MODULE ONE

Krill Oil in Canine and Feline Medicine

Phospholipid delivery, astaxanthin, joint support, and erythrocyte monitoring

High-astaxanthin krill oil for joints and general health

BEFORE WE BEGIN

Learning objectives

01 Explain why dogs and cats are best supplied preformed EPA and DHA (marine or algal) for therapeutic use.

02 Compare triglyceride-bound versus phospholipid-bound omega-3 delivery.

03 Describe astaxanthin mechanisms in joint support and general well-being.

04 Use the Omega-3 Index (OmegaQuant; dogs only) for baseline and serial monitoring.

05 Apply evidence-based EPA + DHA doses and the NRC safe upper limit (dogs).

FOUNDATION

Essential fatty acid biochemistry

Limited conversion

Dogs and cats have limited Δ-6-desaturase activity. ALA → EPA conversion is poor (under 10%) in dogs and essentially absent in cats; DHA conversion is negligible in both species.

Preformed marine LC-PUFA

EPA and DHA are best supplied preformed (marine or algal). Plant ALA is not a reliable substitute in companion animals.

EPA: eicosanoid shift

EPA displaces arachidonic acid from eicosanoid production (PGE_3, LTB_5). DHA-derived resolvins and protectins further help resolve inflammation.

DHA: membrane structure

DHA is a major structural lipid of neuronal and retinal membranes and is the principal fatty acid of interest for Module Two.

Triglyceride versus phospholipid form

MOST FISH OILS · TRIGLYCERIDE

- EPA and DHA esterified to triglycerides
- Require pancreatic lipase and micellar solubilization
- Depend on bile-salt emulsification
- Packaged into chylomicrons → lymphatic transit
- Oil phase separates from gastric contents
- GI upset is dose-limiting at therapeutic doses

KRILL OIL · PHOSPHOLIPID

- EPA and DHA esterified to phospholipids (mainly PC)
- Form micelles readily
- Absorbed in part as lyso-phosphatidylcholine
- May favor phospholipid-pathway absorption
- Delivered within a phospholipid matrix
- GI tolerance not established head-to-head

EVIDENCE

Why phospholipid delivery raises the Omega-3 Index

Micelle formation

Phospholipid-bound fatty acids assemble into mixed micelles more readily than triglycerides, which may improve intestinal uptake.

Canine comparison data

Small controlled canine studies (krill meal and krill oil, largely industry-affiliated) suggest phospholipid-bound omega-3s raise the erythrocyte Omega-3 Index more efficiently than equivalent fish-oil doses.

Dose implication

A lower absolute milligram dose may still meaningfully raise omega-3 status. That matters for palatability, cost, and GI tolerance.

Clinical takeaway

Do not treat ‘mg EPA+DHA on the label’ as interchangeable across chemistries. The carrier may change the tissue result.

Astaxanthin: structure and co-localization

- Xanthophyll carotenoid present naturally in krill oil; content varies by lot — see third-party COAs at www.krilpet.com (3rd Party Testing).
- Among the most potent lipid-soluble antioxidants characterized.
- Esterified within the krill lipid fraction — it does not travel as a free passenger.
- Remains associated with the lipid fraction through intestinal uptake and lipoprotein transport (proposed).
- That co-localization may let astaxanthin protect the cargo it accompanies.

TEACHING POINT

Astaxanthin is not an add-on pigment. In krill oil it is esterified within the same lipid fraction that carries EPA and DHA.

High-astaxanthin krill oil is therefore a dual-payload product: fatty acids plus a membrane-resident antioxidant.

TRANSPORT

Astaxanthin may protect LDL cargo (proposed)

1

Packaging

Phospholipid-bound EPA/DHA enter circulating lipoproteins, including LDL particles.

2

Co-localization

Esterified astaxanthin travels in the same particles as the polyunsaturated cargo.

3

Antioxidation

Astaxanthin may limit oxidation of co-transported PUFAs during LDL transit (proposed mechanism).

4

Delivery intact

Proposed consequence: less-oxidized cargo on arrival — not yet demonstrated in dogs or cats.

Clinical implication. Co-delivery is mechanistically distinct from giving a separate antioxidant — but transport protection has not been demonstrated clinically.

Astaxanthin in mitochondrial membranes

Position
Astaxanthin spans lipid bilayers, including mitochondrial membranes — the electron transport chain is the primary site of superoxide generation.

Action
It scavenges reactive oxygen species at their source rather than downstream in the cytosol.

Consequence
Proposed consequence: ATP production is better preserved and mitochondrial permeability transition is less likely (mechanistic; not demonstrated in vivo).

WHY THIS MATTERS IN JOINTS

Chondrocytes and synoviocytes under inflammatory load generate mitochondrial ROS. Protecting that membrane is not a cosmetic claim — it is energy metabolism in the tissue you are trying to spare.

