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The Gut Microbiome Should Modify Nutritional Decisions Only When It Changes Clinical or Production Action: A Translational Veterinary Framework Connecting Microbial Function, Diet, Metabolism, Disease State, and Host Performance

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ABSTRACT

Veterinary microbiome research increasingly identifies diet-associated microbial taxa, functional genes, metabolites, and dysbiosis patterns. However, detecting a difference does not establish that nutrition should change. Taxonomic composition may diverge from functional capacity; functional potential may not produce measurable metabolites; and metabolite variation may not alter host health, welfare, or production performance. Interpretation is further constrained by species, gastrointestinal compartment, life stage, genotype, disease, medication exposure, production environment, sampling, and analytical method. This original non-empirical article develops a translational framework in which microbiome information modifies nutritional decisions only when it contributes information beyond conventional assessment and changes a feasible clinical or production action. The framework separates microbial measurement, functional interpretation, host-response evidence, decision consequence, and post-intervention reassessment. It proposes that actionability requires a credible connection between a modifiable dietary exposure, a microbiome-mediated or microbiome-indicated process, and an outcome that matters for the animal or production system. Noncompensatory constraints prevent attractive microbial findings from overriding nutritional adequacy, safety, disease-specific requirements, welfare, or practical feasibility. Taxonomic novelty, diversity shifts, predicted pathways, and isolated metabolites remain nonactionable when their direction, persistence, host relevance, or response to diet is uncertain. Potential applications include selecting among otherwise acceptable fibre strategies, interpreting recovery after microbiome-disrupting exposures, and identifying heterogeneous nutritional responses. The framework is not a validated score, diagnostic instrument, or prescribing algorithm. Its use requires species-specific reference resources, standardized measurement, prospective intervention studies, external validation, and evidence that microbiome-informed decisions improve meaningful outcomes compared with decisions made without microbiome information.

Keywords: Veterinary microbiome, Nutritional actionability, Microbial metabolites, Dysbiosis, Precision nutrition, Host performance

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Introduction

Veterinary microbiome studies now describe microbial communities across companion animals and livestock at unprecedented taxonomic and functional resolution. A large integrated analysis of canine and feline stool

metagenomes nevertheless demonstrated substantial variation across populations, datasets, diets, and analytical approaches [1]. Controlled dietary research in healthy dogs further showed that fibre-associated metabolomic responses may be more pronounced than taxonomic responses and remain dependent on the animal's baseline ecological configuration [2]. In pigs, reconstruction of core microbial organisms and regulatory functions illustrates how community organization can extend beyond lists of differentially abundant taxa [3]. Sheep and goat metagenomes likewise indicate that host species, breed, farm environment, and microbial glycan-utilization capacity intersect rather than operate independently [4].

These developments create an interpretive problem. A microbial feature may be statistically associated with diet, disease, metabolism, or performance without providing information that changes feeding. Conversely, a modest compositional change may be important if it reliably indicates a functional process that affects an animal-specific outcome. The clinically relevant question is therefore not whether diet changes the microbiome, but whether microbiome information changes what should be fed, withheld, substituted, monitored, or reassessed.

This article develops a proposed translational nutrition framework separating microbial description from nutritional actionability. It connects taxonomic and functional measurements with metabolites, host metabolism, immune and barrier effects, species and life-stage context, disease state, production conditions, feasible dietary alternatives, and meaningful outcomes. It does not treat the microbiome as an independent nutritional target or presume that compositional normalization improves health or production.

From microbiome association to nutritional actionability

Association answers whether two measured features vary together; actionability asks whether knowing that relationship should alter a decision. These questions require different evidence. Cross-cohort metagenomics can identify recurrent organisms and functions, dietary intervention can establish temporal responsiveness, and genome reconstruction can reveal plausible metabolic capacity [1–3]. None alone demonstrates that changing feed will improve a relevant outcome.

Nutritional actionability is proposed here as a conditional property of a finding rather than an intrinsic property of a taxon, pathway, metabolite, or diversity index. A finding becomes potentially actionable when it identifies a modifiable dietary exposure, provides information not already available from conventional nutritional and clinical assessment, distinguishes between feasible alternatives, and predicts a difference in an outcome important to the animal or production system. Its value should disappear when no credible alternative action exists.

