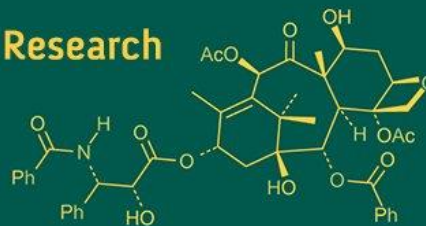


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Postprandial glycemia in dogs: A review of dietary determinants, mechanisms and evidence

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Abstract

Postprandial glycemia the rise in circulating glucose that follows ingestion of a meal is increasingly treated not as a fixed property of the animal but as a modifiable metabolic signal. In dogs the postprandial curve differs from the human one in both shape and timing, reaching its maximum between two and four hours after ingestion rather than at approximately one hour, and its magnitude is determined substantially by what is fed. This review sets out the physiological basis of postprandial glucose regulation in the dog, examines the contribution of each macronutrient class, evaluates carbohydrate and fibre-based formulation strategies, surveys nutraceutical interventions organised by anatomical site of action, and appraises four recent feeding trials in detail. The evidence supports a reasonably firm conclusion about starch: source, structure and processing appear to matter at least as much as quantity, and diets built on slowly digestible starch from pulses or intact whole grains produce lower and flatter glucose responses than those built on refined cereals. Beyond that the picture is less settled than the applied literature usually acknowledges. One trial that held starch content nearly constant across diets recovered significant source-driven differences, while another that varied nitrogen-free extract almost fourfold did not reach conventional significance on any glucose endpoint. Dietary fibre and protein have well-described mechanisms but modest and inconsistently quantified effects in the dog specifically. Nutraceutical evidence is thinner still, and rests heavily on *in vitro* assays, rodent models and human trials rather than canine feeding studies. Four structural weaknesses run through the whole body of work: sample sizes of nine to sixteen animals, feeding periods of one to seven weeks, near-exclusive use of healthy rather than dysglycemic dogs, and reliance on glycaemic index values generated in humans and never validated in this species. The review concludes with a set of research priorities that follow from these gaps.

Keywords: Postprandial glycemia, dog, glycaemic index, slowly digestible starch, dietary fibre, nutraceuticals, insulin, glucagon

1. Introduction

Postprandial glycemia refers to the concentration of glucose in the blood measured after a meal, conventionally at one to two hours after ingestion in human clinical practice (American Diabetes Association Professional Practice Committee, 2024) [2]. That convention does not transfer cleanly to the dog. Canine postprandial glucose typically reaches its zenith between two and four hours after ingestion, and the interval to peak varies with the composition of the meal (Rucinsky *et al.*, 2010) [47]. A measurement protocol imported from human medicine without adjustment therefore risks sampling the canine curve at the wrong moment. This is not a pedantic observation; as will become clear, the sampling schedules adopted in the canine feeding trials reviewed here vary from four postprandial points to sixteen, and the sparser schedules cannot in principle resolve a peak whose timing is itself diet-dependent. The case for treating postprandial glycemia as an endpoint worth managing rests on several distinct arguments, which are worth separating. It reflects metabolic flexibility and the integrity of glucose regulation (Dörner *et al.*, 2025) [13]. It functions as an early indicator of impaired glucose control, detectable before fasting values shift (Thomas & Elliott, 2009) [54]. Although, persistent elevation is associated with metabolic disorders in dogs (Nelson & Couto, 2019) [42]. It is strongly influenced by diet composition and feeding pattern. And, critically, it can be modulated through targeted nutritional strategies (Sidhu & Randhawa, 2019) [49]. The last of these is what distinguishes postprandial glycemia from most other

metabolic markers available to the small-animal clinician: it is not merely diagnostic but tractable, and tractable through a route the food bowl that the owner already controls. The clinical burden gives the question its weight. Diabetes mellitus in dogs has been estimated at a prevalence of approximately one in three hundred (Federation of European Companion Animal Veterinary Associations, 2025) ^[16], and epidemiological work drawn from primary-care veterinary practice indicates a measurable and non-trivial prevalence, with risk rising in association with obesity, advancing age and breed predisposition (Mattin *et al.*, 2014) ^[35]. Onset peaks at seven to nine years, the condition is more common in middle-aged female dogs, and spayed females carry a reported four to ten-fold increased risk (American Veterinary Medical Association, n.d.). Against this, a substantial majority of affected dogs reported in the source material as seventy to eighty percent maintain quality of life under appropriate feeding management and exercise (Fall *et al.*, 2007; O'Neill *et al.*, 2013; Graves & Gilor, 2023) ^[15, 23, 43]. The denominator for that figure is not specified in the material reviewed, and it should be read as indicative rather than precise.

