Biochemistry

Unit 6 · Lesson 3 · Nucleotides, inborn errors and hormones

The notes for Lesson 3 of Unit 6: chapters 6.7 to 6.9 of the unit book, with their figures, worked examples, exam traps and quizzes, in one file. The full unit book, with every chapter, the exam question bank and the glossary, is linked at the end.

Lesson 3 of 3 | 3 chapters | Chapters 6.7 to 6.9

About these notes

These are the notes for **Lesson 3 · Nucleotides, inborn errors and hormones** of the University free n easy video lessons on Biochemistry Unit 6, Lipid, Amino Acid and Nucleotide Metabolism. They are chapters 6.7 to 6.9 of the unit book, unchanged, so a page here is the same page in the full book.

6.7 Purine and pyrimidine biosynthesis and degradation

Every dividing cell needs nucleotides for DNA and RNA, and every cell needs ATP, GTP, NAD^+ and CoA, all of which contain a nucleotide. Cells make nucleotides in two ways: **de novo** (from small molecules: amino acids, CO_2, one-carbon units and ribose 5-phosphate) and by **salvage** (reusing free bases from broken-down nucleic acids). Purines and pyrimidines are built in opposite orders: a **purine** ring is assembled on the ribose, while a **pyrimidine** ring is made first and then attached to ribose. Degradation also differs: purines end as **uric acid**, which can crystallise (gout), while pyrimidines end as soluble products.

You need: base, nucleoside and nucleotide structures from **Unit 4, Purine and pyrimidine bases** and **Nucleosides and nucleotides**; the pentose phosphate pathway from **Unit 5**.

PRPP: the activated ribose

Both pathways and salvage use **5-phosphoribosyl-1-pyrophosphate (PRPP)**, made by PRPP synthetase: ribose 5-phosphate + ATP → PRPP + AMP. Ribose 5-phosphate comes from the pentose phosphate pathway.

Purine synthesis de novo

1. **Committed step.** Glutamine-PRPP amidotransferase replaces the pyrophosphate of PRPP with the amide nitrogen of glutamine, giving 5-phosphoribosylamine. The product's nitrogen becomes N-9 of the purine. This enzyme is inhibited by the end products AMP, GMP and IMP.
2. **Ring assembly on ribose.** In ten steps in all, glycine, a formyl group from N^{10}-formyl-tetrahydrofolate (THF, from folic acid), another glutamine nitrogen, CO_2 and aspartate are added, closing first the five-membered and then the six-membered ring.
3. The first complete purine nucleotide is **inosine monophosphate (IMP)**, whose base is hypoxanthine.
4. **IMP → AMP:** aspartate adds an amino group at C-6 (adenylosuccinate synthetase, uses **GTP**), then fumarate is removed (adenylosuccinate lyase).
5. **IMP → GMP:** IMP dehydrogenase oxidises IMP to XMP (NAD^+); GMP synthetase adds an amino group from glutamine (uses **ATP**).

The cross-use of GTP for AMP and ATP for GMP keeps the two in balance: plenty of one drives synthesis of the other.

