Diabetic Dog Companion

Practical guidance for the first ninety days, and the full research behind it

Tom Sampson

The vet always comes first.

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This book is intended as general information for owners of diabetic dogs. It is not a substitute for veterinary advice, diagnosis, or treatment. Always consult your own veterinarian regarding your dog's specific condition and treatment plan. The vet always comes first.

The companion app referenced throughout this book is available at prodexa.co.uk/diabetic-dog-companion.

Foreword

Bryce was thirteen when he was diagnosed with diabetes. We were in southern Spain at the time, and I found it genuinely challenging to manage well. Diabetes in a dog is a demanding, day-to-day undertaking wherever you are, and doing it for the first time, without the network of experienced owners and vets I'd later find in the UK, made it harder still.

So I did the research myself. Between what I found and what the vets already knew, we got Bryce reasonably under control. It was not straightforward, and there was a fair amount of trial and error along the way, but it worked.

Back in the UK, I found a much wider network to draw on: excellent owner forums, full of people who had already worked out most of what a new owner needs to know, and more people locally who'd managed the condition before. But I still thought there was more that could be done. Building a glucose curve by hand every few months is fiddly and easy to get wrong. Planning a meal that properly accounts for fat, calories, fibre, and protein together is more than most owners want to work out from scratch. And when it comes to the hardest decisions, the ones about whether a dog can keep going, there is very little support at all. So I built an app, prodexa.co.uk/diabetic-dog-companion, a web based version and one that runs on Windows, to help with exactly that kind of practical, ongoing work.

But an app alone did not feel like the whole answer. Some owners need something targeted at the first few weeks, the practical decisions that cannot wait. Others want to understand the condition properly, not just follow instructions. This book is my attempt at both.

Part 1 covers the start of the journey. It is built to work alongside the app, and it assumes nothing except that your dog was diagnosed recently and you need to know what to do. Part 2 goes further. It draws together research carried out by a wide range of organisations

and tries to present it in a form that is thorough without being unreadable.

I am not a vet. I am an owner who did a great deal of reading because Bryce needed me to, with plenty of help along the way from vets and from owners on forums who had been through it before me. This book is free for exactly that reason. It is my way of putting something back. If it saves someone else that same steep first climb, it has done its job.

Tom Sampson

The Companion App

Throughout this book you will see references to the companion app: a DMB fat checker, a Daily Calorie Calculator, a Meal Builder, a Food Finder, a Glucose Log, and a quality of life checker among them. It is available at prodexa.co.uk/diabetic-dog-companion, as a web app and as a downloadable Windows application, and it is built to sit alongside Part 1 of this book in particular, handling the calculations so you do not have to.

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PART ONE: THE FIRST NINETY DAYS

Chapter 1: The Diagnosis

What just happened, what it means, and why this is not the end.

If your dog has just been diagnosed with diabetes mellitus, you are probably feeling one of two things: frightened, or numb. Possibly both. The words landed, the vet said things you tried to follow, and now you are at home with a dog who looks the same as he always has, and a future that suddenly looks very different.

The first thing to say is the one that matters most: this is manageable. Dogs live well with diabetes. Not despite it, not in spite of heroic effort, but because once you understand what the condition requires and build a routine around it, daily life with a diabetic dog is not dramatically different from daily life with a healthy one. The routine becomes normal. The injections become normal. Most owners, a few months in, describe it that way: normal.

The second thing to say is that you will not be ready for all of it immediately, and you don't need to be. This chapter covers what you need to understand right now. The detail comes later, when you have the headspace for it.

What diabetes actually is

Your dog's pancreas has stopped producing enough insulin, or in some cases has stopped producing it altogether. Insulin is the hormone that allows cells to take glucose from the bloodstream and use it for energy. Without it, glucose accumulates in the blood while the body's cells are effectively starving, unable to access the fuel that's right in front of them. The body responds by breaking down fat and muscle instead, and by attempting to flush the excess glucose out through the kidneys, which is why a diabetic dog drinks so much and urinates so frequently. The excessive thirst, the accidents indoors, the weight loss despite a normal appetite: these are all the same mechanism.

Almost all diabetic dogs have what would be called Type 1 diabetes in human medicine: the insulin-producing cells are gone or non-functional, and they are not coming back. This means insulin

replacement for life. There is no dietary fix, no supplement, no alternative. But insulin therapy works. It works well.

Why your dog is still weeing at night

This is usually the thing that feels most urgent in the first days, so it is worth addressing directly.

Starting insulin does not immediately resolve the symptoms. The insulin dose your vet has prescribed is a cautious starting point, deliberately on the low side, because too little insulin causes problems gradually, while too much can cause a hypoglycaemic crisis that can kill a dog within hours. The dose will be adjusted upward over the coming weeks as your vet reviews glucose curves and response. During this period, your dog's blood glucose will still be elevated enough that the kidneys are still working to clear it, and the drinking and night-time urination will continue.

This is worth saying plainly, because it is the point where new owners are most tempted to push for a faster fix: your vet increasing the dose slowly is not caution for its own sake. A dog that is a little too high for a few more weeks is uncomfortable and inconvenient. A dog whose dose is pushed up too fast can crash into a hypoglycaemic crisis with far less warning. If the accidents are still happening after a week or two and you are tempted to ask for a bigger jump in dose, ask instead for a glucose curve: that tells your vet what is actually happening, rather than guessing from symptoms alone.

Most owners see meaningful improvement within one to two weeks. Full resolution typically takes four to six weeks, once the dog reaches a stable, well-controlled dose. It will get better. It is going to require some patience and some floor-cleaning in the meantime.

The prognosis

The honest picture: the highest-risk period is the first six months after diagnosis, while you and your vet are still finding the right dose and routine. Dogs that get through that period stable and well controlled do markedly better: one pilot study following primary-

care dogs found that those who stabilised within the first three months went on to survive roughly eight times longer than those who didn't (Cartwright et al., 2019). Diabetes itself, well managed, generally isn't what shortens a dog's life. What matters more is how well it's controlled, and the complications that come with poor control or bad luck, such as cataracts, recurring infections, and pancreatitis. We cover all of that fully in Chapter 8.

The honest answer to "how long will my dog live?" is: we don't know, and the first six months matter more to that answer than almost anything else. Age, breed, and concurrent conditions matter too. What you are doing right now, by learning how to manage this properly, is giving your dog the best possible chance of getting through the hard part and into the years beyond it. That is all any owner can do.

What the vet has prescribed and why

Your vet will have started your dog on an insulin specifically formulated for dogs. In most of the world this is Caninsulin, a porcine (pig-derived) insulin that closely matches the structure of canine insulin, which is why it works so well in dogs. In the United States it is marketed as Vetsulin. It is the same product. This matters for a reason we cover fully in the next chapter: it is a U-40 concentration insulin, meaning 40 units per millilitre, and it must be used with U-40 syringes. For now, file that detail away, just make sure you use the syringes your vet or pharmacy has supplied, and do not substitute others.

The starting dose will feel tentative. That is intentional. The first few weeks are about establishing how your dog responds, not hitting a target straight away. Expect vet appointments, expect dose adjustments, and expect to be asked to perform a glucose curve at home. Chapter 7 explains exactly how to do that.

What to do today

If you have just come from the vet with a diagnosis and a bottle of insulin, these are the only things that matter today:

- Understand the feeding and injection schedule your vet has given you, and follow it consistently. The same food, the same amount, at the same times. Consistency is the foundation of diabetic control.
- Know the signs of hypoglycaemia, low blood sugar, and know what to do if you see them. This is the one emergency that can develop quickly. We cover it fully in Chapter 3, and it is worth reading that chapter today, before anything else goes wrong.
- Keep a notebook or use the app. Write down every reading, every meal, every injection. You will want this record at every vet appointment, and you will find yourself grateful for it.

Everything else can wait until tomorrow.

Sources for this chapter: AAHA, Today's Veterinary Practice, JAVMA, MSD Animal Health, Cornell University College of Veterinary Medicine; Cartwright H, Wardell-Lane E, Kenny PJ. Pilot study evaluating the monitoring of canine diabetes mellitus in primary care practice. Veterinary Record Open. 2019;6(1):e000250.

Chapter 2: Insulin, What It Is and How to Give It

The injection is the thing most new owners dread most. Within a week, almost all of them have stopped thinking about it.

Dogs tolerate subcutaneous injections, into the loose skin rather than a vein or muscle, remarkably well. The needle is fine, the volume is small, and most dogs barely react. The bigger challenge is not the physical act but understanding what you are injecting and why the details matter. One error in this area can be serious. This chapter covers everything you need before you give the first dose.

What your dog is being injected with

The standard insulin for diabetic dogs across most of the world is porcine insulin, derived from pigs. This is not arbitrary: canine and porcine insulin are chemically identical, which is exactly why it works so well in dogs. It's human insulin that differs from canine, by a single amino acid, at a position called B30. It is sold as Caninsulin in the UK, Europe, Australia, Canada and most other countries, and as Vetsulin in the United States. The product is identical.

Dogs and humans handle insulin differently at a physiological level, not dramatically, but enough that products and equipment built for one don't always translate cleanly to the other. That's part of the reason dog-specific insulins and dog-specific glucose meters exist, and we'll go into exactly why in Part 2. For now, the practical point is simpler: use what your vet has prescribed, and never assume a human product or device will behave the same way in a dog.

This chapter focuses on Caninsulin, which is what the vast majority of owners will be using, but several other insulins are also in regular use, covered shortly.

As a rule of geography: Caninsulin (U-40) is the standard in the UK, the rest of Europe, Australia, Canada and more than thirty other countries. U-100 human analogues turn up almost exclusively in the United States, where Vetsulin isn't always a vet's first choice. If you

were diagnosed outside the US, assume U-40 unless your vet has told you otherwise, and if in doubt, ask.

Other insulins your vet may prescribe

While Caninsulin/Vetsulin is the most widely used insulin for diabetic dogs, several others are in regular veterinary use and your dog may be prescribed one of them, either from the outset or if Caninsulin does not achieve adequate control. The main ones are:

- NPH (Neutral Protamine Hagedorn): a human insulin sold under the brand names Humulin N and Novolin N. U-100, intermediate-acting, given twice daily. Historically widely used in North America, where it's inexpensive and readily available, though its duration of action in dogs is often under twelve hours, meaning less complete coverage and a real risk of a glucose spike between doses, and many specialists now favour other options where they're accessible.
- Detemir (Levemir): a long-acting human insulin analogue, U-100, typically given twice daily in dogs. Notable for being significantly more potent per unit than other insulins in dogs, roughly four times as potent, meaning starting doses are much lower than owners might expect. Used particularly in dogs that are poorly controlled on intermediate-acting insulins, or where concurrent illness complicates regulation. Its use in dogs has been growing as vets look for alternatives to shorter-acting insulins like NPH.
- Glargine (Lantus, U-100): another long-acting human analogue. More commonly used in cats than dogs, and in dogs its response is unpredictable enough that it's generally tried only after other options haven't worked.

Glargine U-300 (Toujeo) and degludec (Tresiba): newer, more concentrated basal insulins, increasingly used as a once-daily alternative that doesn't need to be timed to meals the way Caninsulin does. Growing evidence supports this approach in dogs; see Part 2 for detail. If your vet has prescribed one of these, ask

which of this book's feeding-timing rules still apply to you, some won't.

- ProZinc (Protamine Zinc Insulin): U-40, licensed for veterinary use and FDA-approved for dogs and cats. Can sometimes be given once daily in dogs, which suits some owners and some dogs' insulin requirements.

All of these insulins differ in concentration, duration of action, mixing instructions, and starting dose. The U-40/U-100 distinction covered above applies here too: NPH, detemir, and glargine are all U-100, requiring U-100 syringes. ProZinc is U-40 like Caninsulin. The specific properties of each, and how your vet decides between them, are covered in depth in Part 2.

U-40 and U-100: what this means and why it matters

Caninsulin is a U-40 insulin, 40 units per millilitre. Most human insulins are U-100, 100 units per millilitre. This matters because the syringe you use must be matched to the insulin, and if you use the wrong syringe without accounting for the difference, the dose will be wrong.

In most countries, U-40 syringes are the standard supplied with Caninsulin and are what almost all owners use. Draw up the prescribed number of units on the U-40 syringe and inject. It's straightforward.

U-100 syringes can be used with Caninsulin, but only if you apply the conversion: multiply the intended dose by 2.5 to find where to draw on the U-100 syringe. If the prescribed dose is 10 units, draw to the 25 mark on the U-100 syringe. The dose your dog receives is still 10 units. You are measuring the same amount of a more dilute insulin into a syringe calibrated for a more concentrated one. Some owners end up in this situation because U-40 syringes are not universally available; in parts of Europe and elsewhere they can be difficult to source. Knowing the conversion means you are never stranded.

The critical point either way: never change syringe type without recalculating. If someone else gives your dog an injection, a family

member or a boarding kennel, they need to know which system is in use. A dog receiving 2.5 times the intended dose of insulin can be in serious danger within hours.

The VetPen is a third option: a dial-up pen device designed for Caninsulin cartridges that eliminates syringe drawing entirely. If you find syringe administration difficult, ask your vet whether this suits your situation. Glargine and detemir have their own manufacturer pen devices too, carried over from their use in human medicine, which some owners find easier and more precise than drawing up a syringe; ask your vet whether one is available for your dog's insulin.

Preparing the dose

Before drawing up, the vial must be mixed. Caninsulin is a suspension: the insulin particles settle out of solution and must be redistributed before each use. The manufacturer's instruction is to shake the vial vigorously until the liquid is uniformly milky, then allow the foam to settle before drawing up.

This is different from the guidance for some other insulins: glargine and detemir must be rolled gently and must never be shaken. If your dog is ever switched to a different insulin type, check the mixing instructions for that product specifically before use.

Draw up the dose with a fresh syringe. Hold the vial upside down, insert the needle through the rubber stopper, and draw the plunger back to the prescribed mark. Check for air bubbles: tap the syringe gently and expel them before injecting.

Injection technique

Caninsulin is given subcutaneously, into the fatty layer beneath the skin. The scruff of the neck, the loose skin along the back and sides of the neck, and the flanks are the standard sites. Rotate between sites to avoid scar tissue building up in one area.

1. Tent a fold of skin by pinching it loosely; the scruff works well for most dogs.

1. Insert the needle at roughly a 45-degree angle into the base of the tent, bevel facing up. The bevel is the angled opening at the needle's tip; with it facing up, the sharpest edge slides under the skin rather than punching straight through it, which is noticeably less uncomfortable for your dog.
2. Pull back the plunger slightly. If blood appears, withdraw and start again at a different site with a fresh needle.
3. Depress the plunger smoothly and fully.
4. Hold the needle in place for two or three seconds before withdrawing. This allows the insulin to disperse and prevents it tracking back out along the needle path.
5. Withdraw and release the skin. Gentle pressure on the site afterwards is fine.

On needle choice: a higher gauge number means a finer needle. 32g is very fine and comfortable for the dog. On length, 8mm is sufficient when injecting into a properly tented skin fold on the scruff, where the folded skin brings the subcutaneous layer within reach. For injections into flatter sites on a larger dog, a 12.7mm needle gives more confidence that the insulin lands in the subcutaneous layer rather than the skin itself. If you notice variable glucose control that you cannot otherwise explain, needle length and injection depth are worth reviewing with your vet.

Dispose of used needles in a sharps container. Do not recap and reuse: needles dull after a single use and a blunt needle is more uncomfortable for the dog.

Timing

Caninsulin is given every twelve hours, immediately after a meal. The sequence is always: feed first, confirm the dog has eaten, then inject. The twelve-hour interval matters: drifting the timing progressively erodes glucose control. Choose times you can maintain reliably every day.

Making injections routine

The mechanics of the injection matter less than making it something your dog accepts calmly, twice a day, indefinitely. A dog that tenses, moves, or has to be restrained makes every injection harder and increases the chance of a poor result. A dog that associates the injection with something good makes it easy.

I know of two approaches that work well in practice. The first is to inject while the dog is eating: the distraction of food means most dogs barely register the needle. Many forum owners use this method from day one and find it reliable throughout.

The second approach, and the one I use with Bryce, is to build a specific post-meal ritual. Immediately after he finishes eating, he gets a small treat, I call it jab time, and I give the injection while he is focused on that. It took very little time for him to make the association. Now he comes to me as soon as his bowl is empty, ready for his treat. He initiates it. The injection is almost incidental.

Either method works. What matters is consistency: the same cue, the same sequence, every time. Dogs find routine reassuring, and a dog that knows exactly what is coming is a dog that doesn't resist it.

If twice-daily injections are proving genuinely difficult, for you or for your dog, it's worth knowing there's another option some vets now use: a once-daily basal insulin such as Toujeo or Tresiba, which doesn't need to be given at a fixed mealtime the way Caninsulin does. It isn't right for every dog, and the decision sits with your vet, but if the twice-daily rhythm is the main source of stress in all this, it's a reasonable thing to raise at your next appointment.

One practical advantage of the post-meal treat method: because the dog finishes the meal before the treat appears, you know with certainty how much was eaten before you decide on the dose. If the bowl is empty, full dose. If it isn't, adjust accordingly before reaching for the syringe.

One more thing worth knowing: your dog is reading you as closely as you're reading them. There's good evidence that dogs' stress levels track their owners' over the long term, and that dogs can pick up the actual scent of a stressed person and become more

anxious in response. You don't need to be relaxed every day, that's not realistic, and no one's asking. But for the two moments a day that matter most, the injection and the testing, a calm, unhurried manner genuinely helps your dog as much as it helps you.

The rule you must know before anything goes wrong

If your dog does not eat, give roughly a third of the usual dose, not the full amount and not none at all. If your dog eats half the meal, give half the dose. Below half a meal, the dose doesn't keep dropping in step the same way, food eaten stops being the main thing driving the calculation, so the reduction flattens out rather than continuing straight down toward nothing. The app's insulin calculator works out the exact figure for whatever your dog actually ate, rather than you having to judge it.

This rule, like the timing above, is built around Caninsulin and insulins like it, where the dose and the meal are tied together. It doesn't hold in the same way for a basal insulin such as Toujeo or Tresiba, where skipping a meal doesn't carry the same risk. If you're not certain which applies to your dog, ask your vet how to handle a missed or partial meal for your insulin type.

The dose is always proportional to what was eaten, but it should never drop to zero. A full dose with nothing to offset it can send glucose dangerously low, but withholding insulin completely is not the safe alternative it looks like: the liver keeps releasing glucose into the blood whether or not your dog has eaten, and it is the absence of insulin itself, not just a high reading, that can tip a dog into diabetic ketoacidosis. Chapter 3 covers emergency response in full. Read it today.

A dog that refuses food occasionally is not usually a crisis, provided a reduced dose is still given rather than none. A dog that goes without eating properly for more than twelve hours needs a vet call; reduced appetite can be a sign of something that dose adjustment alone will not fix.

Storing insulin

Unopened Caninsulin should be kept in the fridge at 2 to 8 degrees Celsius. Once opened, it does not need to be returned to the fridge between uses, provided it is stored below 25 degrees Celsius and kept out of direct sunlight, worth using a cool bag in hot weather, since room temperature alone can exceed that in summer in warmer climates. Temperatures above 30 degrees Celsius degrade it significantly faster. Freezing destroys it entirely and a frozen vial must be discarded.

Mark the date of first use on the vial and discard after 42 days, regardless of how much remains. This is the maximum shelf life from opening; the insulin degrades with time whether used or not. At typical doses most vials will be finished well before 42 days, but the rule applies regardless.

Sources for this chapter: AAHA, MSD Animal Health, Merck Animal Health, Canine Diabetes Wiki, ADW Diabetes, VCA, PMC, JAVMA, Zoetis.

Chapter 3: When Things Go Wrong

There are two emergencies a diabetic dog owner needs to recognise, and they're opposites: too much insulin (hypoglycaemia) versus too little (diabetic ketoacidosis, or DKA). They need completely different responses, and confusing them is dangerous. This chapter is built around telling them apart fast, then telling you exactly what to do for each.

Hypoglycaemia: too much insulin, too little glucose

Hypoglycaemia means blood glucose has dropped below roughly 3.3 mmol/L (60 mg/dL). It's the fastest-moving danger in diabetic dog care, onset can go from mild signs to collapse within minutes, which is why recognising it early matters more here than almost anywhere else in this book.