2. Physiological Regulation of Postprandial Glycemia in the Dog

Postprandial glycemia is regulated through coordinated interaction between intestinal absorption, hormonal signalling and peripheral tissue metabolism. It is conventionally decomposed into four sequential phases, and the decomposition is useful because different dietary interventions act at different points in the sequence.

2.1 Intestinal phase: Digestion and absorption

Dietary starch is hydrolysed to glucose by amylase and by brush-border enzymes of the small intestine. The liberated glucose crosses the enterocyte by way of two transporters: SGLT-1, which mediates sodium-coupled uptake at the apical membrane, and GLUT-2, which handles glucose

transport in both intestine and pancreas. Absorbed glucose then enters the portal circulation (Vastolo *et al.*, 2023) ^[56]. This phase is where the rate-limiting properties of the food matrix exert themselves: anything that slows enzymatic access to starch granules, or slows the delivery of digesta to the absorptive surface, acts here.

2.2 Pancreatic phase: Insulin secretion

Rising portal and systemic glucose is sensed by the pancreatic β -cell, which takes up glucose through GLUT-2. Intracellular metabolism raises the ATP:ADP ratio, closing ATP-sensitive potassium channels; the resulting membrane depolarisation opens voltage-gated calcium channels, and calcium influx triggers exocytosis of insulin-containing granules into the circulation (Holst *et al.*, 2016) ^[27]. The sequence is canonical mammalian physiology, and the sources reviewed here do not establish dog-specific quantitative parameters for any step in it.

2.3 Circulatory phase

Blood glucose concentration rises and insulin circulates alongside it. This is the phase that the glucose curve actually measures, and it is worth keeping in view that a postprandial glucose value is a net quantity the balance of appearance from the gut and disappearance into tissue rather than a direct readout of either process.

2.4 Peripheral phase: Utilisation and storage

In muscle and adipose tissue, insulin signalling drives translocation of GLUT-4 to the plasma membrane and consequent glucose uptake, feeding glycolysis for energy production and, in adipose tissue, lipogenesis. In the liver, glycogenesis is upregulated and gluconeogenesis suppressed (Nelson & Couto, 2019) ^[42]. The three metabolic pathways engaged glycolysis, glycogenesis, and inhibition of gluconeogenesis together determine how rapidly the circulating glucose load is cleared, and therefore the descending limb of the curve.

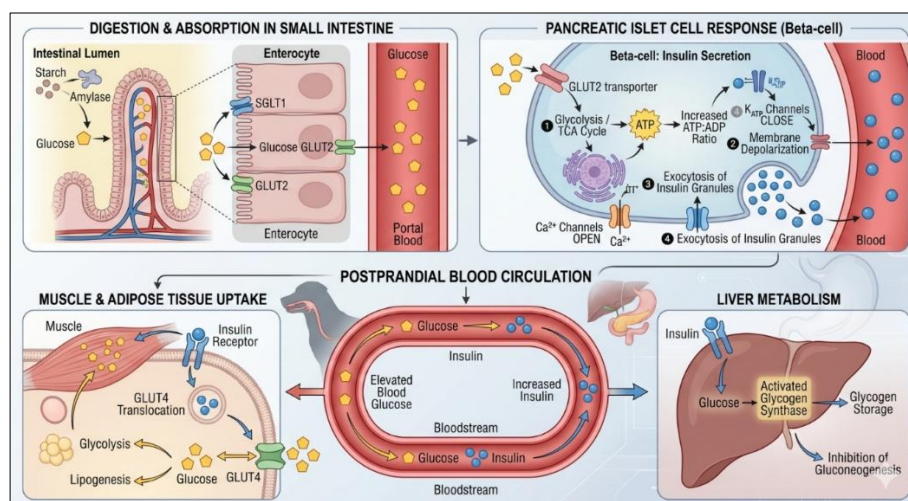


Fig 1: Postprandial glycemia is regulated through coordinated interactions between intestinal absorption, hormonal signalling and peripheral tissue metabolism

