



Physiology at a Glance

Fourth Edition

Jeremy P. T. Ward
Roger W. A. Linden

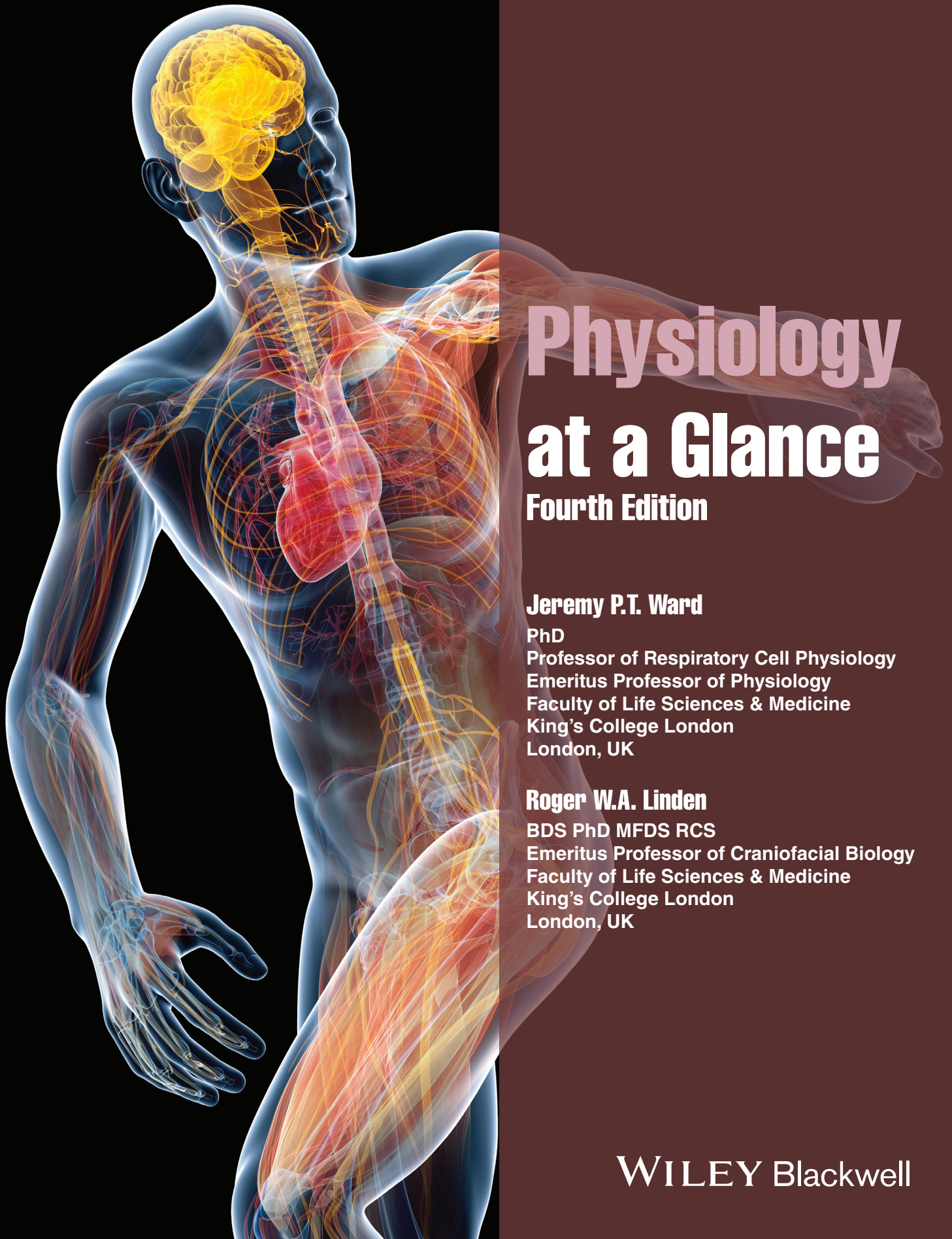


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Physiology at a Glance

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Physiology at a Glance

Fourth Edition

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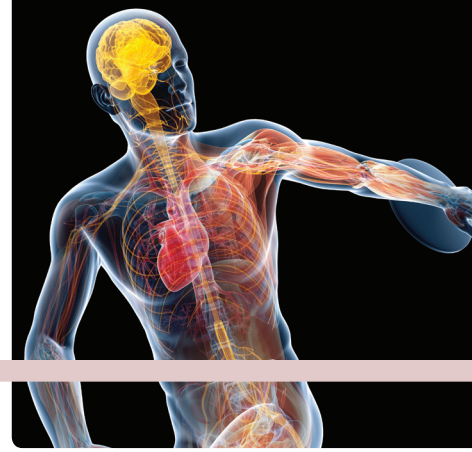
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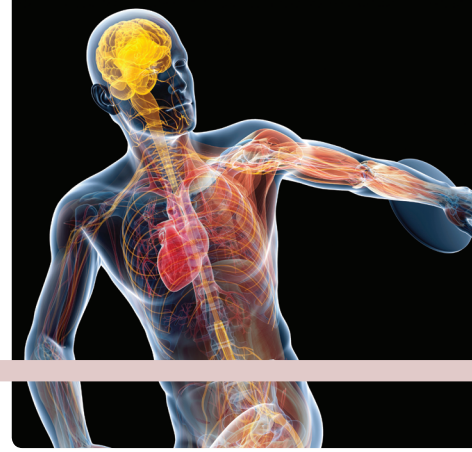
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Preface



Physiology is defined as ‘the scientific study of the bodily function of living organisms and their parts’. There is a natural symbiosis between function (physiology) and structure (anatomy) from which physiology emerged as a separate discipline in the late 19th century. A good understanding of anatomy and physiology is an essential prerequisite for understanding what happens when things go wrong – the structural abnormalities and pathophysiology of disease – and as such underpins all biomedical studies and medicine itself. Following a century of reductionism, where the focus of research has progressively narrowed down to the function of individual proteins and genes, there is now a resurgence in integrative physiology, as it has been realized that to make sense of developments such as the Human Genome Project we have to understand body function as an integrated whole. This is considerably more complex than just the sum of its parts because of the multiplicity of interactions involved. True understanding of the role of a single gene, for example, can only be gained when placed in the context of the whole animal, as reflected by the often unexpected effects of knock-out of single genes on the phenotype of mice.