Recognise it

Signs get more serious as glucose keeps falling:

- Early: lethargy, unusual tiredness, seeming uninterested in what's around them.
- Moderate: wobbliness, trembling, muscle twitching, confusion, struggling to walk straight.
- Severe: seizures, collapse, unconsciousness.

Respond, if your dog is conscious

- If they can eat, offer food immediately: their normal meal if it's to hand, or anything they'll eat.
- If they won't eat, or seem too disoriented to, rub honey, corn syrup, or glucose gel onto the gums and inner cheek: about a tablespoon for a medium or large dog, a teaspoon for a small one. These sugars absorb straight through the gum tissue, so your dog doesn't need to swallow. Apply along the gum line rather than pouring anything into the mouth.
- Expect improvement within 5 to 10 minutes. If there's no change within 15 minutes, go to an emergency vet. Don't keep

waiting and hoping.

Respond, if your dog is unconscious

- Never pour liquid into the mouth of an unconscious dog: there's a real risk it goes into the lungs instead of the stomach.
- Rub honey or glucose gel gently onto the gums with a finger. Even unconscious, your dog can absorb it through the gum tissue.
- Call an emergency vet immediately. Don't wait to see if they come round first.

I keep honey in a wide-nozzle syringe in the car as well as the kitchen. It means I can get a precise amount onto the gums quickly, without spilling it everywhere in a moment that's already stressful enough.

After a hypo episode

Once your dog is alert and eating again, offer a small meal rather than a large one, and keep offering small amounts over the following hours to help hold glucose steady. Keep them calm and still: exercise lowers glucose further, the last thing you need right now. Watch closely for several hours in case symptoms return.

Don't give the next scheduled insulin dose without speaking to your vet first. The episode is telling you the current dose may be too high, and repeating it straight away risks a second crisis. Call your vet as soon as you reasonably can to talk through what happened and review the dose.

The emergency kit

Keep glucose sources somewhere you can reach them fast: the kitchen, the car, your travel bag.

- Honey or corn syrup, ready to hand.
- Glucose gel (sold for pets, pre-measured in a squeeze tube) as a standardised alternative to honey.
- Ask your vet about a glucagon injection: prescription only, but useful if your dog is unconscious and gum sugar isn't working.

Not every owner carries one, but it's worth the conversation.
- Urine ketone test strips: these help you tell a hypo from the very different emergency below.

Why hypos happen

- Your dog didn't eat, but got a full or partial dose anyway.
- The dose is too high for the food or activity that day.
- Unplanned exercise on top of the usual dose.
- Stress and reduced eating, such as travel or a change of routine.
- A dose recently increased, before there's been time to see how your dog responds to it.

It happens to careful owners too. Mine did: a motorway journey, a meal that hadn't been fully eaten, a dose already given before I'd realised. It rarely comes down to one thing; it's usually two or three ordinary factors stacking up at once.

Diabetic ketoacidosis (DKA): too little insulin

DKA is the opposite problem. When there isn't enough effective insulin, the body can't use glucose for fuel and starts burning fat instead, which produces acidic by-products called ketones. Enough ketones and the blood itself becomes abnormally acidic, affecting every organ system. Unlike hypoglycaemia, this isn't a sudden crash. It builds over hours to days.

Recognise it

The earliest signs, increased thirst, more urination, reduced appetite, lethargy, can look just like ordinary, poorly controlled diabetes, which is part of what makes DKA easy to miss early. What sets it apart as it progresses:

- Repeated vomiting: a key signal, and not something you'd expect to see with hypoglycaemia.
- Diarrhoea, real lethargy, abdominal discomfort, rapid weight loss, dry or tacky-feeling gums.
- Sweet or fruity-smelling breath, and laboured or rapid breathing: signs the body is critical and trying to correct its

own acid balance.

The clearest way to tell it apart from a hypo: a hypo happens in minutes, in a dog that seemed fine moments before. DKA builds over hours or days, in a dog that's been "off" for a while, and it comes with vomiting and a high glucose reading, not a low one.

What to do

DKA can't be treated at home. It needs IV fluids, electrolyte correction, and carefully managed insulin in a hospital setting. Don't give extra insulin yourself; this isn't simply a dose problem. Go to an emergency vet straight away if your dog has vomited more than once, is refusing all food, is unusually lethargic, has sweet-smelling breath, or is breathing rapidly. Don't wait to see if it settles on its own. Caught early, dogs do well; the danger is in waiting.

Testing for ketones at home

Urine ketone test strips are cheap and available from veterinary suppliers. Worth testing if glucose has been persistently above about 16.6 mmol/L (300 mg/dL) across several readings, if your dog is vomiting or off their food, or if something just feels wrong and you can't pin down why. Ketones should always read negative in a well-controlled dog. A trace result is worth a call to your vet and some encouragement to drink; anything moderate or high means an emergency vet, now.

Telling the two apart, fast

This is the single most useful thing in this chapter, because the two responses are opposites: sugar for a hypo, the vet for DKA.

- Hypo: sudden, minutes not hours; glucose reading low; your dog was fine just before this.
- DKA: gradual, hours to days; glucose reading high; your dog has seemed "off" for a while; vomiting is a strong signal.

If you're genuinely not sure and your dog is conscious, a small amount of honey on the gums won't make DKA worse, and could save a dog who's actually hypoglycaemic. Either way, get to the vet

once you've done it. When in doubt, don't wait to decide; act, then call.

Sources for this chapter: AAHA, Merck Animal Health, VCA, PMC.

Chapter 4: Feeding Your Diabetic Dog

If there is a single word that defines diabetic dog management, it is consistency. Not perfection. Consistency. The same food, the same quantity, at the same times, every day. This matters more than which brand you choose or whether the food is labelled as diabetic-specific. A dog on an ordinary complete food fed with absolute regularity will almost always be better controlled than a dog on a premium diabetic diet fed erratically.

The reason is straightforward. Caninsulin is given twice daily at fixed times. It begins working at a predictable rate and peaks at a predictable point. When food arrives at the same time in the same quantity, the insulin and the glucose from the meal rise and fall in step. When the food changes, in timing, amount, or composition, the glucose response changes too, and the insulin that was calibrated for the previous routine is now either too much or too little.

This does not mean your dog must eat the same food forever. It means that when you change something, you change one thing at a time, do it gradually, and monitor the response. We will return to this throughout the book.

What to look for in a diabetic dog's food

Three things matter in a food for a diabetic dog: fat content, fibre content, and consistency of composition between batches.

Fat matters because diabetic dogs have an elevated risk of pancreatitis, inflammation of the pancreas that can range from uncomfortable to life-threatening, and high dietary fat is a recognised trigger.

A figure you will meet everywhere is a dry matter basis (DMB) fat content of no more than 14%. It is worth being honest about where that comes from, because I could not find it in any guideline. The AAHA diabetes guidelines give no fat percentage for dogs at all, and state that dogs with diabetes can do well on any diet that is complete and balanced, fed at consistent times in consistent amounts, and palatable enough that the dog reliably eats it. The European

endocrinology bodies do not address diet. The 14% figure appears to be a practical working threshold that has been repeated until it acquired the appearance of a rule.

That does not make it useless. Lower fat is a sensible default for a dog at raised pancreatitis risk, and a threshold gives you something to shop against. But treat it as a rule of thumb rather than a clinical requirement, and do not reject an otherwise suitable food purely because it sits above the line. Your vet's view of your own dog outranks any number in this book.

For dogs with a history of pancreatitis or concurrent pancreatic concerns, the figure that does appear in the veterinary nutrition literature is below 12% DMB. I use 10% in the app as a deliberately conservative margin, not because 10% is itself a published threshold.

The fat percentage on a pet food label is not the figure that matters. It's the as-fed figure, which includes the food's water content, and wet food in particular can look far lower in fat than it actually is once that water is accounted for. The companion app to this book has a DMB checker that converts any label's figures into the true dry matter percentage in seconds. Use it before you commit to a food; the maths behind it is covered in Part 2.

One important exception. If your dog is underweight, which many are at diagnosis, fat restriction is the wrong priority. The AAHA guidelines are explicit that underweight diabetic dogs should be fed a good-quality maintenance diet or a diabetic diet that is not designed for weight loss, with the goal of restoring weight and muscle. Getting the weight back on comes first. See 'A note on weight' later in this chapter.

Fibre is often recommended on the basis that it slows the rise in blood glucose after a meal, and there is some evidence for it. Two small crossover studies, of eleven and seven dogs, found improved glycaemic control on high insoluble-fibre diets. A later and larger study of dogs whose diabetes was already stable found no advantage to high-fibre diets at all.

So the position is that fibre may help, particularly early on, and it is unlikely to hurt. It is not established, and it is not worth agonising over. What matters far more than the fibre figure is that your dog

eats the same food, in the same amount, at the same times. If you want to add fibre, plain canned pumpkin or cooked green beans, added to the existing food in a consistent daily amount, is a reasonable way to do it.

One caution if your dog is underweight: fibre is filling, and a high-fibre diet can leave a dog feeling full before they've eaten enough calories to rebuild lost weight. If weight gain is the priority right now, don't chase fibre figures; that can wait until your dog is back to a healthy weight.

Grain-free foods are not automatically better for diabetic dogs. Some replace grains with high-starch alternatives, often potato or pea starch, that spike glucose just as fast, or faster. This is worth taking further than the grain-free label itself: a food's total digestible carbohydrate matters as much as its fat content for glucose control, and a food engineered to hit a low fat percentage has often made up the calories with extra starch to do it. Fat and fibre are not the whole picture. If a food's ingredient list is heavy with starches, potato, pea, tapioca or rice appearing more than once or high on the list, that's worth weighing too, not just the fat number or the grain-free claim. Part 2 covers why digestible carbohydrate matters as much as fat for glucose control.

Reading a pet food label

The information you need is on every label but not always in a useful form. The guaranteed analysis panel gives fat, protein, moisture, and crude fibre as percentages. That's the source data behind the DMB checker in the app.

Avoid foods where sugar, syrup, molasses, or honey appear in the ingredients list. These are added for palatability and serve no nutritional purpose in a diabetic dog's diet.

Watch the order of ingredients too. They're listed by weight, so whatever comes first makes up the largest share of the food. Unnamed "cereals" at the top of the list is worth noticing: it's a cheap bulk filler that digests quickly to glucose. "Meat and animal derivatives" is another one to watch for; it's a legal category name

that tells you nothing about which parts of which animal are actually in the tin, only that some minimum percentage is present. And keep an eye out for the term "various sugars" specifically. It's a legally permitted catch all that confirms sugar is in there somewhere without telling you what kind. Formulations change too, so it's worth checking the actual label on the bag in front of you rather than relying on what you remember about a brand from a year or two ago.

Treats: the honest answer

The honest answer is that treats complicate diabetic management, and the fewer you give the easier control becomes. Every treat introduces glucose that was not accounted for in the insulin calculation. An occasional carrot stick given at the same time every day is a manageable variable. A handful of commercial dog treats given randomly throughout the day is not.

That said, treats serve real purposes: training, reward, routine, and the quality of life that comes from a dog enjoying something. Removing them entirely is not always necessary or kind. The rules are:

- Small amounts only: treats should represent no more than 10% of daily calorie intake, and what counts as small depends heavily on your dog's size, a treat sized for a Chihuahua is a very different thing from one sized for a German Shepherd.
- Always give after a meal, never on an empty stomach.
- Keep the type and quantity consistent day to day so the glucose impact is predictable.
- Choose low-fat, low-sugar options.

The safest treat options for a diabetic dog are plain vegetables: green beans, carrot sticks, broccoli florets, cucumber. These are low in calories and sugar, high in fibre, and most dogs accept them readily. Small pieces of plain cooked chicken or other lean meat are also suitable. If you are using a commercial dog biscuit, as I do with Bryce for the post-injection ritual described in the previous chapter,

choose the smallest, plainest option available, give it consistently, and account for it as part of the daily intake.

What to avoid in treats: anything containing added sugar, honey, or syrup; high-fat chews; rawhide; anything with xylitol, an artificial sweetener found in some foods and treats that is acutely toxic to dogs even in small quantities. Check treat ingredients as carefully as you check food labels.

Foods that must never be given

Some foods are toxic to dogs regardless of diabetes. These include: chocolate and cocoa products; grapes and raisins; onions and garlic in any form, raw, cooked, or powdered; macadamia nuts; and anything containing xylitol. Diabetes does not change this list, but it adds an additional reason to be scrupulous about what the dog has access to. An upset stomach or episode of vomiting in a diabetic dog disrupts the insulin-food balance and can turn a minor incident into a more serious one.

When to feed

Feed twice daily, twelve hours apart, timed to match the insulin injections, if your dog is on Caninsulin or a similar insulin that works for around twelve hours. The sequence is always food first, then insulin, as covered in Chapter 2. The quantity at each meal should be equal. Splitting the daily ration unevenly, a larger breakfast and a smaller dinner for example, means the glucose load at each meal is different, which works against stable control.

If your dog is on a once-daily basal insulin instead, Toujeo or Tresiba are the two in current use, this section works differently, and in some ways more simply. The injection doesn't need to happen at a meal at all. You can feed two meals, three, or let your dog graze, at whatever times suit your household, without the same risk of mistiming insulin against food. Your vet will tell you whether this applies to your dog; if you're not sure which kind of insulin your dog is on, that's worth asking, since it changes a fair amount of the advice in this chapter.

If your dog is a slow eater or a grazer, pick up the bowl once the meal is finished rather than leaving food down. Free-choice feeding is not compatible with twice-daily insulin. You cannot calibrate the dose if you do not know when or how much the dog has eaten.

Do not feed extra food between meals to try to raise a glucose reading that looks low. A genuinely low reading needs to be treated as a possible hypoglycaemia event. Chapter 3 covers this. Adding food outside the normal schedule disrupts the routine without reliably solving the problem.

A note on weight

Uncontrolled diabetes causes weight loss even when the dog is eating well, because without insulin the body cannot use glucose and turns to breaking down fat and muscle instead. Once insulin therapy begins and control improves, weight typically stabilises and may increase.

An underweight diabetic dog needs adequate calories and adequate protein, not a reduced diet. Protein is particularly important because uncontrolled diabetes accelerates muscle loss, and rebuilding that muscle requires sufficient dietary protein. Older dogs are already prone to muscle wasting as a normal feature of ageing; a diabetic older dog needs protein maintained or increased, not restricted. We return to this in Chapter 8.

An overweight diabetic dog benefits from gradual weight reduction towards an ideal body weight, as excess weight increases insulin resistance and makes control harder. Gradual is the key word: rapid weight loss in a diabetic dog complicates glucose management significantly.

Sources for this chapter: AAHA, Purina Institute, Cornell University College of Veterinary Medicine.

Chapter 5: Building Your Dog's Meal Plan

The core principle

Feed for the weight you want your dog to be, not the weight they are right now. This matters most for overweight diabetic dogs, where feeding to current weight simply maintains the excess weight, and the insulin resistance that comes with it.

Portions need to be measured, not estimated. A kitchen scale, not a measuring cup: volume measures are surprisingly imprecise, especially for kibble, and even small variations in portion size show up in blood glucose stability. Split the daily total into two equal meals, 10 to 12 hours apart, timed to the injections as covered in Chapter 4.

How many calories does your dog need

Vets calculate a diabetic dog's calorie needs the same way they would for any dog of the same size and activity level. Diabetes doesn't change the formula, it just raises the stakes on getting portions consistent. The standard veterinary calculation involves body weight and an activity factor, and isn't simple enough to do reliably in your head. That's exactly what the companion app's Daily Calorie Calculator is for. Enter your dog's weight and activity level and it gives you the daily total and the amount per meal.

What if your dog isn't at their ideal weight

Vets use a Body Condition Score (BCS) to judge this: a 9-point scale based on how easily you can feel the ribs, whether there's a visible waist from above, and an upward tuck to the abdomen from the side. Ideal is a 4 or 5 out of 9. Ask your vet to score your dog at your next visit if they haven't already; it takes moments and matters more than the number on the scale.

If your dog is overweight, calorie calculations should be based on their ideal weight, not their current one. Your vet can help you estimate this, and the app's calculator will do the same once you tell

it the target. Weight loss in a diabetic dog should always be gradual; rapid loss complicates glucose management rather than helping it.

If your dog is underweight, common at diagnosis, from the muscle and fat breakdown covered in Chapter 4, the opposite applies. Calories need to support weight restoration, not restriction. Don't cut back because your instinct says a diabetic dog should eat less; an underweight diabetic dog needs to eat enough to rebuild.

Using the app for this chapter

Once you have your dog's daily calorie target, the Meal Builder lets you combine up to three foods and see the total calories alongside fat, protein, and fibre on a dry matter basis, checked against your target. The Food Finder filters foods by type and fat level and suggests grams per meal once your dog's weight is set. Between them, these replace the arithmetic entirely: this chapter gives you the principles, the app does the maths.

Weighing and switching foods

Weigh your dog monthly and adjust portions if their weight is drifting. A diabetic dog gaining or losing weight without a deliberate change to their diet is worth a call to the vet. It can be an early sign that the dose needs reviewing.

If you do need to switch foods, do it gradually over seven to ten days, mixing old and new in increasing proportions. It's worth letting your vet know when you change food too, even just a mention at the next visit rather than a special trip, since a different food can shift how the dose behaves and they'll want that context if anything needs adjusting. This protects against digestive upset, and it also protects against a sudden, unexplained shift in glucose control from a composition change happening all at once.

There's a second reason gradual mixing works, and it matters just as much in practice. Dogs decide whether to eat something almost entirely by smell, not taste, and a food that smells completely unfamiliar can make a dog hesitant or simply refuse it, however good it might be for them. Mixing the new food in gradually lets

your dog get used to the new smell a little at a time, while the familiar smell of the old food still dominates the bowl. Once your dog has actually eaten and enjoyed the mixture a few times, the new smell stops registering as unfamiliar and the resistance usually fades on its own. If your dog is still refusing a new food even mixed in small amounts, don't force the issue or wait them out: go back to a higher proportion of the old food and slow the transition down further.

Sources for this chapter: AAHA, Merck, VCA, Purina Institute.

Chapter 6: Monitoring at Home

Why home monitoring matters

Testing at the vet's is stressful for a dog, and stress raises blood glucose the same way it does in people, so a clinic reading can run higher than what your dog shows day to day. Testing at home, in familiar surroundings, gives you and your vet a truer picture, and lets you build up a full picture of how your dog responds through the day rather than a single snapshot.

Sometimes you'll test just once, as a spot check: confirming your dog isn't dangerously low or unexpectedly high. But your vet will also ask you to run what's called a glucose curve: a full day of readings, often every one to two hours, that maps exactly how your dog's glucose rises and falls in response to a specific meal and insulin dose. That's what tells your vet whether the dose is working, not one number in isolation. Chapter 7 covers how to run one properly. What follows here is what you need to know before you get there.

Choosing a glucometer

Dogs and humans distribute glucose differently between blood plasma and red blood cells: roughly 87.5% in plasma for a dog, against about 58% in a person. A meter calibrated for human blood doesn't know that, and tends to read a dog's glucose lower than it actually is — AAHA 2018 specifically states the Task Force does not recommend human glucometers due to inaccuracies when reading canine blood. That matters most exactly where accuracy matters most: near the low end, where an underestimate could mask a real hypoglycaemia risk.

The AlphaTrak 3 (Zoetis) is the current veterinary-standard meter, purpose-built for dogs and cats, needs only a tiny blood sample, and gives a result in seconds. Other veterinary meters exist too, such as Glucocalea from Wellion, though AlphaTrak remains the most widely used. If your vet has given you an older AlphaTrak 2, be

aware Zoetis has discontinued its test strips. You'll eventually need to move across to the AlphaTrak 3.

If you do end up using a human glucometer, some owners do, usually on cost, tell your vet. They need to know which device produced the numbers they're interpreting, because the bias is real, even if it is predictable.

Getting a blood sample

The standard site is the inner margin of the ear. Warm the ear first, hold it gently between your palms for about a minute, to bring blood to the surface and make the drop easier to get. Shaving a small patch of the inner ear margin, as I do with Bryce, makes the vein easier to find and the lancet easier to place accurately.