This definition imposes boundaries. A reproducible association may remain nonactionable if directionality is unresolved, the measured compartment is inappropriate, functional activity is unconfirmed, or the proposed diet violates nutritional adequacy. Actionability may also be temporary: a finding obtained during antimicrobial exposure, weaning, acute disease, or diet transition may not apply after the underlying state changes. Accordingly, microbiome-guided nutrition should be formulated as an intervention with a defined objective and reassessment plan, not as permanent correction of an assumed ideal community.

Taxonomic composition versus functional capacity

Taxonomic profiles describe which organisms are detected and their relative representation. Functional measurements address what genes, transcripts, proteins, or metabolites are present or active. These levels are related but non-equivalent because different organisms may perform overlapping functions, while members of the same taxon may differ by strain, substrate access, and metabolic state.

The companion-animal gut metabolome provides a potentially closer bridge between diet, microbial activity, and host physiology, but current evidence remains limited by inconsistent platforms, incomplete metabolite identification, and reliance on biological interpretation transferred from other species [5]. Comparative canine metagenomics has also demonstrated partial similarity between dog and human microbial gene content and dietary responses [6]. Such similarity supports comparative investigation but does not make human nutritional associations directly prescriptive for dogs.

The proposed interpretive hierarchy therefore privileges demonstrated function over taxonomic reputation without treating function as sufficient for action. Relative abundance is descriptive; gene content indicates capacity; transcription or protein expression indicates activity; metabolite concentration reflects production, utilization, absorption, and transit; and host response establishes biological consequence. No level automatically substitutes for the next. A predicted short-chain-fatty-acid pathway, for example, should not be described as increased short-

chain-fatty-acid production without measurement. Even a measured fecal metabolite may incompletely represent production because host absorption and microbial cross-feeding alter the quantity ultimately recovered.

Diet–microbiome interactions

Diet supplies substrates, changes intestinal physicochemistry, modifies transit, and can indirectly alter host secretions and immune activity. Microbial responses therefore depend on more than the nutrient name. Fibre source, fermentability, particle structure, inclusion amount, background diet, energy balance, adaptation period, and prior microbial configuration may all affect the observed response. In healthy dogs exposed to multiple foods, identical broad fibre categories did not generate uniform microbial or metabolomic effects across individuals [2]. Interpretation must also distinguish a diet effect on the microbiome from a microbiome-mediated diet effect on the host. A diet may directly change stool water, digestibility, bile flow, or intake, with microbial changes occurring secondarily. Conversely, microbial metabolism may generate products that contribute to the host response. Demonstrating temporal co-occurrence cannot identify the dominant pathway.

Comparative evidence further cautions against assuming that a substrate produces the same function across hosts merely because similar microbial genes are present [6]. Dietary history, gastrointestinal anatomy, nutrient digestibility before the colon, and community interactions determine whether substrate reaches an organism and whether encoded functions are expressed. Consequently, microbiome-informed feeding should specify the complete nutritional exposure and intended host outcome. Statements that an ingredient "improves the microbiome" are insufficient unless improvement is operationalized independently of taxonomic preference and connected to health, welfare, nutrient use, or production performance.

Microbial metabolites

Microbial metabolites can connect dietary substrates with epithelial, immune, endocrine, and systemic processes more directly than community composition. Yet they differ in evidential value. Some are end products of several organisms; others are transformed further by microbes or host tissues. Fecal concentration reflects the balance among production, degradation, absorption, secretion, and transit rather than production alone.

Mechanistic work in pigs identified Eubacterium siraeum as a candidate organism influencing fat deposition through a tyrosine-related PI3K/AKT pathway and strengthened the association through experimental follow-up [7]. This provides a model for linking organism, molecular process, and host phenotype, but it does not establish a universal microbial target across breeds, diets, or species. Similarly, calf-derived microbial ursodeoxycholic acid improved intestinal homeostasis and attenuated inflammatory injury in experimental infection and colitis systems [8]. The evidence supports biological plausibility for microbial bile-acid signalling while leaving routine dietary reproducibility uncertain.

The proposed framework therefore asks four questions of a candidate metabolite: Was it measured with an appropriate method? Is its microbial contribution distinguishable from dietary and host sources? Does its change precede or track a meaningful host response? Can a nutritionally acceptable intervention modify it predictably? Without these connections, metabolite data may refine biological interpretation but should not independently determine feeding. Functional importance must be established for the particular animal state, not inferred from a metabolite's general reputation.