3. Macronutrient Determinants of the Glycaemic Response

3.1 Carbohydrate: Quantity, digestibility and processing

The amount of digestible carbohydrate in practice, starch is described as the main determinant of postprandial glucose response in dogs (Carciofi *et al.*, 2008; Hewson-Hughes *et*

al., 2011) ^[10, 26]. Higher starch intake raises postprandial glucose concentration; lower digestible carbohydrate lowers the glycaemic response (Hall *et al.*, 2013) ^[24].

Quantity is only half the account. The rate at which starch is digested matters independently. Rapidly Digestible Starch (RDS) characteristic of processed cereals and gelatinised

starch produces a sharp glucose spike, whereas slowly digestible starch (SDS), found in whole grains and minimally processed starch sources, gives a sustained and lower-amplitude release. Processing operations, extrusion in particular, increase starch digestibility and consequently raise the glycaemic response (Singh *et al.*, 2010; Vastolo *et al.*, 2023) [56]. Meynier *et al.* (2015) [38, 51] report that SDS, fat, fibre and RDS collectively explain approximately fifty-three per cent of the variability in glycaemic response, with food-matrix interactions exerting significant influence. That figure deserves more attention than it usually receives. If the

four compositional variables routinely managed in formulation account for slightly over half the variance, then nearly half of the variability in glycaemic response is attributable to something else matrix architecture, particle size, processing history, individual animal variation, or measurement error. A formulation strategy built exclusively on the four measured variables is therefore working with an incomplete model, and the residual should temper confidence in predicting the glycaemic behaviour of a novel diet from its proximate analysis alone.

Table 1: Reported glycaemic index values for carbohydrate ingredients relevant to canine diet formulation

Class	Ingredient	Glycaemic index	Classification
Cereals	Barley (whole)	25-35	Low
	Oats (rolled/whole)	40-55	Low-moderate
	Wheat (whole)	45-60	Moderate
	Wheat (white bread/flour)	70-85	High
	Maize (corn products)	52-70	Moderate
	Rice (white, polished)	70-89	High
Millets	Sorghum (jowar)	62-68	Moderate
	Pearl millet (bajra)	54-68	Moderate
	Finger millet (ragi)	65-84	Moderate-high
Legumes	Lentils	28-38	Low
	Chickpeas	28-36	Low
	Peas	35-48	Low
Vegetables	Potato (processed)	78-95	High

Source: FAO/WHO (1998) [17]; Foster-Powell *et al.* (2002) [18]; Atkinson *et al.* (2021) [5].

3.2 Protein

Protein influences the glucose response through three mechanisms operating in different phases. Amino acids stimulate insulin secretion, which reduces the glucose excursion; protein slows gastric emptying, which delays glucose absorption; and it enhances peripheral glucose uptake, improving clearance. When protein is co-ingested with carbohydrate, the composite glycaemic response is modified relative to carbohydrate alone, with improved post-meal glucose regulation (Banton *et al.*, 2025; Holst *et al.*, 2016) [7, 27]. Protein is thus unusual among the macronutrients in acting on both the appearance and the disposal of the balance simultaneously.

3.3 Fat

Fat slows gastric emptying, delaying delivery of nutrients to the small intestine and reducing the rate at which glucose appears in the circulation; the result is a delayed and blunted glycaemic response (Gentilcore *et al.*, 2006) [21]. The acute effect is therefore favourable. The chronic effect is not: sustained high fat intake is associated with increased risk of insulin resistance. Fat consequently sits awkwardly in any glycaemic management strategy, because the same nutrient that improves the shape of today's postprandial curve may, at sustained high inclusion, degrade the insulin sensitivity on which tomorrow's curve depends. The evidence base compounds the difficulty: Gentilcore *et al.* (2006) [21] studied human subjects with type 2 diabetes, and the magnitude of the acute effect in dogs is not established by the sources reviewed here.