Prick with a lancet. A dedicated lancing device gives a controlled, consistent depth, though a standard human lancet works free-hand too. Squeeze gently around the prick site to raise a small drop, then touch the test strip to the drop rather than smearing it on. Rotate where you prick around the ear margin rather than using the same spot every time.

Some dogs tolerate a paw pad better than the ear, specifically the small, non-weight-bearing pad on the side, not the one they walk on. The skin there is thicker, which is part of why most owners find the ear both easier and better tolerated, but it's worth knowing the alternative exists if your dog objects to ear testing.

What the numbers mean

Blood glucose is measured in mmol/L in the UK and most of the world, and in mg/dL in the US. Multiply mmol/L by 18 to convert between them. In a healthy dog the kidneys hold on to glucose. Once blood glucose climbs past roughly 10 to 12 mmol/L (180 to 200 mg/dL), the kidneys can't reabsorb it all, and it spills into the urine and pulls water with it. That's the mechanism behind the excessive drinking and night-time urination from Chapter 1. Keep glucose consistently below that threshold and the symptoms ease.

The goal isn't a human-normal reading. It's a curve that stays in a safe range without dipping dangerously low. As a guide, a well-controlled dog typically runs somewhere between 5.6 and 13.9 mmol/L, 100 to 250 mg/dL, through the day, with the lowest point of the curve ideally landing around 4.4 to 8.3 mmol/L, 80 to 150 mg/dL (AAHA 2018). Below roughly 4.4 mmol/L, 80 mg/dL, is worth a call to your vet even if your dog seems fine, since it often means the dose needs reducing; Chapter 7 covers this in more detail. Below 3.3 mmol/L, 60 mg/dL, is a hypoglycaemia emergency: Chapter 3 covers exactly what to do.

Flash monitors (FreeStyle Libre and similar)

These sensors measure glucose in the fluid under the skin rather than in blood directly, which means there's a short lag, most noticeable when glucose is rising or falling quickly. In dogs, the sensor sits on the back of the neck or scruff rather than the arm, and can usually stay in place for up to two weeks.

Their use is growing quickly, in some practices they're displacing routine glucometer testing almost entirely, and FurCarer is another sensor brand appearing alongside FreeStyle Libre. They're genuinely useful for spotting trends and cutting down how often you need to prick your dog on a curve day. What they're not good at is the low end. Flash monitors are least reliable exactly in the hypoglycaemia range, so any low reading from one needs confirming with a blood test before you act on it. Think of it as a trend tool that sits alongside blood testing, not a replacement for it. We cover CGMs properly in Part 2.

Sources for this chapter: AAHA, Merck Animal Health, Zoetis, PMC.

Chapter 7: The Glucose Curve

What a curve is, and why home beats the clinic

A glucose curve is a series of readings taken through one full stretch between injections, 12 hours for a dog on twice-daily insulin, mapping how glucose rises and falls in response to food and insulin together. Vets used to run these in clinic, but a stressed dog releases cortisol, which pushes glucose up in its own right; a clinic curve can run well above what your dog actually shows at home, sometimes by several mmol/L. Running the curve at home, using the technique from Chapter 6, gives your vet a truer picture.

How to run one

Take a reading every two hours across the full 12-hour interval, starting immediately before the morning meal and injection, finishing immediately before the evening meal. That's seven readings in total. If any reading drops below roughly 8.3 mmol/L, 150 mg/dL, switch to hourly readings from that point on. The nadir may be close, and it needs tracking more closely than every two hours allows.

Your dog needs to eat normally, same food, same amount, same time, for the curve to mean anything. If they don't eat as usual that morning, stop and try again another day. A curve run around an off day isn't wrong, it's just not telling you anything real.

The nadir, and the mistake almost every owner makes

The nadir is the true lowest point on the curve, and on Caninsulin it typically lands four to eight hours after the injection, not at the twelve-hour, pre-meal mark. The pre-meal reading is simply wherever glucose happens to be by the end of the interval, which may already be climbing back up well after the nadir has passed. Treating the pre-meal number as if it were the nadir is the single most common mistake in reading a curve, and it can lead you or your vet to the wrong conclusion about whether the dose is working.

What a good curve looks like

Glucose starts elevated before the morning dose, falls to a nadir somewhere in the 4.4 to 8.3 mmol/L, 80 to 150 mg/dL (AAHA 2018), range during that four-to-eight-hour window, then climbs back up toward the evening reading. Most readings through the day sitting somewhere in the 5.6 to 13.9 mmol/L, 100 to 250 mg/dL, range from Chapter 6 is the general target, not a human-normal number, just safely clear of both danger zones. A nadir below roughly 4.4 mmol/L, 80 mg/dL, means the dose is likely too high, even if your dog seems perfectly well. That's a call to the vet, not a wait-and-see. A nadir below 3.3 mmol/L, 60 mg/dL, is a hypoglycaemia emergency: treat it immediately using the steps in Chapter 3, then call the vet.

Other shapes, and what they mean

Flat and high all the way through, with no real dip: usually points to a dose that isn't yet high enough. But read the next section before assuming that's the answer. Nadir too low: the dose is too high, and the risk is hypoglycaemia; your vet needs to reduce it. Falls to nadir quickly, then climbs steeply back up well before the twelve-hour mark: the insulin isn't lasting long enough for this dog, which is worth raising with your vet as a timing or insulin-type question rather than a dose one.

Somogyi rebound, and why a high curve is not always undertreatment

Sometimes a dose that's actually too high produces a curve that looks exactly like a dose that's too low, flat and high, with no visible dip. What's really happened is the insulin pushed glucose down sharply enough, between your two-hourly readings, to trigger the body's own emergency response, and that response floods glucose back up hard enough to erase any trace of the low by the time you test again. Read only the numbers on the page and it looks like more

insulin is needed. Give more, and you make a genuinely dangerous situation worse.

The rule that matters: never assume a flat, high curve simply means the dose needs increasing. That decision belongs to your vet, with the full curve in front of them, not something to act on at home.

How often to run one

After any dose change, run a curve five to fourteen days later, giving your dog time to settle on the new dose before you judge it. Once your dog is stable, roughly every three months is standard, or sooner if any of the original symptoms, the thirst, the accidents, the weight loss, come back. Run one immediately if you suspect a hypoglycaemic episode.

Fructosamine, your vet's other tool

Your vet may also run a blood test called fructosamine alongside curves. It reflects average glucose over the previous two to three weeks rather than a single day, so it isn't thrown off by one bad night or a stressful car journey, but it can't show you the shape of a curve, so a dangerous low followed by a Somogyi rebound could still sit behind a perfectly normal-looking result. It's a complement to curves, not a substitute for them. We cover it properly in Part 2.

What to bring to the vet

A log of every reading with date, time, and value; what your dog ate and when; the dose given; and anything unusual that day, vomiting, exercise, heat, stress. A plotted graph is far quicker for a vet to read at a glance than a column of numbers. The app logs all of this as you go, date, time, value, and notes, so you can hand your vet a phone instead of a notebook.

Sources for this chapter: AAHA, Merck Animal Health, MSD Animal Health, VCA.

Chapter 8: The Long Game

The first ninety days are about getting your feet under you. Once you're there, the questions change, not "how do I manage this crisis" but "what does the rest of my dog's life actually look like." This chapter is the honest answer.

The long-term prognosis

The mortality risk in a newly diagnosed diabetic dog is highest in the first six months. That's when concurrent illness, DKA, or the sheer difficulty of the learning curve claims the dogs that don't make it. If your dog gets through that period stable and well controlled, the outlook improves substantially. Well-controlled diabetic dogs with no major concurrent illness routinely live three to five years past diagnosis, and plenty go on for eight to ten, particularly if they were diagnosed relatively young.

The important point, worth holding onto: diabetes itself, properly managed, doesn't shorten a dog's life. What determines the outcome is complications and concurrent illness, which is exactly why the rest of this chapter exists.

Remission, managing expectations

If you've read anything about diabetic cats, you may have come across remission: cats can sometimes come off insulin entirely, in as many as 90% of cases with an early, aggressive approach. That's not what happens in dogs, and it's worth knowing now so it doesn't feel like a failure later. Almost all diabetic dogs have what's effectively Type 1 diabetes. The insulin-producing cells have been destroyed and aren't coming back. Insulin is for life.

There's one genuine exception: diabetes triggered by the hormonal swings of a season in an unspayed female can sometimes resolve after spaying. It's part of why an intact female is routinely spayed at diagnosis.

Cataracts

Somewhere around three-quarters of diabetic dogs develop cataracts within about a year of diagnosis, and it happens regardless of how well you control glucose. Good control slows it, it doesn't prevent it. It can move fast: a dog can lose meaningful vision within days to weeks of the first signs. If it happens to your dog, it isn't a sign you did something wrong.

Surgery, removing the cloudy lens, the same procedure used in humans, has a success rate above 90% in an otherwise healthy dog, with most maintaining good vision for years afterwards, though it does mean a referral to a veterinary ophthalmologist and a real cost, typically a couple of thousand pounds per eye in the UK. If you have pet insurance in place before diagnosis, it's worth checking whether it's covered.

If surgery isn't the right choice for your dog, know that dogs adapt to blindness remarkably well. They navigate by smell and memory, and quality of life stays good as long as the eyes themselves are comfortable. An eye that's become painful, rather than just cloudy, is the thing that needs addressing, regardless of whether sight can be restored.

Pancreatitis

Diabetic dogs carry a higher risk of pancreatitis, and the relationship runs in both directions: pancreatitis can be severe enough to cause diabetes in the first place, and diabetes raises the risk of pancreatitis developing. Watch for vomiting, loss of appetite, abdominal pain, sometimes visible as a "prayer position" with the front end down and the rear end up, and lethargy. Any of these in a diabetic dog is worth a prompt call to the vet, since it can also signal early DKA, and either way it will destabilise glucose control fast.

The low-fat feeding principle from Chapter 4 isn't just about glucose stability. It's your main tool for keeping pancreatitis risk down too.

Urinary tract infections

UTIs are considerably more common in diabetic dogs than in healthy ones. Glucose in the urine feeds bacteria, and high blood glucose blunts the immune cells that would normally clear an infection. The frustrating part is that a diabetic dog can have a UTI with no obvious symptoms at all, and it can still knock control sideways: the inflammation raises stress hormones, those hormones cause insulin resistance, and a dose that was working stops working, for reasons that aren't obvious from the outside.

This is one of the more common explanations for a previously stable dog suddenly not being stable, and the effect can linger for a week or two even after antibiotics finish. It's why a proper urine culture at routine check-ups, not just a dipstick, is worth asking for, even when nothing seems wrong.

Muscle loss in an older diabetic dog

Age-related muscle loss starts in most dogs from around seven or eight onwards. Bryce is fourteen, well into that territory regardless of his diabetes. Diabetes adds to it on top: uncontrolled glucose pushes the body to break down muscle for fuel, and the low-grade inflammation of chronic illness gets in the way of rebuilding it. It can look a lot like arthritis, a reluctance to jump, trouble with stairs, general weakness, and it's easy to misread as joint pain rather than muscle.

The fix is mostly the same as in Chapter 4: enough good-quality protein, not less of it, plus gentle, consistent exercise rather than rest. Good glucose control is itself the best long-term protection here. A stable dog simply isn't breaking its own muscle down for fuel.

Quality of life, what good actually looks like

In one study using a quality-of-life tool built specifically for diabetic dogs, 81% of owners rated their dog's overall quality of life as good. But when asked more specific, diabetes-related questions, 84% of those same owners described some negative impact somewhere.

Both things are true at once, and it's worth knowing that going in: the headline answer and the day-to-day reality aren't quite the same question.

What good actually looks like, in practice: interest in food, engagement with what's going on around them, tail wagging, willingness to play or go for a walk, no obvious pain, normal sleep. A simple scale worth knowing is HHHHHMM: Hurt, Hunger, Hydration, Hygiene, Happiness, Mobility, and more good days than bad, scored out of ten each; above 35 out of 70 generally means a good quality of life. The app has a version of this you can run regularly, which is more useful than trying to judge it from memory on a bad day.

The honest message, and the one worth ending on: a well-controlled diabetic dog is not a dog just getting by. Most owners, once the routine settles, describe their dogs as thriving. The injections become normal. The whole thing becomes normal. That was true in Chapter 1, and it's still true here.

Staying stable, long term

Once your dog is settled, a glucose curve every three months, or sooner if anything changes, and a vet check-up every three to six months covering weight, body condition, a urine culture, and fructosamine, is the routine that keeps things that way.

Watch for the symptoms from Chapter 1 coming back, thirst, accidents, weight loss, appetite changes, vomiting, and treat any of them as a reason to call the vet, not a reason to wait and see. And if your dog picks up any illness, even something minor, it's worth testing glucose a little more often than usual until it's passed; almost anything can knock control sideways temporarily.

You matter in this too

There's a name for what you might be feeling some days, even once the routine is running well: caregiver burden. Researchers who study it in owners of chronically ill dogs and cats have found real, measurable increases in stress, anxiety, and low mood, not because

you're not coping, but because sustained caregiving is genuinely hard, whatever species is on the receiving end of it. Roughly half of owners caring for a pet with a chronic illness show meaningful signs of it.

Two things are worth knowing. First, feeling this way doesn't mean you're managing your dog's diabetes badly. The research found it in owners doing everything right. Second, the same research found that owners who felt confident in their vet and their treatment plan carried less of this burden. That's part of why this book exists: understanding what's happening and why gives you back some of the control that caregiving can quietly take away.

If you're struggling, that's worth taking seriously in its own right, not just as a means to managing your dog better. Talk to your vet, talk to people who understand, the forums exist for a reason, and talk to your own doctor if it's become more than tiredness. Looking after yourself isn't a distraction from looking after your dog. It's part of the same job.

Sources for this chapter: PubMed / JAVMA, Cornell University College of Veterinary Medicine, Merck Veterinary Manual, Purina Institute, dvm360, PetMD, Royal Canin Academy, Kent State University (Spitznagel et al.), Veterinary Record.

PART TWO: THE RESEARCH

Chapter 1: The Physiology of Insulin

This chapter goes underneath Part 1, Chapters 1 and 2, to explain in full what is actually happening inside your dog's cells when insulin does its work, and what goes wrong when it cannot. None of this is necessary for day to day management; Part 1 covers everything needed for that. It is here for anyone who wants to understand the mechanism behind the routine, not just follow it.

What insulin actually does: the cellular mechanism

Insulin's central job is to let cells take up glucose from the bloodstream. The mechanism centres on a protein called GLUT4, short for Glucose Transporter Type 4.

In a healthy dog, GLUT4 proteins are manufactured by muscle and fat cells, but they are not left sitting on the cell surface waiting for glucose to arrive. Instead they are held in reserve inside the cell, packed into specialised compartments called GLUT4 storage vesicles. When blood glucose rises after a meal, the pancreas secretes insulin. Insulin binds to receptors on the cell surface, and that binding triggers a cascade of phosphorylation signals inside the cell, essentially a chain of chemical switches flipping on in sequence. This cascade causes the storage vesicles to migrate to the cell membrane, fuse with it, and deliver GLUT4 proteins to the surface. Once there, GLUT4 acts as a channel, allowing glucose to move from the bloodstream into the cell by facilitated diffusion. No energy is spent by the cell to make this happen; the glucose simply follows its own concentration gradient once the channel is open. Inside the cell, the glucose is then used for energy or stored as glycogen for later use.

Without insulin, none of this happens. The GLUT4 proteins remain locked inside the cell, and the cascade that would normally bring them to the surface never fires. Glucose accumulates in the bloodstream because the cells cannot take it in, even though it is abundant all around them. This is the central paradox of diabetes: the body is simultaneously awash in glucose and starving for it. Blood glucose climbs, cells go without fuel, and the body begins

breaking down fat and muscle for energy instead, since it cannot use the glucose that is actually there. Once blood glucose exceeds the renal threshold, approximately 10 to 12 mmol/L, 180 to 200 mg/dL, in dogs, the kidneys can no longer reabsorb all of it, and the excess spills into the urine. This draws water with it, which is the direct cause of the excessive urination and thirst covered in Part 1, Chapter 1.

Why dogs are almost always Type 1

In human medicine, Type 2 diabetes, in which cells become resistant to insulin but the pancreas still produces some, accounts for around 90% of cases. In dogs the picture is almost the reverse. The overwhelming majority of diabetic dogs have a disease that closely resembles human Type 1 diabetes: their pancreatic beta cells, the cells that manufacture insulin, have been destroyed, leaving an absolute deficiency rather than a resistance problem.

The clearest evidence for this comes from a 2015 study that examined the pancreata of diabetic and non-diabetic dogs directly. It found a near total absence of beta cells even in dogs that had only recently been diagnosed, not dogs years into the disease. This matters because healthy dog pancreata are predominantly composed of beta cells; their destruction in diabetic dogs is close to complete from the outset, and the pattern closely mirrors what is seen in human Type 1 diabetes. It strongly implies the same underlying process: loss of the cells themselves, not resistance to what they produce.

This also explains something many owners struggle with emotionally. Clinical signs typically do not appear until around 90% of beta cell function has already been lost. By the time the classic symptoms show up, the disease has usually been progressing quietly for some time, which is why many dogs are quite unwell by the time they are diagnosed. It was not something that could easily have been caught sooner.

How beta cells are destroyed

The exact route to beta cell destruction is not always identifiable in an individual dog, and several different mechanisms appear to be involved across the population.

Immune-mediated destruction is one route. Evidence of cell-mediated autoimmune attack on beta cells has been found in as many as half of diabetic dogs in some studies, while other studies have found no autoimmune markers at all. The picture is genuinely mixed. Immune-mediated insulitis appears to be a contributing factor in some dogs, but not all.

Pancreatitis-related damage is a second route, and a significant one. Up to 30% of diabetic dogs show evidence of concurrent pancreatitis at post-mortem examination. The relationship between the two conditions is complex. Pancreatitis and diabetes appear to predispose each other, and the direction of cause and effect is not always clear in an individual case. Selective loss of beta cells occurs in dogs with both conditions, which suggests the damage is not simply incidental to inflammation elsewhere in the pancreas but something more targeted. Chronic pancreatitis can progressively impair both the endocrine function of the pancreas, which produces insulin, and its exocrine function, which produces digestive enzymes. In the Miniature Schnauzer, a breed with an elevated risk of diabetes, that risk appears to be mediated largely through the breed's separate predisposition to pancreatitis.

A third mechanism, vacuolar degeneration of beta cells, has also been described in dogs, though it is less studied than the immune-mediated or pancreatitis-linked routes.

Why dogs do not go into remission, and the one exception

The rule for dogs is straightforward and worth stating plainly. Dogs with diabetes require insulin for the rest of their lives. There is no equivalent, in dogs, of the tight-control-and-remission pathway that exists for some diabetic cats.

The reason lies in the difference between the two diseases. Most diabetic cats have a condition closer to human Type 2 diabetes. Their

beta cells are still present, but suppressed or impaired by insulin resistance and by the toxic effect of prolonged high glucose on the pancreas itself, sometimes compounded by amyloid deposits within the pancreatic islets. If that glucose toxicity is reversed early enough, through insulin therapy and careful dietary management, some cats' beta cells recover enough function to sustain normal glucose levels without further insulin. Remission rates in cats given aggressive early treatment have been reported as high as 50 to 70% in some studies.

None of this applies to dogs, because a dog's beta cells are destroyed rather than suppressed. There is nothing left to recover. Once they are gone, they are gone.

There is, however, one genuine exception, and it is worth knowing in detail because it can be life changing information for the right dog. Unspayed female dogs can develop a form of diabetes that is not driven by beta cell destruction at all, but by progesterone induced insulin resistance during dioestrus, the roughly two month period of sustained high progesterone that follows each heat. Progesterone inhibits insulin from binding properly to its receptors and blocks part of the glucose transport pathway described earlier in this chapter. It also stimulates the mammary gland to produce growth hormone, which has its own anti-insulin effect throughout the body. The combined result can be persistent insulin resistance severe enough to progress into overt diabetes.

