医学英语思维的培养。

本系列丛书是参加美国医师执照考试的必备辅导用书, 也可作为我国医学院校从事双语教学的教材和参考用书, 对教师进行英语授课, 学生学习、参加考试具有重要的参考价值。

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This book is dedicated to my father, H. Bruce Wilcox, for endowing me with a passionate love for teaching, and to the freshman medical and dental students at Loma Linda University who for over 40 years have paid tuition at confiscatory rates so that I have never had to go to work.

Preface

High-Yield Biochemistry is based on a series of notes prepared in response to repeated and impassioned requests by my students for a “complete and concise” review of biochemistry. It is designed for rapid review during the last days and hours before the United States Medical Licensing Examination (USMLE), Step 1, and the National Board of Medical Examiners subject exams in biochemistry. Although this book provides information for a speedy review, always remember that you cannot review what you never knew.

Acknowledgments

Dr. John Sands provided invaluable help in reviewing and editing Chapter 11, “Biotechnology,” for the first edition. While preparing for the second edition, Lisa Umphrey, a third-year medical student at Loma Linda University, generously gave me access to her notations in the first edition. Katherine Noyes and Daniel Rogstad, graduate students in Biochemistry at Loma Linda University, gave generously of their time and expertise in assisting with revisions to Chapter 10, “Gene Expression” and Chapter 11, “Biochemical Technology” in the second edition. Daniel Rogstad assisted me again during preparation of the third edition. I am also indebted to Dr. J. Paul Stauffer at Pacific Union College for instruction in the felicitous use of English, to P.G. Wodehouse for continuing and enriching that instruction, and to General U.S. Grant for providing an example of clear and laconic communication.

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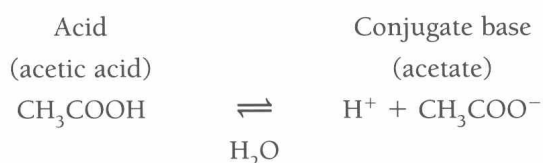
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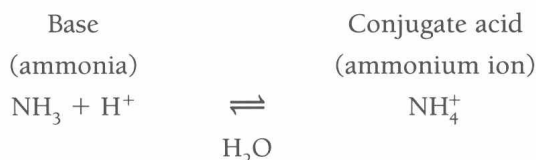
Acid–Base Relationships

I Acidic Dissociation

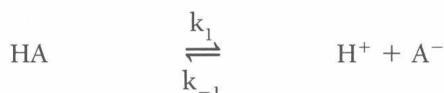
A. An acid dissociates in water to yield a **hydrogen ion** (H^+) and its conjugate base.



B. A base combines with H^+ in water to form its **conjugate acid**.



C. In the more general expression of acidic dissociation, **HA** is the acid (proton donor) and A^- is the **conjugate base** (proton acceptor).



II Measures of Acidity

A. pK_a

1. When acidic dissociation is at equilibrium, the **acidic dissociation constant**, K_a , is defined by:

$$K_a = \frac{[\text{H}^+][\text{A}^-]}{[\text{HA}]}$$

2. pK_a is defined as $-\log[K_a]$.
3. pK_a is a measure of the **strength** of an acid.
4. **Stronger acids** are more completely dissociated. They have **low pK_a values** (H^+ binds loosely to the conjugate base). Examples of stronger acids include the first dissociable H^+ of phosphoric acid ($\text{pK}_a = 2.14$) and the carboxyl group of glycine ($\text{pK}_a = 2.34$).
5. **Weaker acids** are less completely dissociated. They have **high pK_a values**. (H^+ binds tightly to the conjugate base.) Examples of weaker acids include the amino group of glycine ($\text{pK}_a = 9.6$) and the third dissociable H^+ of phosphoric acid ($\text{pK}_a = 12.4$).