This volume is designed as a concise guide and revision aid to core topics in physiology, and should be useful to all students following a first-year physiology course, whether they are studying single honours, biomedical sciences, nursing, medicine or dentistry. It should also be useful to those studying system-based curricula. The layout of *Physiology at a Glance* follows that of the other volumes in the *At a Glance series*, with a two-page spread for each topic (loosely corresponding to a lecture),

comprising a large diagram on one page and concise explanatory text on the other. For this fourth edition we have extensively revised the text and figures, there are three completely new chapters, on Cell signalling, Thermoregulation, and Altitude and aerospace physiology, and we have added a Glossary.

Physiology is a large subject, and in a book this size we cannot hope to cover anything but the core and basics. *Physiology at a Glance* should therefore be used primarily to assist basic understanding of key concepts and as an assistance to revision. Deeper knowledge should be gained by reference to full physiology and system textbooks, and in third-year honours programmes to original peer-reviewed papers. Students may find one or two sections of this book difficult, such as that on the physics of flow and diffusion, and detailed elements of cell signalling. Though such material may not be included in some introductory physiology courses, an understanding of these concepts can assist in learning how body systems behave in the way they do, and in understanding primary research papers.

In revising this fourth edition we have been helped immensely by constructive criticism and suggestions from our colleagues and students, and junior and senior reviewers of the last edition. We thank all those who have given us such advice; any errors are ours and not theirs. We would also like to thank the team at Wiley-Blackwell who provided great encouragement and support throughout the project.

