

Biochemistry, remembered.

NEET-PG + INI-CET

Understand it once. Retrieve it again.

- + 120 topic sheets across the subject
- + 24 original clinical reasoning exercises
- + Handwriting-style notes + annotated pathway sketches
- + Mechanisms, exceptions and close-book recall

WHY?
then WHAT?

This book is for the details that keep slipping away:

enzyme -> substrate -> block -> clinical clue

Use the full book for revision across several sittings.
On the final day, follow the priority route on page 2
and revisit only your weak topics.

Newly written synthesis based on selected topics from
your supplied notes, with original diagrams and cases.
No claim of guaranteed exam questions.

How to use the last day

Pass 1: the backbone

Read your weakest pathways first: glycolysis/PDH/TCA, glycogen, PPP, lipid oxidation, urea and haem. Say the compartment, enzyme and consequence aloud.

Pass 2: volatile contrasts

Compare B12 versus folate; OTC versus UMPS; Pompe versus McArdle; synthesis versus oxidation; competitive versus uncompetitive inhibition; DNA-repair systems.

Pass 3: clinical retrieval

Attempt the cases on pages 125-148 without looking at the explanation. For each wrong answer, return to the matching topic instead of rereading everything.

Pass 4: final sweep

Cofactors, apoproteins, enzyme defects, transporter names, molecular methods and lab formulas. Use the contents pages to jump directly to the item.

Mark each topic: ? unsure ~ partly recalled + secure

Three questions for every metabolic block:

1. What accumulates?
2. What becomes deficient?
3. Why does that produce the clinical finding?

Find the topic / 1

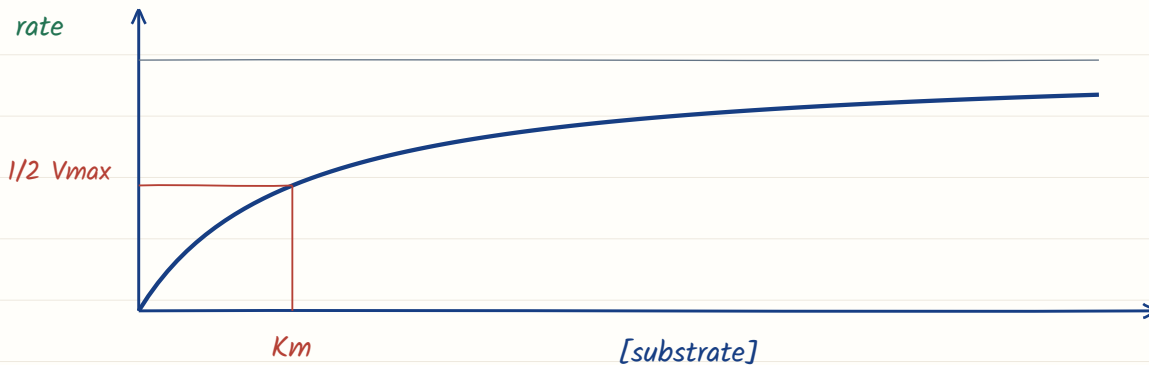
001 Water, pH & buffers	5	031 Liver glycogen disorders	35
002 Amino acids: classify by chemistry	6	032 Muscle & lysosomal glycogen disease	36
003 Protein structure: four levels	7	033 PPP: NADPH and ribose	37
004 Collagen: build, modify, cross-link	8	034 Fructose: benign or dangerous?	38
005 Elastin & connective-tissue clues	9	035 Galactose: milk and the lens	39
006 Haemoglobin: release oxygen	10	036 Fed, fasting and starvation	40
007 Haemoglobin variants & dyshemoglobins	11	037 Lipid classes: identify the backbone	41
008 Membranes & transport	12	038 Fat digestion and transport	42
009 Enzyme classes: recognise the verb	13	039 Beta-oxidation: shorten by two	43
010 Km & Vmax: the meaning	14	040 Odd chains and peroxisomes	44
011 Reversible inhibitors: the pattern	15	041 Fatty-acid synthesis	45
012 Allosteric & feedback control	16	042 Ketones: make, export, use	46
013 Isoenzymes & diagnostic enzymes	17	043 Cholesterol synthesis	47
014 Where does the pathway live?	18	044 Bile acids and enterohepatic cycling	48
015 Free energy & coupled reactions	19	045 Lipoprotein traffic map	49
016 ETC: follow the electrons	20	046 Apoproteins: five anchors	50
017 ETC poisons versus uncouplers	21	047 Dyslipidaemia patterns	51
018 ROS & antioxidant defence	22	048 Sphingolipids & surfactant	52
019 Sugars & bonds	23	049 Lysosomal lipid storage diseases	53
020 Digestion & absorption of sugars	24	050 Eicosanoids: local signals	54
021 Glycolysis: the complete skeleton	25	051 Essential fatty acids	55
022 Hexokinase versus glucokinase	26	052 Ethanol and the redox shift	56
023 PFK-1: the committed switch	27	053 Collecting amino nitrogen	57
024 Pyruvate: four destinations	28	054 Urea cycle: map every step	58
025 PDH & its five helpers	29	055 Hyperammonaemia: locate the block	59
026 TCA cycle: count and connect	30	056 Phenylalanine & BH4	60
027 Gluconeogenesis: bypass the locks	31	057 Tyrosine: several different blocks	61
028 Cori & glucose-alanine cycles	32	058 Branched-chain amino acids	62
029 Glycogen synthesis	33	059 Methionine, SAM & homocysteine	63
030 Glycogen breakdown	34	060 Tryptophan: three useful products	64