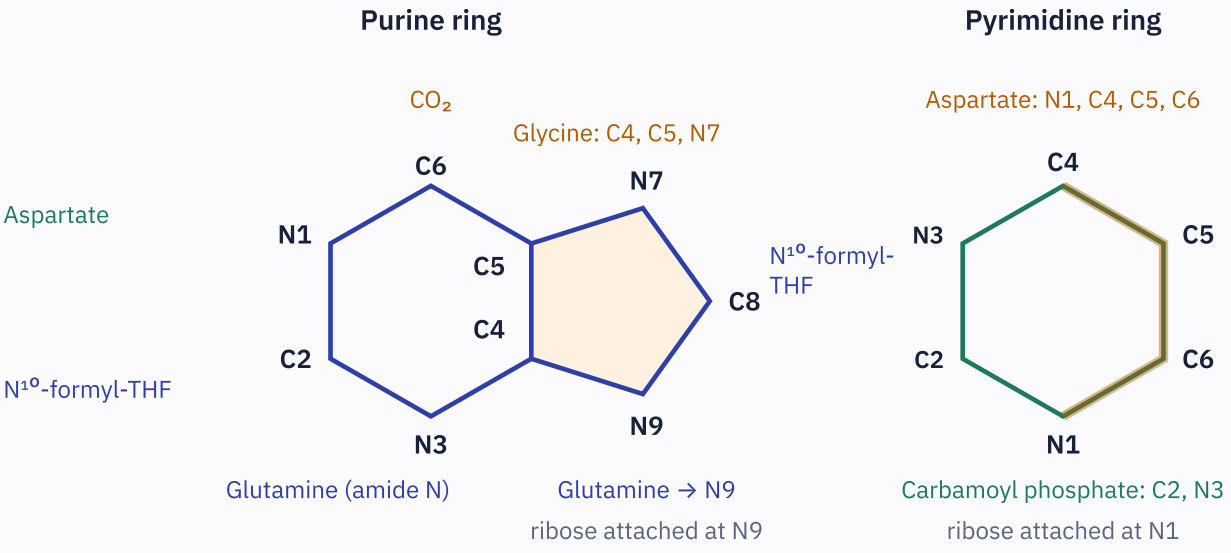


Figure 6.7.1 Where each ring atom comes from. Purine (left): glycine supplies the shaded C4, C5 and N7; two glutamines give N3 and N9; formyl-THF gives C2 and C8; aspartate gives N1; CO_2 gives C6. Pyrimidine (right): carbamoyl phosphate gives C2 and N3; aspartate gives the highlighted N1, C4, C5 and C6.

Pyrimidine synthesis de novo

1. **Carbamoyl phosphate synthetase II (CPS-II)**, cytosol: glutamine + HCO_3^- + 2 ATP → carbamoyl phosphate. Unlike CPS-I of the urea cycle, it uses glutamine and does not need NAG. In animals this is the regulated step (UTP inhibits, PRPP activates).
2. **Aspartate transcarbamoylase (ATCase)**: carbamoyl phosphate + aspartate → carbamoyl aspartate. In *E. coli* this is the regulated step and the textbook example of an allosteric enzyme: CTP inhibits, ATP activates.
3. **Dihydroorotase** closes the ring to dihydroorotate.
4. **Dihydroorotate dehydrogenase**, on the outer face of the inner mitochondrial membrane, oxidises it to **orotate**.
5. **Orotate phosphoribosyltransferase** attaches ribose phosphate from PRPP, giving OMP; **OMP decarboxylase** makes **UMP**.
6. UMP → UDP → UTP by kinases; **CTP synthetase** adds an amino group from glutamine to UTP, giving CTP.

In mammals the first three activities sit on one protein (called CAD) and the last two on another (UMP synthase), so intermediates pass straight from one active site to the next. A defect of UMP synthase causes **hereditary orotic aciduria** (Chapter 6.8).

Deoxyribonucleotides and thymidylate

Ribonucleotide reductase reduces the 2′-OH of ribonucleoside diphosphates (ADP, GDP, CDP, UDP) to give dNDPs, using electrons from NADPH through thioredoxin. **Thymidylate synthase** then methylates dUMP to dTMP, with N^5,N^{10}-methylene-THF as the one-carbon donor; this leaves dihydrofolate, which **dihydrofolate reductase (DHFR)** reduces back to THF. These enzymes are classic anticancer targets: **5-fluorouracil** inhibits thymidylate synthase, **methotrexate** inhibits DHFR and **hydroxyurea** inhibits ribonucleotide reductase.

Salvage and degradation

Salvage saves energy. Hypoxanthine-guanine phosphoribosyltransferase (**HGPRT**) makes IMP or GMP from the free base + PRPP; adenine phosphoribosyltransferase (APRT) does the same for adenine. Its deficiency causes Lesch–Nyhan syndrome (Chapter 6.8).

Purine degradation in humans:

1. AMP → IMP (AMP deaminase), or AMP → adenosine → inosine (**adenosine deaminase**, ADA). ADA deficiency kills developing lymphocytes and causes severe combined immunodeficiency (SCID).

2. Nucleosides lose their ribose to purine nucleoside phosphorylase, giving the free bases hypoxanthine and guanine.
3. Hypoxanthine → xanthine → **uric acid**, both steps by **xanthine oxidase**; guanine → xanthine by guanine deaminase.

