Chapter 3 Workbook

What Goes Wrong in Type 2 Diabetes?

Chapter 3 takes the healthy system you met in Chapter 2 and shows, step by step, how it becomes overloaded: where surplus energy goes, why safe storage runs out, how cells defend themselves, and why the pancreas eventually falls behind. Every part of the plan in Part 2 is a response to this process. The better you understand it, the more sense the plan will make, and the harder it becomes for anyone to sell you a shortcut.

These pages ask more of you than rereading would, and that is deliberate. Pulling an idea out of your own head, getting it slightly wrong and then putting it right fixes it in memory far better than reading it a second time. The questions start easy and climb, and every answer explains its reasoning. If this is your first workbook, read the one-page guide before you start.

Contents

	Each set has a question page followed by its answer page	Pages
	How to use these pages	2
1	Energy in, fat stored Excess energy from any source ends up as fat · about 12 minutes	3–4
2	The buffer and the threshold The protective buffer phase · Personal fat threshold · about 12 minutes	5–6
3	Spillover Energy toxicity and spillover · about 12 minutes	7–8
4	Insulin resistance Insulin resistance – cellular self-defence · about 12 minutes	9–10
5	When the pancreas can't keep up Finally, your pancreas can't keep up and sugar surges · about 15 minutes	11–12
6	Join it all up Draws on the whole chapter · about 20 minutes	13–14
✓	Return ticket Spaced review, checklist and a final word	15

BEFORE YOU STA RT

How to use these pages

Read this once.
It takes a minute.

1. Close the book. Answer from memory. Write it down, say it aloud, or think it through fully before you look. Recalling an idea strengthens it far more than rereading it, even when your recall is shaky.
2. Note your confidence. Circle S (sure), U (unsure) or G (guess) beside each question, or just note it in your head.
3. Turn the page to check. Every question page is followed by its answer page, with matching question numbers.
4. Mark yourself. Give each question ✓ ✓ (right), ✓ (partly right) or ✗ (wrong). A question with several parts counts as one, and its answer tells you when to give yourself the mark. The brain dump and the Look ahead questions are not marked.
5. Learn most from confident mistakes. Read every explanation, but pay most attention to answers you were sure of and got wrong. Once corrected, those errors tend to stick best of all.
6. Come back later. A gap before you revisit makes the second attempt harder and the learning stronger. The Return ticket at the end shows how.

The five levels

●○○○○	Recall	Bring a fact back from memory.
●●○○○	Understand	Explain what it means or why it is so.
●●●○○	Connect	Link ideas from different parts of the book.
●●●●○	Apply	Use the ideas on a real situation.
●●●●●	Look ahead	Predict what later chapters will say. You are not expected to know these yet, and they are not marked - but trust the process and try them anyway

On paper or on a screen

On paper: print on A4, double-sided, flipping on the long edge. Each answer page then falls on the back of its questions, so the answers stay out of sight while you work. A pencil is all you need.

On a screen: view one page at a time on a tablet or computer. Think each answer through fully, or say it aloud, before you move on. Nodding along doesn't count. A phone works too, but the pages are A4, so expect to zoom and drag it around a lot.

Pace: Doing these right after you finish and then again a day after reading the chapter works very well indeed.

Don't skip the easy questions. Fast, confident recall of the basics is what frees your thinking for the harder ones.

Your progress

Set	Date	Right	Sure but wrong	Redone (date)
Set 1 Energy in, fat stored		___ / 7		
Set 2 The buffer and the threshold		___ / 8		
Set 3 Spillover		___ / 7		
Set 4 Insulin resistance		___ / 8		
Set 5 When the pancreas can't keep up		___ / 8		
Set 6 Join it all up		___ / 5		

The methods here come from the research on learning summarised in Make It Stick (Brown, Roediger and McDaniel, 2014): retrieval practice, spacing, mixing topics, explaining in your own words, and checking your confidence against your results.

1 Energy in, fat stored

About 12 minutes
Close the book first

Chapter section: Excess energy from any source ends up as fat

Start with the brain dump: it shows you what you remember before any question gives you clues.

1.1 Three-minute brain dump. Close the book. Take a blank sheet of paper or a notebook and write down everything you remember from Chapter 3. Words, short notes, arrows and sketches are all fine. Don't worry about order or neatness. Stop after three minutes. ●○○○○ RECALL

Prefer not to write? Explain everything you remember aloud instead.