Find the topic / 2

061 Neurotransmitter precursor map	65	091 Protein-synthesis inhibitors	95
062 Creatine, creatinine & muscle energy	66	092 Gene expression and epigenetics	96
063 Glucogenic versus ketogenic carbon	67	093 Mutations: predict the consequence	97
064 Amino-acid transport disorders	68	094 Inheritance: read the pedigree	98
065 Porphyrin chemistry & haem roles	69	095 Mitochondrial DNA & heteroplasmy	99
066 Haem synthesis: two mitochondrial ends	70	096 PCR, RT-PCR and qPCR	100
067 Porphyrins: attack or photosensitivity?	71	097 DNA sequencing methods	101
068 Bilirubin: production to excretion	72	098 Blots, gels and separation	102
069 Jaundice: biochemical patterns	73	099 ELISA and assay logic	103
070 Iron: absorb, carry, store	74	100 Recombinant DNA, FISH & CRISPR	104
071 Copper: Wilson versus Menkes	75	101 Vitamin A: sight and differentiation	105
072 GAGs & lysosomal targeting	76	102 Vitamin D: activation and feedback	106
073 Nucleotides: name the parts	77	103 Vitamin E: protect membranes	107
074 Purine synthesis: build on ribose	78	104 Vitamin K: activate binding sites	108
075 Purine salvage & Lesch-Nyhan	79	105 B1: energy failure clues	109
076 Purine breakdown & urate	80	106 B2 and B3: redox vitamins	110
077 ADA deficiency & immune cells	81	107 B5 and B7: carry versus carboxylate	111
078 Pyrimidines: build ring first	82	108 B6: nitrogen and neurotransmitters	112
079 Orotic aciduria: the key split	83	109 Folate: DNA synthesis clues	113
080 Making DNA building blocks	84	110 B12: two reactions, two clues	114
081 Antimetabolites: match the target	85	111 Vitamin C: connective-tissue clues	115
082 One-carbon metabolism	86	112 Calcium, phosphate & trace elements	116
083 DNA organisation & chromatin	87	113 Nutrition: energy and nitrogen balance	117
084 DNA replication: direction rules	88	114 Malnutrition and refeeding	118
085 Polymerases and telomeres	89	115 Hormone signalling: receptor families	119
086 DNA repair: match the lesion	90	116 Diabetes: the biochemical pattern	120
087 Transcription: DNA to RNA	91	117 Acid-base calculations	121
088 RNA processing and splicing	92	118 Liver and kidney test interpretation	122
089 Genetic code and tRNA	93	119 Pre-analytical errors & lab quality	123
090 Translation: the ribosome journey	94	120 Calculated results: use the assumptions	124