Jeremy Ward
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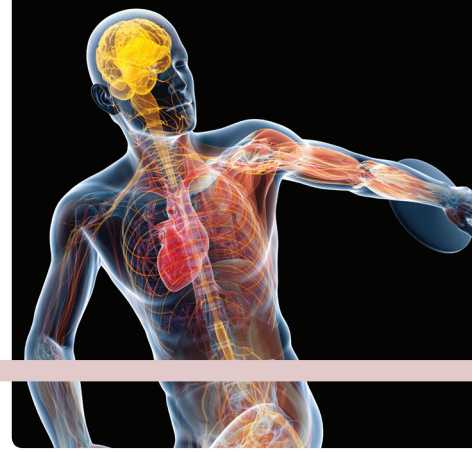
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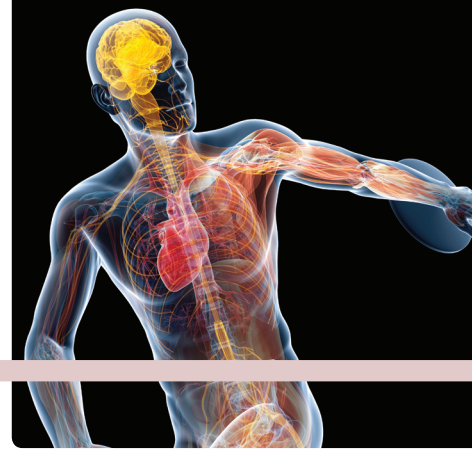
Abbreviations



1,25-(OH)₂D	1,25-dihydroxycholecalciferol	DNA	deoxyribonucleic acid
2,3-DPG	2,3-diphosphoglycerate	DOPA	dihydroxyphenylalanine
5-HT	5-hydroxytryptamine; serotonin	E_(ion)	equilibrium potential for ion (e.g. K ⁺ , Na ⁺ , Ca ²⁺ or Cl ⁻)
μG	micro-gravity (weightlessness)	ECF	extracellular fluid
ACE	angiotensin-converting enzyme	ECG (EKG)	electrocardiogram (or graph)
ACh	acetylcholine	EDP	end diastolic pressure
ACTH	adrenocorticotrophic hormone	EDV	end diastolic volume
ADH	antidiuretic hormone (also called vasopressin)	EGF	epidermal growth factor
ADP	adenosine diphosphate	E_m	membrane potential
AIDS	acquired immune deficiency syndrome	EMG	electromyogram
AMH	anti-Müllerian hormone	EPO	erythropoietin
AMS	acute mountain sickness	EPP	end plate potential
ANP	atrial natriuretic peptide	ERV	expiratory reserve volume
ANS	autonomic nervous system	ESV	end systolic volume
AP	action potential	ETC	electron transport chain
APC	active protein C <i>or</i> antigen presenting cell	F_{ab}	hypervariable region of antibody molecule
ATP	adenosine triphosphate	F_c	constant region of antibody molecule
ATPase	enzyme that splits ATP	FEV₁	forced expiratory volume in 1 s
AV node	atrioventricular node (heart)	FFA	free fatty acids
AVAs	arteriovenous anastomoses	FGF	fibroblast growth factor
BAT	brown adipose tissue	FN₂ (F_{O2})	fractional concentration of nitrogen (oxygen) in a gas mixture
BSA	body surface area	FRC	functional residual capacity
BTPS	body temperature and pressure, saturated with water	FSH	follicle-stimulating hormone
CaM	calmodulin	FVC	forced vital capacity
CaM-kinase	calcium-calmodulin kinase	G-LOC	G-forces induced loss of consciousness
cAMP	cyclic adenosine monophosphate	G-protein	GTP-binding protein
CaSR	calcium-sensing receptor (protein)	GDP	guanosine diphosphate
CCK	cholecystokinin	GFR	glomerular filtration rate
CDI	central diabetes insipidus	GH	growth hormone
cGMP	cyclic guanosine monophosphate	GHRH	growth hormone-releasing hormone
CICR	Ca ²⁺ -induced Ca ²⁺ release	GI	gastrointestinal
CNS	central nervous system	GIP	gastric inhibitory peptide
CO	cardiac output	GLP-1	glucagon-like peptide 1
COMT	catechol-O-methyl transferase	GLUT-1, 2 or 4	glucose transporters
COX	cyclooxygenase	GnRH	gonadotrophin-releasing hormone
CRH	corticotrophin-releasing hormone	GPCR	G-protein-coupled receptor
CSF	cerebrospinal fluid	GRP	gastrin-releasing peptide
CVP	central venous pressure	GTP	guanosine triphosphate
Da	Dalton (unit for molecular weight)	GTPase	enzyme that splits GTP
DAG	diacylglycerol	HACE	high-altitude cerebral oedema
DHEA	dehydroepiandrosterone	HAPE	high-altitude pulmonary oedema
D_LO₂	O ₂ diffusing capacity in lung; transfer factor	[Hb]	haemoglobin concentration