3.4 Dietary fibre

Fibre alters ingesta viscosity, modulates gastrointestinal motility, and influences nutrient digestibility and absorption. Five mechanisms are described in the material reviewed. Soluble fibre forms a gel in the intestine that slows glucose

diffusion and delays carbohydrate digestion and absorption (Thorne *et al.*, 2020) [55]. Increased viscosity reduces enzyme-substrate interaction and thereby the rate of glucose appearance in blood (Meynier *et al.*, 2015) [38]. Delayed gastric emptying slows nutrient delivery to the intestine and blunts the postprandial peak. Fermentation yields short-chain fatty acids, principally acetate and propionate, which enhance peripheral glucose utilisation and improve insulin sensitivity (de Godoy *et al.*, 2013) [12]. And fibre modulates the gut microbiota, promoting beneficial populations and their production of metabolites that regulate glucose metabolism (Moreno *et al.*, 2022) [40]. The five mechanisms are not equally well evidenced in the dog, and they are not additive in any simple way. The first three are physical and operate acutely within a single meal; the last two are microbial and metabolic, operate over days to weeks, and depend on fermentability rather than viscosity. A diet formulated for acute curve-flattening and one formulated for chronic insulin sensitisation would therefore select different fibre sources, and conflating the two objectives under the single heading of "fibre" obscures a real formulation decision.

4. Dietary Strategies

4.1 Carbohydrate-based strategies

Six carbohydrate-directed strategies emerge from the mechanistic account. The first is ingredient selection: preferring low-glycaemic sources such as barley, oats and legumes, and limiting maize, rice and wheat flour, which are high-glycaemic when processed (Atkinson *et al.*, 2021) [5]. The second is deliberate incorporation of slowly digestible starch from pulses and whole grains to promote gradual glucose release (Meynier *et al.*, 2015) [38]. The third is limitation of rapidly digestible starch by avoiding highly processed and gelatinised starch and controlling the inclusion of refined cereals (Augustin *et al.*, 2015) [6]. The

fourth is optimisation of processing: coarse grinding in preference to fine, and avoidance of excessive extrusion and gelatinisation, since processing raises starch digestibility and with it the glycaemic response (Singh *et al.*, 2010) ^[51]. The fifth is nutrient balancing combining carbohydrate with fibre, fat and protein to slow absorption and improve the insulin response (Gannon & Nuttall, 2010) ^[20]. The sixth is control of total carbohydrate inclusion, avoiding excessive carbohydrate density and adjusting to metabolic status (Elliott *et al.*, 2012) ^[14].

These are formulation heuristics derived from mechanism, and it is worth being explicit that most of them have not been tested head-to-head in dogs. Of the six, only the first two receive reasonably direct support from the canine trials examined in Section 6, and the fourth, the processing recommendation rests substantially on food-science work conducted outside the species.

4.2 Fibre-based strategies

Five fibre-directed strategies are described. Soluble and viscous fibres such as psyllium, β -glucans from oats and barley and pectins are identified as most effective for glycaemic control (Jenkins *et al.*, 2002) ^[29]. Moderately fermentable fibres such as beet pulp, inulin and fructo-oligosaccharides support metabolic regulation (Swanson *et al.*, 2002) ^[53]. Fibre types should be balanced, combining

soluble fibre for glycaemic control with insoluble fibre for gut motility (Reynolds *et al.*, 2019; de Godoy *et al.*, 2013) ^[20, 45]. Inclusion level should be optimised, since moderate levels give the best glycaemic response while excess fibre depresses nutrient digestibility (de Godoy *et al.*, 2013) ^[12]. Finally, functional additives including prebiotics and resistant starch may be used to engage the gut-metabolic axis (Roberfroid *et al.*, 2010) ^[46].