Host metabolism

Microbial metabolism becomes nutritionally important when it changes nutrient availability, energy harvest, host signalling, substrate competition, or metabolic waste in a direction relevant to the animal. This relationship may be beneficial, neutral, or harmful depending on physiological state. Greater fermentation can supply useful metabolites but may also alter stool quality, gas production, luminal acidity, or energy extraction. Accordingly, more microbial activity is not necessarily better.

In healthy dogs, fibre-responsive metabolomic patterns depended on both food composition and individual microbial configuration [2]. Pig evidence connecting a candidate organism to fat deposition further shows that host phenotype may reflect a particular microbial pathway rather than community diversity as a whole [7]. Neither observation justifies optimizing the microbiome independently of total energy balance, digestibility, amino-acid supply, body condition, or production objective.

The framework treats host metabolism as a translation boundary. Before that boundary, evidence concerns microbial potential or activity. Beyond it, evidence concerns consequences for the animal. Crossing the boundary

requires a measured or defensible connection to outcomes such as nutrient utilization, body composition, metabolic control, recovery, growth, or feed efficiency. An observed taxonomic or metabolite shift that does not change these outcomes may remain scientifically informative but nutritionally nonactionable. Where conventional measures already determine the appropriate diet, microbiome testing must demonstrate incremental decision value rather than merely concordance with information already available.

Immune and barrier effects

Microbial communities interact with mucus, epithelial cells, microbial products, immune signalling, and colonization resistance. Nutritional interventions may modify these processes through direct nutrient effects, microbial metabolism, or both. In broilers, Lactobacillus plantarum supplementation was accompanied by microbial and metabolomic changes, altered intestinal morphology and barrier-related measures, and improved growth in the tested setting [9]. The result supports multi-domain concordance but remains specific to the strain, diet, population, and production conditions.

Fermented liquid feeding in grower-finisher pigs similarly affected microbial diversity, nutrient digestibility, intestinal morphology, barrier-associated gene expression, and performance [10]. Because feed form, fermentation products, intake, digestibility, and microbial ecology changed together, the study cannot assign the host outcome exclusively to the microbiome.

For nutritional adjudication, immune and barrier evidence should therefore be partitioned into distinct patterns. A compositional change without barrier or host evidence remains mechanistically incomplete. A barrier response without demonstrated microbial mediation may still justify a diet, but not specifically as microbiome-guided nutrition. Concordant microbial, functional, barrier, and outcome responses provide stronger translational support, although they do not eliminate confounding or prove mediation.

The distinctions required to prevent immune or barrier observations from being converted automatically into feeding recommendations are organized below. **Table 1** presents joint immune–barrier patterns that do and do not support microbiome-informed feeding.

Table 1. Joint immune–barrier patterns that do and do not support microbiome-informed feeding

Cross-domain configuration	Immune domain	Barrier domain	Non-microbial alternative	Nutritional option allowed	Test before or after diet change
Taxonomic shift alone	Unmeasured	Unmeasured	Diet and baseline ecology	Do not change diet solely for “balance”	Confirm function and host relevance
Host response without mediation	Changed marker	Changed permeability or morphology	Direct nutrient effects possible	Diet may be justified conventionally	Do not label effect microbiome-mediated
Cross-domain concordance	Directionally aligned	Directionally aligned	Species, strain, dose, life stage	Consider among nutritionally acceptable options	Reassess clinical or production outcome
Discordant response	Improved or worsened	Opposing or unchanged	Timing and compartment	Withhold microbiome-based inference	Repeat measures and examine alternatives
Proposed actionability state	Relevant and persistent	Relevant and persistent	Feasibility and nutritional adequacy	Conditional diet modification	Reverse if meaningful benefit is absent

Species dependence

Veterinary microbiome interpretation cannot be separated from digestive anatomy and feeding ecology. Ruminants depend on foregut microbial fermentation before small-intestinal absorption, whereas dogs, cats, pigs, and poultry differ in nutrient digestion, hindgut fermentation, bile-acid metabolism, transit, and dietary adaptation. A microbial function valuable in the rumen may have a different consequence when expressed in the colon, and a fecal sample may represent neither the location nor the timing at which the decisive process occurred.

Comparative metagenomic similarity is therefore useful for generating hypotheses, not bypassing species-specific validation. Canine and human gut microbiomes share portions of their functional gene repertoire and may show parallel dietary responses, but their hosts, diets, and gastrointestinal environments remain distinct [6]. Sheep and

goat data likewise demonstrate that microbial composition and functional capacity vary with both host and rearing environment [4].