HbA	adult haemoglobin	PLC	phospholipase C
HbF	fetal haemoglobin	PMCA	plasma membrane Ca ²⁺ ATPase
hCG	human chorionic gonadotrophin	P_{O₂}	partial pressure of oxygen
HIV	human immunodeficiency virus	PRR	pattern recognition receptor
HMWK	high molecular weight kininogen	PTH	parathyroid hormone
ICF	intracellular fluid	Ras, Rho	small monomeric GTPases
IgA, E, G, M	immunoglobulin A, E, G or M	ROC	receptor-operated channels
IGF-1 or 2	insulin-like growth factor (1 or 2)	ROMK	renal outer medullary potassium channel
IL-1β or 6	interleukin-1 β or 6	RPF	renal plasma flow
IP₃	inositol trisphosphate	RTK	receptor tyrosine kinase
IRS-1	insulin receptor substrate 1	RV	residual volume <i>or</i> right ventricle
IRV	inspiratory reserve volume	SA node	sinoatrial node
ISF	interstitial fluid	SERCA	smooth endoplasmic reticulum Ca ²⁺ ATPase
JAK	Janus kinase		
JGA	juxtaglomerular apparatus	SH2	Src-homology 2
LH	luteinizing hormone	SMAD	intracellular protein associated with streptokinases
MAO	monoamine oxidase		
MAP	mean arterial pressure	SOC	store-operated channels
MAPK(K)	mitogen-activated protein kinase (kinase)	SP	Substance P
		SR	sarcoplasmic reticulum
MEPP	miniature end plate potentials	Src	a non-receptor tyrosine kinase
MHC I, II	major histocompatibility complex I or II	SST	somatostatin
MIH	melanotrophin-inhibiting hormone	STAT	signal transduction and activation of transcription (protein)
MLC	myosin light chain		
MLCK	myosin light chain kinase	STIM	stromal interaction molecule
MLCP	myosin light chain phosphatase	STPD	standard temperature and pressure, dry gas
mRNA	messenger RNA		
MSH	melanotrophin-stimulating hormone	SV	stroke volume
Na⁺ pump	Na ⁺ -K ⁺ ATPase	SWVP	saturated water vapour pressure
NAD⁺ or (NADH)	nicotinic adenine dinucleotide (oxidized and reduced forms)	T₁ or 2	mono- or di-iodotyrosine
		T₃	tri-iodothyronine
NCX	Na ⁺ -Ca ²⁺ exchanger	T₄	thyroxine
NDI	nephrogenic diabetes insipidus	T_C	Core temperature
NGF	nerve growth factor	TF	tissue factor
NK	natural killer (cells)	TGFβ	transforming growth factor β
NMJ	neuromuscular junction	TH	thyroid hormone
NO	nitric oxide	TLC	total lung capacity
NOS	nitric oxide synthase	T_m	tubular transport maximum (kidney)
P2Y or P2X	purinergic receptor type 2Y or 2X	TNF	tumour necrosis factor
PAH	<i>para</i> -aminohippuric acid	TNZ	thermoneutral zone
PAMP	pathogen-associated molecular pattern	tPA	tissue plasminogen activator
		TPR	total peripheral resistance
P_B	barometric pressure	TRa	thyroid hormone receptor
PDGF	platelet-derived growth factor	TRE	thyroid response element
PEFR	peak expiratory flow rate	tRNA	transfer RNA
PGE₂	prostaglandin E ₂	TSH	thyroid-stimulating hormone
PGI₂	prostacyclin (prostaglandin I ₂)	TUC	time of useful consciousness
PI-3 kinase	phosphatidylinositol-3 kinase	TV	tidal volume
pK	negative log of dissociation constant (buffers)	TXA₂	thromboxane A ₂
PKA	protein kinase A	UCP-1, 2 or 3	uncoupling protein-1, 2 or 3
PKC	protein kinase C	V_A/Q mismatch	ventilation-perfusion mismatch (lungs)
PKG	protein kinase G	VC	vital capacity
PLA₂	phospholipase A2	VIP	vasoactive intestinal polypeptide
		vWF	von Willebrand factor