Humans and other apes lack urate oxidase, so uric acid is the end product. It is poorly soluble; a high blood level (hyperuricaemia) can deposit sodium urate crystals in joints, which is **gout**. **Allopurinol**, a hypoxanthine analogue, inhibits xanthine oxidase and lowers uric acid.

Pyrimidine degradation opens the ring: cytosine and uracil give **β-alanine**, CO_2 and NH_4^+; thymine gives **β-aminoisobutyrate**. These are water-soluble, so pyrimidine breakdown causes no crystal disease.

WORKED EXAMPLE

Question (6 marks). A researcher grows liver cells with (a) [^{15}N]-glycine, (b) [^{14}C]-bicarbonate and (c) [^{15}N]-aspartate. Which ring atoms of newly made AMP and UMP will be labelled in each case?

1. (a) Glycine gives C4, C5 and N7 of the purine; only its nitrogen is ^{15}N, so **N7 of AMP** is labelled. Glycine is not used for pyrimidines: **UMP unlabelled**.
2. (b) CO_2 gives **C6 of the purine**. For UMP, the bicarbonate taken up by CPS-II becomes **C2**. The CO_2 released by OMP decarboxylase came from aspartate, not from bicarbonate, so no label is lost there.
3. (c) Aspartate gives **N1 of the purine ring** and, in AMP, also the **6-amino group** (from adenylosuccinate). In UMP, aspartate's nitrogen becomes **N1**.

EXAM TRAPS

- Purines: ring built on PRPP. Pyrimidines: ring first, then PRPP. A favourite "differentiate" question.
- CPS-II (pyrimidines, cytosol, glutamine) is not CPS-I (urea cycle, mitochondria, NH_4^+, NAG).
- The first purine formed is IMP, not AMP; the first pyrimidine nucleotide formed is OMP, then UMP.
- Uric acid is the end product of purines only. Pyrimidines give β-alanine and β-aminoisobutyrate.
- Ribonucleotide reductase acts on diphosphates (NDPs), not monophosphates.

CHECK YOURSELF

– REMEMBER **Name the committed step of purine synthesis and two of its inhibitors.** 2 marks

Glutamine-PRPP amidotransferase; inhibited by AMP, GMP and IMP.

– REMEMBER **Which enzyme does allopurinol inhibit?** 1 mark

Xanthine oxidase.

– UNDERSTAND **Why do methotrexate and 5-fluorouracil kill dividing cells faster than resting cells?** 4 marks

Dividing cells need large amounts of dTMP for DNA replication. 5-FU blocks thymidylate synthase directly; methotrexate blocks DHFR, so THF is not regenerated and thymidylate (and purine) synthesis stops. Resting cells make little DNA and are affected less.

– UNDERSTAND **How does the cell keep AMP and GMP in balance?** 3 marks

IMP → AMP uses GTP and IMP → GMP uses ATP; also AMP and GMP each inhibit the first enzyme of their own branch and, with IMP, the amidotransferase.

– APPLY **Which atom of UMP is labelled if cells are given [^{15}N]-amide glutamine?** 2 marks

N3, via carbamoyl phosphate made by CPS-II.

– ANALYZE **Compare the end products of purine and pyrimidine breakdown and explain why only one causes a crystal disease.** 4 marks

Purines are oxidised to uric acid, which humans cannot break down (no urate oxidase) and which is poorly soluble, so it can crystallise as sodium urate (gout, kidney stones). Pyrimidine rings are opened and reduced to β-amino acids, which are soluble and can be further metabolised.

BEYOND THE SYLLABUS

Nucleotide metabolism is a frontline of drug design: antivirals such as remdesivir and sofosbuvir are nucleotide analogues, mycophenolate (an IMP dehydrogenase inhibitor) suppresses lymphocytes after organ transplant, and ADA-SCID was among the first diseases treated by gene therapy. The newer urate-lowering drug febuxostat also inhibits xanthine oxidase. Search "nucleotide analogue antivirals mechanism". Not for the exam.