The framework requires every microbiome-informed nutritional claim to declare its species, gastrointestinal compartment, health state, diet, and intended outcome. “Veterinary microbiome” should not operate as a single transferable evidence category. Even closely related livestock species may differ in fibre use, mucin degradation, growth targets, and environmental exposures. Species dependence also limits universal definitions of a healthy microbiome: absence of a taxon considered beneficial elsewhere may represent normal host adaptation rather than deficiency. Cross-species evidence should inform mechanism selection while remaining outside the final decision unless functional and outcome relevance are demonstrated in the target species.

Life-stage dependence

Microbial communities and nutritional requirements change during neonatal colonization, milk feeding, weaning, growth, adulthood, reproduction, and aging. These transitions can create time-limited associations that should not be mistaken for stable biomarkers. In calves, early colostrum processing altered selected microbial and resistance-gene trajectories across repeated sampling, demonstrating that initial feeding conditions can shape development without implying a fixed adult state [11].

Intervention resistance is equally important. High maternal dietary zinc produced little detectable effect on neonatal-calf microbiome composition or resistome despite clear temporal changes in the cows [12]. A biologically plausible exposure may therefore fail to alter the microbial outcome, and developmental time may explain more variation than supplementation. In Beagles, age groups showed selected taxonomic and short-chain-fatty-acid differences without evidence of wholesale restructuring of microbial diversity [13]. Because that comparison was cross-sectional and breed-restricted, age could not be isolated completely from cohort or environmental effects.

A life-stage claim should specify whether it concerns developmental establishment, transient adaptation, maintenance, or loss of resilience. The proposed framework does not treat early-life deviation from an adult reference as dysbiosis, nor does it assume that an age-associated feature warrants correction. Diet should change only when the microbial information distinguishes a life-stage-appropriate action and predicts an outcome beyond what age, growth, reproductive status, and conventional nutritional assessment already indicate. Reassessment timing must reflect the expected developmental trajectory.

Production-system dependence

Production relevance is determined by the biological system in which microbial activity occurs and by the outcome being optimized. Reconstruction of rumen microbial genomes revealed extensive functional capacity for feed degradation and fermentation that cannot be represented adequately by fecal taxonomic profiles [14]. Such capacity is central to ruminant nutrition but cannot be transferred directly to monogastric animals, in which substrates reach different compartments and microbial metabolites encounter different absorptive conditions.

Production phenotypes also arise from interacting host and microbial determinants. In dairy cattle, host genetics and rumen microbial composition were jointly associated with methane emissions [15]. In pigs, relationships between microbial features and feed efficiency varied with host genotype and microbial interaction structure [16]. These findings weaken the premise that a universally favorable community can be selected independently of breed or production context.

Microbiome-informed feeding must therefore declare its production objective: growth, feed conversion, product quality, methane mitigation, resilience, or disease control may impose different priorities. A microbial pattern associated with one objective may be neutral or undesirable for another. Housing, feed processing, antimicrobial exposure, climate, biosecurity, and available ingredients further affect whether an intervention is reproducible. The framework consequently treats the production system as an effect modifier and feasibility boundary, not as background. Microbial evidence becomes actionable only when it improves a decision within the actual feeding system and when performance, welfare, safety, environmental, and economic consequences can be reassessed together.

Disease-associated dysbiosis

Dysbiosis describes a departure from a reference microbial state, but the term often combines several distinct phenomena: loss of particular organisms, expansion of facultative or potentially harmful groups, altered microbial

functions, reduced ecological stability, and disrupted metabolite production. These changes need not occur together. A composition outside a reference interval may retain important functions, whereas apparently modest taxonomic change may conceal substantial metabolic disruption.

Disease also changes the conditions under which the microbiome is measured. Reduced intake, altered macronutrient exposure, vomiting, diarrhea, intestinal transit, inflammation, malabsorption, medication, and therapeutic diets can all modify fecal communities. Consequently, a disease-associated profile may reflect the disease, its treatment, its nutritional consequences, or their interaction. “Normalization” is not a sufficient nutritional objective unless the reference state is appropriate for the species, life stage, diet, compartment, and analytical platform.

The proposed framework distinguishes dysbiosis as a state marker from dysbiosis as an actionable nutritional mechanism. A state marker may support characterization or monitoring without identifying a diet. A mechanistically relevant pattern requires evidence that a modifiable nutritional exposure changes the implicated function and that this change relates to a meaningful outcome. Where disease-specific nutritional requirements already determine feeding, a microbiome result should not override them. Its legitimate role is to refine selection among otherwise acceptable options, identify response heterogeneity, or indicate when reassessment is warranted.