10

K_m & V_{max}: the meaning



Must remember

- Michaelis-Menten: $v = V_{max}[S]/(K_m + [S])$. At $[S] = K_m$, v is half V_{max} .
- At low substrate, rate is approximately proportional to substrate. At saturation, adding more substrate gives little extra rate.
- Doubling enzyme concentration doubles V_{max} under otherwise identical conditions; K_m remains unchanged.

Why / clinical clue

K_m is an apparent kinetic parameter, not universally the binding dissociation constant. For simple exam comparisons under the same mechanism, a lower K_m often suggests higher apparent substrate affinity. Allosteric enzymes may not obey the simple hyperbolic model.

Do not mix up:

V_{max} is a rate, not an amount of substrate. Units must remain consistent when comparing values.

Close the book:

At $[S] = 3K_m$, what fraction of V_{max} ?

Check: $3/(1+3) = 3/4$, or 75%.

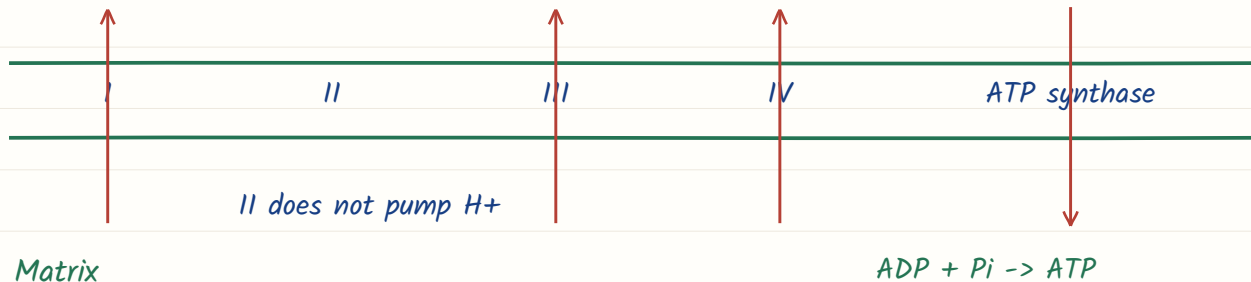
Margin memory

Quick fractions: $[S] = K_m \rightarrow 50\%$; $3K_m \rightarrow 75\%$; $9K_m \rightarrow 90\%$ of V_{max} .

16

ETC: follow the electrons

Intermembrane space: H^+ accumulates



Must remember

- Complex I accepts electrons from NADH; complex II includes succinate dehydrogenase. Both feed coenzyme Q.
- Complexes I, III and IV pump protons; complex II does not. Oxygen is reduced to water at complex IV.
- ATP synthase uses proton return to the matrix. Modern approximate P/O values are 2.5 ATP per NADH and 1.5 per $FADH_2$ -linked input.

Why / clinical clue

Electron transfer builds an electrochemical gradient. ATP synthesis depends on an intact inner membrane and coupling, not merely on electrons moving. Cytosolic NADH needs a shuttle because NADH itself does not freely cross the inner membrane.

Do not mix up:

Use the ATP convention stated in the question. Older totals of 3 and 2 per reducing equivalent give different answers.

Close the book:

Why does FAD-linked entry yield less ATP?

Check: Entry downstream of complex I bypasses one proton-pumping site.

Margin memory

ATP shuttles: malate-aspartate preserves NADH-equivalent entry; glycerol-3-P shuttle enters at the FAD-linked level.

25

PDH & its five helpers



Must remember

- PDH needs TPP (B1), lipoate, CoA (B5), FAD (B2), and NAD⁺ (B3).
- PDH kinase phosphorylates and inactivates the complex; PDH phosphatase activates it by dephosphorylation.
- Abundant NADH and acetyl-CoA favour inhibition. Calcium supports activation in contracting muscle.