6.8 Inborn errors of metabolism

An **inborn error of metabolism** is an inherited disease in which one enzyme (or transporter) of a pathway is missing or faulty because of a gene mutation. The substrate before the block piles up, often spilling into abnormal side-pathways, and the product after the block runs short. The physician Archibald Garrod coined the term in 1908 from his study of alkaptonuria, an early clue to the "one gene, one enzyme" idea. Most are **autosomal recessive**. Each is rare, but together they are an important cause of illness in newborns. The other universities meet the same diseases inside the pathway chapters.

Reading any inborn error

Every disorder can be explained in four lines, and examiners expect all four: **the block** (which enzyme), **what accumulates** (substrate and side products, often seen in urine), **what is deficient** (product), and **the treatment** (usually restricting the substrate in the diet or supplying the product).

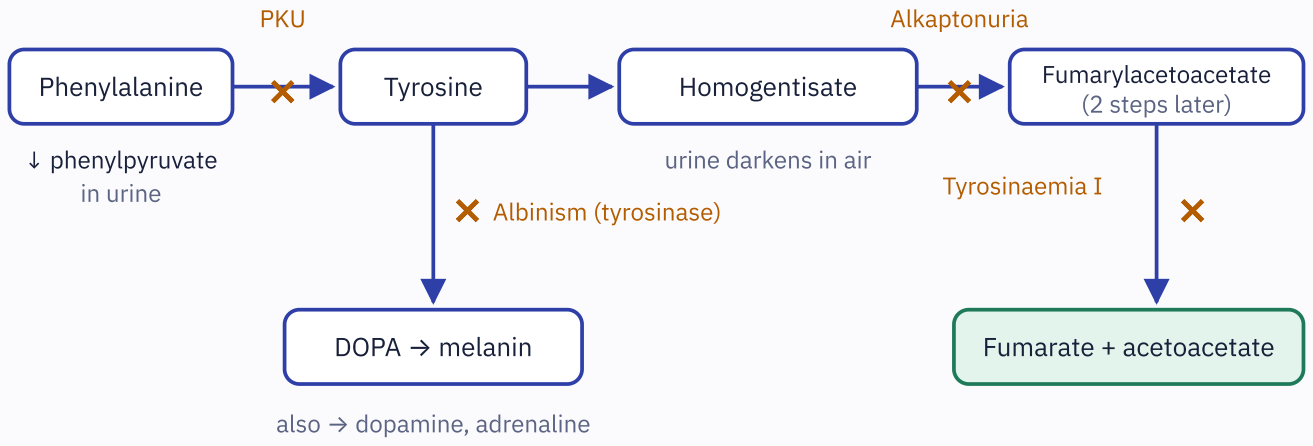


Figure 6.8.1 Four classic disorders on one pathway. Each ✕ is a missing enzyme. In PKU phenylalanine is diverted to phenylpyruvate; in alkaptonuria homogentisate builds up; in albinism melanin is not made. Tyrosine becomes an essential amino acid in PKU.

The classic disorders

DISORDER	ENZYME MISSING	ACCUMULATES OR IS LACKING	KEY POINT
Phenylketonuria (PKU)	Phenylalanine hydroxylase (or its cofactor tetrahydrobiopterin)	Phe, phenylpyruvate, phenyllactate, phenylacetate	Intellectual disability, mousy odour; low-Phe diet from birth

DISORDER	ENZYME MISSING	ACCUMULATES OR IS LACKING	KEY POINT
Alkaptonuria	Homogentisate oxidase	Homogentisic acid	Urine darkens on standing; dark connective tissue (ochronosis), arthritis
Albinism (oculocutaneous)	Tyrosinase	No melanin	Pale skin and hair, light sensitivity
Maple syrup urine disease	Branched-chain α-keto acid dehydrogenase	Leu, Ile, Val and their keto acids	Sweet-smelling urine; brain damage if untreated
Galactosaemia	Galactose 1-phosphate uridylyltransferase	Galactose 1-phosphate	Liver damage, cataract; remove milk sugar
Urea cycle defects (OTC most common)	A urea cycle enzyme (6.6)	NH_3; orotic acid in OTC deficiency	Hyperammonaemia; low-protein diet, nitrogen scavengers
Lesch–Nyhan syndrome	HGPRT (6.7), X-linked	Uric acid (salvage fails, PRPP rises)	Gout, self-injury, neurological signs
Hereditary orotic aciduria	UMP synthase (6.7)	Orotic acid; low pyrimidines	Anaemia, poor growth; treated with uridine
MCAD deficiency	Medium-chain acyl-CoA dehydrogenase (6.1)	Medium-chain acyl-carnitines	Low blood sugar without ketones in fasting; avoid fasting

WORKED EXAMPLE

Question (8 marks). A six-month-old infant has delayed development, fair skin and a musty smell in the urine. Plasma phenylalanine is very high and tyrosine is low. Identify the disorder and explain the biochemistry, diagnosis and treatment.