1.2 Roughly how much glycogen can your muscles and liver store between them? ●○○○○ RECALL S U G

○ A about 50 g ○ B about 500 g ○ C about 2 kg ○ D about 10 kg

1.3 You eat a meal rich in carbohydrate and fat. What happens to the fat after you finish eating it? S U G

●●○○○ UNDERSTAND

○ A It is turned into glycogen in much the same way as the carbohydrate
○ B Less of it is absorbed while carbohydrate is present
○ C More of it is stored, because less fat is burned while carbohydrate is being used
○ D The liver converts it into glucose for later use

1.4 How much does turning carbohydrate into new fat contribute to body fat? S U G

●●○○○ UNDERSTAND

○ A A lot, as it is the main destination of carbohydrate in the body
○ B It happens mainly when glycogen stores are empty, so quite a lot
○ C It happens only in the livers of people with diabetes, but in large amounts
○ D It happens, but much less than is commonly believed

1.5 Which statement about insulin and storing the fat you eat fits? ●●●○○ CONNECT S U G

○ A Insulin makes fat storage easier, but fat can be stored even at low insulin levels
○ B Fat can be stored only when insulin spikes after a carbohydrate-rich meal
○ C Eating less carbohydrate lowers insulin and prevents ‘fat storage mode’
○ D Fat eaten without carbohydrate is burned for energy rather than stored

1.6 Someone eats a whole packet of butter on its own. Their blood glucose and insulin barely move. What happens to the energy, and what does this tell you about the idea of a ‘fat storage mode’? S U G

●●●●○ APPLY

1.7 True or false? “Protein can't make you gain fat, because the body uses it for building and repair, not energy.” If false, correct it. ●●○○○ UNDERSTAND S U G

1.8 The chapter compares fuels to several doors leading into the same hotel lobby. What is the point of the comparison, and what decides whether body fat grows? ●●●○○ CONNECT S U G

Turn the page to check your answers.

SET 1 OF 6 · A NSW ERS

1 Energy in, fat stored

Mark each question ✓, ½ or ✗

1.1 Not marked. The point is to find out what you can recall before any question gives you clues. The gaps you noticed are useful: they show you where to look. Keep your sheet, or jot down a few words about what you managed aloud. You will repeat the brain dump on the 'return ticket' and can compare.

1.2 B. Around 400 g in muscle and 100 g in the liver (~1 lb and ~¼ lb), so about 500 g in all. That is a small store compared with the fat you can carry, which is why glycogen fills up quickly when carbohydrate keeps arriving.

1.3 C. When plenty of carbohydrate is available, the body burns more carbohydrate and less fat, which leaves more of the incoming fat free to be stored. A and D describe conversions the chapter doesn't describe, and B confuses what happens after absorption with absorption itself.

1.4 D. It does happen, particularly when carbohydrate intake stays very high and glycogen is already full, but much less than is commonly believed. B has it backwards. Carbohydrate mostly adds to body fat indirectly, by being burned in place of fat (1.3).

1.5 A. Insulin makes fat storage more favourable after eating, but it isn't an on/off switch. Fat from food travels in small fat-rich particles and is released to tissues such as fat and muscle as they pass, and it can be stored even at the low background insulin levels between meals. That is why C is wrong: there is no ‘fat storage mode’.

1.6 Model answer: The butter supplies far more energy than the body needs while it is digesting it, so the surplus is stored as body fat, even though insulin hardly rises. Fat storage doesn't need an insulin spike, so there is no special ‘fat storage mode’ to avoid. What decides storage is energy surplus, not the insulin response to a single meal.

1.7 False. Protein can be used as fuel. Eating more than you need adds to your daily energy intake, and burning protein for energy also means burning less of the carbohydrate and fat arriving at the same time. Either way, a repeated surplus ends up in fat stores.

1.8 Model answer: Fuels arrive by different routes and are handled differently at first, but they all end up in the same shared energy pathways, where the body decides whether to use the energy now or store it. What decides whether fat grows is whether energy coming in repeatedly exceeds energy used. In the chapter's words, your metabolism does not see what was on your plate.

> FROM ANDREW
>
> *If one idea from this set follows you into the supermarket, make it 1.5. Plenty of products and plans are sold on the promise of keeping you out of ‘fat storage mode’. There is no such mode, and the energy arithmetic doesn't care which aisle the surplus came from.*

Marked questions right: ____ out of 7 Sure but wrong: ____ (redo these after a day or two)

Several wrong? Try this set again in a day or two, after rereading the section Excess energy from any source ends up as fat

2 The buffer and the threshold

About 12 minutes
Close the book first

Chapter sections: The protective buffer phase · Personal fat threshold

Your fat tissue's first jobs are storage and protection. This set is about how long it can keep that up, and why the answer differs from person to person.