About the companion website



This book is accompanied by a companion website:



www.ataglanceseries.com/physiology

The website features:

- Interactive multiple choice questions
- Revision notes
- Interactive self-test flashcards





Introduction

Part 1

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1

Homeostasis and the physiology of proteins

Figure 1.1 Elements of a negative feedback system

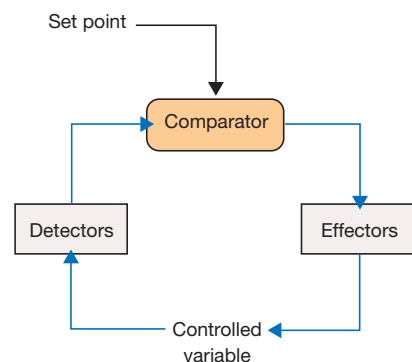


Figure 1.2 Operation of a negative feedback system

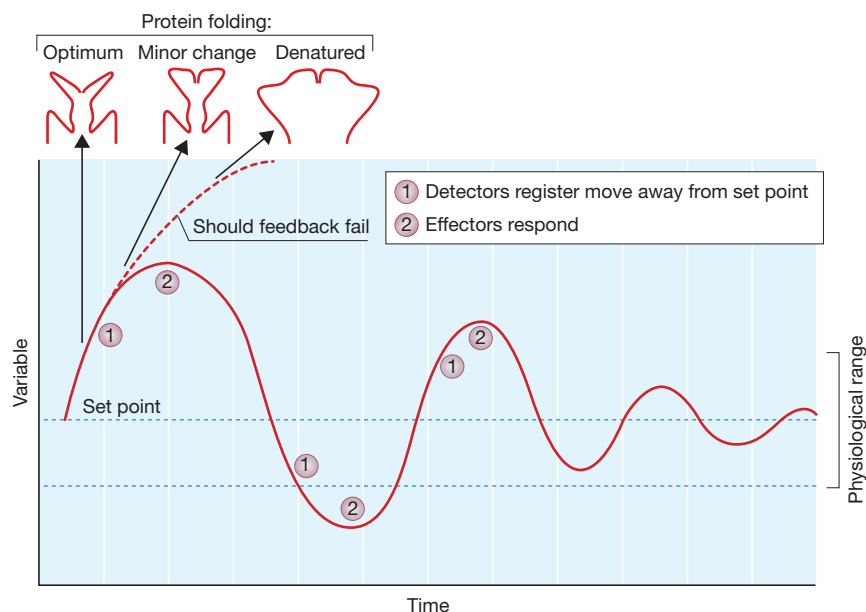


Figure 1.3 Primary protein structure (hypothetical)