B. pH

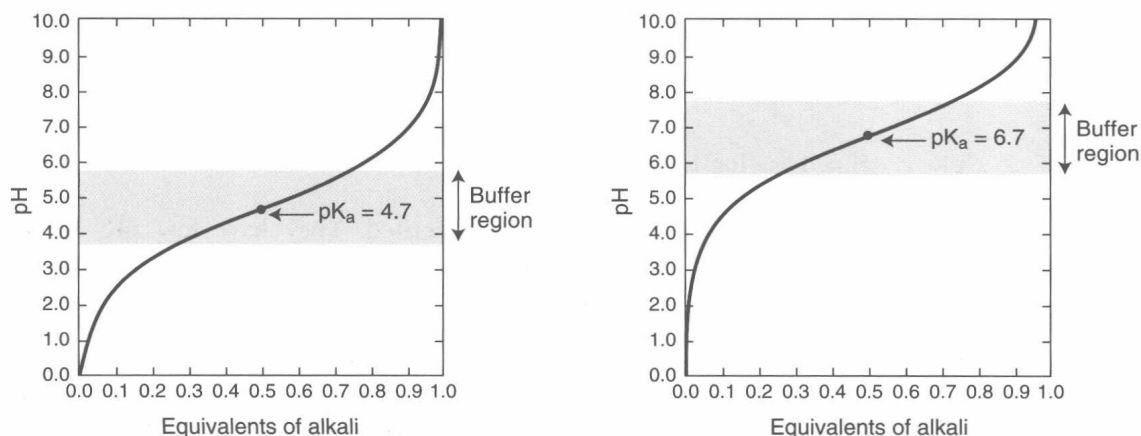
1. When the equation defining K_a is further rearranged and expressed in logarithmic form, it becomes the **Henderson-Hasselbalch equation**:

$$\text{pH} = \frac{\text{p}K_a + \log [A^-]}{[HA]}$$

2. pH is a measure of the acidity of a solution.
 - a. By definition, **pH equals $-\log[H^+]$** .
 - b. A **neutral solution** has a pH of 7.
 - c. An **acidic solution** has a pH of less than 7.
 - d. An **alkaline solution** has a pH of greater than 7.

III Buffers

- A. A **buffer** is a solution that contains a mixture of a weak acid and its conjugate base. It resists changes in $[H^+]$ on addition of acid or alkali.
- B. The **buffering capacity** of a solution is determined by the **concentrations** of weak acid and conjugate base.
 1. The **maximum buffering effect** occurs when the concentration of the weak acid $[HA]$ is equal to that of its conjugate base $[A^-]$.
 2. If $[A^-] = [HA]$, then $[A^-]/[HA] = 1$.
 3. When the buffer effect is at its maximum, the **pH of the solution equals the $\text{p}K_a$ of the acid**.
- C. The buffering effect is readily apparent on the titration curve for a weak acid such as $H_2PO_4^-$ (Figure 1-1).
 1. The **shape** of the titration curve is the same for all weak acids.
 2. At the **midpoint** of the curve, the **pH equals the $\text{p}K_a$** .
 3. The **buffering region** extends **one pH unit** above and below the $\text{p}K_a$.



● **Figure 1-1** Titration curves for acetic acid (CH_3COOH) (left) and phosphoric acid ($H_2PO_4^-$) (right). $H_2PO_4^-$ is the more effective buffer at physiologic pH.

IV

Acid-Base Balance

- A. Because pH strongly affects the stability of proteins and the catalytic activity of enzymes, **biological systems usually function best near neutrality**, that is, near $\text{pH} = 7$. Under normal conditions, blood pH is 7.4 (range, 7.37–7.42).
- B. The acid-base pair **dihydrogen phosphate (H_2PO_4^-)-monohydrogen phosphate (HPO_4^{2-})** is an **effective buffer at physiologic pH** (see Figure 1-1). Phosphate is an important buffer in the **cytoplasm**.
- C. The **carbon dioxide (CO_2)-carbonic acid (H_2CO_3)-bicarbonate (HCO_3^-) system** is the **principal buffer in plasma and extracellular fluid (ECF)**.

Carbonic anhydrase



1. CO_2 from tissue oxidation reactions dissolves in the blood plasma and ECF.
2. CO_2 combines with H_2O to yield H_2CO_3 . This reaction is catalyzed in red blood cells by **carbonic anhydrase**.
3. H_2CO_3 dissociates to yield H^+ and its conjugate base, HCO_3^- .
4. In this system, CO_2 is behaving like an acid, so the Henderson-Hasselbalch equation can be written:

$$\text{pH} = 6.1 + \log [\text{HCO}_3^-] / (0.0301) \text{ PCO}_2$$

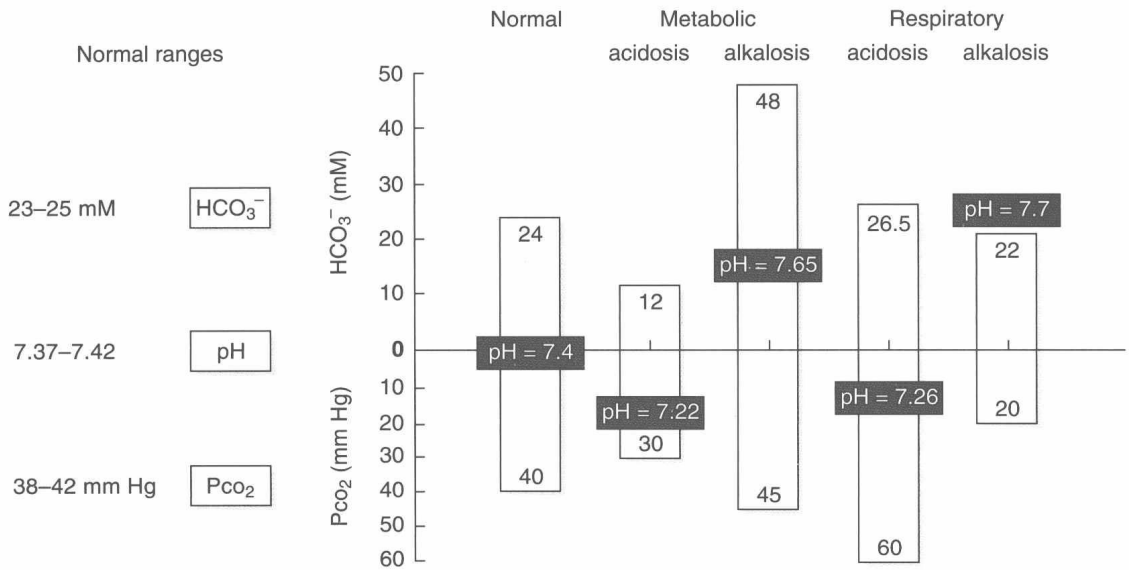
where $[\text{HCO}_3^-]$ is in mM and PCO_2 is in mm Hg.

- D. The CO_2 - H_2CO_3 - HCO_3^- buffer system is effective around the physiologic pH of 7.4, even though the pK_a is only 6.1, for four reasons:
1. The supply of CO_2 from oxidative metabolism is unlimited, so the effective concentration of CO_2 is very high.
 2. Equilibration of CO_2 with H_2CO_3 (catalyzed by carbonic anhydrase) is very rapid.
 3. The variation in CO_2 removal by the lungs (respiration) allows for rapid changes in the concentration of the H_2CO_3 .
 4. The kidney can produce or excrete HCO_3^- , thus changing the concentration of the conjugate base.

V

Acid-Base Disorders

- A. **ACIDOSIS** occurs when the pH of the blood and ECF falls below 7.35. This condition results in **central nervous system depression**, and when severe, it can lead to coma and death.
1. In **metabolic acidosis**, the $[\text{HCO}_3^-]$ **decreases** as a consequence of the addition of an acid stronger than H_2CO_3 to the ECF.
 2. In **respiratory acidosis**, the **partial pressure of CO_2 (PCO_2) increases** as a result of **hypoventilation** (Figure 1-2).
- B. **ALKALOSIS** occurs when the pH of the blood and ECF rises above 7.45. This condition leads to **neuromuscular hyperexcitability**, and when severe, it can result in tetany.
1. In **metabolic alkalosis**, the $[\text{HCO}_3^-]$ **increases** as a consequence of excess acid loss (e.g., vomiting) or addition of a base (e.g., oral antacid preparations).
 2. In **respiratory alkalosis**, the PCO_2 **decreases** as a consequence of **hyperventilation**.



● **Figure 1-2** Bar chart that demonstrates prototypical acid–base states of extracellular fluid (ECF). HCO_3^- is plotted up from zero, and PCO_2 is plotted down from zero.