The fourth of these that the dose-response is non-monotonic, with an optimum rather than a ceiling is the practically important one, and it is also the least well quantified. No inclusion level is specified in the material reviewed, and the trade-off point between glycaemic benefit and digestibility loss will depend on fibre source, the rest of the diet, and the metabolic status of the animal.

5. Nutraceutical Interventions

Nutraceutical candidates are usefully organised by anatomical site of action, since this determines both the expected mechanism and the kind of evidence that would be required to demonstrate efficacy. Table 2 summarises the compounds described, grouped into intestinal modulators, agents acting on pancreatic insulin secretion, systemic agents acting on hepatic and whole-body metabolism, and peripheral agents acting on muscle and tissue uptake.

Table 2: Nutraceutical candidates for modulation of postprandial glycemia, by site of action

Ingredient	Active compound	Mechanism	Reported effect	Reference
Intestinal modulators				
Moringa	Peptides, polyphenols	α -Amylase and α -glucosidase inhibition	↓ PPG peak	Wang <i>et al.</i> (2025) ^[57]
Basil	Flavonoids	Amylase inhibition; ↑ insulin	↓ glucose (~31%)	Abdelrahman <i>et al.</i> (2020) ^[1]
Jamun	Jamboline	Delays starch digestion	↓ PPG	Kochhar <i>et al.</i> (2005) ^[32]
Fenugreek	Galactomannan	Gel formation slows absorption	↓ glycemia	Singletary (2017) ^[51]
Gymnema	Gymnemic acids	↓ intestinal glucose absorption	↓ glucose	Mayyas <i>et al.</i> (2025) ^[36]
Dietary fibre	β -Glucan, pectin	↑ viscosity, ↓ absorption	↓ PPG spike	de Godoy <i>et al.</i> (2013) ^[12]
FOS	Fructans	SCFA production	Indirect ↓	Makki <i>et al.</i> (2018) ^[34]
Flaxseed	Lignans, fibre	Delays glucose absorption	↓ spikes	Moreira <i>et al.</i> (2022) ^[39]
Pancreatic (insulin response)				
Fenugreek	4-Hydroxyisoleucine	↑ insulin secretion	↓ glycemia	Singletary (2017) ^[51]
Basil	Eugenol, flavonoids	↑ insulin levels	↓ glucose	Abdelrahman <i>et al.</i> (2020) ^[1]
Chromium	Trace mineral	Enhances insulin signalling	↑ glucose tolerance	Gupta <i>et al.</i> (2019) ^[23]
Zinc	Trace mineral	Insulin storage and secretion	Indirect ↓	Jansen <i>et al.</i> (2009) ^[28]
Systemic (liver and whole-body metabolism)				
Cinnamon	Procyanidins	Improves insulin sensitivity	Mild ↓	Gupta <i>et al.</i> (2019) ^[23]
Turmeric	Curcumin	Anti-inflammatory, insulin sensitiser	Indirect ↓	Suemantham <i>et al.</i> (2022) ^[52]
Rosemary	Rosmarinic and carnolic acid	Antioxidant, metabolic regulation	↓ glucose	Abdelrahman <i>et al.</i> (2020) ^[1]
Polyphenols (general)	Flavonoids, tannins	Multi-target metabolic effects	↓ glycemia	Ruiz-Cano & Arnao (2024) ^[48]
Peripheral (muscle and tissue uptake)				
Berberine	Isoquinoline alkaloid	AMPK activation	↑ glucose uptake	Freitas <i>et al.</i> (2025) ^[19]
Green tea	Catechins	↑ glucose uptake	Mild ↓	Li <i>et al.</i> (2020) ^[33]
Magnesium	Enzyme cofactor	Improves insulin sensitivity	Indirect ↓	Barbagallo & Dominguez (2015) ^[8]
Vanadium	Trace element	Insulin-mimetic action	↓ glucose	Kim <i>et al.</i> (2006) ^[31]

PPG = postprandial glycemia; FOS = fructo-oligosaccharides; SCFA = short-chain fatty acids.