1. **Disorder:** phenylketonuria, a deficiency of phenylalanine hydroxylase (autosomal recessive).
2. **Block:** Phe + O_2 + tetrahydrobiopterin → Tyr cannot occur. Phe accumulates and is transaminated to **phenylpyruvate** (a "phenylketone", hence the name), then to phenyllactate and phenylacetate, which give the musty odour.
3. **Deficiency:** less tyrosine, so less melanin (fair skin) and possibly less dopamine and noradrenaline. High Phe also competes with other large neutral amino acids for transport into the brain; the damage is to the developing brain.
4. **Diagnosis:** newborn screening of a heel-prick blood spot, once by the Guthrie bacterial inhibition assay and now mostly by tandem mass spectrometry, confirmed by plasma Phe.
5. **Treatment:** a low-phenylalanine diet started in the first weeks of life, with tyrosine supplied (it is now essential), and avoidance of the sweetener aspartame, which contains phenylalanine.

EXAM TRAPS

- In PKU tyrosine becomes essential; do not write that tyrosine accumulates.
- Alkaptonuria is not "black urine at passing": the urine darkens on standing in air as homogentisate oxidises.
- Lesch–Nyhan and OTC deficiency are X-linked; most other inborn errors are autosomal recessive.

CHECK YOURSELF

– REMEMBER **Who coined "inborn errors of metabolism", and from which disease?** 1 mark

Archibald Garrod, from alkaptonuria.

– REMEMBER **Which enzyme is missing in maple syrup urine disease?** 1 mark

Branched-chain α-keto acid dehydrogenase complex.

– UNDERSTAND **Why do PKU patients often have fair skin?** 2 marks

Less tyrosine is made, so less melanin; high phenylalanine also inhibits tyrosinase.

– APPLY **A child has high blood ammonia and orotic acid in urine. Which enzyme is likely deficient?** 2 marks

Ornithine transcarbamoylase: carbamoyl phosphate builds up and enters pyrimidine synthesis.

– ANALYZE **Why does MCAD deficiency cause low blood glucose with low ketones during fasting?** 4 marks

Fatty acids cannot be fully β-oxidised, so the liver lacks acetyl-CoA for ketogenesis and lacks the ATP and NADH that drive gluconeogenesis. Both ketone bodies and glucose fall together.

BEYOND THE SYLLABUS

Expanded newborn screening by tandem mass spectrometry tests one blood spot for dozens of disorders, and some Indian states and hospitals now run such programmes. Enzyme replacement therapy treats lysosomal storage disorders such as Gaucher disease, and an enzyme (pegvaliase) that degrades phenylalanine is approved for adult PKU in some countries. Search "expanded newborn screening India". Not for the exam.

6.9 Overview of hormones and metabolic regulation

Each pathway in this unit and in Unit 5 has a pair: glycolysis and gluconeogenesis, fatty acid synthesis and β-oxidation, glycogen synthesis and breakdown. If both members of a pair ran at once, the cell would waste ATP in a **futile cycle**. Hormones prevent this by switching the whole body between two states: the **fed state** (store fuel) under **insulin**, and the **fasting or stress state** (release fuel) under **glucagon**, **adrenaline** (epinephrine) and **cortisol**.

You need: the regulated enzymes of each chapter in this unit, and **Unit 5, Glycolysis: steps, energetics and regulation**.

Four ways a cell controls a pathway

1. **Allosteric control** (seconds): a metabolite binds away from the active site. Examples: citrate activates acetyl-CoA carboxylase (6.2); malonyl-CoA inhibits CPT-I (6.1); N-

acetylglutamate activates CPS-I (6.6).