Spaying removes the source of that progesterone. In an intact female dog whose diabetes is driven by dioestrus in this way, spaying can dramatically improve, or in some cases fully resolve, the condition. This is not beta cell recovery. It is the removal of the hormone that was working against the insulin the dog still has. It is the closest thing to true remission that occurs in dogs, and it applies only to intact females with this specific, hormonally driven form of the disease. Because this distinction is not widely known among owners, it is worth stating clearly: any intact female dog newly diagnosed with diabetes warrants an urgent conversation with the vet about spaying, since the outcome for her may be very different from a typical case.

Confirming it in practice

No single test proves in advance that a case will reverse, but several tools together build a clear picture. A serum progesterone test is the key first step: a raised level confirms the dog is in dioestrus, the phase during which this form of diabetes develops, and vaginal cytology can support the same diagnosis by showing the cell pattern typical of that stage of the cycle. Growth hormone itself is difficult to measure directly, since assays for it are not widely available, so vets instead measure IGF-1, a more stable marker that rises when progesterone is driving growth hormone excess. In a small number of documented cases, dogs with this form of diabetes have also shown physical signs of the growth hormone excess itself, including enlarged paws, a widened space between the teeth, and thickened skin, a picture sometimes described as dioestrus induced acromegaly. A vet will also want to rule out pyometra, an infection of the womb that is common in intact females, causes its own insulin resistance, and is a medical emergency in its own right regardless of its effect on diabetes.

Even with all of this evidence, the closest thing to a definitive test is often the outcome of spaying itself. In the largest retrospective study of this question, 57 diabetic bitches with some form of ovarian related condition were spayed, and 6 went into full remission, close to 1 in 10. The underlying cause varied within that group, including dioestrus, pregnancy, an ovarian remnant left behind after an earlier incomplete spay, and pyometra, so this figure describes the wider population of reproductively driven diabetes rather than dioestrus alone. Remission has been reported even in dogs spayed many weeks after diagnosis, and in dogs who first presented in a severe crisis. What consistently matters most is timing: the longer a dog is left undiagnosed or unspayed, the more likely the beta cells are to become permanently exhausted from prolonged high glucose, turning a case that might have reversed into one that cannot. Where remission does happen, it tends to happen quickly, with a median of around ten days after surgery and a documented range of one to fifty one days. One recent review put it plainly: spaying in these

cases should not be treated as an elective procedure, but as an urgency.

Which dogs are at higher risk

Sex plays a measurable role on its own. Intact females carry a significantly higher risk than males or spayed females, for the progesterone related reasons described above, and the overall sex ratio among diabetic dogs skews female in most studies.

Breed also plays a role, and the differences in relative risk are substantial. The following breeds show elevated risk compared with mixed breed dogs, based on large scale insurance and hospital data:

- Australian Terrier: around 32 times the risk of a mixed breed dog, the highest relative risk recorded in the major studies.
- Samoyed: around 12 times the risk, with an inherited predisposition strongly suspected given insulin dependent diabetes in closely related breeds.
- Miniature Schnauzer: elevated risk, most likely mediated through the breed's separate predisposition to pancreatitis, as described earlier in this chapter.
- Toy and Miniature Poodle: elevated risk, consistent across multiple independent studies.
- Bichon Frise: elevated risk.
- Keeshond: elevated risk.
- Swedish Elkhound: very high risk in Scandinavian data, with dioestrus associated diabetes particularly well documented in the breed.

Age matters as much as breed. Most dogs are diagnosed in middle to older age, typically between seven and ten years old. For dogs outside the higher risk breeds listed above, a dog's own age and individual biology, rather than any particular genetic predisposition, explains the great majority of cases.

Why the starting dose is always cautious

Part 1, Chapter 2 introduced this principle in practical terms. This section explains the reasoning behind it in full.

Every dog's sensitivity to insulin is different, and there is no way to know in advance exactly where an individual dog will sit on that spectrum. The dose that stabilises one dog could cause life threatening hypoglycaemia in another of similar size and breed. The 2018 AAHA guidelines recommend a starting dose of 0.25 IU per kilogram every twelve hours, rounded to the nearest whole unit, with a typical stabilisation dose around 0.5 IU per kilogram and an overall range across the diabetic dog population of roughly 0.2 to 1.0 IU per kilogram. This is worth contrasting with the Vetsulin and Caninsulin product label itself, which specifies 0.5 IU per kilogram given once daily as the starting dose. AAHA's guidance is more conservative on both counts, a lower starting dose, and split across two daily injections rather than one, reflecting a preference for a slower, safer path to initial stabilisation.

The reasoning behind that caution is a matter of asymmetric risk. Undertreatment leaves a dog with persistent high blood glucose, which is uncomfortable and, over time, damaging, but is survivable in the short term while the dose is corrected. Overtreatment causes hypoglycaemia, which can be fatal within hours. Part 1, Chapter 3 covers emergency response to this in full; Chapter 2 of this Part covers the related phenomenon of Somogyi rebound, in which a dose that is already too high produces a curve that looks, misleadingly, like a dose that is too low. Given that asymmetry, it is always safer to start low and build up gradually than to start high and risk overshooting. Once a baseline curve has been established, dose adjustments are made incrementally, typically in steps of 0.5 to 1 IU, and never on the strength of a single reading.

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Chapter 2: Insulin Pharmacokinetics, and the Two Opposite Emergencies

This chapter goes underneath Part 1, Chapters 2, 3, and 7. It explains why Caninsulin behaves the way it does across a twelve hour cycle, what the other insulins offer and why they are used less often, and the full clinical detail behind the two emergencies at opposite ends of the insulin axis: hypoglycaemia, where there is too much effective insulin, and diabetic ketoacidosis, where there is too little.

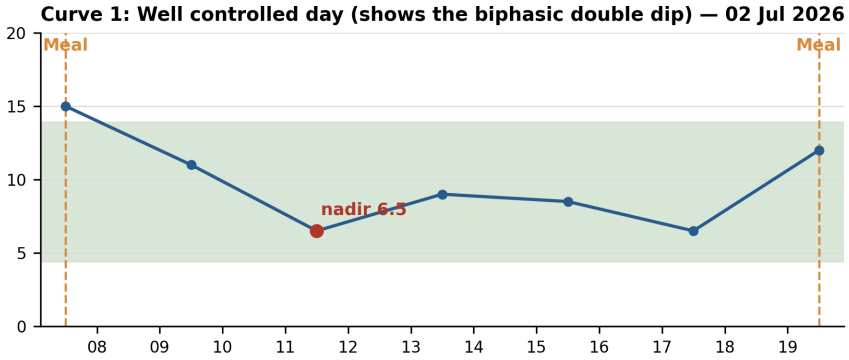
How Caninsulin actually works: the biphasic profile

Caninsulin, sold as Vetsulin in the United States and Canada, is a 40 IU per millilitre suspension of highly purified porcine insulin. What makes it behave differently from the human analogue insulins sometimes used in dogs is its composition. It is not a single, uniform preparation, but a fixed mixture of two distinct particle types, each dissolving and acting at a different rate.

Roughly 35% of the mixture is an amorphous fraction, made of small particles that dissolve quickly. This fraction reaches its peak effect at around 4 hours after injection, and its influence lasts for roughly 8 hours. The remaining 65% is a crystalline fraction, made of larger, slower dissolving particles, with a later, broader peak at around 11 hours after injection. Together these two fractions give Caninsulin a biphasic action profile. It begins working within the first hour, produces an early peak around 4 hours in, a second, broader peak around 11 hours in, and continues acting for somewhere between 14 and 24 hours in total.

This is the reason Caninsulin is given twice daily. The combined duration of the two fractions provides overlapping coverage across the full day, but the two peaks are real, and they matter diagnostically. The early, 4 hour peak is when the risk of hypoglycaemia is highest on any given dose, and it usually corresponds to the nadir seen on a glucose curve, as covered in Part 1, Chapter 7. The later, 11 hour peak can produce a second, smaller

dip in some dogs' evening readings, which can look puzzling on a curve until this second peak is understood.



Curve 1: a well controlled day, showing the biphasic double dip.

The other insulins: where they sit and why they matter

Part 1, Chapter 2 introduced the other insulins a vet might prescribe. This section sets out the pharmacokinetic detail behind each.

- NPH, sold as Humulin N or Insulatard: a human intermediate acting insulin. Onset around 1 hour, a single peak at 4 to 6 hours, and a duration of around 12 hours. Some vets use it in dogs, typically given twice daily, though its shorter duration compared with Caninsulin can leave gaps in coverage.
- Detemir, sold as Levemir: a human long acting analogue. Onset is slower and more variable, between 2 and 11 hours, with a broad peak around 8 to 10 hours and a duration that can reach 24 hours. Detemir is roughly four times more potent per unit in dogs than Caninsulin or NPH, which is a critical practical point. A vet switching a dog from Caninsulin to detemir must account for this difference directly, since the same number of units would deliver roughly four times the effective dose. For this reason detemir is generally reserved as a rescue option for refractory cases that have not responded well to first line treatment, not used as a starting insulin.
- Glargine is sold under one name but as two genuinely different products, and this book previously ran them together, which needed correcting.

Glargine U-100, sold as Lantus, is the original formulation, widely used in human Type 1 diabetes and in diabetic cats. In dogs its onset and profile are much less predictable than in cats, and one early study found two of nine dogs treated with it showed no meaningful glucose-lowering effect at all (Fleeman and Rand, 2009). Because of this, the AAHA guidelines do not recommend U-100 glargine in dogs unless every other option has failed.

Glargine U-300, sold as Toujeo, is a different, more concentrated formulation: low in potency and slow enough in onset to act as a genuine basal insulin, working steadily in the background rather than peaking around a particular meal. A 2024 multi-centre trial of 95 dogs found good or excellent glycaemic control in 92% of them on once-daily Toujeo (Tardo et al., 2024). Its most significant practical difference from Caninsulin or Lantus is that it uncouples the injection from feeding: meals can be given at flexible times, in variable amounts, or occasionally skipped, without the same hypoglycaemia risk that applies to twice-daily insulins timed to meals. This is a genuinely different management style, not just a different product. Some vets prescribe it once daily, others split it into two smaller doses for steadier coverage in a particular dog; either way, the uncoupling from feeding holds, since it comes from the insulin's flat, prolonged release, not from how many times a day it's given. If your vet has prescribed it, most of the rigid feeding-timing advice elsewhere in this book, particularly in Part 1, doesn't apply to you in the same way; ask your vet what does.

Degludec, sold as Tresiba, is the other basal option now in occasional use in dogs, sharing the same uncoupling of injection from feeding as Toujeo, whether given once or twice daily, though with less canine-specific evidence published to date.

- ProZinc: a recombinant protamine zinc insulin developed primarily for cats, with a slow onset and an extended, prolonged action. It is occasionally tried as a once daily option in dogs, though it is not a first choice.

Somogyi rebound: the overdose that looks like underdose

This is one of the most important, and most misunderstood, concepts in canine diabetes management, and it deserves to be understood in full, because acting on the wrong interpretation of it can make a dangerous situation considerably worse.

When insulin drives blood glucose down too rapidly, or into a genuinely low range, the brain detects the falling glucose as a threat and triggers a cascade of counter regulatory hormones. Adrenaline is released first, followed by cortisol, glucagon, and growth hormone. Glucagon and adrenaline stimulate the liver to release its stored glucose. Cortisol and growth hormone promote the manufacture of new glucose from other sources. Together, these hormones also make the body's cells temporarily more resistant to insulin. The combined effect is a sharp rebound rise in blood glucose, sometimes climbing very high, and sometimes persisting for days.

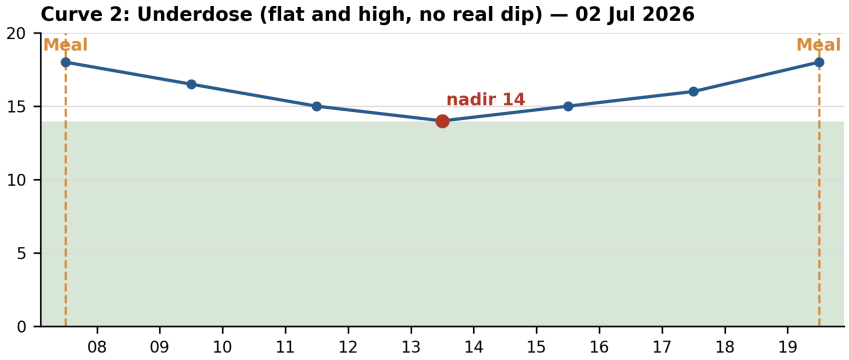
The clinical picture this produces is genuinely confusing if you do not know what you are looking at. Glucose does not need to fall all the way to visibly dangerous levels to trigger this response. In dogs, the counter regulatory cascade can be triggered once glucose drops below roughly 3.3 mmol/L (60 mg/dL) (AAHA 2018), a threshold that can be reached and recovered from entirely between two scheduled readings, meaning the owner may never see a genuinely low number on the meter at all. What they see instead is morning readings that are high, a curve that stays persistently elevated through the day, and a dog that may seem hungry, restless, or lethargic. Taken at face value, this looks exactly like a dose that is not high enough.

This is the dangerous moment, and the one a vet must actively guard against. The natural response to a curve like this is to raise the dose further. Doing so makes the underlying hypoglycaemic dip deeper, triggers an even stronger counter regulatory response, and produces even higher rebound readings on the next curve, reinforcing the mistaken impression that more insulin is still needed. The correct response, once Somogyi rebound is suspected, is the opposite: reduce the dose.

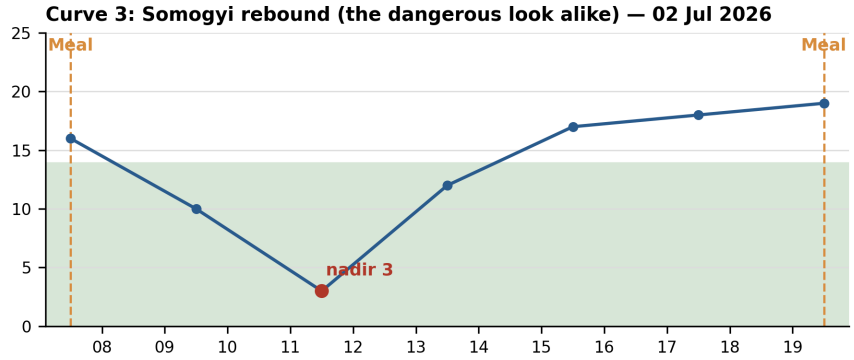
The rebound itself is not brief. This has a real practical consequence. Raising the dose, seeing persistently high readings over the following days, and raising the dose again in response compounds the problem substantially before a proper curve is ever run.

Distinguishing Somogyi rebound from straightforward undertreatment requires a full twelve hour curve, with readings at least every two hours, and ideally hourly readings if glucose approaches the lower ranges. Somogyi rebound shows a dip to low or low normal values at some point in the curve, followed by a rebound rise. Straightforward undertreatment, by contrast, shows glucose that stays high throughout, with only a modest, appropriately timed fall and rise around the insulin's peak, and no dip below safe levels at any point.

It is worth noting that canine evidence for the full Somogyi phenomenon dates mainly to a 1982 study by Nelson and colleagues, and no modern continuous-glucose-monitoring study in dogs has been identified. In cats, AAHA 2018 notes (citing Roomp and Rand, 2016) that hypoglycaemia does not always trigger a rebound response, and that insulin resistance does not always follow; the same may be true in dogs.



Curve 2: underdose, flat and high with no real dip.



Curve 3: a high, flat curve — possible causes include underdosing, Somogyi rebound, or stress hyperglycaemia. Discuss with your vet before changing the dose.

Hypoglycaemia and DKA: opposite poles of the same axis

Somogyi rebound sits at one end of a spectrum. At the far opposite end sits diabetic ketoacidosis, known as DKA. The two conditions could not be more different in what they need from you, and confusing them is dangerous, so it is worth setting them out one after the other, with the contrast between them made deliberately explicit.

Hypoglycaemia: too much effective insulin

Part 1, Chapter 3 covers what to do if you suspect hypoglycaemia. This section covers the clinical thresholds behind that advice in full.

Hypoglycaemia in dogs is defined diagnostically as blood glucose below 3.3 mmol/L, or 60 mg/dL. Clinical signs, however, typically do not appear immediately at that threshold. Below 3.3 mmol/L, 60 mg/dL, a dog may still appear entirely normal. Once glucose falls further, into the range of roughly 2.2 to 2.8 mmol/L, 40 to 50 mg/dL, clinical signs typically begin: a change in behaviour, restlessness or unusual lethargy, hunger, trembling, muscle twitching, and unsteadiness on the feet. Below roughly 2.0 mmol/L, 36 mg/dL, hypoglycaemia becomes severe, and can produce seizures, collapse, stupor, and loss of consciousness.

One important nuance is worth knowing. A dog whose glucose has run chronically low over time can adapt somewhat, showing fewer or milder signs at a given reading than a dog experiencing that

level for the first time. This means clinical signs alone can understate how serious a given reading actually is.

The emergency response described in Part 1 rests on straightforward physiology. Rubbing glucose, in the form of honey or glucose gel, onto the gums works because the mucous membrane of the mouth absorbs glucose directly into the bloodstream, without requiring the dog to swallow. This matters most in exactly the situation where it is needed most: when a dog cannot coordinate normally, or is semi conscious, and cannot be relied upon to eat or drink in the usual way.

Diabetic ketoacidosis: too little effective insulin

DKA sits at the opposite end of the same axis. It develops not from too much insulin, but from a shortage of effective insulin, whether because a dog's diabetes has not yet been diagnosed, because the current dose is too low, or because a concurrent illness has caused severe insulin resistance that the existing dose can no longer overcome.

The underlying biochemistry follows directly from the mechanism described in Chapter 1 of this Part. Without sufficient insulin, cells cannot take up and use glucose for energy, so the body shifts to breaking down fat stores instead. The liver converts the resulting fatty acids into ketone bodies, principally acetoacetate, beta hydroxybutyrate, and acetone. In small quantities, ketones can serve as a genuine alternative fuel source. In DKA, production runs out of control, and ketones accumulate faster than the body can use or excrete them. Ketones are acidic, and once the body's bicarbonate buffering system is overwhelmed, the result is metabolic acidosis, a fall in blood pH that disrupts enzyme function, electrolyte balance, and blood flow to the organs.

The clinical picture progresses in stages. Early on, the signs are increased thirst, increased urination, reduced appetite, and lethargy, indistinguishable from ordinary poorly controlled diabetes. As it progresses, vomiting, dehydration, weakness, and abdominal pain appear. In advanced DKA, breathing becomes rapid and laboured, a

pattern called Kussmaul respiration, in which the body is attempting to blow off carbon dioxide to compensate for the acid load in the blood. The breath itself may take on a sweet or acetone smell, similar to nail polish remover, and the dog may collapse.

DKA is more common at diagnosis than many owners realise. In the largest published outcome study of canine DKA, 65% of cases were the first presentation of diabetes in that dog altogether, meaning the dog was not previously known to be diabetic at all when it went into DKA. DKA is often the event that reveals the diagnosis, not a complication that develops afterwards in a dog already being managed.

Concurrent illness is common alongside DKA. In the same study, 69% of dogs with DKA had one or more other conditions present at the same time: acute pancreatitis in 41%, a urinary tract infection in 20%, and hyperadrenocorticism, also known as Cushing's disease, in 15%. Each of these conditions can independently cause enough insulin resistance to tip an otherwise reasonably managed dog into DKA.

DKA is a genuine medical emergency, and it is fatal without treatment. In that same study, of 121 dogs who were treated, 89, or 70%, survived to be discharged from hospital, with a median hospital stay of six days. The dogs with the poorest outlook were those with severe anaemia, low ionised calcium, low blood pH, and a large base deficit on bloodwork. Treatment requires intravenous fluids, insulin, correction of electrolyte imbalances, and treatment of any concurrent illness. It cannot be managed at home.

Telling them apart

The two conditions sit at opposite ends of the same axis, and the practical distinction is worth holding in mind as a single, memorable contrast.

- Insulin: too much in hypoglycaemia, too little in DKA.
- Blood glucose: low in hypoglycaemia, high in DKA.
- Ketones: absent in hypoglycaemia, present in DKA, often above 3 mmol/L, roughly 31 mg/dL, in urine or blood.