The table conceals a steep gradient in evidence quality, and reading across it uncritically would be a mistake. Only a minority of the entries rest on canine feeding studies. Abdelrahman *et al.* (2020)^[1] worked in Rottweilers, Li *et al.* (2020)^[33] in dogs with diet-induced obesity, Suemanotham *et al.* (2022)^[52] in dogs with diabetes mellitus, and Wang *et al.* (2025)^[57] in beagles. Kim *et al.* (2006)^[31] studied dogs, but using alloxan-induced diabetes, a chemical model whose relationship to spontaneous canine diabetes is not straightforward. Moreira *et al.* (2022)^[39] is a human crossover trial in type 2 diabetes. Mayyas *et al.* (2025)^[36] is a computational and network-pharmacology study with no animal feeding component at all. Makki *et al.* (2018)^[34] and Barbagallo and Dominguez (2015)^[8] are reviews of general mechanism rather than canine trials. A compound's presence in Table 2 therefore indicates that a plausible mechanism has been proposed, not that efficacy has been demonstrated in the target species.

The moringa data illustrate why the distinction matters quantitatively as well as categorically. Wang *et al.* (2025)^[57] report that moringa leaf peptide inhibited α -amylase with a half-maximal inhibitory concentration of 2.29 ± 0.10 mg/mL and α -glucosidase at 2.80 ± 0.04 mg/mL, against acarbose values of 0.06 mg/mL and 1.02×10^{-3} mg/mL respectively. Expressed as a ratio, the peptide is roughly thirty-eight-fold less potent than acarbose against α -amylase and something on the order of three orders of magnitude less potent against α -glucosidase. Describing the two as showing "similar inhibition" with the pharmaceutical merely having "higher potency" understates the gap considerably. The *in vivo* effect that was reported at three per cent dietary inclusion is therefore unlikely to be attributable to pharmacological enzyme blockade at concentrations achievable in the gut lumen, and some other mechanism matrix effects, viscosity, or displacement of digestible starch should be considered.

6. Experimental Evidence

Four studies form the empirical core of this review. They differ in design, in the dietary contrast tested, and importantly in the density of postprandial sampling, which bears directly on what each can and cannot establish.

6.1 Grain-free versus cereal-based diets

Vastolo *et al.* (2023)^[56] used a crossover design with repeated measures in fifteen healthy dogs, feeding each of three diets for fifty days and sampling blood at baseline and at 120, 240 and 360 minutes post-feeding for glycemia and insulin. The three diets were closely matched in energy are 3990, 3990 and 3997 kcal/kg metabolisable energy and more importantly, in starch content: 25 per cent in the first

grain-free diet (sweet potato), 28 per cent in the second grain-free diet (pea starch), and 27.9 per cent in the cereal-based diet (oats and spelt). The protein and fat backbone was essentially identical across all three, based on boneless chicken, dehydrated chicken protein, chicken fat, dried eggs, herring and herring protein, fish oil and dried carrot.

The first grain-free diet produced the lowest glucose area under the curve and the lowest glucose nadir, together with a low insulin area under the curve and the longest time to peak insulin. The second grain-free diet produced the strongest insulin response, with the highest insulin peak. The cereal-based diet produced the highest mean glucose and the highest glucose area under the curve, alongside high insulin values and the lowest insulin nadir; it represented the poorest glycaemic control of the three.

Because starch content was held nearly constant, the differences recovered are attributable to starch source and structure rather than to starch quantity. This is the study's most valuable feature and it cuts against the framing under which it is often cited. The second grain-free diet was also grain-free, and it produced the strongest insulin response of the three; grain-free status per se therefore did not confer glycaemic advantage. What distinguished the best-performing diet was its carbohydrate source sweet potato, not the absence of cereal. Formulators and clinicians reading the "grain-free versus cereal-based" headline should note that the operative variable was botanical origin and starch architecture.

The design has one significant limitation. Postprandial sampling at 120-minute intervals cannot resolve a peak that the same literature places somewhere between two and four hours, and whose timing shifts with diet composition. The true zenith on any of the three diets may have fallen between sampling points, and the reported peak values are consequently lower bounds. The fifty-day feeding period, by contrast, is the longest of the four studies and offers reasonable confidence that adaptation had occurred.