2.1 When you regularly eat more energy than you need, where does most of the excess go at first? S U G

● ○ ○ ○ ○ RECALL

- ○ A Visceral fat around the organs
- ○ B Fat inside the liver
- ○ C Muscle glycogen
- ○ D Subcutaneous fat cells

2.2 As subcutaneous fat tissue fills, it expands because… ● ● ○ ○ ○ UNDERSTAND S U G

- ○ A cells enlarge and new cells form
- ○ B cells enlarge; no new cells form
- ○ C new cells form; size stays the same
- ○ D it moves stored fat into muscle

2.3 True or false? “During the buffer phase, growing fat tissue makes your metabolic health worse.” If false, correct it. ● ● ○ ○ ○ UNDERSTAND S U G

2.4 During the buffer phase… ● ● ○ ○ ○ UNDERSTAND S U G

- ○ A glucose is in the diabetic range
- ○ B visceral and organ fat rise a little
- ○ C fat tissue is insulin resistant
- ○ D no surplus fat reaches the organs

2.5 According to the chapter, what mainly determines your personal fat threshold? ● ○ ○ ○ ○ RECALL S U G

- ○ A The fat content of your diet
- ○ B Your current body weight
- ○ C Your genes
- ○ D Your activity level

2.6 True or false? Write T or F beside each statement about the personal fat threshold. S U G

● ● ○ ○ ○ UNDERSTAND

□	a. It can be measured with a routine blood test.
□	b. It is better thought of as a gradual transition than a sharp line.
□	c. Two people at the same weight can be on opposite sides of it.
□	d. It describes how much fat there is in your diet.
□	e. Family history and your blood results over the years can hint at which side you're on.
□	f. Crossing it helps explain why diabetes can suddenly speed up after years of slow change.

2.7 In Chapter 2, insulin told fat cells to do two things. Once overfilled, subcutaneous fat responds less to insulin, Which of those jobs weakens, and what is the result? ● ● ● ○ ○ CONNECT S U G

2.8 Sam and Alex both weigh 82 kg. Sam has type 2 diabetes, while Alex's blood glucose is normal. Use the personal fat threshold to explain how this can happen, and why it doesn't mean Sam's weight is irrelevant. ● ● ● ● ○ APPLY S U G

Turn the page to check your answers.

2 The buffer and the threshold

Mark each question ✓, ½ or ✗

2.1 D. Most of the excess is first stored as fat in subcutaneous fat cells, which gives it a safe place to go. Visceral and liver fat (A, B) begin to rise later and more slowly, and glycogen (C) fills within a day or so.

2.2 A. Individual fat cells grow larger, and new fat cells are made to help out. Together, this lets subcutaneous fat act as a buffer that protects the rest of the body.

2.3 False. For a while, fat tissue is solving the problem by growing, not causing it. It keeps surplus fuel away from organs where it would interfere with normal function. The trouble starts when that capacity runs out.

2.4 B. Visceral fat starts to increase, and small amounts of ectopic fat collect in the liver, muscles and pancreas, but while subcutaneous fat still has room the organs stay fairly well protected. Metabolic health is starting to decline in this preclinical phase, before anyone has noticed. A and C come later.

2.5 C. Mainly genetics, and to a lesser extent life history. A is the classic mix-up: the threshold is about your capacity to store body fat safely, not the fat content of your diet.

2.6 a F · b T · c T · d F · e T · f T There is currently no way to measure or predict the threshold directly, which is why the clues in e are useful. Mark: five of the six.

2.7 Model answer: Insulin tells fat cells to take up and store fuel after meals, and also to hold on to the fat they already contain. As the strained tissue becomes less responsive, the second job weakens: the restraint on fat breakdown loosens and more free fatty acids escape into the blood. Storage space is running short and stored fat is leaking at the same time.

2.8 Model answer: Each person's capacity to store fat safely is different. Alex's subcutaneous fat may still be storing the surplus safely, while Sam's has passed its limit, so surplus is spilling into the organs. Weight still counts for Sam: what matters is fat relative to his own threshold, so losing fat can bring him back below it, as Mika's story shows in Set 6.

Marked questions right: ____ out of 8 Sure but wrong: ____ (redo these after a day or two)

Several wrong? Try this set again in a day or two, after rereading the sections The protective buffer phase and Personal fat threshold

3 Spillover

About 12 minutes
Close the book first

Chapter section: Energy toxicity and spillover

Safe storage has run out. Now the surplus goes where it shouldn't, and the organs that control your glucose suffer.