Why / clinical clue

When pyruvate cannot efficiently become acetyl-CoA, more can be converted to lactate. PDH deficiency and severe thiamine deficiency therefore connect neurological energy failure with lactic acidosis. Arsenite can interfere with lipoate-dependent enzyme complexes.

Do not mix up:

The PDH reaction is irreversible; it is not a gluconeogenic bypass. The alpha-ketoglutarate dehydrogenase complex shares the same five cofactor types.

Close the book:

Which vitamin connects PDH, alpha-KGDH and transketolase?

Check: Thiamine, through TPP.

Margin memory

CoA carries acyl groups; lipoate shuttles reaction intermediates; NAD⁺ is the final soluble electron acceptor in the complex.

54

Urea cycle: map every step



Must remember

- Mitochondria: CPS-I forms carbamoyl phosphate; OTC adds its group to ornithine, forming citrulline.
- Cytosol: argininosuccinate synthetase adds aspartate; lyase releases fumarate; arginase generates urea and regenerates ornithine.
- Urea has nitrogen from free ammonia and aspartate; three ATP molecules supply four high-energy phosphate bonds.

Why / clinical clue

N-acetylglutamate activates CPS-I. Fumarate links nitrogen disposal with central carbon metabolism. The liver is the major site of complete ureagenesis; products and intermediates must cross the mitochondrial boundary.

Do not mix up:

CPS-I uses ammonia in mitochondria; CPS-II uses glutamine nitrogen for cytosolic pyrimidine synthesis.

Close the book:

Which step releases fumarate?

Check: Argininosuccinate lyase.

Margin memory

Nitrogen donors: ammonia + aspartate. Carbon donor: bicarbonate/CO₂. Energy: 3 ATP molecules, 4 high-energy bonds.

109

Folate: DNA synthesis clues

Folate $\longrightarrow$ THF derivatives $\longrightarrow$ Purines + dTMP

Must remember

- Folate supplies one-carbon units for nucleotide synthesis. Deficiency causes impaired DNA synthesis and can produce megaloblastic anaemia.
- Homocysteine rises; methylmalonic acid is usually normal in isolated folate deficiency.
- Pregnancy increases folate needs; adequate periconceptional folate reduces neural-tube-defect risk.

Why / clinical clue

Rapidly dividing marrow cells reveal nucleotide insufficiency through nuclear-cytoplasmic maturation mismatch. Macrocytosis has many causes, so the blood film and clinical setting remain important. Folate status and B12 status should not be interpreted as interchangeable.

Do not mix up:

Folate can improve the anaemia of B12 deficiency without correcting B12-related neurological injury.

Close the book:

Which metabolite helps distinguish isolated folate deficiency from B12 deficiency?

Check: MMA is typically normal in folate deficiency and raised in B12 deficiency, with renal-function caveats.

Margin memory

Folate deficiency: DNA problem + high homocysteine. B12 adds a second reaction and can raise MMA.

110

B12: two reactions, two clues

Methylmalonyl-CoA



Succinyl-CoA

Homocysteine



Methionine

Must remember

- B12 supports methylmalonyl-CoA mutase and methionine synthase.
- Food-bound B12 is released, binds haptocorrin, then intrinsic factor after pancreatic processing; the intrinsic-factor complex is absorbed in distal ileum.
- Deficiency can raise MMA and homocysteine and cause neurological findings with or without anaemia.

Why / clinical clue

Pernicious anaemia impairs intrinsic-factor-mediated absorption. Ileal disease can also reduce uptake. MMA is useful but can rise with renal impairment; homocysteine is less specific because folate and other factors also affect it.

Do not mix up:

Normal haemoglobin does not exclude neurological B12 deficiency. Serum B12 and functional markers require contextual interpretation.

Close the book:

Which gastric cell makes intrinsic factor?

Check: The parietal cell.

Margin memory

Methylcobalamin: methionine synthase. Adenosylcobalamin: methylmalonyl-CoA mutase.