6.2 High-protein, low-carbohydrate diets

Banton *et al.* (2025)^[7] employed a Latin square design in nine adult dogs fed three diets over forty-two days: a high-protein, low-carbohydrate diet (52.1 per cent crude protein, 19 per cent crude fat, 10.7 per cent nitrogen-free extract), a moderate-protein, moderate-carbohydrate diet (28.7 per cent crude protein, 14.5 per cent fat, 40 per cent nitrogen-free extract), and a high-fibre metabolic diet (27.2 per cent crude protein, 12.5 per cent fat, 10.2 per cent crude fibre, 16 per cent total dietary fibre, 34.2 per cent nitrogen-free extract). Postprandial glucose, insulin, glucagon and amino acids were measured.

Table 3: Postprandial responses to three diets differing in protein, carbohydrate and fibre (Banton *et al.*, 2025)^[7]

Parameter	Measure	HPLC	MPMC	MET	P
Glucose (mmol/L·min)	iAUC	236.9±79.0	397.8±75.5	386.5±74.9	0.103
	NetAUC	129.3±95.0	361.8±90.4	346.7±89.8	0.057
Glucagon (pg/mL·min)	iAUC	346,481±40,073 ^a	102,086±38,411 ^b	115,168±38,129 ^b	<0.001
	NetAUC	344,946±39,992 ^a	93,697±38,178 ^b	111,955±37,900 ^b	<0.001
Insulin (μIU/mL·min)	iAUC	23,080±5,046	25,190±4,872	22,392±4,844	0.820
	NetAUC	22,887±5,135	24,962±4,956	22,370±4,928	0.844
HOMA-IR	iAUC	5,562±1,476	7,112±1,429	6,179±1,421	0.502
	NetAUC	5,515±1,497	7,059±1,449	6,175±1,442	0.521
Glucose: Insulin	iAUC	1,691±663	3,229±629	2,048±624	0.093
	NetAUC	-1,032±715 ^a	-2,906±692 ^b	-1,979±687 ^{ab}	0.039

HPLC = high protein, low carbohydrate; MPMC = moderate protein, moderate carbohydrate; MET = high-fibre metabolic diet. Values sharing a superscript letter within a row do not differ.

The way these results are usually summarised does not survive contact with the P-values. It is commonly stated that the high-protein, low-carbohydrate diet delivered the best glycaemic control and the moderate-protein diet the poorest. Directionally the glucose data point that way the incremental area under the curve on the high-protein diet was roughly forty per cent lower than on the moderate-protein diet but neither the incremental nor the net glucose area reached conventional significance, at $P = 0.103$ and $P = 0.057$ respectively. Insulin showed no differences whatever ($P = 0.82$ and $P = 0.844$), and neither did HOMA-IR ($P = 0.502$ and $P = 0.521$). The only endpoints attaining significance were glucagon, which was roughly three-fold higher on the high-protein diet ($p < 0.001$), and the net glucose-to-insulin ratio ($P = 0.039$).

The defensible reading is therefore narrower than the usual one. The robust finding is a large glucagon response to the high-protein diet, which is what one would expect from a substantial amino-acid load and is not in itself evidence of improved glycaemic control. The glucose trend is suggestive and, with nine animals, the study is plainly underpowered to detect effects of the size observed; a difference of that magnitude might well reach significance in a larger trial. But a review should report a non-significant trend as a non-significant trend rather than converting it into a conclusion. There is also an internal inconsistency in the reported fibre fractions worth flagging: for all three diets, insoluble fibre exceeds total dietary fibre (10.9 against 7.0; 8.4 against 7.9; 16.5 against 16.0), which cannot be correct as stated and suggests a transcription or units error somewhere in the chain. Those values should be checked against the primary publication before being used.

6.3 Pulse-based high-fibre diets

Quilliam *et al.* (2022) [44] used a randomised crossover design in eight adult beagles, feeding each of six diets for seven days with a three-day washout between periods. Blood was collected after an overnight fast and then at sixteen postprandial points were 5, 10, 15, 20, 30, 45, 60, 90, 120, 150, 180, 210, 240, 270 and 300 minutes making this by a wide margin the best-resolved glucose curve among the four studies. The diets contrasted a rice-based control against lentil, smooth pea, fava bean and two wrinkled pea formulations.