VI Clinical Relevance: Diabetic Ketoacidosis

- A. Uncontrolled **insulin-dependent diabetes mellitus** (type I diabetes) involves decreased glucose utilization, with hyperglycemia, and increased fatty acid oxidation.
- B. **PATHOGENESIS OF KETOACIDOSIS**
 1. **Increased fatty acid oxidation** leads to excessive production of acetoacetic and 3-hydroxybutyric acids and of acetone, which are known as **ketone bodies**.
 2. Acetoacetic and 3-hydroxybutyric acids dissociate at body pH and release H^+ , leading to a **metabolic acidosis**.
- C. The combination of high blood levels of the ketone bodies and a metabolic acidosis is called **ketoacidosis**.
- D. The clinical picture involves **dehydration, lethargy, and vomiting**, followed by drowsiness and coma.
- E. **THERAPY** consists of correcting the hyperglycemia, dehydration, and acidosis.
 1. **Insulin** is administered to correct the hyperglycemia.
 2. **Fluids** in the form of physiologic saline are administered to treat the dehydration.
 3. In severe cases, intravenous **sodium bicarbonate** ($\text{Na}^+\text{HCO}_3^-$) may be administered to correct the **acidosis**.

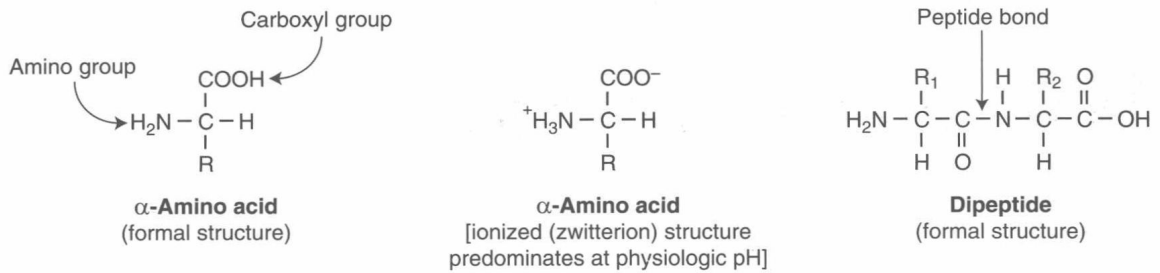
Amino Acids and Proteins

I Functions of Proteins

- A. Specific binding to other molecules
- B. Catalysis
- C. Structural support
- D. Coordinated motion

II Proteins as Polypeptides

- A. Proteins are **polypeptides**: polymers of amino acids linked together by peptide bonds (Figure 2-1).
 - 1. Proteins are synthesized from **20 different amino acids**.
 - 2. Some of the amino acids are modified after incorporation into proteins (e.g., by hydroxylation, carboxylation, phosphorylation, or glycosylation). This is called **post-translational modification**.
- B. The amino acids are called **α -amino acids** because they have an amino ($-\text{NH}_2$) group, a carboxyl ($-\text{COOH}$) group, and some other “R-group” attached to the α -carbon (see Figure 2-1).
 - 1. **Aliphatic R-groups** that are **nonpolar (uncharged, hydrophobic)** (see Figure 2-2) are characteristic of alanine, valine, leucine, isoleucine, and proline, which is an imino acid (a secondary amine). Glycine has hydrogen ($-\text{H}$) as its R-group.
 - 2. **Aromatic R-groups** are components of phenylalanine, tyrosine, and tryptophan (see Figure 2-2). Phenylalanine and tryptophan are nonpolar. Tyrosine contains a polar hydroxyl group.
 - 3. **Hydroxyl-containing R-groups** that are **mildly polar (uncharged, hydrophilic)** are part of serine and threonine (see Figure 2-2).
 - 4. **Sulfur-containing R-groups** are characteristic of cysteine (a good reducing agent) and methionine (see Figure 2-2).
 - 5. **Carbonyl-containing R-groups** include the **carboxylates** aspartic acid and glutamic acid and their **amides** asparagine and glutamine. The carboxylates are **negatively charged and polar**, and their amides are **uncharged and mildly polar** (see Figure 2-2).
 - 6. **Basic R-groups**, which are **positively charged and polar (hydrophilic)**, are characteristic of lysine, arginine, and histidine (see Figure 2-2).



● **Figure 2-1** Structure of an α -amino acid and a dipeptide.