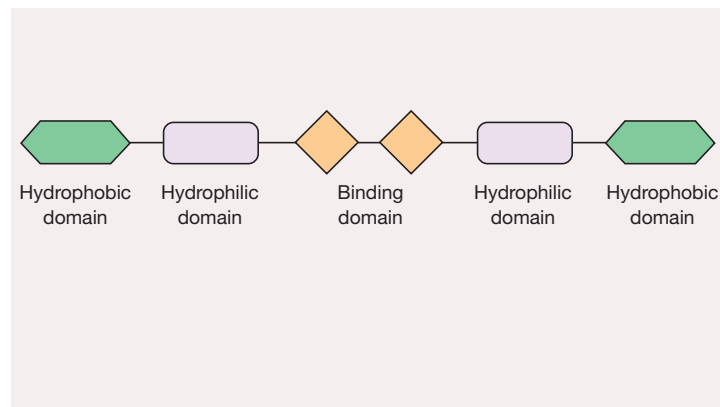
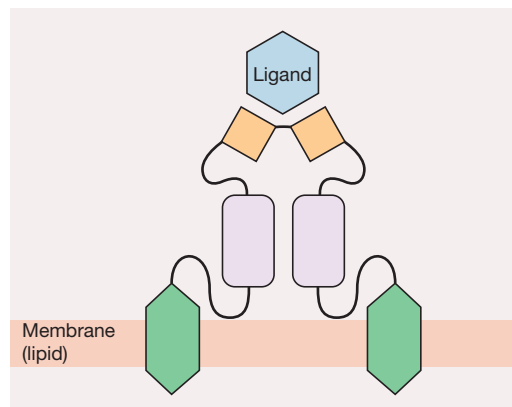


Figure 1.4 Folded (tertiary) structure



Claude Bernard (1813–1878) first described ‘le milieu intérieur’ and observed that the internal environment of the body remained remarkably constant (or in equilibrium) despite the ever changing external environment. The term **homeostasis** was not used until 1929 when **Walter Cannon** first used it to describe this ability of physiological systems to maintain conditions within the body in a relatively constant state of equilibrium. It is arguably the most important concept in physiology.

Homeostasis is Greek for ‘staying the same’. However, this so-called **equilibrium** is not an unchanging state but is a dynamic state of equilibrium causing a **dynamic constancy** of the internal environment. This **dynamic constancy** arises from the variable responses caused by changes in the external environment. Homeostasis maintains most physiological systems

and examples are seen throughout this book. The way in which the body maintains the H^+ ion concentration of body fluids within narrow limits, the control of blood glucose by the release of insulin, and the control of body temperature, heart rate and blood pressure are all examples of homeostasis. The human body has literally thousands of control systems. The most intricate are genetic control systems that operate in all cells to control intracellular function as well as all extracellular functions. Many others operate within organs to control their function; others operate throughout the body to control interaction between organs. As long as conditions are maintained within the normal physiological range within the internal environment, the cells of the body continue to live and function properly. Each cell benefits from homeostasis and in turn, each cell contributes its share towards the maintenance of homeostasis. This reciprocal

interplay provides continuity of life until one or more functional systems lose their ability to contribute their share. Moderate dysfunction of homeostasis leads to sickness and disease, and extreme dysfunction of homeostasis leads to death.

Negative feedback control

Most physiological control mechanisms have a common basic structure. The factor that is being controlled is called the **variable**. Homeostatic mechanisms provide the tight regulation of *all* physiological variables and the most common type of regulation is by **negative feedback**. A negative feedback system (Figure 1.1) comprises: **detectors** (often neural **receptor cells**) to measure the variable in question; a **comparator** (usually a neural assembly in the central nervous system) to receive input from the detectors and compare the size of the signal against the desired level of the variable (the **set point**); and **effectors** (muscular and/or glandular tissue) that are activated by the comparator to restore the variable to its set point. The term 'negative feedback' comes from the fact that the effectors always act to move the variable in the opposite direction to the change that was originally detected. Thus, when the partial pressure of CO₂ in blood increases above 5.3 kPa (40 mmHg), brain stem mechanisms increase the rate of ventilation to clear the excess gas, and *vice versa* when CO₂ levels fall below 5.3 kPa (Chapter 32). The term 'set point' implies that there is a single optimum value for each physiological variable; however, there is some tolerance in all physiological systems and the set point is actually a narrow *range* of values within which physiological processes will work normally (Figure 1.2). Not only is the set point not a point, but it can be reset in some systems according to physiological requirements. For instance, at high altitude, the low partial pressure of O₂ in inspired air causes the ventilation rate to increase. Initially, this effect is limited due to the loss of CO₂, but, after 2–3 days, the brain stem lowers the set point for CO₂ and allows ventilation to increase further, a process known as **acclimatization** (Chapter 14).