Cause, consequence, and correlation

The canine dysbiosis index illustrates both the value and limitation of composite microbial measurement. Its qPCR panel distinguished healthy dogs from dogs with chronic inflammatory enteropathy in development and validation subsets [17]. This supports classification of an altered microbial state, not the conclusion that the indexed organisms caused disease or that moving the index toward a reference value will improve outcome.

Temporal perturbation offers stronger directional information. In healthy cats, metronidazole markedly changed stool characteristics, bacterial diversity, the dysbiosis index, metabolites, and bile-acid profiles; subsequent diet affected selected recovery variables but not every microbial endpoint [18]. The study demonstrates that medication and time may dominate observed change and that compositional recovery, functional recovery, and dietary response can diverge.

Cross-sectional disease comparisons remain more ambiguous. Cats with chronic enteropathy showed fecal microbial and lipid, sterol, and bile-acid differences from controls [19]. These findings identify biologically relevant coexistence but cannot determine whether microbial alterations initiated disease, resulted from inflammation or altered intake, or reflected treatment and transit.

Causal language therefore requires more than temporality or statistical adjustment. Credible inference should combine controlled exposure, appropriate comparator, repeated measurement, functional evidence, host response, and preferably mediation or perturbation testing. Where those elements are absent, microbiome findings should be described as associated indicators and should not independently trigger nutritional change.

Evidence thresholds for changing diet

A microbiome result should cross several non-interchangeable thresholds before altering diet. First, the signal must be technically credible and interpretable against an appropriate species, life-stage, health, diet, and compartment reference. Second, taxonomic change must connect to measured function or a defensible mechanism. Third, the function must relate to a host or production outcome rather than only to another microbial endpoint. Fourth, at least one nutritionally adequate and feasible alternative action must exist. Fifth, expected benefit must justify cost, burden, palatability risk, production disruption, and uncertainty. Finally, the response must be reassessable.

These thresholds are proposed as noncompensatory conditions, not additive points. A strong metabolomic association cannot compensate for an nutritionally inadequate alternative. Likewise, a plausible mechanism cannot compensate for an outcome irrelevant to the animal. Controlled dietary evidence shows that microbial and metabolic responses may vary substantially among apparently comparable animals [2]. Multi-domain responses involving microbial, intestinal, and performance measures strengthen interpretation, but do not by themselves establish microbial mediation [9]. Perturbation evidence further shows that time and medication can outweigh dietary effects [18].

Table 2 presents noncompensatory evidence gates before a microbiome result can change diet.

Table 2. Noncompensatory evidence gates before a microbiome result can change diet

Gate reached	Evidence available	Nutritional action permitted	Blocking condition	Continuation or exit test
State: Valid signal only **Meaning:** Descriptive difference	Reproducible measurement	No diet change	Function unknown	Repeat if state changes
State: Functional but host-uncertain **Meaning:** Plausible process	Function or metabolite confirmed	Monitor or investigate	Outcome relevance absent	Add host endpoint
State: Action-linked concordance **Meaning:** Conditional relevance	Diet, function, and meaningful outcome align	Trial an acceptable alternative	Mediation may remain uncertain	Prespecify benefit and stopping rule
State: Constraint failure **Meaning:** Nonactionable	Nutritional inadequacy, safety, or infeasibility	Reject proposed change	Cannot be offset by microbial evidence	Seek another option
State: Sustained response **Meaning:** Provisionally actionable	Meaningful benefit persists	Continue with monitoring	Context-specific	Withdraw or revise if benefit disappears

A microbiome-to-nutrition decision framework

The proposed framework begins with a defined animal or production problem, not a stool profile. Conventional assessment first establishes nutritional adequacy, disease requirements, performance objectives, current diet, medication exposure, welfare constraints, and feasible alternatives. Microbiome information enters only when uncertainty remains between acceptable actions.

Three parallel evidence domains are then adjudicated. The microbial domain asks whether the signal is technically credible, persistent, and functionally interpretable. The host domain asks whether metabolism, barrier function, immune state, clinical condition, or production performance changes in a relevant direction. The decision domain asks whether the information distinguishes among feasible diets and whether benefit can be monitored. Noncompensatory constraints—including nutritional inadequacy, safety concern, disease incompatibility, poor intake, or operational infeasibility—block progression regardless of microbial attractiveness.