3.1 Once subcutaneous fat can no longer contain the surplus, the chapter describes two related problems. Which pair? ●●○○○ UNDERSTAND S U G

- ○ A glycogen overflow; liver stops storing anything
- ○ B fat cells die; stored fat is lost
- ○ C less room; stored fat held less tightly
- ○ D insulin stops; glucagon takes over

3.2 Visceral fat adds to the burden in several ways. Which of these does the chapter **not** say about it? ●●○○○ UNDERSTAND S U G

- ○ A It produces inflammatory signals
- ○ B It releases fatty acids between meals
- ○ C Some of it drains straight to the liver
- ○ D It stores fat safely, shielding the organs

3.3 Someone says, “Fatty liver must mean a person eats too much fat.” Why is that conclusion wrong? Describe two other ways fat can build up in the liver. ●●●○○ CONNECT S U G

3.4 In the oshiya picture, the passenger-pushers and the crowded carriages stand for… ●●○○○ UNDERSTAND S U G

- ○ A insulin; overloaded muscle cells
- ○ B glucagon; overloaded liver cells
- ○ C muscle cells; the bloodstream
- ○ D fatty acids; fat cells

3.5 Fat droplets build up inside and between muscle cells. As described in the chapter, what is the real problem that comes along with this? ●●●○○ CONNECT S U G

- ○ A droplets physically block glucose
- ○ B fat buildup causes cellular malfunction
- ○ C cells turn the fat into glucose
- ○ D fat replaces the muscle fibres

3.6 The overflowing kitchen returns. Match each picture to what it stands for. Write the number in the box. ●●●○○ CONNECT S U G

In the kitchen	In your body
□ Cupboards full and expanded as far as the kitchen allows	1. Visceral and ectopic fat
□ Countertop piling up with new and fallen-out items	2. The next meal adding to the surplus
□ Items on the floor and behind the appliances	3. Subcutaneous fat is past its safe capacity
□ Cupboards creaking under the strain	4. Raised glucose and fatty acids in the blood
□ Knock knock… another delivery	5. Strained fat tissue adds to background inflammation

3.7 The chapter calls this state **energy toxicity**. Why do these changes tend to push blood glucose higher both after meals and between them? ●●●○○ CONNECT S U G

3.8 A muscle cell keeps receiving more fuel than it can use, while insulin keeps telling it to take in more. Predict what a sensible cell might do to protect itself. ●●●●● LOOK AHEAD S U G

Turn the page to check your answers.

SET 3 OF 6 · A NSW ERS

3 Spillover

Mark each question ✓, ½ or ✗

3.1 C. Incoming energy has less safe storage space, while fat already in storage is held less effectively (Set 2, question 2.7). The result is more fatty acids circulating and more fat collecting in visceral and ectopic sites. B is wrong: fat cells are struggling, not disappearing.

3.2 D. That describes healthy subcutaneous fat. Visceral fat produces inflammatory signals that worsen insulin resistance, releases fatty acids in the fasted state, and partly drains into the portal circulation, so some of what it releases goes straight to the liver.

3.3 Model answer: Fat from meals is only one source. Fatty acids released from your body's fat stores travel in the blood and are taken up by the liver; in tracer studies this was the largest single source, and it grows as strained fat tissue leaks and visceral fat drains straight to the liver. The liver can also make new fat from glucose and fructose (and alcohol), especially when carbohydrate intake stays **very** high. So fat in the liver is not a record of what you ate recently: it reflects the whole body's energy surplus. Mark: two of these sources correctly described.

3.4 A. Insulin keeps trying to push glucose out of the crowded blood and into muscle cells that are already crammed with fuel, while extra fatty acids keep arriving at the same time.

3.5 B. The droplets themselves are not necessarily the harmful part. Chronic fat oversupply leads to **abnormal handling of fat inside the cells**, which interferes with insulin signalling. A is the intuitive picture, but the actual problem is a blockage of signalling, not of physical space.

3.6 Full cupboards 3 | Countertop 4 | Floor, appliances 1 | Creaking 5 | Delivery 2 Mark: four of the five.

3.7 Model answer: With buffering overwhelmed, fuels stay in the blood for longer. After meals, overloaded muscle takes up glucose less readily. Between meals, a fat-laden liver regulates glucose less well and releases too much. At first, the pancreas may compensate by releasing extra insulin, so blood glucose can stay close to normal even as the underlying problem grows (Set 4). Looking ahead: these two pressures become the spike and the floor of Chapter 7.

3.8 Not marked. Set 4's answer: it becomes less responsive to insulin's message, which reduces the pressure to keep accepting fuel. That is insulin resistance, and it comes at a cost to the rest of the body.

