Commonality in features observed as early responses in healthy dogs fed a legume-rich diet and in dogs diagnosed with dilated cardiomyopathy 2018-2020

Anne Marie Bakke¹, Prof. Dr.med.vet. (DVM, PhD)  
Wood J¹, Gilham M¹, Kuhlman G², Bierer T², Allaway D¹, Butterwick R¹, O’Flynn C¹  
¹Waltham Petcare Science Institute, Leicestershire, UK  
²Mars Pet Nutrition NA, Tennessee, US

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Background

In 2018, the FDA announced their investigation of reports of DCM in dogs given certain foods in the "grain-free“ category across breeds and age groups. A multi-factorial etiology has been suggested (Freeman et al. 2018)

We therefore

1. Initiated a pilot trial investigating early responses in healthy, adult large breed dogs when fed a high-legume test diet for 30 days

and subsequently

2. Attempted to gain an understanding of the clinical relevance of data from the pilot trial by interrogating the database of electronic medical records at BANFIELD™


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1) Pilot trial

Main hypotheses:
Legume-rich diet will impact:
– Sterol metabolism and thereby taurine status
– Other components/nutrients vital for heart health, e.g. carnitine, K, Cu, Zn, Se, Vit. E, thiamine

As always, routine health parameters i.e. hematology, clinical biochemistry, urinalysis, were also measured
1) Pilot trial – Design

Parallel design

30-day feeding trial

Eleven healthy, adult Labrador retrievers

- Routine hematology and clinical biochemistry
- Ultrasound scans

Diets

- **Control** (n=5) – mainstream, low-legume formulation
- **Test** (n=6) – 20% split peas, 40% red & green lentil mix, 6% flaxseed
  - With the exception of fibre, similar in proximate composition
  - Nutrient composition of both compliant with NRC, AAFCO and FEDIAF guidance
1) Pilot trial – Results

Test compared to Control diet-fed dogs (p<0.05)

- Reduced RBC counts, HCT, total HGB
- Increased plasma phosphate
- Reduced protein, carbohydrate, K and Na apparent digestibilities
- Taurine status
- Fecal bile acids
  - Increased for primary bile acids
  - Decreased for secondary bile acids
1) Pilot trial - Results

- Progressive reductions

Hemolysis? Changes in plasma or urinary bilirubin were not observed
1) Pilot trial - Results

• Progressive elevation

→ Hyperphosphatemia (>1.60 mmol/L)

WAL ref. range 0.80-1.60 mmol/L
1) Pilot trial - Results

Primary bile acids
A. Cholic acid (CA)
B. Chenodeoxycholic acid (CDCA)

Secondary bile acids
C. Deoxycholic acid (DCA)
D. Lithocholic acid (LCA)
E. Hyodeoxycholic acid (HDCA)
1) Pilot trial - Conclusions

Some rather surprising results were observed
Pathophysiological implications for DCM development were not understood

• Anemia as a cause of DCM?
• Hyperphosphatemia a result of hemolysis? Or a product of Ca and P regulatory changes?

Further investigations needed
• To verify the clinical relevance

Therefore, we compared our results with electronic medical records of dogs with DCM in the diagnosis at Banfield™ clinics and hospitals
2) Clinical relevance
BANFIELD™ database interrogation

OBJECTIVE

- Investigate and identify the most informative features to separate DCM cases from controls

RAW DATA PULL from the electronic medical records (EMRs) of dogs at BANFIELD™ clinics and hospitals in 2018-2020
2) Clinical relevance

Dataset and Processing Steps

Dataset

- 39,169 visit records (38,602 healthy control and 567 cases that have/develop DCM) between 2018 and early 2020
- All cases of DCM

Processing Steps

- 32 features of demographics (BW and age), routine blood biochemistry and haematology
- 390 rows with missing values

Refinement of the dataset, balancing it using propensity scoring, with cases and controls matched for breed, gender and age categories is underway
2) Clinical relevance - Results

Hematocrit (Hct)

Ref. range: 40-55%

BANFIELD™ data

Pilot trial data

Ref. range: 40-55%

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2) Clinical relevance - Results
Plasma inorg. phosphate (Phos)

Pilot trial data

BANFIELD™ data

WAL ref. range 0.80-1.60 mmol/L

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2) Clinical relevance - Results
Alkaline phosphatase (ALP)

Bone-specific ALP isoform is currently being analysed.
2) Clinical relevance - Results
Plasma Creatinine

BANFIELD™ data

Pilot trial data

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Preliminary conclusions

Similar features observed in both studies

The refined dataset has so far confirmed the significance of the raw data and identified further parameters of potential interest

Further investigation is needed to verify whether high dietary inclusion of split peas, lentils and/or flaxseed cause these changes and their contribution to the development of DCM

• Anemia as a cause of DCM?
• Hyperphosphatemia a result of hemolysis? Or a product of Ca and P regulatory changes?
• Creatinine’s role?
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Thank you
Questions?

anne.marie.bakke@effem.com

@waltham_science
linkedin.com/showcase/waltham-science/
facebook.com/walthamscience

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