In healthy dogs, fibre and biotic feeding changed digestibility, fecal metabolites, microbiota, IgA, and serum lipids, although responses were not uniformly aligned [20]. A randomized plant- versus animal-based diet comparison changed selected canine microbial features without establishing superior health [21]. Peri-weaning galactooligosaccharide supplementation in pigs connected hindgut changes with intestinal-health and growth outcomes, while microbial mediation remained incomplete [22]. Together, these patterns support conditional adjudication rather than taxonomic prescribing.

Every decision is revisable. Absence of meaningful benefit, emergence of harm, or change in disease, life stage, feed availability, or production objectives returns the case to reassessment.

The architecture in **Figure 1** compresses this non-linear decision logic while preserving the boundaries between microbial measurement, host consequence, and permissible nutritional action.

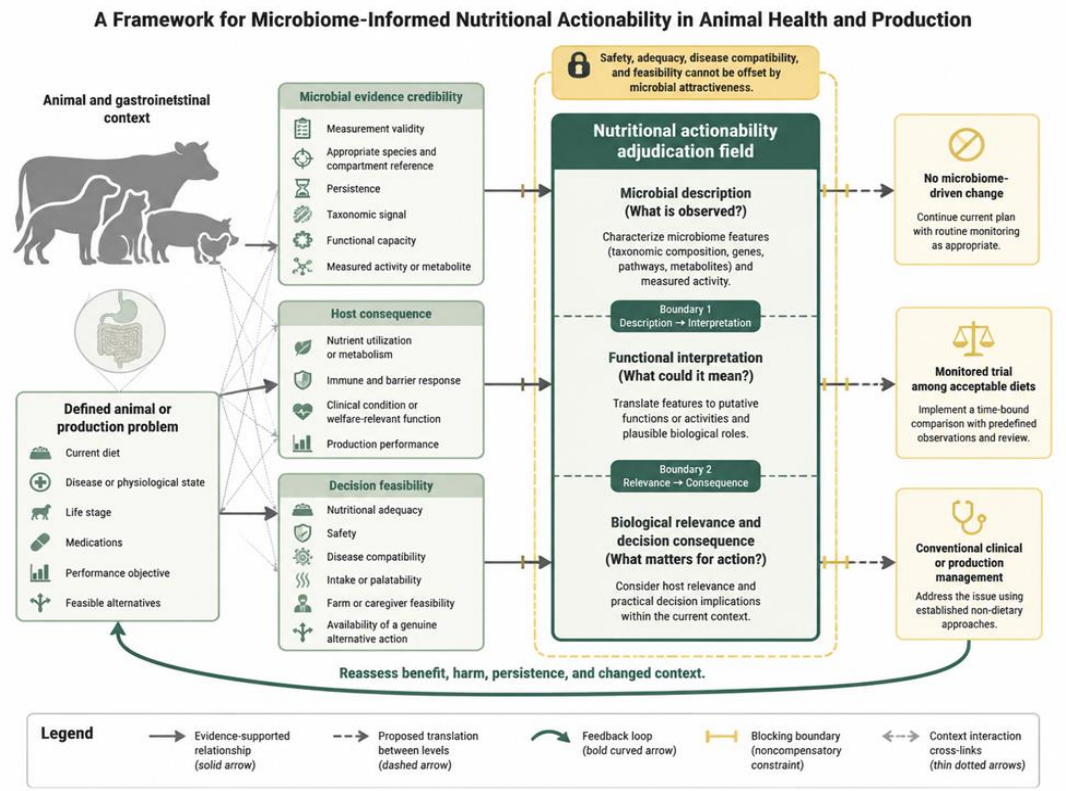

Figure 1. The actionability boundary that prevents a microbial signal from becoming an automatic diet prescription

Actionable versus nonactionable microbiome findings

An actionable finding changes a choice. It identifies which acceptable diet to select, whether to initiate a monitored dietary trial, which response to measure, or when to stop or revise an intervention. Actionability therefore depends on the decision context as much as on biological evidence. The same microbial result may be actionable when two nutritionally adequate alternatives exist and nonactionable when only one safe diet is feasible.