- Breath: normal in hypoglycaemia, often sweet or acetone smelling in DKA.
- Immediate response: glucose by mouth or on the gums for hypoglycaemia; intravenous fluids and insulin in hospital for DKA.
- Speed of onset: minutes to hours for hypoglycaemia; hours to days for DKA.

Continuous glucose monitoring: the Libre and its limits

The FreeStyle Libre measures interstitial glucose, the glucose present in the fluid between cells, rather than blood glucose directly. Glucose diffuses from the bloodstream into this interstitial fluid, and that diffusion takes a measurable amount of time.

In dogs, the sensor is typically applied to the scruff at the back of the neck, or to the upper part of the chest, held in place with a medical adhesive. It reads continuously for up to fourteen days and is checked by scanning it with a phone or a dedicated reader.

The time lag this creates matters most when blood glucose is changing quickly. Because the sensor is measuring a fluid compartment that trails behind the blood itself, during a rapid fall in blood glucose, such as the fall produced by insulin at its peak, the Libre can show a reading that is higher than the true blood glucose value at that moment. In other words, during precisely the period when a dog is at greatest risk, a rapid drop toward hypoglycaemia, the Libre tends to give an unduly optimistic reading.

Accuracy varies considerably by range. In the hyperglycaemic and normal ranges, the Libre performs reasonably well and is genuinely useful for spotting trends. In the hypoglycaemic range, accuracy drops sharply. One study of diabetic dogs found the correlation between blood glucose and interstitial glucose in this range was only 0.43, a weak relationship, with the Libre tending to understate how severe a low actually was. A separate study, examining rapidly changing glucose in non-diabetic dogs, found the Libre failed to reliably detect hypoglycaemia at all when glucose was falling quickly.

The practical implication follows directly. The Libre is genuinely useful for watching trends across the day and for reducing how often a dog needs an ear or paw prick test. It is least reliable in exactly the situation where accuracy matters most, when a dog may be heading toward a dangerous low. Any low reading from a Libre sensor should always be confirmed with a blood test, using an AlphaTrak 3, before it is acted on.

AlphaTrak 3: the blood glucose standard

The AlphaTrak 3, made by Zoetis, is the current recommended blood glucose meter for home monitoring in diabetic dogs. Its predecessor, the AlphaTrak 2, has been discontinued, as its test strips are no longer manufactured, and the AlphaTrak 3 is its direct successor.

Species specific calibration matters because human and canine blood are not the same. Dogs have a different haematocrit, and a different distribution of glucose between blood plasma and red blood cells, compared with humans. A meter built and calibrated for human blood typically underestimates a dog's true blood glucose, sometimes significantly, which means a reading that looks reassuringly normal on a human meter could actually represent dangerous hypoglycaemia in a dog.

The AlphaTrak 3 is calibrated specifically for dogs and cats. In a 2024 validation study, it met the ISO 15197:2013 analytical accuracy standard used to assess human glucose meters. All of the hypoglycaemic and normal range samples tested fell within 15 mg/dL, or 0.83 mmol/L, of the reference laboratory analyser, with a strong positive correlation to laboratory reference methods.

The practical message is simple. Use the AlphaTrak 3, not a human meter, for any reading you plan to act on, and use it to confirm any low reading from a Libre sensor before treating it as a genuine hypoglycaemic event.

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Chapter 3: The Diet Science

This chapter goes underneath Part 1, Chapters 4 and 5. It sets out the full evidence behind the dry matter calculation, the fat and protein targets, the role of fibre, and what glycaemic index does and does not tell you about a dog's diet.

Why dry matter basis is the only honest comparison

Pet food labels state nutrient content on an as fed basis, meaning the figures include all the water present in the food. A wet food showing 10% protein on the label and a dry food showing 30% protein cannot be compared directly, because the wet food might be 78% water while the dry food is only 10% water. Once that water is accounted for, the actual protein content per gram of food tells a very different story from the one the label comparison suggests.

The conversion is straightforward. Dry matter basis percentage equals the labelled nutrient percentage divided by one hundred minus the moisture percentage, multiplied by one hundred.

Worked example, wet food: a tin showing 8% fat and 78% moisture converts to 8 divided by 22, multiplied by 100, which comes to 36.4% fat on a dry matter basis. A food that looks like a modest 8% fat on the label is, in real terms, nearly 37% fat once the water is removed from the calculation, close to three times the recommended ceiling for a diabetic dog.

Worked example, dry food: a kibble showing 14% fat and 10% moisture converts to 14 divided by 90, multiplied by 100, which comes to 15.6% on a dry matter basis. Because there is comparatively little water to remove, the dry matter figure looks similar to the label value, though it is still slightly above the diabetic target.

This matters specifically for diabetic dogs because three things need to be judged accurately from a label: fat, because of pancreatitis risk; protein, because of muscle preservation; and digestible carbohydrate, because of glucose spikes. None of these three assessments can be made reliably from an as fed label alone. Tufts University's Petfoodology programme describes comparing as fed

values across different foods as comparing apples to oranges, and this is not a fringe opinion; it reflects standard veterinary nutrition teaching. The companion app includes a working DMB fat checker that performs this calculation instantly from the figures on any label.

Why dogs resist new food, and what actually helps

Owners of diabetic dogs run into this constantly, and it is worth understanding properly, because the standard advice, transition gradually over seven to ten days, is usually explained only in terms of digestion. That explanation is correct, but it is incomplete. There is a second mechanism operating at the same time, and it is arguably the more immediate one for a dog who simply will not eat the new food in the first place.

The sensory arithmetic

Dogs have approximately 1,700 to 2,000 taste buds, against a human's roughly 9,000. Their olfactory receptor cell count runs to somewhere between 220 and 300 million, against a human's 5 to 6 million. The more important finding is structural rather than just numerical: the olfactory cortex occupies a proportionally much larger share of a dog's brain than a human's, and dogs use a dual olfactory pathway, assessing a food's volatile compounds through the nose before approaching it, and then again retronasally, from inside the mouth, while chewing. Smell runs both the decision to approach a food and the experience of eating it.

Smell decides before tasting begins

The most directly relevant study gave dogs a short period of visual and nasal access only, no eating, to investigate two food options, then measured which food they went on to eat more of. Dogs ate more of the food they had selected by sniffing alone 89% of the time, without having tasted either option first. The researchers concluded that dogs do not need to taste a food to make an acceptance decision. It is worth adding a caveat the original study itself flagged: a separate, stricter experiment in the same paper, using an isolated

odour port with no food or eating involved, did not find dogs distinguishing between different food odours, only a preference for food odour in general over no odour at all. The headline finding, that smell alone can drive a real eating decision, still holds; the follow up experiment is a reminder that isolating exactly what dogs are smelling for is harder than the main result suggests.

This is also why the pet food industry sprays palatants onto dry kibble as the final step of manufacture, while it is still warm and porous: specific blends of volatile compounds, including animal digest and Maillard reaction products, engineered to drive olfactory acceptance at the bowl. The spray changes almost nothing about the food's nutritional content. It changes how the food smells.

Food neophobia in dogs is documented, not folk belief

Genuine food neophobia in dogs, a reluctance around unfamiliar food, has been confirmed behaviourally, though it does not appear uniformly across all dogs or breeds. Documented feeding studies show dogs eating measurably more slowly, hesitating longer, and showing more distraction specifically on the first day of a new diet, with all three effects fading over the following days as the food becomes familiar. Separately, one frequently cited early study found that puppies raised on a single food for their first six months later rejected novel food even when they had been left without food beforehand, meaning the rejection was driven by unfamiliarity itself rather than physical need. If it were purely a digestive issue, hunger would be expected to override it more readily than it does.

A counterintuitive finding is worth knowing alongside this. Dogs fed the same diet for a long period can develop an enhanced preference for novel food, sometimes called the novelty effect. Neophobia appears to operate most strongly around early life experience with food; in an adult dog who has eaten the same restricted diet for months, novelty itself can become attractive rather than threatening. A diabetic dog who has been on the same food for a long stretch may, on this basis, be less resistant to a change than

the general pattern would suggest, though this has not been tested specifically in diabetic dogs.

Smell gets the dog to try it, taste confirms it is safe

An older but carefully designed study offers the clearest picture of how the two senses divide the work. Dogs were given a choice between a bland diet with meat odour blown across it and the same bland diet with no odour. Preference for the meat scented food started at 70%. By the third week, it had fallen to 52%, statistically negligible. The conclusion drawn at the time was that odour alone, without an accompanying taste experience, cannot sustain a preference on its own.

This matters directly for a food transition. It suggests that smell is what gets a dog to approach and try a new food, but a single positive taste experience is what locks the association in. Once a dog has eaten the new food, even mixed into a familiar bowl, and found it acceptable, the pairing between that smell and safety appears to form, and the new smell stops signalling unfamiliar in the way it did before.

Olfactory habituation: the likely mechanism

Separately from feeding studies, olfactory habituation research has found that repeated exposure to a novel odour measurably reduces the neophobic response to it and increases how pleasant it is subsequently rated, meaning familiarity with a smell genuinely lowers the threshold for accepting it. Related work in cats has found that a familiar food odour tends to suppress further feeding interest, while a novel food odour triggers renewed feeding motivation, two mechanisms, a familiar smell read as safe and a novel smell read as worth alerting to, operating at the same time, with the balance between them shaping behaviour. Masking a new food with a familiar smell or flavour is, separately, a named strategy in the wider food neophobia literature covering several species, though it does not appear to have been tested specifically as a mechanism in dogs.

Put together, gradually mixing a familiar food with a new one plausibly does three things at once. The familiar smell dominates the bowl at first, so the safe food signal outweighs the small amount of novel smell present. Repeated low level exposure to the new smell, paired each time with an actual eating experience, builds olfactory habituation before the new smell ever dominates the bowl on its own. And once the dog has eaten the mixture and found it acceptable, taste confirmation locks the new smell in as safe, the same mechanism the 1978 meat odour study pointed to.

Two real mechanisms, not tested apart

The gut microbiome side of gradual transition is independently well established: abrupt dietary change measurably alters the composition of the gut microbiota and increases the rate of diarrhoea, while a gradual transition reduces both, with the microbial community typically stabilising within about six days of a dietary change. That is the case for gradual transition as digestive management, and it is solid on its own terms.

The case made in this section is that the same seven to ten day mixing period is very likely also serving a second purpose: letting the dog's sense of smell learn that the new food is safe before it fully replaces the old one, quite separately from anything happening in the gut. Both mechanisms are almost certainly operating during the same transition window, and nobody appears to have designed a study that separates their individual contributions. That is a genuine, currently open research gap rather than a settled fact, and it is worth saying so plainly rather than presenting the smell based explanation as more proven than it is.

Fat: the threshold question

Diabetic dogs carry an elevated risk of pancreatitis through several overlapping routes. Many dogs develop diabetes through, or alongside, an episode of pancreatitis in the first place. Insulin deficiency itself causes hypertriglyceridaemia, elevated fat levels in the blood. And obesity, common in recently diagnosed dogs, is

independently a risk factor for pancreatitis. High dietary fat sits at the centre of this picture.

The mechanism runs along two main pathways. The first is free fatty acid toxicity. Excess circulating triglycerides provide fuel for enzymes called intravascular lipases, which break the triglycerides down into free fatty acids. At high concentrations, these free fatty acids damage the pancreas's acinar cells directly: they interfere with mitochondrial function, disrupt calcium signalling within the cell, trigger the release of inflammatory cytokines, and ultimately cause cell death. Damaged acinar cells then release their own pancreatic lipase into the surrounding tissue, which breaks down still more fat, generating still more free fatty acids in a self amplifying loop. The second pathway is vascular injury. High concentrations of triglyceride rich particles in the blood increase its viscosity and can impair blood flow through the small vessels that supply the pancreas, causing localised oxygen starvation followed by injury as blood flow returns.

The actual fat threshold to aim for is an area where the sourcing deserves more transparency than it usually gets. A dry matter fat ceiling of around 10% is widely cited in veterinary practice as the very low fat, pancreatitis safe threshold. The Purina Institute's own guidance on canine diabetes mellitus recommends below 12% dry matter fat, equivalent to below 30% of metabolisable energy, specifically for diabetic dogs with concurrent pancreatitis or persistent hypertriglyceridaemia. Ten percent, in other words, is the conservative end of this pancreatitis caution range, and it traces back to a genuine primary source. A ceiling of 14% dry matter fat is also widely used as a general diabetic dog recommendation, reflecting broader veterinary dietary guidance for diabetic dogs without specific concurrent pancreatitis, but its precise origin is harder to pin to one primary source. It appears consistently across veterinary nutrition resources as a practical working threshold rather than a single named guideline's number.

A caveat worth stating plainly rather than glossing over: a 2026 review published in the Journal of the American Veterinary Medical Association examined the evidence linking dietary fat to pancreatitis

directly, and found it weaker than the long standing recommendation to restrict fat would suggest. The early, frequently cited studies behind that recommendation relied on experimental models, small sample sizes, inconsistent diagnostic methods, and diets that do not reflect current commercial formulations, and more recent work has generally failed to confirm a straightforward, consistent link between dietary fat content and the onset of pancreatitis. This does not mean fat restriction is pointless. It means the practical recommendation to limit fat in a diabetic dog remains clinically sensible and represents genuine professional consensus, but it should be understood as sound clinical judgement built on an imperfect evidence base, not as an absolute, proven fact.

For a diabetic dog with no history of pancreatitis, keeping fat below 14% dry matter is a sensible general target. For a dog with a history of pancreatitis, documented hypertriglyceridaemia, or a breed predisposition such as the Miniature Schnauzer, targeting somewhere between 10 and 12% dry matter is the more appropriate, better evidenced goal. When in doubt, this is a conversation for the vet.

Protein: why diabetic dogs need more, not less

Insulin's role goes well beyond moving glucose into cells. It also has a direct anabolic effect on skeletal muscle: it promotes the synthesis of new muscle protein and actively inhibits the breakdown of existing muscle protein. In absolute insulin deficiency, which describes most diabetic dogs, this anabolic signal is simply absent.

The consequence follows a specific, self reinforcing sequence. Muscle protein is broken down, a process called proteolysis, at an accelerated rate. The amino acids released by that breakdown are then used by the liver for gluconeogenesis, the manufacture of new glucose, which compounds the very hyperglycaemia the dog is already dealing with. Muscle mass decreases as a direct result, and the dog loses condition, weakens, and struggles increasingly to stand or exercise normally. Studies of the equivalent absolute insulin deficiency state in humans show increased urinary nitrogen loss,

elevated free amino acids in the blood, and accelerated amino acid breakdown, all markers of this catabolic state. Encouragingly, these effects are largely reversible once exogenous insulin is given correctly and consistently.

This matters for reasons beyond the muscle loss itself. Muscle is the body's primary reservoir for disposing of glucose after a meal. Less muscle means less capacity to buffer a post meal glucose spike, which creates a direct negative feedback loop within diabetes management: less muscle makes glucose harder to control, and harder to control glucose accelerates further muscle loss. Preserved muscle mass, conversely, genuinely improves insulin sensitivity and glycaemic control in its own right.

This mechanism also explains something that is easy to misread in an older dog. Muscle atrophy causes difficulty rising, stumbling on stairs, reluctance to exercise, and a general impression of stiffness and weakness, all of which owners understandably attribute to joint pain. In a senior dog already being treated for arthritis, the contribution of diabetes related muscle loss on top of that can go unrecognised for months, because every symptom already has an explanation that seems to fit.

The AAHA 2018 guidelines and other veterinary nutrition sources reference protein targets meaningfully above ordinary maintenance levels for diabetic dogs. The general target is above 30% of metabolisable energy from protein, roughly equivalent to between 25 and 28% on a dry matter basis. For dogs who also need to lose weight, the target rises further, to 40% or more of metabolisable energy, to protect lean muscle mass during the calorie restriction. For underweight dogs specifically, both protein and overall calorie density need to be prioritised together, and fibre should not be pushed too high, since it fills the stomach before enough calories have actually been consumed.

Fibre: the science of slowing glucose

Both soluble and insoluble fibre slow the absorption of glucose from the gut, but they do so through different mechanisms. Soluble fibre

dissolves in water and forms a viscous gel within the intestine; this gel physically slows the diffusion of glucose across the intestinal wall, blunting the glucose spike that follows a meal. Insoluble fibre does not dissolve. Instead it adds bulk to the food, slows gastric emptying, and reduces the overall rate at which digested food reaches the small intestine.

Three studies form the core evidence base here. Two studies from Richard Nelson's group at UC Davis, published in 1991 [alloxan-induced experimental diabetes model; not naturally occurring disease] and 1998, showed improved glycaemic control in dogs fed a high insoluble fibre diet, at around 12% cellulose, compared with a low insoluble fibre diet. The most directly relevant study, by Kimmel, Michel, Hess, and Ward in 2000, fed seven dogs with naturally occurring insulin dependent diabetes three different diets in a crossover design: low fibre, high insoluble fibre, and high combined soluble and insoluble fibre. Mean and maximum blood glucose, and the area under the glucose curve, were all significantly lower on the high insoluble fibre diet than on either of the other two, and the study concluded that a dry, high insoluble fibre diet aids glycaemic control in naturally diabetic dogs. A more recent 2025 study compared a moderate fibre diet, at 7% dry matter, against a high fibre diet, at 15% dry matter, in dogs with spontaneous insulin deficient diabetes, and found both improved HbA1c and 24 hour mean glucose relative to each dog's own baseline diet, without one being dramatically superior to the other.

Taken together, the evidence currently points most strongly toward insoluble fibre for glycaemic benefit specifically. Soluble fibre offers a different, additional benefit: gut bacteria ferment it into short chain fatty acids, including propionate and acetate, which have their own metabolic effects, among them reducing how much glucose the liver releases. In practice, the combination of both types, as found in most whole foods and high fibre commercial diets, is a reasonable practical choice. It's worth being clear about the limits of that evidence, though. A later, larger study of dogs whose diabetes was already stable found no advantage to high-fibre, moderate-carbohydrate diets over the dogs' existing diets at all (Fleeman, Rand

and Markwell, 2009). Taken together, the picture is that fibre may help, particularly in the period after diagnosis, but it is not settled science, and a dog doing well on a lower-fibre diet has no obvious reason to change.

There is one exception that is consistently missed in owner facing resources, and it deserves to be stated clearly. Fibre is filling. An underweight diabetic dog placed on a high fibre diet can reach a feeling of fullness before consuming enough calories to maintain or rebuild their weight. For an underweight dog, lower dietary fibre is the more appropriate choice, despite the diabetes, because rebuilding muscle and body condition takes priority over fibre based glucose control in this specific situation.

Glycaemic index in dogs: useful but limited

Glycaemic index ranks foods by how quickly they raise blood glucose relative to a reference food, typically pure glucose or white bread. It is well established and widely used in human diabetes nutrition, but its application in canine diabetes is considerably more limited.

The concept does genuinely apply to dogs, unlike cats, whose carbohydrate metabolism works quite differently; dogs process and respond to dietary carbohydrate in a broadly similar way to humans. The total amount of digestible carbohydrate in a meal is the main determinant of how much glucose rises afterwards in dogs, more so than the specific source of that carbohydrate, and a diet lower in digestible carbohydrate overall produces a lower post meal glucose rise than traditional diabetic or standard adult maintenance diets. However, among individual starch sources and commercial extruded foods, some studies have found no meaningful differences in glycaemic index at all, with the variation between individual dogs proving as significant as the variation between ingredients.

In practice, glycaemic index is a useful concept for explaining why highly refined carbohydrates, such as white rice or sugary treats, spike glucose faster than complex, whole food carbohydrates. But choosing between, say, brown rice and sweet potato on the strength

of their glycaemic index is probably less important than controlling the total amount of digestible carbohydrate and keeping portions consistent from day to day. It is worth being frank about the state of the evidence here: the glycaemic index database for canine foods is sparse compared with the equivalent for human foods, and an owner trying to apply glycaemic index principles precisely to their dog's diet is working from genuinely incomplete data. Total carbohydrate control and fibre content are better evidenced levers than glycaemic index for most diabetic dogs.

Reading the ingredients list: what to watch for and why

Everything so far in this chapter concerns the nutrients a label states directly. This section looks at what else is in the bag or tin, the sugars, colourings, and preservatives that serve purposes other than nutrition, and why they deserve particular attention in a diabetic dog's diet.