> FROM A NDREW
>
> *The central problem in this set is too much fuel for too long, in a body whose safe storage has run out. That is why the plan in Part 2 is built around draining the excess rather than banishing a particular food.*

Marked questions right: ____ out of 7 Sure but wrong: ____ (redo these after a day or two)

Several wrong? Try this set again in a day or two, after rereading the section Energy toxicity and spillover

4 Insulin resistance

About 12 minutes
Close the book first

Chapter section: Insulin resistance – cellular self-defence

All the threads meet here. Expect at least one answer to surprise you: insulin resistance is not quite the villain it is usually made out to be.

4.1 When a cell or tissue is insulin resistant, it… ●○○○○ RECALL (SUG)

- ○ A makes less insulin than it should
- ○ B responds less to the same insulin
- ○ C breaks insulin down before it can act
- ○ D refuses glucose, whatever the insulin

4.2 Why can insulin resistance be partly protective for an overloaded cell? ●●○○○ UNDERSTAND (SUG)

- ○ A It lowers the cell's need for energy
- ○ B It lets the cell release its stored fat
- ○ C It allows the pancreas to rest
- ○ D It eases the pressure to keep accepting fuel

4.3 Insulin resistance can affect different tissues. Match each tissue to what goes wrong there. (SUG)

●●●○○ CONNECT

□ Muscle	a. Insulin is less able to turn down its glucose output
□ Liver	b. Insulin's restraint on the release of stored fatty acids weakens
□ Fat tissue	c. It takes up less glucose in response to insulin

4.4 In the kitchen analogy, insulin resistance is like… ●○○○○ RECALL (SUG)

- ○ A ordering fewer deliveries
- ○ B fitting a bigger cupboard
- ○ C putting chewing gum on the door knocker
- ○ D clearing the floor behind the appliances

4.5 As cells become insulin resistant, what does the pancreas do at first? ●●○○○ UNDERSTAND (SUG)

- ○ A Releases less insulin, to protect its beta cells
- ○ B Releases more glucagon, to clear the extra glucose
- ○ C Releases insulin only after the largest meals
- ○ D Releases more insulin, to hold glucose down

4.6 Pat's fasting glucose has been normal at three yearly check-ups, yet insulin resistance has gradually been getting worse. What does a normal fasting glucose result tell us, and what does it fail to tell us? ●●●●○ APPLY (SUG)

4.7 Use GLUT4 (Chapter 2) to explain why an insulin-resistant muscle needs more insulin to take up the same amount of glucose. ●●●○○ CONNECT (SUG)

4.8 Insulin resistance helps the overloaded cell but harms the body as a whole. Explain how both can be true. ●●●●○ APPLY (SUG)

Turn the page to check your answers.

SET 4 OF 6 · A NSW ERS

4 Insulin resistance

Mark each question ✓, ½ or ✗

4.1 B. Resistance is about the response, not the supply. A describes reduced insulin production, a different problem that comes later (Set 5). Early on, people with insulin resistance usually have more insulin than normal, not less.

4.2 D. A cell already struggling with more fuel than it can use or store is protected by responding less to insulin's 'take in more' message. The cost is that more glucose stays in the blood.

4.3 Muscle c | Liver a | Fat tissue b When several important tissues respond poorly at once, we can call the whole body insulin resistant. Mark: all three.

4.4 C. Muffling the knocker means it takes a much harder, more persistent knock to be heard, just as resistant cells need more insulin. Ignored deliveries pile up outside the door, as glucose does in the blood.

4.5 D. The pancreas responds mainly to blood glucose and can't see the overload inside cells. As glucose starts to run higher, the beta cells release more insulin, then more again. At first this keeps glucose normal.

4.6 Model answer: It tells us glucose was being kept in range at the moment it was measured. It doesn't tell us how hard the body was working to achieve that. Pat's pancreas may be releasing much more insulin than before to hold glucose there, and because insulin itself is seldom measured, that growing strain stays hidden, sometimes for years. Looking ahead: Chapter 5 returns to why the problem starts well before diagnosis. Mark: both halves: what the result shows, and the hidden extra insulin.

4.7 Model answer: Insulin signals muscle cells to move GLUT4 transporters to the surface so they can take in glucose. In an insulin-resistant cell, the same amount of insulin produces a weaker response, so fewer GLUT4s reach the surface. A higher insulin concentration is needed to get the same glucose uptake.

4.8 Model answer: For the cell, responding less to insulin limits how much more fuel it is pushed to accept, which protects it. For the body, that same refusal leaves more glucose in the blood, which damages tissues over time and forces the pancreas to work harder and harder. Both are true because the cell and the whole body have different problems to solve.