Several common findings remain nonactionable in isolation: increased or decreased diversity; abundance of a taxon labelled beneficial or harmful; a predicted pathway without expression evidence; a fecal metabolite without source or host interpretation; and movement toward a population reference range without meaningful outcome improvement. The plant- versus animal-based canine comparison demonstrates why a detectable microbial difference cannot be equated with health superiority [21]. Conversely, coordinated changes in diet exposure, functional readout, and relevant host response may justify a reversible trial, provided conventional nutritional requirements remain satisfied [20].

Actionability is also outcome-specific. A finding that informs stool management may not inform body composition, immune disease, feed efficiency, or methane mitigation. The framework consequently requires the intended consequence to be declared before interpreting the test. Failure to prespecify the decision invites retrospective storytelling in which any microbial change is described as beneficial. Proposed actionability should instead be judged by whether the result would lead a reasonable clinician, nutritionist, producer, or researcher to choose differently and whether that choice can subsequently be evaluated.

Failure modes and overinterpretation

Microbiome evidence can fail before biological interpretation begins. Differential-abundance methods applied to the same datasets may identify materially different microbial features [23]. Sampling, storage, extraction, sequencing depth, contamination control, reference databases, compositional structure, multiplicity, and reporting choices further influence results; established best-practice guidance therefore treats the complete workflow as part of the biological claim [24]. Benchmarking also shows that confounding and analytic assumptions can generate apparently persuasive biomarkers that do not generalize [25].

Interpretive failures occur when taxonomy is equated with function, gene presence with activity, fecal concentration with production, association with causation, or microbial normalization with benefit. Biological failures include unmeasured host absorption, strain variation, ecological redundancy, cross-feeding, adaptation, and opposing effects across tissues or outcomes. Translational failures occur when human evidence is applied directly to animals, one species or compartment substitutes for another, or a controlled research diet is treated as operationally equivalent to commercial feeding.

These failure modes are not repaired by combining several weak markers. Across methodological comparisons, pipeline choice, confounding control, and reference suitability jointly determine whether an apparent signal survives reinterpretation [23–25]. **Figure 2** shows how separate error channels can converge on a false actionability conclusion and where reassessment can interrupt that progression.