2. **Covalent modification** (seconds to minutes): mostly phosphorylation by kinases and removal by phosphatases, set by hormones. Phosphorylation inactivates acetyl-CoA carboxylase and HMG-CoA reductase but activates glycogen phosphorylase and hormone-sensitive lipase.
3. **Enzyme amount** (hours to days): hormones change gene transcription. A high-protein diet raises urea cycle enzymes (6.6); fasting raises mitochondrial HMG-CoA synthase (6.3); SREBP raises HMG-CoA reductase (6.4).
4. **Compartmentation**: opposing pathways sit in different places, such as fatty acid synthesis in the cytosol and β-oxidation in mitochondria.

How hormones reach the enzymes

Hormones are grouped by chemistry, which decides where their receptor is:

- **Peptide and protein hormones** (insulin, glucagon) and **catecholamines** (adrenaline) are water-soluble. They bind receptors on the cell surface and act through second messengers within minutes.
- **Steroid hormones** (cortisol) and **thyroid hormones** are lipid-soluble and enter the cell. They bind intracellular (nuclear) receptors that act as transcription factors, so they act over hours by changing enzyme amounts.

Glucagon (on liver) and **adrenaline** (on liver, muscle and adipose tissue, through β-adrenergic receptors) use the same cascade: receptor → G-protein (Gs) → adenylyl cyclase → **cAMP** → **protein kinase A (PKA)** → phosphorylation of target enzymes. Each enzyme in the cascade activates many copies of the next, so a tiny amount of hormone produces a large response; this is **amplification**.

Insulin binds a **receptor tyrosine kinase**. The receptor phosphorylates itself and insulin receptor substrate (IRS) proteins, which switch on the PI3-kinase–Akt pathway. The results include moving **GLUT4** glucose transporters to the membrane of muscle and fat cells, activating phosphatases that dephosphorylate enzymes, and lowering cAMP by activating a phosphodiesterase. So insulin largely reverses the phosphorylations made by PKA.

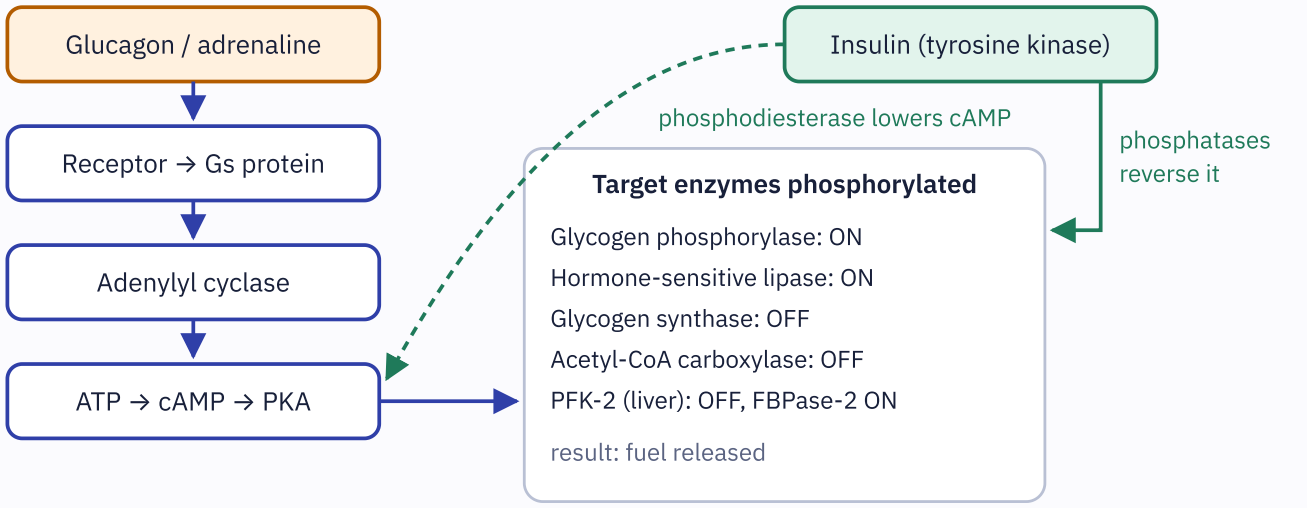


Figure 6.9.1 The cAMP cascade used by glucagon and adrenaline (left) and how insulin opposes it (green). Notice that one kinase, PKA, turns breakdown enzymes on and synthesis enzymes off at the same time, so the pairs never run together.