- C. Each protein has a characteristic shape, or **conformation**.
1. **The function of a protein is a consequence of its conformation.** The conformation of a functional protein is also called its **native structure**.
 2. The **amino acid sequence** of a protein determines its conformation.
 - a. The **rigid, planar nature of peptide bonds** dictates the conformation that a protein can assume.
 - b. The **nature and arrangement of the R-groups** further determine the conformation.

III Protein Structure

Four levels of hierarchy in protein conformation can be described.

- A. PRIMARY STRUCTURE** refers to the order of the amino acids in the peptide chain (Figure 2-3).
1. The **free α -amino group**, written to the left, is called the **amino-terminal** or **N-terminal** end.
 2. The **free α -carboxyl group**, written to the right, is called the **carboxyl-terminal** or **C-terminal** end.
- B. SECONDARY STRUCTURE** is the arrangement of hydrogen bonds between the peptide nitrogens and the peptide carbonyl oxygens of different amino acid residues (Figure 2-4; see also Figure 2-3).
1. In **helical coils**, the hydrogen-bonded nitrogens and oxygens are on nearby amino acid residues (see Figure 2-3).
 - a. The most common helical coil is a right-handed **α -helix**.
 - b. **α -keratin** from hair and nails is an α -helical protein.
 - c. **Myoglobin** has several α -helical regions.
 - d. Proline, glycine, and asparagine are seldom found in α -helices; they are “helix breakers.”
 2. In **β -sheets (pleated sheets)**, the hydrogen bonds occur between residues on neighboring peptide chains (see Figure 2-3).
 - a. The hydrogen bonds may be on different chains or distant regions of the same chain.
 - b. The strands may run **parallel** or **antiparallel**.
 - c. **Fibroin in silk** is a **β -sheet** protein.
- C. TERTIARY STRUCTURE** refers to the **three-dimensional arrangement** of a polypeptide chain that has assumed its secondary structure (see Figure 2-3). Disulfide bonds between cysteine residues may stabilize tertiary structure.

Aliphatic, nonpolar					
$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{H} \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_3 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH} \\ / \quad \backslash \\ \text{H}_3\text{C} \quad \text{CH}_3 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{CH} \\ / \quad \backslash \\ \text{H}_3\text{C} \quad \text{CH}_3 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{HC}-\text{CH}_3 \\ \\ \text{CH}_2 \\ \\ \text{CH}_3 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_2\text{N}^+-text{C}-\text{H} \\ \quad \backslash \\ \text{CH}_2 \quad \text{CH}_2 \\ \quad \quad \\ \text{H}_2\text{C} \quad \text{CH}_2 \end{array}$
Glycine (Gly)	Alanine (Ala)	Valine (Val)	Leucine (Leu)	Isoleucine (Ile)	Proline (Pro)
Aromatic			Sulfur-containing		
$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{C}_6\text{H}_5 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{C}_6\text{H}_4\text{OH} \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{C}_8\text{H}_6\text{N} \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{SH} \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{S} \\ \\ \text{CH}_3 \end{array}$	
Phenylalanine (Phe)	Tyrosine (Tyr)	Tryptophan (Trp)	Cysteine (Cys)	Methionine (Met)	
Hydroxyl, polar		Basic, polar			
$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{OH} \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{H}-\text{C}-\text{OH} \\ \\ \text{CH}_3 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{NH}_3^+ \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{NH} \\ \\ \text{C} = \text{NH}_2^+ \\ \\ \text{NH}_2 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{C}-\text{NH} \\ \quad \backslash \\ \text{C} \quad \text{CH} \\ \backslash \quad / \\ \text{C} \quad \text{N}^+-\text{H} \end{array}$	
Serine (Ser)	Threonine (Thr)	Lysine (Lys)	Arginine (Arg)	Histidine (His)	
Acidic, polar					
	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{COO}^- \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{C}=\text{O} \\ \\ \text{NH}_2 \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{COO}^- \end{array}$	$\begin{array}{c} \text{COO}^- \\ \\ \text{H}_3\text{N}^+-\text{C}-\text{H} \\ \\ \text{CH}_2 \\ \\ \text{CH}_2 \\ \\ \text{C}=\text{O} \\ \\ \text{NH}_2 \end{array}$	
	Aspartic acid (Asp)	Asparagine (Asn)	Glutamic acid (Glu)	Glutamine (Gln)	

● Figure 2-2 The 20 amino acids found in proteins, grouped by the properties of their R-groups.