INDICATION

Astaxanthin and EPA in joint support

NF-κB	Suppresses transcription of IL-1β, TNF-α, and IL-6 in cultured joint cells (in vitro).
Eicosanoids	Lowers NO and prostaglandin output in cultured chondrocytes and synoviocytes; EPA shifts $PGE_2 \rightarrow PGE_3$.
MMPs	Inhibits MMP-1, MMP-3, and MMP-13 in vitro — enzymes that degrade type II collagen and aggrecan.
RNS / ROS	Scavenges peroxynitrite from activated neutrophils and macrophages in the joint.
Additivity	Astaxanthin is complementary to EPA's eicosanoid modulation, not a substitute for it. Astaxanthin mechanisms shown are in-vitro and rodent-model findings; EPA/DHA joint support benefit is supported by clinical trials.

BEYOND THE JOINT

Astaxanthin and general well-being

Skin and coat

Lipid-soluble antioxidant support for integument; coat quality is often the first change owners report (anecdotal).

Exercise recovery

Mitochondrial ROS scavenging is relevant to muscle after work; reduced soreness is a plausible owner-facing benefit (preclinical).

Immune tone

Modulation of inflammatory transcription is not joint-specific; leukocyte membranes take up astaxanthin (in-vitro and rodent data).

Photoprotection

Carotenoid accumulation in skin may provide a modest buffer against UV oxidative load (extrapolated from human data).

What the Omega-3 Index measures

- The Omega-3 Index is EPA + DHA as a percentage of total erythrocyte fatty acids.
- It reflects roughly 120 days of membrane turnover in dogs (about 70 days in cats) — the circulating life of the red cell.
- Plasma omega-3s swing within hours of a meal and are a poor guide to tissue status.
- RBC membranes are the better baseline and the better serial marker for krill-oil administration.

STABILITY

Plasma = hours.
RBC index ≈ 3–4 months (dogs).

USE IT FOR

Baseline status before you start.
Confirmation that the dose actually changed membranes.

HOW TO USE IT

OmegaQuant protocol in practice (dogs)

01

Baseline

Draw before the first dose. You cannot interpret a later index without knowing where the patient started.

02

Start krill oil

Begin the high-astaxanthin product at a conservative therapeutic dose and titrate if GI signs appear.

03

Recheck 8–12 weeks

This window matches the clinical joint support response; the index continues climbing toward its new level over 3–4 months (dogs).

04

Adjust, don’t guess

If the index has not moved, check compliance, diet contribution, and product content before simply doubling the dose.

NUMBERS THAT CHANGE PRACTICE

Therapeutic dosing for joint support

joint support STUDY DOSES	CATS	NRC CEILING
EPA 48–100 mg/kg/day — pooled canine and feline studies DHA 20–32 mg/kg/day — pooled canine and feline studies Common canine target ≈ 100 mg/kg total EPA+DHA	Total EPA+DHA ≈ 120 mg/kg/day (feline joint support trials) Titrate from a lower start. Watch stool quality closely.	370 mg total EPA+DHA (food + supplements) per kg^0.75 per day (dogs only) No NRC feline safe upper limit exists A safe upper limit for dogs — not a target.

Titrate upward. Monitor for loose stool or vomiting. Clinical joint support improvement typically requires 8–12 weeks. Subtract omega-3 already present in therapeutic diets. OmegaQuant recheck: dogs only.

WHAT TO PUT ON THE SHELF

Product selection for this module

Verified content

EPA, DHA, and astaxanthin amounts on the label should match a certificate of analysis — not a marketing range.

Oxidation testing

Ask for peroxide and anisidine values. Rancid oil is not a phospholipid success story.

Diet accounting

Joint diets already carry marine oil. Add, don’t blindly stack.

Sustainability

Krill harvest is regulated; still require a documented source.

This product

High-astaxanthin krill oil is the form discussed for joint and general-health use in Module One.

Not this module

Concentrated phospholipid / cognition product is Module Two. Do not collapse the two labels.

TAKE HOME

Module One in five lines

1. Dogs and cats cannot make meaningful EPA or DHA from ALA — supply them preformed (marine or algal).
2. Phospholipid-bound EPA/DHA may be absorbed more efficiently than triglyceride forms in dogs (small studies).
3. Astaxanthin is esterified in the krill lipid fraction; proposed mechanisms include co-transport protection and membrane antioxidant activity.
4. Joint benefit is dual: EPA changes eicosanoids; astaxanthin adds NF-κB, MMP, and ROS/RNS control.
5. OmegaQuant (RBC Omega-3 Index; dogs only) is the monitoring tool. Baseline, then 8–12 weeks.

Selected references

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Astaxanthin mechanisms: Sun K, et al. (2019). Astaxanthin protects against osteoarthritis via Nrf2. Aging 11 — https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6914430; krill astaxanthin ester composition — https://pmc.ncbi.nlm.nih.gov/articles/PMC8226823/ (interpret species and model carefully).