The hormones and what they do

HORMONE	SIGNAL FOR	MAIN METABOLIC EFFECTS
Insulin (β-cells of pancreas)	High blood glucose, fed state	Glucose uptake into muscle and fat; glycogen, fat and protein synthesis; inhibits gluconeogenesis, lipolysis and ketogenesis
Glucagon (α-cells)	Low blood glucose	Liver only: glycogen breakdown, gluconeogenesis, β-oxidation and ketogenesis; inhibits glycolysis and fat synthesis
Adrenaline (adrenal medulla)	Stress, exercise	Glycogen breakdown in liver and muscle; lipolysis in adipose tissue
Cortisol (adrenal cortex)	Long-term stress, prolonged fasting	Muscle protein breakdown; more gluconeogenic enzymes in liver
Thyroid hormones (T_3, T_4)	Long-term metabolic rate	Raise basal metabolic rate and oxygen use in most tissues

Muscle has no glucagon receptors, so glucagon cannot break down muscle glycogen; only adrenaline can, and muscle keeps that glucose for itself. Inside every cell, **AMP-activated protein kinase (AMPK)** acts as a fuel gauge: when AMP rises it switches off fat and cholesterol synthesis and switches on fat oxidation.

WORKED EXAMPLE

Question (8 marks). A student skips dinner and breakfast and writes an exam 16 hours after the last meal. Predict the hormone levels and the state (on or off) of each pathway in the liver, with the enzyme that sets it.

1. **Hormones:** insulin low, glucagon high (cortisol slightly up). Liver cAMP and PKA activity are high.
2. **Glycogen:** liver glycogen is largely used up by now; phosphorylase was on, synthase off.
3. **Gluconeogenesis: ON.** Low fructose 2,6-bisphosphate releases fructose 1,6-bisphosphatase and slows PFK-1 (Unit 5). Alanine from muscle (6.5) and glycerol from adipose tissue are substrates.
4. **Fatty acid synthesis: OFF.** ACC is phosphorylated, so malonyl-CoA falls (6.2).
5. **β-Oxidation: ON.** With little malonyl-CoA, CPT-I is free; hormone-sensitive lipase in adipose tissue supplies fatty acids (6.1).
6. **Ketogenesis: ON.** Oxaloacetate is used for gluconeogenesis, and HMG-CoA synthase is induced (6.3).
7. **Urea cycle:** active, as amino acids from muscle protein are used for gluconeogenesis (6.6).

EXAM TRAPS

- Glucagon does not act on muscle; adrenaline does.
- Phosphorylation is not always activation: it turns phosphorylase and hormone-sensitive lipase on but glycogen synthase and ACC off.
- Steroid and thyroid hormones act on nuclear receptors (gene expression), not through cAMP.
- Insulin's receptor is a tyrosine kinase, not a G-protein-coupled receptor.

CHECK YOURSELF

– REMEMBER **Name the second messenger of glucagon and the kinase it activates.** 2 marks

cAMP; protein kinase A.

– REMEMBER **List four levels of metabolic regulation.** 2 marks

Allosteric control, covalent modification, change in enzyme amount, compartmentation.

– UNDERSTAND **What is a futile cycle and how is it avoided?** 3 marks

Two opposing pathways running at once, with net hydrolysis of ATP and no useful product. Reciprocal regulation (one hormone signal turning one pathway on and its opposite off) prevents it.

– UNDERSTAND **Why does a lipid-soluble hormone act more slowly than glucagon?** 3 marks

It works by changing gene transcription through a nuclear receptor, so new enzyme protein must be made (hours). Glucagon acts by phosphorylating existing enzymes (minutes).

– APPLY **State the effect of insulin on ACC, CPT-I activity and HMG-CoA reductase.** 3 marks

ACC activated (dephosphorylated); CPT-I inhibited indirectly through higher malonyl-CoA; HMG-CoA reductase activated.