Added sugars

Dogs have relatively few taste receptors for sweetness compared with humans, so sugar is not added to dog food because dogs crave it. It is added for a different reason entirely. Sugar reacts with amino acids under high heat, in what is called the Maillard reaction, to produce appetising smells and browned surfaces, the same reaction that makes bread crusts and roast meat appealing to us. Applied to a lower quality pet food, it makes the product smell attractive enough that the dog eats it readily. The sugar is serving the manufacturer's palatability target, not the dog's nutritional needs.

Under UK and EU labelling regulations, ingredients are listed in descending order by weight, and sugars can appear under several different names, individually or grouped under the permitted category term various sugars: sucrose, glucose, glucose syrup, dextrose, fructose, high fructose corn syrup, maltose, maltodextrin, molasses, caramel, honey, agave, and fruit juice concentrate, which is natural in origin but functions as a concentrated sugar all the same. The category term various sugars is legally permitted and tells you

sugar is present without telling you what type, or how quickly it will be broken down.

For a healthy dog, moderate added sugar is metabolically manageable. For a diabetic dog, any added sugar is a direct problem. Sucrose, glucose, and maltose are absorbed rapidly into the bloodstream and cause a sharp post meal glucose spike. These spikes complicate insulin timing, since an injection calibrated to a consistent meal suddenly has to cope with an unpredictable additional glucose load, and even small, repeated additions accumulate across two meals a day, every day.

Fructose deserves particular attention, because there is direct experimental evidence behind the concern. Fructose is metabolised differently from glucose. It is processed almost entirely by the liver, where it is converted into lipids, driving fat accumulation in the liver, which in turn causes peripheral insulin resistance, meaning the body's cells become less responsive to insulin generally. A 2023 experimental study fed dogs a high fat, high fructose diet, with fructose making up 17% of total energy, and found a 50% decrease in insulin sensitivity within the first four weeks, along with impairment of the dogs' own remaining insulin secretion. The metabolic pattern that resulted closely mirrored human Type 2 diabetes and impaired glucose tolerance. This was an extreme dietary intervention, well beyond the trace amounts found in commercial food, but it demonstrates that fructose has a measurably damaging effect on canine insulin sensitivity at elevated doses, which matters when a diabetic dog's food contains added sugars including fructose daily, at every meal, for years.

Artificial colourings

Dogs see the world in shades of blue and yellow. They are red green colour blind, with far fewer colour receptors than humans, which means the bright reds, oranges, and yellows in some dog foods are not seen as distinct colours by the dog eating the food at all. These colours exist to appeal to the owner's eye at the point of purchase, signalling variety, vitality, and wholesome ingredients through a

visual cue the dog cannot perceive. This is worth stating plainly: colouring in dog food serves the person holding the shopping basket, not the dog eating from the bowl.

In 2007, a study by McCann and colleagues at the University of Southampton showed that a mixture of six artificial food colourings, combined with the preservative sodium benzoate, increased hyperactive behaviour in children aged three and eight to nine. The six colours, now known collectively as the Southampton Six, are tartrazine, quinoline yellow, sunset yellow, carmoisine, ponceau 4R, and allura red. Following the study, the UK Food Standards Agency called on manufacturers to voluntarily phase these colours out, and EU regulation now requires a mandatory warning label on any product containing one or more of them, stating that they may have an adverse effect on activity and attention in children. The European Food Safety Authority reviewed the evidence and did not revoke approval for any of the six, but did subsequently lower the acceptable daily intake for three of them, a tacit acknowledgement of concern even without a formal ban.

Beyond the hyperactivity link in children, animal studies, primarily in rodents and guinea pigs, have raised further questions. Tartrazine has been linked to DNA damage in the colon, liver, and kidneys, and to embryotoxic effects at high doses. Sunset yellow and carmoisine in combination have shown pro-inflammatory effects in short-term rat studies. Allura red has shown some evidence of carcinogenicity at high doses in rodents, though no studies specifically in dogs have been identified.

A caveat is worth stating plainly rather than glossed over: there are no controlled clinical studies specifically examining the effects of these colourings on blood glucose or insulin function in dogs. The evidence of harm sits in rodent studies at high doses, with some extrapolation from the Southampton study in human children. The case against colourings in a diabetic dog's diet is principled rather than directly proven: they provide no nutritional benefit whatsoever, they may contribute to low-level inflammation, and inflammation worsens insulin resistance. That is a reasonable basis for avoiding them, but it should not be overstated as settled canine evidence.

Chemical preservatives

BHA, butylated hydroxyanisole, BHT, butylated hydroxytoluene, and ethoxyquin are synthetic antioxidants added to dog food primarily to prevent the fat content from turning rancid, extending shelf life by inhibiting oxidation. At high doses in rodent studies, all three have shown carcinogenic effects: BHA caused stomach carcinomas, BHT promoted bladder cancer and possibly thyroid cancer, and ethoxyquin significantly increased stomach and colon tumours. At the levels typically used in dog food, however, no convincing evidence of harm has been established specifically in dogs, and the rodent carcinogenicity appears to be both species specific and dose dependent. BHT is nonetheless banned from food use in Japan, Romania, Sweden, and Australia, and barred from infant foods in the United States, bans that reflect precautionary policy rather than proven canine toxicity.

The most recent dedicated review of BHA and BHT in dog and cat food, published in 2024, concluded there is no convincing evidence that these antioxidants, at the levels typically used in dry dog food, are harmful. No studies have specifically examined whether BHA or BHT affect insulin sensitivity or glycaemic control in dogs. The precautionary case for reducing unnecessary chemical load in a metabolically compromised animal is a reasonable position, but it is not currently an evidence based one, and the book should be honest about that rather than overstate the risk.

Advanced glycation end products: the processing problem

This is the finding most directly relevant to diabetic dogs specifically, and it is almost entirely absent from owner facing resources. When proteins or fats react with sugars under heat, the same Maillard reaction described earlier in this section, they form advanced glycation end products, or AGEs: irreversible molecular structures that accumulate in tissue over time and impair normal cell function.

In diabetes, this same process happens endogenously, inside the body, because persistently high blood glucose provides an abundant

supply of sugar for these reactions. A diabetic dog is therefore already generating AGEs internally at an elevated rate, simply as a consequence of the disease itself.

Dry commercial dog food is almost entirely produced by extrusion, a process that forces ingredients through a die under extremely high heat and pressure before drying the output. This process is itself a significant AGE generating event: high heat, combined with protein and the sugars present in starchy ingredients, produces substantial AGE formation. A 2024 peer reviewed study compared dogs fed diets at four different processing levels and found that dogs fed extruded kibble had measurably higher plasma and urinary AGE concentrations than dogs fed minimally processed diets, a difference significant enough to be detectable as a biomarker across the study period.

This matters particularly for a diabetic dog because AGEs ingested from food add directly to the AGE burden the disease is already generating internally. AGEs accumulate in the kidneys, reducing filtration function; in blood vessel walls, increasing cardiovascular risk; and in the pancreas itself, further impairing whatever residual function remains. They also trigger chronic low grade inflammation through a specific receptor pathway, and that inflammation worsens insulin resistance in turn. The net effect is that a diabetic dog fed a high AGE, heavily processed diet is receiving an additional daily dose of the same molecular insult that diabetes is already generating from within.

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Chapter 4: What the Numbers Really Mean

This chapter goes underneath Part 1, Chapter 7, and the interpretation sections of Part 1, Chapter 6. It sets out in full what each type of reading on a glucose curve actually tells you, what fructosamine and the newer HbA1c test add beyond a single reading, and why urine testing cannot substitute for either.

The three types of reading, and why they are not interchangeable

The pre-injection reading

The pre-injection reading is taken immediately before giving insulin and feeding, after the previous dose has been active for the full twelve hours and is largely spent. Because insulin's effect is winding down and glucose has typically been rising for several hours by this point, this is usually the highest reading of the entire cycle. It marks the starting point of the curve, not a verdict on how well the previous dose worked.

The AAHA 2018 target for a well-controlled dog is a pre-injection reading below 17 mmol/L, 300 mg/dL. A reading consistently above that suggests the previous dose is not providing adequate overnight coverage. A reading that is consistently very low at this point suggests the opposite problem: the dose may still be active at the twelve hour mark, raising the risk that two doses overlap in their effect once the next one is given.

The nadir

The nadir is the lowest blood glucose value reached anywhere in the twelve hour cycle, occurring when insulin's action is at its peak. For Caninsulin, this typically falls four to eight hours after injection, corresponding to the amorphous fraction described in Chapter 2 of this Part, with a possible secondary dip around eleven hours, corresponding to the crystalline fraction. The AAHA 2018 target nadir is 4.4 to 8.3 mmol/L, 80 to 150 mg/dL.

The nadir is the single most clinically important value on a glucose curve, for three reasons. It defines the safety margin for any dose increase, since a vet cannot safely raise a dose without first knowing the nadir sits in a safe range. It cannot be assumed to fall at any particular measured time point, which means a standard two hourly reading schedule can miss it entirely if it happens to occur between two scheduled readings. And only a full, structured curve, not a single reading, can identify it at all.

The nadir and the pre-injection reading sit at opposite ends of the same twelve hour cycle, and it is worth stating this distinction as plainly as possible, because confusing the two is the single most common error owners make when reading a curve. The pre-injection reading is the high point, taken when insulin is spent. The nadir is the low point, occurring when insulin is at peak effect. They describe two different moments in the cycle and cannot be the same reading.

Random or spot readings

A random or spot reading is any single reading taken outside the structure of a full curve, and it comes with real limitations. The stress of a veterinary visit, travel, restraint, and an unfamiliar environment can raise a dog's blood glucose significantly, producing a reading that does not reflect how that same dog runs at home. A single elevated reading cannot, on its own, confirm poor control, and a single normal reading cannot, on its own, confirm good control. Fructosamine, covered later in this chapter, is the tool that can distinguish genuine persistent hyperglycaemia from a stress induced spike, something a single glucose reading is simply not equipped to do.

Curve shapes: what each pattern means

The illustrated glucose curves referred to throughout this book, well controlled, underdose, overdose or Somogyi, and short duration, appear in Chapter 2 of this Part, alongside the full mechanism behind Somogyi rebound. This section sets out what defines each shape and what response each one calls for.

The AAHA 2018 targets for a full curve are as follows. The majority of readings through the day should sit between 5.6 and 13.9 mmol/L, 100 to 250 mg/dL. The nadir should fall between 4.4 and 8.3 mmol/L, 80 to 150 mg/dL. The pre-injection reading should sit below 17 mmol/L, 300 mg/dL.

Well controlled

Pre-injection below 17 mmol/L, 300 mg/dL, falling to a nadir of 4.4 to 8.3 mmol/L, 80 to 150 mg/dL, at four to eight hours, then rising gradually to an evening pre-meal reading also below 17 mmol/L, 300 mg/dL. The overall shape is a smooth arc with a clear mid cycle trough.

Underdose

Pre-injection high, glucose falls from that starting point but the nadir remains elevated, above 10 mmol/L, 180 mg/dL, and the curve never enters the target range at any point, staying consistently elevated throughout. The appropriate response is a modest dose increase, but only after the vet has confirmed the nadir is genuinely safe, not simply assumed to be.

Overdose, or Somogyi rebound

The full mechanism is covered in Chapter 2 of this Part. On paper, this shape produces persistently high readings that look identical to underdose. The distinguishing feature is that somewhere in the cycle, glucose may have fallen below roughly 3.3 mmol/L (60 mg/dL) per AAHA 2018, triggering a counter regulatory hormone release and a rebound. Because that low point may fall between two scheduled readings and go uncaptured, distinguishing Somogyi from genuine underdose requires hourly readings around the suspected nadir, not the standard two hourly schedule. The correct response is a dose reduction, the opposite of the underdose response, which is exactly why confusing the two shapes is dangerous.

Short duration of action

The nadir occurs early, within the first four to six hours, and glucose then rises back to high values well before the end of the twelve hour cycle. The insulin is working, but it is not lasting long enough for this particular dog. The evening reading ends up as high as, or higher than, the morning reading. This is worth discussing with the vet as a question of insulin duration or dosing frequency, not simply a question of dose size.

Prolonged duration, or overlap risk

The nadir occurs at twelve hours or later, meaning glucose is still falling when the next dose is due. This carries its own risk, since it raises the possibility of two doses being active simultaneously at peak effect. The response here is a dose reduction, with adjusted timing under the vet's guidance.

Fructosamine: what it measures and where it falls short

Fructosamine, sometimes written as serum fructosamine or sFA, forms when glucose binds irreversibly to albumin, the main protein in blood serum. The higher a dog's average blood glucose has been over recent weeks, the more fructosamine accumulates. It is measured from a single blood sample and requires no fasting beforehand.

Fructosamine reflects approximately two to three weeks of average blood glucose, though some sources cite a window closer to seven to fourteen days. The variation reflects differences in laboratory methodology and in individual albumin turnover rates, not a genuine disagreement about the underlying biology.

Its key advantage is that it is not affected by acute stress. A dog with a high reading at the vet, caused by travel or an unfamiliar environment, will still show a normal fructosamine if its average glucose at home has genuinely been well controlled. This makes fructosamine the most useful tool available for distinguishing persistent hyperglycaemia from a stress induced spike, which, as noted above, a single glucose reading cannot do on its own.

Reference values vary between laboratories and should always be taken from the reporting laboratory's own range rather than a general figure, but as a guide, non-diabetic dogs typically fall in the region of 225 to 374 micromoles per litre. A 2023 Veterinary Record study of 75 diabetic dogs across 321 visits found a mean fructosamine of 506 micromoles per litre on visits assessed as well controlled, compared with 584 micromoles per litre on visits assessed as uncontrolled, a statistically significant difference.

Fructosamine's accuracy, however, is only moderate, not excellent. A 2022 Veterinary Record study found fructosamine had an area under the curve of 0.71 for distinguishing well controlled from poorly controlled diabetic dogs, meaning it performs better than chance but is far from a definitive test on its own. Individual trends across repeated measurements over time are considerably more clinically useful than any single absolute value taken in isolation.

Several conditions can distort a fructosamine result independently of actual glucose control, and it is worth knowing what they are so an unexpected result is not automatically read as a diabetes problem. Hypoalbuminaemia, low blood protein, produces a falsely low result, because there is less albumin available to bind glucose in the first place, which underestimates the true average glucose. Hyperglobulinaemia, excess blood protein, produces a falsely high result through the opposite mechanism. Hyperlipidaemia and azotaemia, kidney disease, both reduce the accuracy of the assay itself through interference. Hypothyroidism can alter the result by affecting protein metabolism more broadly, and a haemolysed blood sample can distort the result as a simple laboratory artefact rather than reflecting anything about the dog. A thin, protein depleted diabetic dog in particular may show a falsely reassuring fructosamine despite genuinely poor control, which is why a result that contradicts the clinical picture in front of you is always worth raising with the vet rather than accepted at face value.

Canine HbA1c: the longer term marker now becoming available

HbA1c, glycated haemoglobin, is the standard long-term marker used in human diabetes, but it was not traditionally used in dogs, because no standardised commercial canine assay existed and results were not comparable between laboratories.

That is changing. Canine red blood cells have a lifespan of approximately 100 to 120 days, considerably longer than the two to three week window fructosamine reflects, which means a validated canine HbA1c test can offer a picture of average blood glucose across roughly 110 days, a substantially longer horizon than any other currently available test. A commercial canine assay, marketed as A1Care by Baycom Diagnostics, is now available.

The evidence for its usefulness is striking. A 2023 study found canine HbA1c achieved 100% diagnostic accuracy for distinguishing diabetic from non-diabetic dogs in the cohort studied, compared with fructosamine's 84.4% accuracy in that same group. A separate 2020 study found significant negative correlations between HbA1c and haematocrit, haemoglobin, and red blood cell count, meaning an anaemic dog can show a falsely elevated HbA1c, because each remaining red blood cell ends up carrying a proportionally higher glycated fraction than it would in a dog with a normal red cell count. This is a genuine limitation worth knowing, particularly in an older dog where mild anaemia is not uncommon.

HbA1c is not yet standard practice in most UK veterinary practices, and fructosamine remains the routine test for most dogs. For an owner, it is worth specifically asking the vet about HbA1c in cases where control has been complex or inconsistent, and where a longer term picture, beyond what fructosamine or a single curve can offer, would genuinely change the picture.

The renal glucose threshold: why urine testing cannot replace blood monitoring

The kidneys reabsorb glucose from filtered blood up to a capacity limit of approximately 10 to 12 mmol/L, 180 to 200 mg/dL, the renal threshold introduced in Part 1, Chapter 1. Above that threshold, glucose spills into the urine. Below it, a urine test for glucose comes back negative.

A negative urine test result only tells you that blood glucose was below the renal threshold at some point while urine was accumulating in the bladder. It cannot distinguish between a blood glucose of 3 mmol/L, 54 mg/dL, dangerous hypoglycaemia, and 9 mmol/L, 160 mg/dL, genuinely good control. These two situations call for opposite responses, and a urine dipstick has no way to tell them apart.

A positive urine test result tells you that blood glucose was above the renal threshold at some point during the same period. In a diabetic dog, this is expected during parts of an ordinary day and does not, on its own, indicate poor control.

For these reasons, AAHA and Merck no longer recommend urine testing as a primary monitoring method. It cannot detect hypoglycaemia. It cannot safely guide a dose increase. It cannot identify Somogyi rebound. It gives a time averaged result, reflecting accumulated urine rather than a dog's current glucose at any given moment. And individual dogs vary in exactly where their own renal threshold sits, so the same urine result can mean different things in different dogs.

The one genuine remaining clinical use is this: if urine glucose is consistently negative across repeated tests, blood glucose may be running persistently low, and that is worth raising with the vet as a prompt to discuss a possible dose reduction, not as a reason to increase the dose.

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Chapter 5: Concurrent Conditions and Insulin Resistance

This chapter goes underneath every point in Part 1 and this Part where a previously stable dog suddenly stops responding to a dose that used to work. Chapter 1 of this Part introduced the intact female as a special case; Chapter 2 noted that most dogs admitted with DKA had a concurrent condition present at the same time. This chapter develops each of those conditions in full, along with several others, and sets out a practical framework for working through unexplained resistance systematically rather than guessing.

Why concurrent conditions matter disproportionately

A well-regulated diabetic dog's glucose control depends on a stable, repeatable relationship between the insulin dose and the dog's insulin sensitivity on any given day. Any condition that alters insulin sensitivity, upward as resistance or downward as enhanced sensitivity, disrupts that relationship and makes the existing dose either too low or, more dangerously, too high. In a non-diabetic dog, the pancreas compensates by adjusting its own insulin output. A diabetic dog has no functional beta cells to call on. Every change in sensitivity falls entirely on the current dose, which cannot self-adjust.

This is why concurrent illness destabilises diabetic control so reliably, and why a dog who was perfectly stable last month can suddenly show persistently elevated glucose readings with no obvious cause. In most of those cases, something has changed in the dog's biology, not in the insulin.

Urinary tract infections

Why diabetic dogs are uniquely vulnerable

Glucose in the blood spills into the urine once blood glucose exceeds the renal threshold, around 10 to 12 mmol/L, 180 to 200 mg/dL, in dogs, introduced in Part 1, Chapter 1. Glucose-rich urine is an

exceptionally good bacterial growth medium. Bacteria that would normally be cleared by the flow of dilute urine find in a diabetic dog both the nutrients and the suppressed immune environment, since high glucose impairs neutrophil function directly, to establish persistent infections. Diabetic dogs face a double disadvantage: they produce the conditions bacteria thrive in, and they produce less effective immune defence against them. [Mayer-Roenne 2007 was a study in diabetic cats; findings are extrapolated to dogs here]

The silent infection problem

This is one of the most clinically important, and most under-recognised, aspects of canine diabetes management. In non-diabetic dogs, a UTI usually produces obvious signs: straining, frequent urination, discomfort, bloody urine. In diabetic dogs, the same signs can be masked almost completely. Diabetic dogs are already urinating frequently and in larger quantities than normal as a direct consequence of the disease. A UTI on top of that baseline is often clinically silent, with no additional symptoms the owner can detect.