Marked questions right: ____ out of 8 Sure but wrong: ____ (redo these after a day or two) Several wrong? Try this set again in a day or two, after rereading the section Insulin resistance – cellular self-defence

5 When the pancreas can't keep up

About 15 minutes
Close the book first

Chapter section: Finally, your pancreas can't keep up and sugar surges

For years the pancreas covers for everyone else. This set is about why it eventually can't, and what that looks like from the outside.

5.1 As type 2 diabetes develops, strained fat tissue and an increasingly fatty liver can both increase the fat reaching the pancreas. Explain what each one releases. ●●○○○ UNDERSTAND S U G

5.2 True or false? “Under fat overload, beta cells mostly die, so they stop making insulin.” If false, correct it. ●●○○○ UNDERSTAND S U G

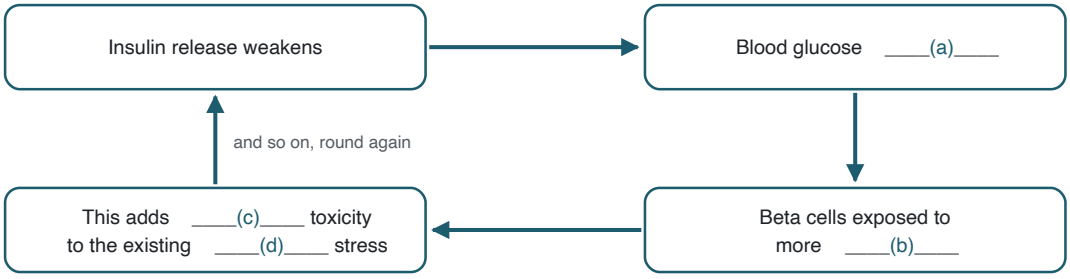
5.3 The first-phase insulin response normally… ●●○○○ UNDERSTAND S U G

- ○ A holds glucose up between meals
- ○ B clears fat before glucose arrives
- ○ C triggers digestive enzyme release
- ○ D restrains the liver's glucose output

5.4 As the first-phase response weakens, blood glucose after meals… ●●●○○ CONNECT S U G

- ○ A rises slower, peaks the same
- ○ B dips too low before rising
- ○ C rises higher and stays up longer
- ○ D unchanged if later release is normal

5.5 The vicious circle. Fill in the four blanks. ●●●○○ CONNECT S U G



5.6 Name three or more symptoms as blood glucose begins to rise. ●○○○○ RECALL S U G

5.7 By diagnosis, four defects are feeding into one another. In a few words, say what has gone wrong in each. ●●●○○ CONNECT S U G

Fat tissue	
Liver	
Muscles	
Pancreas	

5.8 A diagnosis of type 2 diabetes often feels sudden. Explain why it usually isn't. ●●●●○ APPLY S U G

Turn the page to check your answers.

SET 5 OF 6 · A NSW ERS

5 When the pancreas can't keep up

Mark each question ✓, ½ or ✗

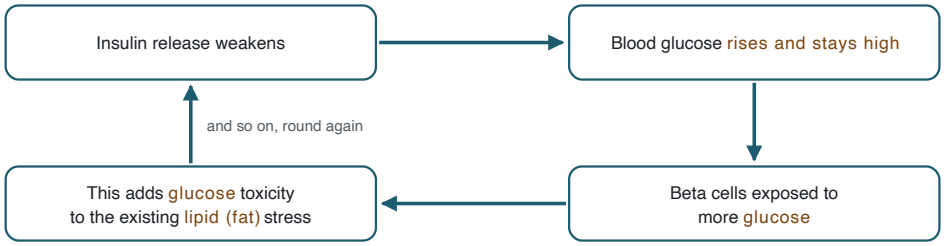
5.1 Insulin-resistant fat tissue holds its stores less tightly and releases more fatty acids into the blood. A fatty liver packages and exports more triglyceride, adding to the fat delivered to other tissues, including the pancreas. Mark: both.

5.2 False. Beta cells can become functionally impaired without necessarily dying. They respond less to rising glucose and lose some of their ability to release the right amount of insulin at the right time. Impaired is not the same as gone, which is why recovery is possible.

5.3 D. The rapid burst of insulin within minutes of eating restrains the liver's own glucose output just as meal glucose begins to arrive, and helps other tissues get ready to take glucose up. A describes glucagon's job.

5.4 C. Without the early burst, the liver keeps releasing glucose while meal glucose is arriving, and other tissues are slower to respond, so glucose rises higher after meals and stays raised for longer.