– ANALYZE **In untreated type 1 diabetes, blood glucose and ketone bodies are both high. Explain using hormone actions.** 4 marks

No insulin: muscle and fat cannot take up glucose (no GLUT4 movement), and glucagon is unopposed, so liver gluconeogenesis and glycogen breakdown continue. Unchecked lipolysis floods the liver with fatty acids, and with low malonyl-CoA and induced HMG-CoA synthase, ketogenesis runs fast.

BEYOND THE SYLLABUS

Newer diabetes and obesity drugs act on these signals: GLP-1 receptor agonists such as semaglutide raise insulin release and reduce appetite, and dual GLP-1/GIP agonists go further. Researchers now model whole-body regulation with genome-scale metabolic models and measure pathway activity directly with isotope-tracer "fluxomics". Search "hormonal regulation of fuel metabolism integration" or "metabolic flux analysis". Not for the exam.

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
PRPP	Activated ribose for purine, pyrimidine and salvage synthesis	6.7
Xanthine oxidase	Hypoxanthine → xanthine → uric acid; allopurinol target	6.7
Inborn error	Inherited single-enzyme defect: block, build-up, deficiency, treatment	6.8
cAMP cascade	Glucagon/adrenaline → Gs → adenylyl cyclase → cAMP → PKA	6.9

Glossary (terms used in this lesson)

Acetyl-CoA carboxylase Biotin enzyme that makes malonyl-CoA; committed step of fatty acid synthesis.

AMPK AMP-activated protein kinase; energy sensor that stops synthesis and starts fuel oxidation.

β-Oxidation Mitochondrial pathway that removes two-carbon acetyl-CoA units from fatty acyl-CoA.

Carbamoyl phosphate synthetase CPS-I (mitochondria, NH_4^+, urea cycle) or CPS-II (cytosol, glutamine, pyrimidines).

Futile cycle Two opposing pathways running together, wasting ATP.

HMG-CoA β-Hydroxy-β-methylglutaryl-CoA; intermediate of both ketogenesis and cholesterol synthesis.

Inborn error of metabolism Inherited deficiency of one enzyme or transporter of a pathway.

IMP Inosine monophosphate, the first purine nucleotide made de novo.

Ketone bodies Acetoacetate, β-hydroxybutyrate and acetone.

Malonyl-CoA Three-carbon donor of fatty acid synthesis and inhibitor of CPT-I.

N-acetylglutamate Obligate activator of carbamoyl phosphate synthetase I.

PRPP 5-Phosphoribosyl-1-pyrophosphate, the activated ribose of nucleotide synthesis.

Urea cycle Liver pathway that converts NH_4^+ and aspartate nitrogen into urea.

Uric acid	End product of purine breakdown in humans; its crystals cause gout.

The full unit book

Every chapter of Unit 6, the unit summary, the exam question bank with model answers and the full glossary: Biochemistry · Unit 6 · Lipid, Amino Acid and Nucleotide Metabolism (one file, works offline).

Sources and further reading

- D. L. Nelson and M. M. Cox, *Lehninger Principles of Biochemistry*, W. H. Freeman: chapters on fatty acid catabolism, amino acid oxidation and urea production, lipid biosynthesis, nucleotide biosynthesis and hormonal regulation (from memory; check chapter numbers in your edition).
- J. M. Berg, J. L. Tymoczko, G. J. Gatto and L. Stryer, *Biochemistry*, W. H. Freeman (from memory; check).
- D. Voet and J. G. Voet, *Biochemistry*, Wiley (from memory; check).
- U. Satyanarayana and U. Chakrapani, *Biochemistry*, Elsevier India; widely prescribed in Indian universities, uses the older P/O values of 3 and 2 in ATP calculations (from memory; check).
- V. W. Rodwell and others, *Harper's Illustrated Biochemistry*, McGraw-Hill; clinical correlations and inborn errors (from memory; check).
- StatPearls, Physiology, Urea Cycle, NCBI Bookshelf (opened; confirms N-acetylglutamate as the obligate activator of CPS-I and OTC deficiency as the most common, X-linked urea cycle defect).
- StatPearls, Biochemistry, Ketogenesis, NCBI Bookshelf (opened; confirms that the liver lacks thiophorase and that urine dipsticks mainly measure acetoacetate).

Syllabus: the University free n easy universal syllabus, built from top universities' syllabi.

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