The practical consequence is significant. A dog with a bacterial infection can run persistently elevated glucose readings that resist adjustment for weeks without any apparent reason, until a urine culture comes back positive. The infection is causing insulin resistance; the insulin resistance is producing persistently high glucose; but the owner sees only a dog whose glucose refuses to respond to what should be adequate treatment.

The antibiotic lag

Even after a UTI has been successfully treated with an appropriate antibiotic course, glucose control does not immediately return to its previous baseline. The inflammatory response triggered by the infection, the release of cytokines including interleukin-6 and tumour necrosis factor-alpha, alters insulin receptor signalling at a cellular level. That signalling disruption does not resolve the moment the bacteria are cleared. In clinical practice, the effect can persist for one to two weeks after completing a full antibiotic course.

This one to two week figure is worth a note on its own sourcing. It is consistent across clinical veterinary references, including the AAHA guidelines and Merck Veterinary Manual entries on diabetic monitoring, and it fits the general immunometabolism literature on cytokine-mediated insulin resistance. It has not been traced to a single named primary study in dogs specifically. It should be read as established clinical consensus rather than a single-study finding.

How to monitor

AAHA's 2018 guidelines recommend routine urinalysis, ideally with bacterial culture, in diabetic dogs at every recheck visit, even in the absence of clinical signs, specifically because silent UTIs are so common and so reliably disruptive to control. Urine sediment examination alone is not sufficient; a bacterial culture with sensitivity testing is needed to identify the organism and guide antibiotic choice. E. coli is the most commonly identified organism in diabetic dog UTIs, consistent with its general prevalence in canine urinary infections, but antibiotic resistance patterns mean culture-guided treatment is preferable to empirical antibiotic choice. This recommendation is not without debate; more recent evidence has questioned whether culturing every diabetic dog regardless of signs is actually warranted. Chapter 6 of this Part covers that more contested picture in full.

Cushing's disease (hyperadrenocorticism)

What it is

Hyperadrenocorticism, known as Cushing's disease when caused by a pituitary tumour and Cushing's syndrome in the broader sense, results from chronically elevated cortisol levels. In approximately 80 to 85% of cases in dogs, the cause is a benign microadenoma of the pituitary gland producing excess ACTH, adrenocorticotrophic hormone, which then drives the adrenal glands to produce cortisol continuously. In roughly 15 to 20% of cases, the cause is a tumour of

the adrenal gland itself producing cortisol independently of pituitary control.

Why it causes insulin resistance: the mechanism

Cortisol acts at multiple points in the glucose metabolism pathway, all in the same direction: it pushes blood glucose up and makes cells less responsive to insulin.

The primary mechanism is gluconeogenesis. Cortisol stimulates the liver to manufacture new glucose from amino acids and other non-carbohydrate precursors, and this happens continuously in a dog with chronically elevated cortisol, not just during acute stress. At the same time, cortisol directly reduces GLUT4 translocation to cell membranes, the same mechanism described in Chapter 1 of this Part, but now operating as a persistent pharmacological effect rather than simply an absence of insulin signalling. Cortisol also increases lipolysis, releasing free fatty acids into circulation that then compete with glucose for cellular uptake through the Randle cycle. The net effect is a form of insulin resistance that exists in parallel with, and compounds, the absolute insulin deficiency the diabetic dog already has.

The diagnostic challenge: signs shared with diabetes

Cushing's is particularly difficult to identify in a diabetic dog because the two conditions share almost every major clinical sign. Polydipsia and polyuria, increased thirst and urination, are classic manifestations of both. Weight redistribution, a pot belly with wasted limbs, occurs in both. Lethargy is common in both.

The signs more specific to Cushing's, and therefore most clinically useful, are: bilateral symmetrical alopecia, hair loss in a coat-sparing pattern rather than one driven by itching, skin thinning with visible superficial blood vessels, comedones (blackheads along the back), calcinosis cutis (calcium deposits in the skin), a pot-bellied appearance due to liver enlargement and abdominal muscle weakness, excessive panting disproportionate to heat or exertion, and a pendulous abdomen with muscle atrophy of the limbs.

The combination of these signs together, particularly alopecia, skin changes, and excessive panting in a dog already known to be diabetic, should prompt investigation.

Breeds at elevated risk

Poodle (all sizes), Dachshund, Yorkshire Terrier, Beagle, Boxer, Boston Terrier, and Staffordshire Bull Terrier appear consistently across published epidemiological data as breeds with elevated risk of hyperadrenocorticism.

Diagnostic tests

Three tests are used clinically. The urine cortisol to creatinine ratio (UCCR) is a useful screening tool; a normal result largely rules out Cushing's, but a positive result requires confirmation, since many non-Cushing's conditions, including stress and concurrent illness, also elevate UCCR.

The low dose dexamethasone suppression test (LDDS) is the most sensitive test and the current ACVIM consensus first-line test for diagnosing hyperadrenocorticism in dogs. In a normal dog, dexamethasone, a synthetic glucocorticoid, suppresses ACTH production and therefore cortisol; in a dog with Cushing's, this suppression fails. Reported sensitivity for pituitary-dependent disease ranges from roughly 85 to 95% across studies.

The ACTH stimulation test is less sensitive for diagnosing Cushing's overall, reported at roughly 60 to 85% depending on the study, but it is the test of choice for monitoring treatment response in dogs already being treated with trilostane, since trilostane's mechanism makes LDDS results uninterpretable in treated dogs.

Why concurrent Cushing's and diabetes is particularly difficult to manage

A dog with both conditions typically requires substantially higher insulin doses than a straightforward diabetic dog, often described in clinical teaching as two to three times the typical range, simply to overcome the continuous cortisol-driven insulin resistance. Treating

the diabetes without addressing the Cushing's leads to a frustrating situation in which the glucose curve never responds as expected.

This two to three times figure appears consistently in veterinary endocrinology teaching, including Feldman and Nelson's reference text, and in clinical experience, but it has not been traced to a single named study reporting a precise multiplier. It is best read as clinical consensus rather than a specific measured statistic.

The correct management sequence is to diagnose and begin treating the Cushing's, with trilostane (Vetoryl) in the UK and EU as first-line treatment, or with mitotane as an alternative, and then reassess insulin requirements as cortisol levels fall. As Cushing's treatment takes effect and cortisol returns toward normal, insulin sensitivity improves, and the dose that was previously insufficient can suddenly become an overdose. Close monitoring during the transition period is essential.

Hypothyroidism (underactive thyroid)

What it is and why it matters in diabetic dogs

Hypothyroidism, underactive thyroid function, is the most common endocrine disorder in dogs after diabetes itself. It results from destruction or atrophy of the thyroid gland, most commonly through immune-mediated lymphocytic thyroiditis, the canine equivalent of Hashimoto's disease in humans, or idiopathic thyroid atrophy. The result is insufficient thyroid hormone, thyroxine or T4, production.

Thyroid hormone plays a central role in regulating metabolic rate, glucose absorption from the gut, and peripheral glucose uptake. Hypothyroidism does not cause insulin resistance through the same cortisol-mediated mechanism as Cushing's, but it slows metabolism in ways that affect glucose handling: reduced gut glucose absorption, reduced GLUT4 expression in muscle cells, and impaired lipolysis all alter the glucose to insulin relationship. The effect is more moderate than Cushing's, but it is clinically significant, particularly in a dog whose control is already precarious.

Hypothyroidism also causes weight gain, which independently increases insulin resistance through adipokine-mediated pathways, the same mechanism described in the obesity section below.

Clinical signs

The signs of hypothyroidism are broad and often develop gradually over months. The most consistent are weight gain despite no increase in food intake, lethargy and general dullness, exercise intolerance, and bilateral symmetrical alopecia, particularly along the trunk and tail, with the head and limbs relatively spared. Skin thickening and a sad, tragic facial expression caused by oedema of the face are characteristic; this is a genuine clinical descriptor used in veterinary textbooks for the myxoedematous facial appearance, not a subjective characterisation. Bradycardia, an abnormally slow heart rate, and cold intolerance are also common, though less reliably reported by owners than the coat and weight changes.

Breeds at elevated risk

Golden Retriever, Dobermann, Irish Setter, Cocker Spaniel, Airedale Terrier, and Boxer appear consistently in epidemiological studies as breeds with elevated hypothyroidism risk.

Diagnosis, and the euthyroid sick syndrome pitfall

Total T4 (tT4) is the standard screening test. In a hypothyroid dog, tT4 is low. However, and this is a clinically important caveat, tT4 can also be suppressed by concurrent illness, medication, particularly glucocorticoids and some anti-epileptic drugs, and by the metabolic state of a poorly regulated diabetic dog itself. This phenomenon, called euthyroid sick syndrome or non-thyroidal illness syndrome, can produce a falsely low tT4 result in a dog whose thyroid is actually functioning normally. Diagnosing hypothyroidism in a diabetic dog on tT4 alone therefore carries a real risk of false positives.

Free T4 (fT4) measured by equilibrium dialysis is more specific and less affected by non-thyroidal illness. TSH measurement

alongside fT4 provides the most reliable picture: a dog with true hypothyroidism will show both a low fT4 and an elevated TSH, as the pituitary attempts to drive the thyroid harder. A dog with euthyroid sick syndrome will typically show a low tT4 but normal or low-normal TSH and normal fT4.

Treatment and diabetes interaction

Hypothyroidism is treated with oral levothyroxine, typically once or twice daily. As thyroid hormone levels normalise, metabolic rate increases, which means the body's calorie requirements increase, and the relationship between food, glucose, and insulin changes. A dog being treated for both hypothyroidism and diabetes will typically need glucose curve reassessment once levothyroxine has reached steady state, usually four to eight weeks after starting, since insulin requirements may shift as the metabolism normalises.

Obesity

Mechanism: adipokines and insulin resistance

Excess body fat, particularly visceral adipose tissue around the abdominal organs, is not metabolically inert. Adipose tissue secretes a range of signalling molecules called adipokines, including leptin, resistin, and adiponectin. In obesity, leptin and resistin secretion increase, and adiponectin, which normally promotes insulin sensitivity, falls. Leptin resistance, where the body's cells stop responding to leptin's satiety signal, compounds this further.

Resistin directly impairs insulin signalling in liver cells, reducing glucose uptake and increasing hepatic glucose output. Visceral adipose tissue also secretes pro-inflammatory cytokines, particularly tumour necrosis factor-alpha and interleukin-6, which interfere with the insulin receptor signalling cascade, the same phosphorylation cascade described in Chapter 1 of this Part. The net effect is peripheral insulin resistance that is directly proportional to the amount of excess fat, and particularly visceral fat, the dog carries.

Why many diabetic dogs arrive overweight, and why it matters

Dogs are commonly overweight or obese at the time of diabetes diagnosis. This is not coincidental. Obesity is an independent risk factor for developing diabetes in the first place, and many diabetic dogs have been overweight for months or years before diagnosis. This pre-existing insulin resistance, layered on top of the beta cell destruction that produces the diabetes, creates a more severe and harder-to-manage disease from the outset.

The priority in an overweight diabetic dog is a carefully managed calorie deficit that targets fat loss while preserving muscle mass, the distinction explained in detail in Chapter 3 of this Part. Calorie restriction without adequate protein increases muscle catabolism, worsening the sarcopenic cycle that diabetes already drives. The combination of reduced fat, higher protein, and consistent exercise, matched to the dog's physical condition, is the appropriate goal.

Exercise as an active lever on insulin sensitivity

In dogs as in humans, regular moderate exercise improves insulin sensitivity independently of weight loss, through increased GLUT4 expression in skeletal muscle and upregulation of the insulin signalling cascade. This is particularly relevant for a diabetic dog who cannot lose weight quickly; exercise can improve glucose handling even before significant weight loss has occurred.

The caveat for diabetic dogs is timing. Because exercise lowers blood glucose directly, by increasing glucose uptake into working muscle, exercising a diabetic dog that has recently received insulin can push glucose into the hypoglycaemic range. The standard practical guidance, drawn from AAHA and MSD/Merck clinical sources rather than a single named primary study on exercise timing specifically, is to keep exercise consistent in timing, duration, and intensity relative to meals and injections, and to test glucose before and after exercise until a reliable pattern is established.

Unspayed females

The hormonal mechanism, progesterone driving growth hormone excess, growth hormone causing insulin resistance, is covered in full in Chapter 1 of this Part, along with the critical practical points: that prompt spaying may allow remission, that remission is uncommon at approximately 1 in 10 of spayed bitches with ovarian-related conditions, and that timing matters because delayed spaying allows beta cell exhaustion. Those points are established there and are not repeated here.

For the purposes of this chapter, concurrent oestrus or dioestrus in an intact female is, mechanistically, a form of concurrent endocrine condition. Progesterone is the hormone disrupting insulin sensitivity, in the same way that cortisol does in Cushing's and thyroxine deficiency does in hypothyroidism. Any intact female in dioestrus who shows unexplained insulin resistance, or any newly diagnosed intact female, should be discussed with the vet for progesterone testing and spaying as a priority.

Cortisol and the acute stress response

The counter-regulatory hormone cascade triggered by hypoglycaemia, in which cortisol, glucagon, adrenaline, and growth hormone together push glucose back up, is covered in full in Chapter 2 of this Part in the context of Somogyi rebound. The same hormones are also released in response to any significant physical or emotional stress: pain, fear, infection, hot weather, veterinary visits, and changes in routine.

The practical implication for monitoring is important. A single blood glucose reading taken at the vet's surgery, or immediately after a stressful car journey, cannot be taken at face value. Stress-elevated glucose readings exist on the same background physiology as Somogyi-elevated readings. Fructosamine, covered in Chapter 4 of this Part, is the appropriate tool for distinguishing stress spikes from genuinely persistent hyperglycaemia, precisely because it reflects a two-to-three-week average rather than a single moment.

For owners managing at home: sustained stress, a period of pain, a concurrent illness, consistent anxiety, can shift a dog's glucose control gradually upward without any single dramatic event. If glucose readings start trending higher over days without an obvious dietary or dose explanation, a concurrent condition or a sustained stress response is the first place to look.

Pancreatitis as a concurrent condition

The fat-pancreatitis biochemistry and dietary management are covered in Chapter 3 of this Part. From the perspective of insulin resistance: acute or chronic pancreatitis produces significant systemic inflammation, with the same cytokine-mediated insulin resistance pathway as UTI and other infections. A dog in an acute pancreatitis episode typically shows severe insulin resistance and may need substantially higher insulin doses during the acute phase, followed by reassessment once the inflammation resolves. The same antibiotic-lag logic described earlier in this chapter applies here too: insulin sensitivity does not restore immediately when inflammation resolves.

DKA in the context of pancreatitis is covered in Chapter 2 of this Part, including the finding that 41% of DKA-presenting dogs had concurrent acute pancreatitis.

A practical framework for unexplained resistance

The following sequence, adapted from AAHA 2018 and MSD clinical guidance, is what the clinician works through when a well-established diabetic dog's glucose control deteriorates without obvious cause:

1. Rule out dietary change: different food, different portion, inconsistent feeding times.
1. Rule out UTI, using a urine culture, not a dipstick.
1. Rule out acute or chronic infection elsewhere: skin, ears, mouth, respiratory tract.
1. Consider cortisol-elevating concurrent conditions: Cushing's, hypothyroidism, pain, stress.

1. Consider intact female status and reproductive cycle.
1. Consider drug interactions; glucocorticoids are the most common iatrogenic cause of insulin resistance.
1. Reassess insulin storage, expiry, and injection technique.

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Chapter 6: Complications, the Science

This chapter goes underneath the long-term outlook material in Part 1, Chapter 8. It sets out the mechanisms behind the complications that develop over months and years of living with diabetes, rather than the acute emergencies covered earlier in this Part. Cataracts, urinary tract infections as a chronic burden rather than an acute disruption, pancreatitis as a bidirectional relationship with diabetes, sarcopenia's functional consequences, and diabetic neuropathy, an area where the evidence in dogs is genuinely weaker than in humans, and this chapter says so plainly rather than overstating it.

Cataracts

Why dogs are particularly vulnerable

Cataracts are by far the most common complication of diabetes in dogs, and they develop faster and more universally in dogs than in any other species. In a 1999 retrospective cohort study of 200 diabetic dogs, 75% developed cataracts within a year of diagnosis. Across multiple subsequent studies and clinical references, the consensus figure is that approximately 80% of diabetic dogs develop cataracts within 16 months of diagnosis, regardless of how well the diabetes is managed. Even dogs with good glycaemic control are at substantial risk.

The speed and frequency are explained by the lens's unique biochemistry. The crystalline lens lacks blood vessels and draws its nutrients entirely from the aqueous humour. Glucose enters the lens not by active transport but by simple and facilitated diffusion, a process that does not require insulin and therefore cannot be switched off when insulin is absent or reduced. When blood glucose is elevated, the lens is flooded with glucose proportionally.

The aldose reductase (polyol) pathway

Once inside the lens, excess glucose enters a two-step enzymatic pathway. First, aldose reductase converts glucose to sorbitol. Second,

sorbitol dehydrogenase converts sorbitol to fructose. This is called the polyol pathway; both sorbitol and fructose are polyols.

The problem is exit. Sorbitol is highly polar and cannot cross the lens cell membrane readily. It accumulates inside the lens fibres. As sorbitol concentration rises, it draws water into the cells by osmosis. The lens fibres swell, disrupt, and ultimately rupture. This swelling and fibre disruption is what produces the visible opacity, the cataract.

A secondary consequence is that fructose produced by the second step is also available for advanced glycation end product (AGE) formation through the Maillard reaction, the same process described from the dietary angle in Chapter 3 of this Part. AGEs accumulate within lens proteins, called crystallins, further altering their structure and contributing to opacity through a distinct mechanism from the osmotic swelling. Dogs have substantially higher aldose reductase activity in their lenses than humans, which explains why diabetic cataracts progress so much more rapidly in dogs.

Lens-induced uveitis: the complication within the complication

A maturing or hypermature cataract releases lens proteins into the aqueous humour, where the immune system recognises them as foreign. This triggers lens-induced uveitis (LIU), an inflammatory response inside the eye that is painful and, if chronic, damages the eye beyond the lens itself, causing glaucoma, retinal detachment, and in serious cases permanent vision loss independent of the cataract.

This is why the timing of cataract surgery matters. The standard veterinary advice is to refer to an ophthalmologist early, before LIU becomes established, rather than waiting until the cataract is visually obvious. Once active LIU is present, the prognosis for surgical recovery of useful vision is significantly worse.

Post-surgical vision outcome is good if LIU is controlled and surgery is timely. A 2023 study of phacoemulsification specifically in diabetic cataracts, 150 eyes across 76 dogs, found 94.8% of eyes in the

surgery group retained vision at last follow-up, compared with only 7.6% of eyes managed with topical medication alone. Diabetic dogs do still face somewhat worse long-term outcomes than non-diabetic dogs undergoing the same surgery, partly because the underlying metabolic disease continues to stress the eye after surgery, but the case for early surgical referral over medical management alone is strong.

Prevention, and realistic expectations

No dietary or pharmacological intervention has been shown to reliably prevent diabetic cataracts in dogs in clinical practice, though aldose reductase inhibitors have been studied experimentally. Rapid glucose control after diagnosis theoretically reduces the rate of sorbitol accumulation, but the speed of cataract development in dogs means this window is narrow. The more important message for owners is early ophthalmologist referral for surgical assessment before LIU establishes.

Urinary tract infections as a long-term complication

Chapter 5 of this Part covered how UTIs destabilise glycaemic control and why diabetic dogs are more vulnerable to infection. This section takes the complementary angle: UTIs are not just an acute disruption but a chronic, recurring complication that carries long-term consequences of its own.

The chronic burden

A diabetic dog whose glucose is persistently elevated will chronically produce glucose-rich urine, which creates a permanently altered urinary environment. This is not something that resolves with better control on a given day; it reflects the average glucose level over time. A dog who is moderately well controlled, with pre-injection readings consistently in the 12 to 16 mmol/L, 220 to 290 mg/dL, range, is still producing measurably glucose-enriched urine for many hours of every day. The result is an elevated UTI risk that persists throughout the dog's life with the disease.

