# Doubts on the Use of Industrial Substitutes Such as Gel for Insectivorous Birds

## A Technical Positioning Report on Processed Diets for Strict Aerial Insectivores (Common Swift, Apus apus)

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In the rehabilitation of strict aerial insectivores such as the common swift (*Apus apus*), the replacement of whole arthropods with processed products like the Wildiets Insectivore Gel presents severe biochemical and anatomical contradictions. This report compiles 8 technical and professional questions backed by comparative physiological studies and the unique ecology of these birds, with the objective of re-evaluating the mandatory use of this preparation in wildlife recovery centres.

> **Question 1: Sugar and Initial Dehydration:** How is it justified to feed a gel containing high amounts of sugar (dextrose) to birds that arrive dehydrated and weak, knowing that this sugar 'steals' water from their bodies in the gut and can trigger fatal diarrhoea?

**Clinical Basis:** In birds entering with acute dehydration and hypovolemic shock, introducing highly hyperosmolar monosaccharides like dextrose before fully restoring volemia is lethal. If dextrose exceeds the absorption capacity of the mucosa, it accumulates in the gut and reverses the osmotic gradient, extracting water from the interstitial space into the lumen of the digestive tract. This causes fulminant osmotic diarrhoea that accelerates renal perfusion collapse. Furthermore, introducing simple carbohydrates in extremely fasting patients induces an insulin spike that massively shifts electrolytes into the intracellular space, triggering iatrogenic Refeeding Syndrome and patient death within a few hours.

> **Question 2: Complex Sugar They Cannot Digest:** Why are strict insectivores forced to feed on maltodextrin, knowing that through evolution these birds lack the necessary enzymes to digest and process this complex sugar?

**Clinical Basis:** Strict insectivorous birds have evolved under an evolutionary paradigm free of starches or complex sugars, resulting in a drastic loss or reduction of maltase activity in the brush border of their intestinal mucosa. Because maltodextrin cannot be absorbed or digested in the foregut, it reaches the hindgut intact, causing a profound imbalance in the microbiome. The presence of non-natural polysaccharides promotes the exponential adhesion of pathogenic strains of *Escherichia coli* and *Salmonella* to the epithelium, destroying the enteric immune system, simulating avian Inflammatory Bowel Disease (IBD), and blocking the absorption of calcium and essential amino acids.

> **Question 3: Gizzard Atrophy from Lack of Rigid Food:** How is the complete absence of insect skins and hard shells compensated for, when we know that without this mechanical stimulus the stomach shrinks rapidly and the bird will lose the ability to digest insects upon release?

**Clinical Basis:** The gizzard of an insectivore requires the mechanical resistance of whole arthropod exoskeletons to maintain its musculature and the proper thickness of the koilin layer through work hypertrophy. Administering soft, pasty, or gel-like diets such as Wildiets offers no frictional resistance to the digestive wall muscles, inducing rapid gizzard atrophy (which can reduce its mass by up to 60% in a few days) and causing a loss of intestinal muscle tone. When the bird is released and attempts to hunt wild dipterans or coleopterans, its atrophied digestive tract is incapable of generating the mechanical force to fragment the exoskeletons, resulting in severe gastrointestinal impactions and death by starvation.

> **Question 4: Vegetable Fats and Land Mammal Residues:** What is the logical explanation for giving vegetable oils and land mammal meals to birds adapted solely to digesting insect fats and proteins? Are we not damaging their liver and kidneys?

**Clinical Basis:** The use of ingredients described as 'terrestrial animals and their derived products' (mammalian by-products) introduces an alien amino acid profile that subjects the bird's organism to an extraordinary catabolic effort, drastically increasing the glomerular filtration rate and causing renal stress, while promoting secondary hepatic lipidosis. Concurrently, the refined sunflower and linseed oils in the product differ from insect lipids, hindering lipolysis mediated by avian pancreatic lipase, slowing absorption, and modifying the composition of fats deposited in flight feather follicles, which severely compromises their waterproofing in the wild.

> **Question 5: Evolutionary Rigidity of Insectivorous Birds:** Since swifts are birds made exclusively to eat insects and their bodies lack the ability to adapt to other foods, why do we feed them a soft artificial paste that slides through their gut without activating digestive mechanisms?

**Clinical Basis:** According to Karasov's digestion optimization models, strict specialist birds, unlike generalists, have extremely reduced physiological flexibility and lack the ability to adapt their intestinal biochemistry to diets outside their evolutionary norm. Soft or gel-like diets transit through the foregut at an excessive speed, preventing the glandular stomach and gizzard from executing the necessary chemical and retention stimuli. This prevents the activation of hormonal and enzymatic feedback loops indispensable for coordinated digestion, leading the bird into a state of chronic malabsorption and severe undernutrition.