A common operational feature of all negative feedback systems is that they induce oscillations in the variable that they control (Figure 1.2). The reason for this is that it takes time for a system to detect and respond to a change in a variable. This delay means that feedback control always causes the variable to overshoot the set point slightly, activating the opposite restorative mechanism to induce a smaller overshoot in that direction, until the oscillations fall within the range of values that are optimal for physiological function. Normally, such oscillations have little visible effect. However, if unusually long delays are introduced into a system, the oscillations can become extreme. Patients with congestive heart failure sometimes show a condition known as **Cheyne–Stokes' breathing**, in which the patient undergoes periods of deep breathing interspersed with periods of no breathing at all (**apnoea**). This is partly due to the slow flow of blood from the lungs to the brain, which causes a large delay in the detection of blood levels of CO₂.

Some physiological responses use **positive feedback**, causing rapid amplification. Examples include initiation of an action potential, where sodium entry causes depolarization which further increases sodium entry and thus more depolarization

(Chapter 5), and certain hormonal changes, particularly in reproduction (Chapter 53). Positive feedback is inherently unstable, and requires some mechanism to break the feedback loop and stop the process (an off switch), such as time-dependent inactivation of sodium channels in the first example and the birth of the child in the second.

Protein form and function are protected by homeostatic mechanisms

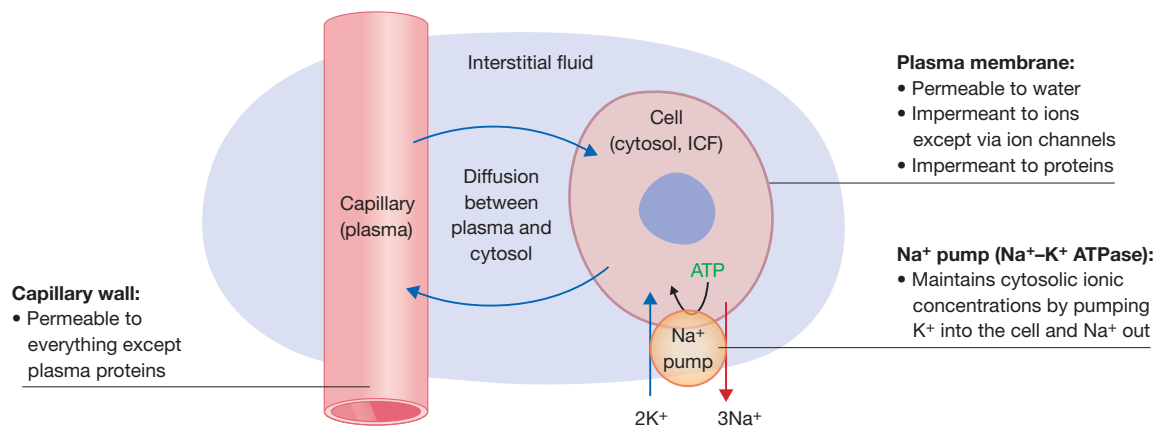
The homeostatic mechanisms that are described in detail throughout this book have evolved to protect the integrity of the protein products of gene translation. Normal functioning of proteins is essential for life, and usually requires binding to other molecules, including other proteins. The specificity of this binding is determined by the three-dimensional shape of the protein. The **primary structure** of a protein is determined by the sequence of amino acids (Figure 1.3). Genetic mutations that alter this sequence can have profound effects on the functionality of the final molecule. Such gene **polymorphisms** are the basis of many genetically based disorders. The final shape of the molecule (the **tertiary structure**), however, results from a process of **folding** of the amino acid chain (Figure 1.4). Folding is a complex process by which a protein achieves its lowest energy conformation. It is determined by electrochemical interactions between amino acid side-chains (e.g. hydrogen bonds, van der Waals' forces), and is so vital that it is overseen by **molecular chaperones**, such as the **heat shock proteins**, which provide a quiet space within which the protein acquires its final shape. In healthy tissue, cells can detect and destroy misfolded proteins, the accumulation of which damages cells and is responsible for various pathological conditions, including **Alzheimer's disease** and **Creutzfeldt–Jakob disease**. Folding ensures that the functional sequences of amino acids (**domains**) that form, e.g. binding sites for other molecules or hydrophobic segments for insertion into a membrane, are properly orientated to allow the protein to serve its function.