5.5



Poorer insulin release lets glucose rise, and high glucose makes effective insulin release harder still. Mark: three of the four.

5.6 Any three of: increased thirst, tiredness, frequent urination and blurred vision. A continuous glucose monitor would also show high readings after eating.

5.7 Fat tissue: can't contain the surplus and, being insulin resistant, releases too many fatty acids. Liver: overloaded with fat and insulin resistant, so it releases too much glucose. Muscles: insulin resistant, so they take up less glucose. Pancreas: can no longer compensate; beta-cell function is impaired just when more insulin is needed. Mark: three of the four.

5.8 Model answer: The process develops silently for years. Insulin resistance builds while the pancreas compensates with extra insulin, so standard glucose tests stay near normal. Only when beta-cell function falters does glucose rise noticeably, so diagnosis marks the final stage of a long process, not its start.

> FROM A NDREW
>
> *The phrase I would underline in this set is 'without necessarily dying'. Beta cells struggling under fat and glucose overload are not the same as beta cells that are gone, and that difference is a large part of why remission is possible. In most people, beta cells can recover substantially.*

Marked questions right: ____ out of 8 Sure but wrong: ____ (redo these after a day or two) Several wrong? Try this set again in a day or two, after rereading the section Finally, your pancreas can't keep up and sugar surges

SET 6 OF 6 · QUESTIONS

6 Join it all up

About 20 minutes
Close the book first

Draws on the whole of Chapter 3, especially Lean diabetics have the same problem differently and There is a silver lining.

Everything mixed together, as you will meet it in real life. Expect this set to be the hardest; that is the point of it.

6.1 Put the story in order. Number these events 1–7 in the broad order used in the chapter. Some stages overlap or unfold differently between people, so look for the main pattern rather than an exact timetable. ● ● ● ○ ○ CONNECT S U G

□	Beta cells falter, the first-phase response fades, and glucose rises
□	Subcutaneous fat stores the surplus safely
□	Muscle and liver become insulin resistant
□	The personal fat threshold is crossed
□	The pancreas compensates with extra insulin; glucose looks normal
□	Fat spills into visceral and ectopic sites
□	Strained fat tissue becomes insulin resistant and leaks fatty acids

6.2 In Figure 3.1's bucket model, what does the tap stand for, why does each bucket have a drain on it, and why do you think the pancreas has the smallest drain? ● ● ● ● ○ APPLY S U G

6.3 Mika is slimmer than her friends, thinks she eats well, and has an HbA1c of 7.3%. Explain how she can have type 2 diabetes, and why a modest weight loss of about 6 kg helped. ● ● ● ● ○ APPLY S U G

6.4 Spot the errors. Gary is back, and he has been reading online again. Find the seven mistakes and correct them. ● ● ● ● ○ APPLY S U G

> *"Type 2 diabetes is caused by eating sugar, because sugar spikes your insulin and puts you into fat storage mode. Fat can't make you fat, because it barely raises insulin. Insulin resistance just means your pancreas has stopped making insulin. If your fasting glucose is normal, your metabolism is fine. Slim people can't get type 2 diabetes. And once you've got it, your pancreas is broken for good."*

6.5 Explain it to a friend. In five sentences or fewer, and with no jargon you don't explain, describe how type 2 diabetes develops. ● ● ● ○ ○ CONNECT S U G

6.6 Look ahead. Guess boldly; these are not marked. ● ● ● ● ● LOOK AHEAD S U G

a. Chapter 4 covers lifestyle drivers such as poor sleep, stress and inactivity. Pick one and predict how it could push someone towards diabetes even without extra food.

b. Part 2 includes resistance training. Using Chapters 2 and 3, predict why.

Turn the page to check your answers.

SET 6 OF 6 · A NSW ERS

6 Join it all up

Mark each question ✓, ½ or ✗

6.1 1 Subcutaneous fat stores the surplus safely. 2 The threshold is crossed. 3 Strained fat tissue becomes insulin resistant and leaks fatty acids. 4 Fat spills into visceral and ectopic sites. 5 Muscle and liver become insulin resistant. 6 The pancreas compensates; glucose looks normal. 7 Beta cells falter and glucose rises. This is a teaching sequence, not a timetable: neighbouring stages often overlap, and some can come in a different order in different people. Mark: the broad order, allowing one swap of neighbouring stages.

6.2 Model answer: The tap is incoming food energy. Each bucket has its own drain because they all dispose of the stored fuels they have onboard and thie drain represents using up fuels by each organ. The pancreas has the smallest drain because it is usually the slowest to clear stored fat and needs a sustained period without being topped up to do so. (Look at the size of the drain on the liver and subcutaneous fat buckets!)

