KoeFormula — Mechanism & Formulation Summary

For veterinary professionals · August 2026

Premise. Roughly two-thirds of canine epilepsy is classified idiopathic — no structural cause found within a standard workup. This formulation addresses one candidate mechanism within that population: a rate limitation in the conversion of glutamate to GABA, secondary to impaired absorption and activation of the B vitamins that conversion depends on. It is a nutritional adjunct, not an anticonvulsant.

THE MECHANISTIC CHAIN

1. Seizure activity reflects imbalance between excitatory (glutamate) and inhibitory (GABA) neurotransmission. **2.** GABA is synthesised from glutamate by glutamic acid decarboxylase (GAD), which requires pyridoxal-5′-phosphate (the active form of B6) as an obligate cofactor. **3.** Activation of B6, folate and B12 depends on methylation, which depends in turn on adequate riboflavin and B12 status. **4.** Chronic dysbiosis — classically post-antibiotic — degrades both absorption and microbial contribution to this pathway. The proposition is that in a subset of dogs the limiting factor is not the enzyme but its cofactor supply, and that this is nutritionally addressable.

WHAT THE LITERATURE SUPPORTS

- **Ketogenic/MCT diets reduce seizure frequency in dogs.** Law et al. (2015), a 6-month randomised, double-blinded, placebo-controlled crossover trial in 21 ASD-treated dogs with IE: significantly lower seizure frequency on the MCT diet; 3 dogs achieved seizure freedom and 7 more had ≥50% reduction. Replicated as an oil supplement by Berk et al. (2020), n=28, with a smaller effect — 2 seizure-free, 3 with ≥50% reduction, 11 with no change or worse.
- **Canine IE is associated with fecal dysbiosis.** García-Belenguer et al. (2021): drug-naïve epileptic dogs showed significantly reduced abundance of GABA-producing and SCFA-producing taxa versus household controls; 30 days of phenobarbital or imepitoin did not alter composition. Since replicated (Animal Microbiome 2025; Sci Rep 2025).
- **PLP dependence of GAD** is settled biochemistry, and pyridoxine-dependent epilepsy is an established, if rare, human entity.

WHAT IS INFERENCE, NOT EVIDENCE

- **No controlled trial of this formulation exists.** The evidence base is one dog followed with a dated log, plus early field reports. Treat every claim below accordingly.
- The chain *antibiotics → dysbiosis → impaired B-vitamin status → limited GAD throughput → seizure threshold* is mechanistically coherent and each link has support in isolation. The chain as a whole has not been demonstrated in dogs.
- Association between dysbiosis and IE does not establish direction. Seizures, drugs and diet all plausibly alter the microbiome.
- Dietary glutamate load as a proximate trigger is drawn from human observation, not canine data.

COMPOSITION — PER 2.5 G SCOOP

COMPONENT	PER SCOOP	RATIONALE
Vitamin B6 (pyridoxal-5′-phosphate)	3.6 mg	Obligate cofactor for GAD; supplied pre-activated
Riboflavin (riboflavin-5′-phosphate)	7.3 mg	Required to activate B6 and support folate cycling
Folate (L-5-MTHF)	242 mcg	Methylation; bypasses MTHFR-dependent conversion
Vitamin B12 (methylcobalamin)	242 mcg	Methylation; commonly low with chronic enteropathy
Betaine anhydrous (TMG)	145 mg	Alternate methyl donor via BHMT
Taurine (free-form)	121 mg	Reported to increase GAD activity; binds GABA-A directly
Glycine	121 mg	Inhibitory neurotransmitter; glutathione precursor
Magnesium (whole-food, buckwheat/chard base)	11.7 mg	Voltage-dependent NMDA receptor block
Ground wild herring	carrier	Long-chain omega-3; palatability — dogs self-consume
Acacia fiber	carrier	Prebiotic; flow agent

Dosing is by body weight: <10 lb ½ scoop · 10–25 lb 1 · 26–50 lb 2 · 51–75 lb 3 · 76 lb+ 4, once daily on food. Manufactured in small batches in an FDA-registered animal food facility (FFR 15758047648).

Two things you will notice, answered plainly. Magnesium is low at 11.7 mg/scoop — this is inherent to the whole-food source and is not a magnesium-loading strategy; a dog requiring therapeutic magnesium needs it dosed separately. **Glycine is an NMDA co-agonist** as well as an inhibitory transmitter, so its net effect in a high-glutamate state is arguable. It is included at a modest dose for its inhibitory and glutathione roles; that trade-off is open, and informed disagreement is welcome.

POSITIONING — AND WHAT IS BEING ASKED

This is an adjunct. It is not an anticonvulsant, it does not replace antiseizure medication, and no owner should reduce or discontinue a prescribed drug on the basis of it. The candidates it is likeliest to suit are dogs with a normal workup, a recurring interval of roughly 4–8 weeks, and a history of prolonged antibiotic exposure or chronic GI signs. Dogs with paroxysmal dyskinesia — conscious throughout, immediate recovery, no post-ictal phase — are outside the proposed mechanism entirely and should not be enrolled.

A six-month case series is open and recruiting through koeformula.com. Dated owner-kept seizure logs, no change to existing medication, monthly or quarterly interim reporting. Referrals from practices willing to co-monitor are the most useful thing at this stage — and negative results are as publishable as positive ones.

References. Law TH et al. A randomised trial of a medium-chain TAG diet as treatment for dogs with idiopathic epilepsy. *Br J Nutr* 2015;114:1438–47. · Berk BA et al. A multicenter randomized controlled trial of medium-chain triglyceride dietary supplementation on epilepsy in dogs. *J Vet Intern Med* 2020;34:1248–59. · García-Belenguer S et al. Gut microbiota in canine idiopathic epilepsy: effects of disease and treatment. *Animals* 2021;11:3121. · Idiopathic epilepsy in dogs is associated with dysbiotic faecal microbiota. *Animal Microbiome* 2025. · The fecal metabolome and microbiome are altered in dogs with idiopathic epilepsy. *Sci Rep* 2025. · Exploring gut microbiota-targeted therapies for canine idiopathic epilepsy. *Int J Mol Sci* 2025;26:1742.

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