The rice diet produced the highest peak blood glucose, at approximately 5.0 mmol/L, and the highest glycaemic response by area under the curve, at approximately 810 mmol/L·min. The lentil diet produced the lowest peak, approximately 4.4 mmol/L, and the lowest area under the curve, approximately 726 mmol/L·min. Fasting blood glucose was lowest on the lentil diet at 3.5 ± 0.1 mmol/L and on the wrinkled pea diet at 3.6 ± 0.1 mmol/L.

Two qualifications apply. The first is confounding: the six diets did not differ only in carbohydrate source. Flour inclusion ranged from 23.12 per cent in the rice diet to 58.65 per cent in one wrinkled pea diet, with chicken by-product meal falling correspondingly from 37.83 per cent to 8.85 per cent. The comparison therefore contrasts whole formulations differing simultaneously in carbohydrate source, carbohydrate load and protein source, and the pulse effect cannot be cleanly isolated from the accompanying reduction in animal protein. The second qualification

concerns magnitude. The difference between the best and worst diets amounts to roughly 0.6 mmol/L in peak glucose and approximately ten per cent in area under the curve, in healthy beagles whose glucose remained within a narrow physiological band throughout. Whether an effect of this size is clinically meaningful in a dysglycemic animal is a separate question that the study, conducted in healthy dogs, cannot address.

6.4 Moringa oleifera leaf peptide

Wang *et al.* (2025) [57] combined *in vitro* enzyme inhibition and digestion work with an *in vivo* feeding trial. *In vitro*, moringa leaf peptide showed dose-dependent inhibition of α -amylase, reaching 90.98 per cent at 40 mg/mL, and of α -glucosidase, reaching 98.44 per cent at 20 mg/mL, with the half-maximal inhibitory concentrations already discussed in Section 5. In simulated digestion, starch hydrolysis at 180 minutes was 79.58 per cent for the control snack against 65.82 per cent for the peptide-containing snack, and the estimated glycaemic index was significantly lower in the peptide group ($p < 0.01$).

The *in vivo* component used sixteen beagles allocated to a control group ($n = 8$) and a peptide group ($n = 8$) fed snacks with three per cent inclusion, with blood glucose measured at 0, 15, 30, 60, 90, 120 and 180 minutes. Blood glucose was significantly lower at 30 and 60 minutes ($p < 0.01$), with a reduced peak, a longer time to peak indicating slower glucose absorption, reduced total and incremental area under the curve, and a significantly lower glycaemic index ($p < 0.01$).

The *in vivo* design is the weakest of the four in one specific respect: it is a parallel-group comparison rather than a crossover, so between-animal variation in glucose handling is uncontrolled, and with eight dogs per arm the design has limited capacity to absorb it. The test matrix is also carbohydrate-dense rice flour at 27.93 per cent, corn flour at 9.31 per cent and potato starch at 8.38 per cent in the control snack, so what is being demonstrated is attenuation of a high-starch challenge rather than performance within a diet already formulated for glycaemic control. Finally, incorporating the peptide at three per cent displaced a proportional quantity of every other ingredient, so the two snacks are not strictly iso-nutrient; the observed effect includes a small reduction in digestible starch alongside whatever the peptide itself contributed.

7. Conclusions

Postprandial glycemia in the dog is diet-dependent and dynamic and it is modifiable by nutritional means. Carbohydrate is the central lever, but the useful formulation of that claim is more specific than the usual one: the source, structure and processing history of dietary starch appear to matter at least as much as its quantity and the single best-supported strategy to emerge from this literature is substitution of rapidly digestible refined-cereal starch with slowly digestible starch from pulses or minimally processed whole grains. Protein, fat and dietary fibre modulate the response through mechanisms that are well described qualitatively and poorly quantified in this species. Nutraceutical interventions are mechanistically plausible and, with a small number of exceptions, not yet demonstrated in dogs at feeding-relevant doses.

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