6.3 Model answer: Mika's personal fat threshold is low, so surplus spills into her liver, pancreas and muscle at a body weight that looks slim. Lifestyle factors can also drive insulin resistance (Chapter 4). Losing about 6 kg (14 lb), cutting snacking, starting resistance training and improving sleep are the kinds of change that drain that fat and improve insulin sensitivity. Her HbA1c fell from 7.3% to 6.0% (56 to 42 mmol/mol) in five months.

6.4 Corrections: 1 Sugar alone doesn't cause it. A major route into type 2 diabetes is a long-running energy surplus, from any food, that builds up fat in the organs, but people differ in how susceptible they are, and other factors such as inactivity, poor sleep and stress contribute too. 2 There is no ‘fat storage mode’. 3 Dietary fat is stored even at low insulin; surplus from fat still becomes body fat. 4 Insulin resistance means tissues respond less to insulin; early on, the pancreas usually makes more. 5 Normal glucose can hide years of compensation. 6 Slim people can develop it, especially with a low personal fat threshold. 7 Beta-cell function can recover when the excess fat is cleared. Mark: five or more; ½ for three or four.

6.5 Example: In one major route into type 2 diabetes, a long-running energy surplus is stored as fat under the skin until that store can no longer take more safely. The extra then collects around and inside organs such as the liver, pancreas and muscles, where it disrupts how they work. Overloaded cells respond less to insulin, the hormone that moves glucose into them, so the pancreas releases more and more insulin, keeping blood glucose near normal, sometimes for years. Eventually the insulin-making cells of the pancreas, strained by fat and rising glucose, can't keep up, and blood glucose climbs into the diabetic range. People differ in how much fat they can store safely and how vulnerable their pancreas is, and inactivity, poor sleep and stress push towards insulin resistance too, which is why some people develop diabetes at much lower body weights than others.

6.6 Not marked. Likely answers: a Poor sleep, stress and disrupted daily rhythms push glucose control off course through hormones such as cortisol, and inactivity shrinks your biggest glucose sink. b Muscle is the main destination for glucose after meals, and working muscle takes up glucose by a route that doesn't rely on insulin. Resistance training also helps you keep muscle while you lose fat.

Marked questions right: ____ out of 5 Sure but wrong: ____ (redo these after a day or two)

SPA C ED REVIEW

Return ticket

Use this page twice, after a gap each time

Memory fades fastest in the first few days, and each successful recall after a gap slows the fading. Come back to this page a day or two after finishing the sets, then again about a week later, wherever you have got to in the book. There are no answers here. Answer from memory, tick the round, then check against the answer named.

	Question	First return	Second return
R1	What happens to surplus energy from protein? See 1.7	☐	☐
R2	Does storing the fat you eat require a large rise in insulin? See 1.5	☐	☐
R3	What mainly sets your personal fat threshold? See 2.5	☐	☐
R4	Name two ways visceral fat adds to the burden. See 3.2	☐	☐
R5	Define insulin resistance in one sentence. See 4.1	☐	☐
R6	What can a normal fasting glucose result fail to tell you? See 4.6	☐	☐
R7	What does the first-phase insulin response do, and what happens when it fades? See 5.3 and 5.4	☐	☐
R8	Name the four defects that feed into one another by diagnosis. See 5.7	☐	☐
R9	Three-minute brain dump: close the book and, on a blank sheet or aloud, recall everything you can from Chapter 3. Then compare it with your first attempt to see how much more you can now recall. See 1.1	☐	☐

Can you do these without the book?

- ☐ I can explain why surplus energy from any food can end up as body fat.
- ☐ I can explain why there is no ‘fat storage mode’.
- ☐ I can describe the buffer phase and the personal fat threshold.
- ☐ I can explain energy toxicity and where spilled-over fat ends up.
- ☐ I can define insulin resistance and say why it is partly protective.
- ☐ I can explain how the pancreas compensates, and why glucose can look normal.
- ☐ I can describe how beta cells falter and the vicious circle that follows.
- ☐ I can name the four defects present at diagnosis.
- ☐ I can explain why lean people can develop type 2 diabetes.

FROM ANDREW

Chapter 3 is the perhaps hardest stretch of the book, and it ends with the most useful news. The damage in type 2 diabetes is driven by fat in the wrong places, and that fat can be drained. When it is, beta cells can recover and the liver and muscles can respond properly to insulin again. Everything in Part 2 is built on that. Any box you couldn't tick points to the set to revisit first.