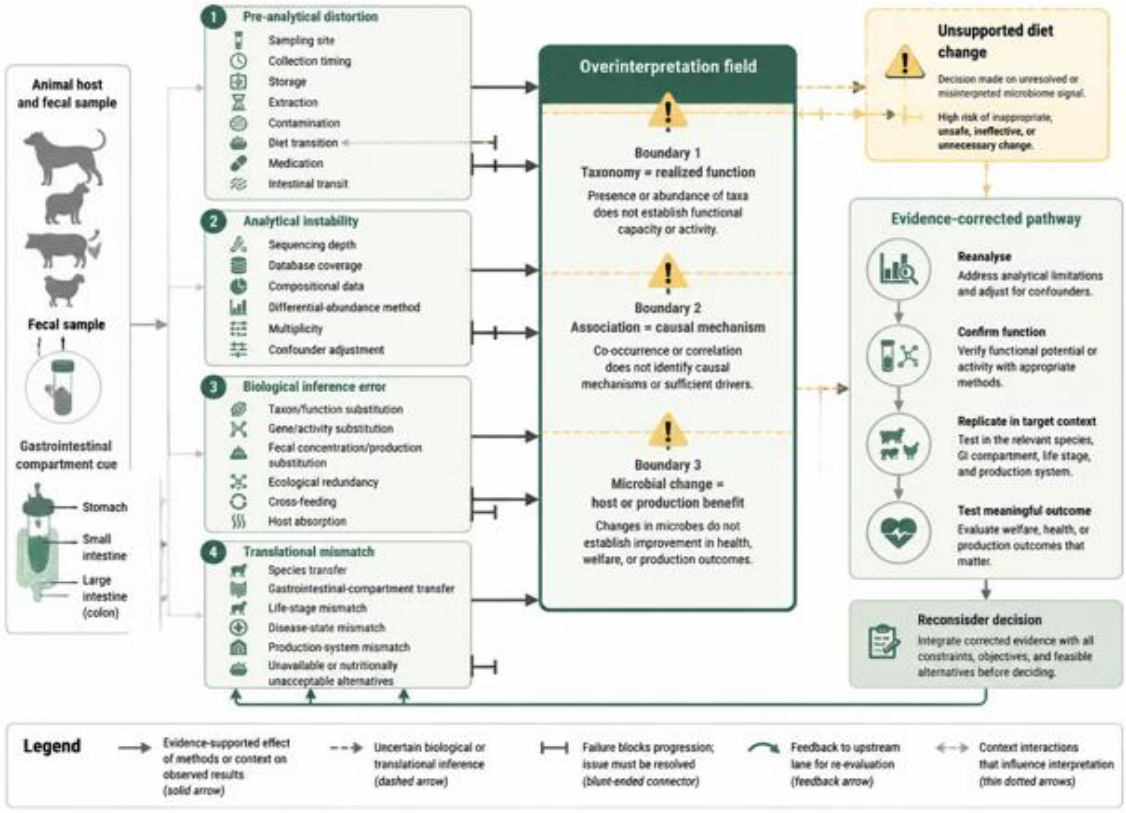

Figure 2. Where microbiome evidence can fail before it reaches a feeding decision

Precision-nutrition applications

The most defensible near-term application is not unrestricted individualized feeding from a single sample. It is conditional selection among diets already acceptable on nutritional, clinical, welfare, and practical grounds. Repeated canine fibre exposures demonstrate substantial individual variation in microbial and metabolomic response, creating a rationale for identifying responders without assuming that every difference is beneficial [2]. In production pigs, host genotype–microbiome interactions similarly caution against population-wide microbial prescriptions for feed efficiency [16].

A second application is functional monitoring after disruption. Following medication, disease, weaning, transport, or diet transition, repeated microbial and metabolite measurements may help distinguish persistent functional disturbance from taxonomic fluctuation. Such monitoring is useful only if it changes duration, composition, or withdrawal of a nutritional intervention.

A third application is mechanism-informed formulation. Controlled canine feeding can connect fibre or biotic exposure with digestibility, microbial features, metabolites, immune markers, stool characteristics, and systemic measures [20]. This supports richer response phenotyping, but not automatic personalization.

Precision nutrition should therefore be defined by precision of question, context, intervention, and reassessment—not merely resolution of sequencing. A high-resolution profile with no alternative action is less useful than a modest functional measure that resolves a genuine feeding choice. Implementation also requires turnaround time,

analytical reproducibility, accessible diets, interpretability, and proof that microbiome information improves decisions beyond conventional clinical or production assessment.

Validation priorities

Validation should begin with species-appropriate reference resources. A canine catalogue constructed from long- and short-read metagenomics substantially expanded known taxa and functional genes and improved mapping in an independent dog cohort [26]. This reduces dependence on human-biased databases, but gene recovery still establishes potential rather than expression, metabolite production, or clinical relevance.

Earlier canine metagenomic work similarly identified taxonomic, metabolic, and nutritional capacities that cannot be recovered reliably from broad taxonomic profiling alone [27]. Future studies should connect such capacity to metatranscriptomics, metaproteomics, targeted metabolites, substrate use, and host outcomes under controlled dietary exposure. Sampling should cover relevant gastrointestinal compartments where feasible and distinguish transient response from stable adaptation.

External validation must preserve major effect modifiers. Multi-breed pig evidence shows that microbiome–feed-efficiency relationships vary with breed and growth stage [28]. Replication should therefore test transportability across genotype, age, disease, geography, housing, feed formulation, season, laboratory, and analytical pipeline rather than reproduce only internal discrimination.

The proposed framework itself requires prospective testing. Studies should compare decisions made with and without microbiome information, prespecify the alternative actions and meaningful outcomes, and record cases in which constraints block intervention. Validation should assess calibration, decision change, benefit, harm, feasibility, persistence, and reversibility. Negative and discordant results are essential because a useful framework must identify when microbiome evidence should not change nutrition, not merely when a change can be rationalized.

Conclusion

The veterinary gut microbiome should influence nutrition only when it changes a defensible clinical or production action. Taxonomic differences, predicted functions, metabolites, dysbiosis measures, and diversity shifts occupy different evidential levels and should not be treated as interchangeable proxies for benefit. The framework proposed here places nutritional adequacy, safety, disease compatibility, species and life-stage context, host response, production feasibility, and meaningful outcomes around a noncompensatory actionability boundary. It permits microbiome evidence to refine choices among acceptable options while preventing microbial attractiveness from overriding established nutritional priorities. Decisions remain conditional and reversible because diet, disease, medication, environment, and microbial function change over time. Species-specific reference catalogues, standardized measurement, functional confirmation, prospective comparisons, external validation, and explicit decision-impact testing are required before routine implementation. Precision nutrition will depend less on generating increasingly detailed microbial descriptions than on demonstrating when those descriptions justify doing something different for the animal.

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