The relatively weak nature of the forces that cause folding renders them sensitive to changes in the environment surrounding the protein. Thus, alterations in acidity, osmotic potential, concentrations of specific molecules/ions, temperature or even hydrostatic pressure can modify the tertiary shape of a protein and change its interactions with other molecules. These modifications are usually reversible and are exploited by some proteins to detect alterations in the internal or external environments. For instance, nerve cells that respond to changes in CO₂ (chemoreceptors; Chapter 32) possess **ion channel** proteins (Chapter 4) that open or close to generate electrical signals (Chapter 5) when the acidity of the medium surrounding the receptor (CO₂ forms an acid in solution) alters by more than a certain amount. However, there are limits to the degree of fluctuation in the internal environment that can be tolerated by proteins before their shape alters so much that they become non-functional or irreversibly damaged, a process known as **denaturation** (this is what happens to egg-white proteins in cooking). Homeostatic systems prevent such conditions from arising within the body, and thus preserve protein functionality.

2

Body water compartments and physiological fluids

Figure 2.1 Physiological fluid compartments



Constituents of physiological fluids (approximate values, intracellular varies between tissues)				
	Plasma	Interstitial	Intracellular	Unit
Water: % total body water (volume in a 70 kg person)	13% (3.5)	22% (9.5)	65% (27)	% L
Osmolality	290	290	290	mosmol/kg H ₂ O
Cations: Na ⁺	140	140	10	mmol/L
K ⁺	4	4	140	mmol/L
Ca ²⁺ (free)	1	1	0.0001	mmol/L
Anions: Cl ⁻	108	129	3–30	mmol/L
HCO ₃ ⁻	26	26	9	mmol/L
Proteins ⁻	10	1	50	mmol/L
Other anions (mainly PO ₄ ³⁻ , SO ₄ ³⁻)	3	0	60–88	mmol/L

Notes: Ca²⁺ (and Mg²⁺) tend to bind to plasma proteins, and their free concentrations are about 50% of the total. Ionic concentrations are sometimes given in mEq/L to reflect the amount of charge, where an equivalent (Eq) is 1 mole of charge. So 1 Eq of a monovalent ion such as Na⁺ = 1 mole, but 1 Eq of Ca²⁺ = 0.5 mole

Figure 2.2 Effects of ingesting fluids of differing osmotic potential

Hypertonic fluid

Plasma volume expands
Plasma becomes concentrated
Free movement of water and ions from plasma leads to expansion of interstitial fluid

Water movement

Plasma



ISF



ICF

Interstitial fluid becomes concentrated
Osmotic potential draws water out of cells

Cells lose water and shrink
Intracellular fluid concentrated

Isotonic fluid

Plasma volume expands
Free movement of water and ions from plasma leads to expansion of interstitial fluid

Water movement

Plasma



ISF



ICF

Fluid is isotonic so no osmotic potential generated between ISF and cells

Intracellular fluid unaffected

Hypotonic fluid

Plasma diluted
Oncotic pressure reduced
Water moves into interstitial fluid

Water movement

Plasma



ISF



ICF

Interstitial fluid diluted
Osmotic potential generated between ISF and cells
Water moves into cells

Intracellular fluid diluted
Cells swell