> **Question 6: The Gut Unprotected Against Infections:** Did you know that the chitin fiber from whole insect shells helps maintain a healthy and strong intestinal mucosa? Why do we deprive them of this natural protection by using industrially ground meals?

**Clinical Basis:** Although the gel contains Tenebrio meal, industrial grinding and emulsification eliminate the biomechanical structure of the exoskeleton. Under natural conditions, whole chitin reaches the hindgut, acting as a prebiotic that stimulates beneficial bacteria (such as *Bacteroides plebeius* or *Christensenella minuta*) to produce short-chain fatty acids, especially butyrate. Butyrate is the metabolic fuel of colonocytes and activates the transcription of tight junction proteins (claudin-1 and occludin), reducing intestinal permeability and blocking systemic pathogenic translocation. Depriving the bird of this natural protection mechanism exponentially stimulates the adhesion of pathogenic bacteria.

> **Question 7: Lack of Critical Nutrients and Weak Feathers:** How can an artificial food produce strong feathers, if the lack of real nutrients from live insects forces the chick to disintegrate its own muscles to survive, filling its feathers with stress marks?

**Clinical Basis:** The development of a swift chick demands extremely high levels of taurine (essential for cardiac function and retinal development) and sulfur amino acids such as methionine and cysteine (fundamental for keratin synthesis in flight feathers). Experiencing microdeficiencies in these nutrients due to different kinetics and low bioavailability of artificial mixes, the metabolism initiates destructive deamination, catabolizing its own pectoral musculature to obtain the necessary energy. This severe malnutrition triggers the release of corticosterone, which interrupts keratogenesis and induces the formation of 'Fault Bars' (stress marks devoid of keratin)—extremely fragile flight feathers that break under the aerodynamic pressure of high-performance dynamic flight.

> **Question 8: The Fallacy of the Gel's 'Easy Digestion':** It is claimed that the gel is beneficial because it is 'very easy to digest,' but is it not precisely this lack of hardness that makes the bird's body lazy and prevents it from absorbing nutrients and growing strong?

**Clinical Basis:** The commercial premise of 'superior digestibility' as a benefit is physiologically incorrect for highly specialized birds. Aerial insectivorous birds are evolutionarily adapted to a digestive transit where the hardness and acid exposure time of the hemolymph and exoskeleton autonomously regulate retention speed. The absence of physical resistance in gelatinous diets halts digestive motility, reduces proventricular gastric juice secretion, and depresses bile secretion. This causes a disordered digestive transit that disables real nutrient absorption and accelerates fecal elimination without actual metabolic assimilation.

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## PHYSIOLOGICAL & PATHOLOGICAL RISKS SUMMARY MATRIX

| Problematic Component | Direct Clinical Effect | Pathological Outcome / Risk |
|---|---|---|
| Dextrose (Simple Sugar) | Monosaccharide hyperosmolar overload in weak patients. | Reverses osmotic gradient, causing lethal osmotic diarrhoea. Triggers Refeeding Syndrome. |
| Maltodextrin (Complex Sugar) | Undigested polysaccharides reach hindgut due to lack of maltase. | Alters microbiome, favoring exponential growth of E. coli & Salmonella; simulates avian IBD. |
| Soft/Gel Consistency & Lack of Hard Shells | Absence of mechanical/frictional stimulation on digestive walls. | Induces up to 60% gizzard muscle atrophy and loss of muscle tone; post-release impaction. |
| Refined Vegetable Oils | Incorrect lipid structure relative to insect-derived fats. | Hinders lipolysis, slows assimilation, and alters flight feather follicle lipids (loss of waterproofing). |
| Land Mammal By-products | Alien amino acid profile introduced to a strict insectivore. | Elevates glomerular filtration rate, causing acute renal stress and secondary hepatic lipidosis. |
| Industrially Ground Chitin | Destruction of the physical, prebiotic fiber structure. | Suppresses short-chain fatty acids (butyrate), weakening tight junctions (claudin-1/occludin); leaky gut. |
| Low Bioavailability of Critical Nutrients | Microdeficiencies in taurine, methionine, and cysteine. | Forces pectoral muscle catabolism for energy; triggers corticosterone, causing Fault Bars and fragile feathers. |
| Ultra-soft Gel Premise | Absence of physical resistance, depressing gastric secretions. | Slows gastrointestinal motility and reduces bile/stomach acid; food passes unassimilated. |