A 2023 study of 107 diabetic dogs found 14% had a positive urine culture, and of those, the substantial majority (73.3%) showed no clinical signs at all, meeting the definition of subclinical bacteriuria. This is worth reading alongside a genuinely important nuance: the same study found no statistical association between clinical signs and culture results, and its authors concluded that routine urine culture in diabetic dogs without any signs of lower urinary tract disease may not be warranted, since much of what is found is subclinical bacteriuria that resolved without treatment or did not require it. This represents a meaningful shift from older guidance recommending culture at every recheck regardless of signs, and reflects a broader move in both human and veterinary medicine away from treating asymptomatic bacteriuria reflexively. Current practice varies, and this is worth discussing directly with your vet rather than assuming either position is settled.

Ascending infection and pyelonephritis

The more serious long-term concern is that recurrent or inadequately treated lower UTIs, when they are genuinely symptomatic, can ascend to the kidneys, causing pyelonephritis. Pyelonephritis is painful, causes significant systemic illness, and can cause irreversible renal damage. In a diabetic dog already at risk of renal AGE accumulation, covered in Chapter 3 of this Part, additional renal insult from recurrent upper UTI is a clinically meaningful concern.

Signs of pyelonephritis differ from simple lower UTI: fever, back or flank pain, vomiting, and systemic illness alongside urinary signs. Diagnosis requires urine culture, ideally a cystocentesis sample, plus renal imaging. Treatment requires a longer antibiotic course than lower UTI, typically six weeks rather than the two to four weeks standard for lower urinary tract infection in dogs.

Pancreatitis as a long-term complication and bidirectional relationship

Chapter 3 of this Part covered the dietary fat to free fatty acid mechanism for pancreatitis risk. Chapter 2 noted that 41% of dogs

presenting with DKA had concurrent acute pancreatitis. This section covers pancreatitis as a long-term complication of poorly controlled diabetes, and the two-way relationship between the two conditions.

How diabetes perpetuates pancreatitis risk

Insulin deficiency directly causes hypertriglyceridaemia, elevated triglycerides in the blood, through a specific mechanism: in the absence of insulin, lipoprotein lipase, an enzyme that breaks down circulating triglycerides, is not activated at the capillary wall. Triglyceride-rich particles therefore accumulate in the bloodstream rather than being cleared. As Chapter 3 described, high circulating triglycerides feed the free fatty acid toxicity pathway that damages acinar cells. Poor glycaemic control therefore perpetuates the very pancreatitis risk the dog started with, independent of dietary fat intake.

Exocrine pancreatic insufficiency (EPI) as an outcome

Repeated or chronic pancreatitis can destroy not only the endocrine tissue, the beta cells producing insulin, already gone in most diabetic dogs, but also the exocrine tissue: the acinar cells that produce digestive enzymes. When enough exocrine tissue is lost, the dog develops exocrine pancreatic insufficiency (EPI), in which the pancreas can no longer produce adequate digestive enzymes.

EPI produces a characteristic set of signs: large volumes of pale, foul-smelling, fatty stools, known as steatorrhoea, weight loss despite a ravenous appetite, and progressive nutritional deficiency despite eating adequately. In a dog already managing diabetes, the additional challenge of EPI, which alters nutrient absorption and therefore the glycaemic impact of every meal, makes management substantially more difficult.

EPI is diagnosed by serum TLI, trypsin-like immunoreactivity, a specific and sensitive test for exocrine pancreatic function in dogs. Treatment involves lifelong supplementation with pancreatic enzyme powder added directly to the food. It's worth noting that the key evidence linking EPI to pancreatitis as a specific end-stage

outcome comes from a small case series of four dogs; the underlying mechanism is well established, but the scale of the primary evidence in diabetic dogs specifically is limited, and most supporting evidence is extrapolated from the broader pancreatitis and EPI literature.

Sarcopenia (muscle wastage) as a long-term complication

Chapter 3 of this Part covered the protein catabolism mechanism: how insulin deficiency removes the anabolic signal that maintains muscle mass, and how accelerated proteolysis and gluconeogenesis perpetuate hyperglycaemia. This section covers the functional consequences of progressive muscle loss over time.

The progressive trajectory

Sarcopenia in a diabetic dog is not a one-time event. It is a progressive process that continues throughout the dog's life unless insulin therapy restores adequate anabolic signalling. A dog diagnosed at age seven may already have lost significant muscle mass by the time diabetes is identified; clinical signs typically appear only after approximately 90% of beta cell function is lost, meaning hyperglycaemia and its catabolic consequences have often been developing quietly for months before anyone knew the disease was present.

Under insulin therapy, the catabolic process slows substantially, but it does not fully reverse in dogs that were already significantly sarcopenic at diagnosis, and any period of poor glycaemic control resumes the catabolic cycle. The net effect over years of disease management is typically progressive muscle atrophy, particularly in the hindquarters, even in dogs who are otherwise doing well.

Functional consequences in the older dog

The hindlimb muscle groups primarily responsible for rising, climbing stairs, and maintaining balance are disproportionately affected. This produces a clinical picture, difficulty rising from lying, reluctance to climb stairs, stumbling or weakness on longer walks,

that is almost indistinguishable from osteoarthritis in an older dog. In a dog already being treated for joint disease, the contribution of diabetes-related sarcopenia can go unrecognised for extended periods.

This matters because the two conditions require somewhat different management responses. Joint disease is managed with analgesia, controlled exercise, and joint supplementation. Sarcopenia requires dietary protein adequacy, consistency of insulin therapy, and appropriate muscle-maintaining exercise. Getting one right without recognising the other leaves the dog underserved.

The muscle-glucose-insulin feedback loop

Chapter 3 noted that muscle is the body's primary post-meal glucose sink. Less muscle means less capacity to buffer post-meal glucose spikes, which makes glucose harder to control, which accelerates further sarcopenia through the catabolic pathway. This loop is worth restating here from the complications angle: sarcopenia is not just a consequence of diabetes, it actively worsens the disease it results from. It is self-reinforcing unless actively countered.

The practical intervention, higher dietary protein alongside consistent insulin therapy, with appropriate exercise, was covered in Chapter 3. From a complications standpoint, the point worth emphasising is that catching and addressing muscle loss early produces better long-term outcomes than waiting until it is clinically obvious.

Diabetic neuropathy

This section needs to open with a statement about the strength of the evidence, because the honest picture in dogs is genuinely different from the picture in humans or cats, and overstating it would do readers a disservice.

Diabetic peripheral neuropathy in humans is one of the best-characterised long-term complications of diabetes, affecting over 50% of people with long-standing disease and producing sensory loss, neuropathic pain, and autonomic dysfunction. In diabetic cats,

a clinical peripheral neuropathy causing a plantigrade stance, cats walking on their hocks rather than their toes, is well recognised and frequently referenced in veterinary literature.

In dogs, the evidence is materially weaker. Subclinical neurophysiological changes have been documented in diabetic dogs, but clinically symptomatic peripheral neuropathy, the kind that presents as obvious gait changes, pain, or measurable sensory loss, is not established as a common or well-defined complication in dogs with the same degree of confidence as it is in humans. This chapter reflects that honestly rather than implying diabetic neuropathy is a common, well-characterised complication in dogs.

What is established: subclinical electrophysiological changes

Several studies have used nerve conduction velocity testing and electromyography in diabetic dogs and found measurable slowing of nerve conduction and abnormal findings compared with non-diabetic controls. These changes are consistent with the pattern seen in early human diabetic neuropathy. They confirm that the disease process, high glucose causing sorbitol accumulation in Schwann cells and nerve fibres through the same polyol pathway as cataracts, combined with AGE accumulation in myelin proteins, is active in the dog's nervous system.

The mechanism

The polyol pathway operates in Schwann cells, the cells that form the myelin sheath around peripheral nerve fibres, in the same way it operates in lens cells, accumulating sorbitol and causing osmotic stress. Myelin sheath damage slows nerve conduction velocity. AGE formation in myelin proteins alters their structural integrity. Impaired blood flow to peripheral nerves, through the same triglyceride-mediated vascular mechanism described in the pancreatitis section above, causes ischaemic nerve damage independently of the polyol pathway.

The result is progressive damage to both large myelinated fibres, which carry touch, vibration, and proprioception signals, and small unmyelinated fibres, which carry pain and temperature information.

What owners may observe, framed with appropriate uncertainty

Given the evidence available, the following are what may represent early neuropathic change in a diabetic dog, though none of these signs is pathognomonic and all have other, far more common explanations: altered sensation in the paws, such as flinching at light touch or a reduced withdrawal reflex; postural instability or delayed repositioning of the feet when placed in an abnormal position; and unexplained weakness not attributable to sarcopenia, joint disease, or concurrent orthopaedic disease.

These signs should be evaluated by a vet for their more common causes first, sarcopenia, joint disease, spinal disease, and only attributed to neuropathy if those have been adequately excluded. There is no large-scale prevalence study of clinical neuropathy in spontaneously diabetic dogs. The condition may be underdiagnosed, undertreated, or genuinely less prevalent in dogs than in humans or cats; the honest answer is that nobody currently knows which.

Autonomic neuropathy

Autonomic neuropathy in human diabetes causes gastroparesis, bladder dysfunction, orthostatic hypotension, and impaired heart rate variability. In dogs, autonomic neuropathy as a consequence of diabetes has been described in case reports but is not established as a recognised clinical complication in population-level data.

The one exception worth knowing: a dog with chronic vomiting, delayed gastric emptying, or unexplained oesophageal or gastric dysmotility alongside diabetes could, in principle, have an autonomic component. This is speculative rather than established in dogs, and is mentioned here with that caveat rather than as settled fact.

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Chapter 7: Prognosis, Quality of Life, and the Long View

This chapter goes underneath Part 1, Chapter 8. It sets out the actual survival data behind the reassurance given there, what predicts a better or worse outcome, what the quality-of-life research says in detail, and the evidence base for caregiver burden, the psychological weight of managing a chronic condition, which turns out to be measurable, common, and directly reducible by understanding what you are doing and why.

Survival data: what the studies actually show

The large population study

The most robust survival data for canine diabetes comes from a 2007 Swedish study examining 180,000 insured dogs across a multi-year period. This study, Fall T, Hansson Hamlin H, Hedhammar A, Kampe O, Egenvall A, provides the largest and most epidemiologically robust survival dataset for the condition. Incidence was highest in middle to older aged dogs, with most diagnoses between seven and ten years of age. Survival curves showed the steepest mortality in the period immediately following diagnosis. Breed effects were substantial, and are detailed in Chapter 1 of this Part.

A referral hospital cohort with precise outcome data

A 2019 single-centre study, Tardo AM, et al., published in Veterinary Record, followed 68 dogs with newly diagnosed diabetes at a referral teaching hospital and found a median survival time of 964 days, with a range of 22 to 3140 days. This is the source of the 964-day figure referenced in Part 1, Chapter 8; it works out to approximately 2.6 years. The study also found that a higher haematocrit and higher serum phosphate concentration at diagnosis were independently associated with shorter survival, and that the

presence of concurrent pancreatitis was not necessarily associated with a worse outcome.

What the survival curve actually means

The clinical pattern is consistent across studies. Mortality risk is highest in the first six months after diagnosis. This is when DKA, acute concurrent illness, the management learning curve, and the owner's decision to discontinue treatment account for most early deaths. Dogs that stabilise through this initial period have substantially improved prognosis. The Part 1 framing, three to five years, plenty go on for eight to ten, is consistent with the published data for well-managed dogs without major concurrent disease.

Dogs euthanased at diagnosis or within the first few weeks drag down the population median substantially, which means a median survival figure can understate the realistic expectation for an owner who commits to management. A 2017 survey of 1,192 veterinarians worldwide, Niessen SJM, Hazuchova K, Powney SL, et al., found a median of one in ten diabetic dogs is euthanased at the point of diagnosis, and a further one in ten within the first year, most commonly because of concurrent disease, cost, the animal's age, difficulty achieving control, or the impact on the owner's lifestyle, not because of a poor biological prognosis on its own. This matters for reading the survival statistics correctly: a meaningful share of the dogs pulling the population median downward were never given the chance to respond to treatment at all. The relevant figure for an owner whose dog has just been stabilised is the conditional survival, survival given that the dog has already survived the first month, which is considerably more optimistic than the raw population median.

Outcome predictors

Several factors have been consistently identified across clinical studies as influencing survival in diabetic dogs.

Concurrent disease at diagnosis is the most powerful predictor. A dog diagnosed with diabetes alone has substantially better prognosis

than a dog diagnosed with concurrent pancreatitis, Cushing's disease, or hypothyroidism. Chapter 5 of this Part covered how each condition complicates management; from a prognosis standpoint, the impact is not just on short-term glucose control but on survival duration.

DKA at presentation carries a meaningful mortality risk during that hospitalisation, approximately 30% in the largest published outcome study, covered in full in Chapter 2 of this Part. Of those who survive to discharge, long-term prognosis is not necessarily worse than dogs diagnosed without DKA; surviving the initial crisis is the key threshold.

Age at diagnosis matters in an arithmetic rather than a management sense. Older dogs have shorter expected remaining lifespan regardless of diabetes; a dog diagnosed at twelve has less capacity for the three-to-five-year survival range simply because of age. This is not a failure of management. The relevant question for an older dog is the quality of the remaining life, not its duration.

Sex carries the specific exception discussed fully in Chapter 1 of this Part: progesterone-driven diabetes may resolve after spaying. For other dogs, sex does not independently predict survival once concurrent disease is accounted for.

Glycaemic control quality is the factor most directly within an owner's and vet's influence. Chronic poor control drives the complications covered in Chapter 6 of this Part, cataracts largely independent of control, UTI burden, pancreatitis perpetuation through hypertriglyceridaemia, and sarcopenia, which in turn shorten functional survival. Dogs with consistently good glucose curves live longer and better than dogs with persistently elevated or erratic readings.

Quality of life: what the research shows

The QoL instrument for diabetic dogs

Part 1, Chapter 8 cited the headline findings: 81% of owners rated their dog's overall quality of life as good, while 84% reported some

diabetes-specific negative impact. The study behind these figures, Niessen SJM, Powney S, Guitian J, Niessen APM, Pion PD, Shaw JA, Church DB, developed and validated a diabetes-specific quality-of-life tool, rather than adapting a generic animal QoL scale, by combining owner questionnaire data across multiple domains: physical health, emotional state, ability to perform normal activities, and owner-dog relationship quality. The tool was validated in a population of diabetic dogs in the UK, making it directly relevant to this book's audience.

The apparent contradiction between the 81% overall positive rating and the 84% reporting some negative impact reflects a genuine phenomenon in chronic disease management: people assess overall quality of life holistically, as a gestalt, while domain-specific questions reveal impacts that are real but do not, taken individually, tip the overall assessment negative. Both findings are honest. They are measuring different things.

The HHHHHMM scale

The HHHHHMM quality of life scale, mentioned in Part 1, Chapter 8, was developed by Alice Villalobos primarily in the context of oncology and end-of-life decision making, and has been widely adopted across chronic disease contexts. The seven domains are: Hurt, Hunger, Hydration, Hygiene, Happiness, Mobility, and more good days than bad. Each is scored from 1 to 10, with a score above 35 out of 70 generally indicating an acceptable quality of life.

Its strength is that it converts a subjective, emotionally loaded question, is my dog suffering, into a structured, repeatable assessment that an owner can run at intervals, comparing over time rather than relying on memory of a bad day. Its limitation is that it was designed for oncology, and some domains map less naturally to a well-managed diabetic dog than to a dog with terminal cancer. Hunger and hydration, for example, are directly affected by diabetic symptoms in ways that do not straightforwardly indicate suffering; a diabetic dog may drink excessively because of the disease, not from dehydration.

What good actually looks like in a stable diabetic dog

The Niessen et al. 2012 study found something worth knowing on its own terms: owners consistently underestimated, in retrospect, how distressing they had expected the management to be compared with what it turned out to be in practice. The adjustment burden, learning to give injections, run curves, interpret readings, was consistently heavier in the early weeks, and eased substantially once routine was established. This is not spin. It reflects genuine, documented adaptation, not simply owners becoming resigned to a difficult situation.

Caregiver burden

The anchor study and what it established

The foundational research on caregiver burden in companion animal ownership, and the study that places this experience in a formally measured scientific context, is Spitznagel MB, Jacobson DM, Cox MD, Carlson MD, published in Veterinary Record in 2017. This study adapted the Zarit Burden Interview, the gold-standard human caregiver burden measurement tool, for veterinary use and applied it to 238 owners of animals with confirmed chronic illness. It found measurable caregiver burden comparable in magnitude to human caregiver burden studies, levels previously assumed to belong only to the human context.

The single most actionable finding from this study: owner satisfaction with their veterinarian and with their treatment plan was a statistically significant predictor of lower caregiver burden. Owners who felt confident in the clinical management, who understood what was being done and why, carried measurably less burden than those who felt uncertain or unsupported. This finding has been replicated in subsequent studies across different chronic animal conditions.

This is the evidence base for why explanation matters. Understanding insulin pharmacokinetics, reading a glucose curve, knowing what fructosamine is and what it cannot tell you: these are

not academic exercises. They measurably reduce the psychological weight of caregiving.

Subsequent replications

The Spitznagel group published several follow-on studies that confirmed and extended the 2017 findings. A 2018 study, published in the Veterinary Journal, found that the animal's visible clinical signs and behavioural problems were the strongest predictors of owner burden, more than disease severity on objective clinical measures. In practical terms, an animal who looks unwell is more distressing for an owner than one who is ill but appears normal day to day.

The prevalence of caregiver burden

Part 1, Chapter 8 stated that roughly half of owners caring for a pet with a chronic illness show meaningful signs of caregiver burden. A 2019 validation study by the same research group, published in the Journal of Veterinary Internal Medicine, states directly that approximately half of owners providing care for a seriously ill companion animal demonstrate significant caregiver burden, consistent with the figure used in Part 1.

What caregiver burden looks and feels like

The adapted Zarit Burden Interview covers domains including time burden, how much of the day is consumed by management, emotional burden, anxiety, grief, guilt, role burden, the sense that caregiving is taking over other aspects of life, and social burden, isolation, inability to travel or maintain social routines normally. A diabetic dog's twice-daily injection and feeding schedule, the need for home testing, the monitoring for hypoglycaemia, and the avoidance of travel that disrupts the regime all map directly onto these burden domains.

Guilt is a specific feature worth addressing explicitly. Because diabetes in dogs is associated with obesity in some cases, and because insulin management errors can have serious consequences,

owners frequently experience guilt: about the diet that may have contributed, about an injection that caused a hypoglycaemic episode, about a glucose curve that stayed high despite their efforts. The research context is clear on this point: caregiver burden and feelings of guilt occur in owners doing everything correctly, not as markers of mismanagement.

What helps

The evidence from the Spitznagel research programme points to three things. First, veterinary communication and confidence in the treatment plan. Owners who understood their dog's management, who had been given clear explanations and felt their vet's guidance was trustworthy, showed lower burden scores. This is an actionable finding: seeking a second opinion if the first explanation felt unclear, or specifically asking for more explanation at the next visit, has a measurable likely benefit.

Second, peer support. Owner forums and support communities function as a practical coping resource. The normalising effect of discovering that other owners face the same challenges, that a glucose curve that baffled them also baffled experienced owners initially, that their dog's resistance to ear testing is standard rather than a personal failure, reduces isolation and perceived burden.

Third, knowing that the animal's quality of life is good. The 2018 finding that perceived animal wellbeing is the strongest predictor of burden has a converse implication: a dog who appears well, engaged, and comfortable provides its owner with direct reassurance that the caregiving effort is worthwhile. This is the most honest and practical case for good glycaemic control, not just the dog's health, but the owner's mental health in managing it.

The longer view: what owners report

A consistent pattern across QoL studies, forum data, and clinical reports is that diabetic dog ownership, after the initial adjustment period, normalises. The injection becomes routine. The twice-daily schedule structures the day rather than overwhelming it. Owners

who were frightened by the diagnosis at week one describe the management as no big deal by month three, not because the effort has decreased but because it has become part of life rather than an emergency.

This is not spin. It reflects genuine adaptation, documented in the research: the same Niessen et al. 2012 finding that owners consistently underestimated, in retrospect, how distressing they had expected the management to be compared with what it turned out to be in practice.

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