

Chronic Pancreatitis: Diagnosis & Management Tips to Improve Outcomes

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CONFLICT OF INTEREST DISCLOSURE: CRIDGE

I have financial interest, arrangement or affiliation with:

- Blue Buffalo: Speaker honorarium
- CEVA Animal Health: Speaker honorarium, grant recipient & consultant
- IDEXX Laboratories: Speaker honorarium
- Nutramax: Speaker honorarium
- Royal Canin: Speaker honorarium, grant recipient (via CGS)



CONFLICT OF INTEREST DISCLOSURE: STEINER

● Gastrointestinal Laboratory	Director
● IDEXX Laboratories	Paid Consultant
● Nutramax Laboratories	Paid Consultant
● ISK	Paid Consultant
● CEVA Animal Health	Paid Consultant
● Glycosbio	Paid Consultant
● Bond Pet Care	Paid Consultant
● Royal Canin, Nutramax Laboratories, Hill's Pet Care, IDEXX Laboratories	Paid Speaker
● Hill's Pet Care, Nutramax Laboratories	Grant Support



Clinical Characteristics

Acute Pancreatitis

Acute onset of moderate to severe clinical signs

Hypoechoic enlarged pancreas with hyperechoic mesentery

Elevated pancreatic lipase

Chronic Pancreatitis

Waxing and waning mild gastrointestinal signs

Vague sonographic features

Mildly elevated pancreatic lipase



Histopathology

Acute Pancreatitis

Neutrophilic inflammation

No fibrosis or acinar cell atrophy

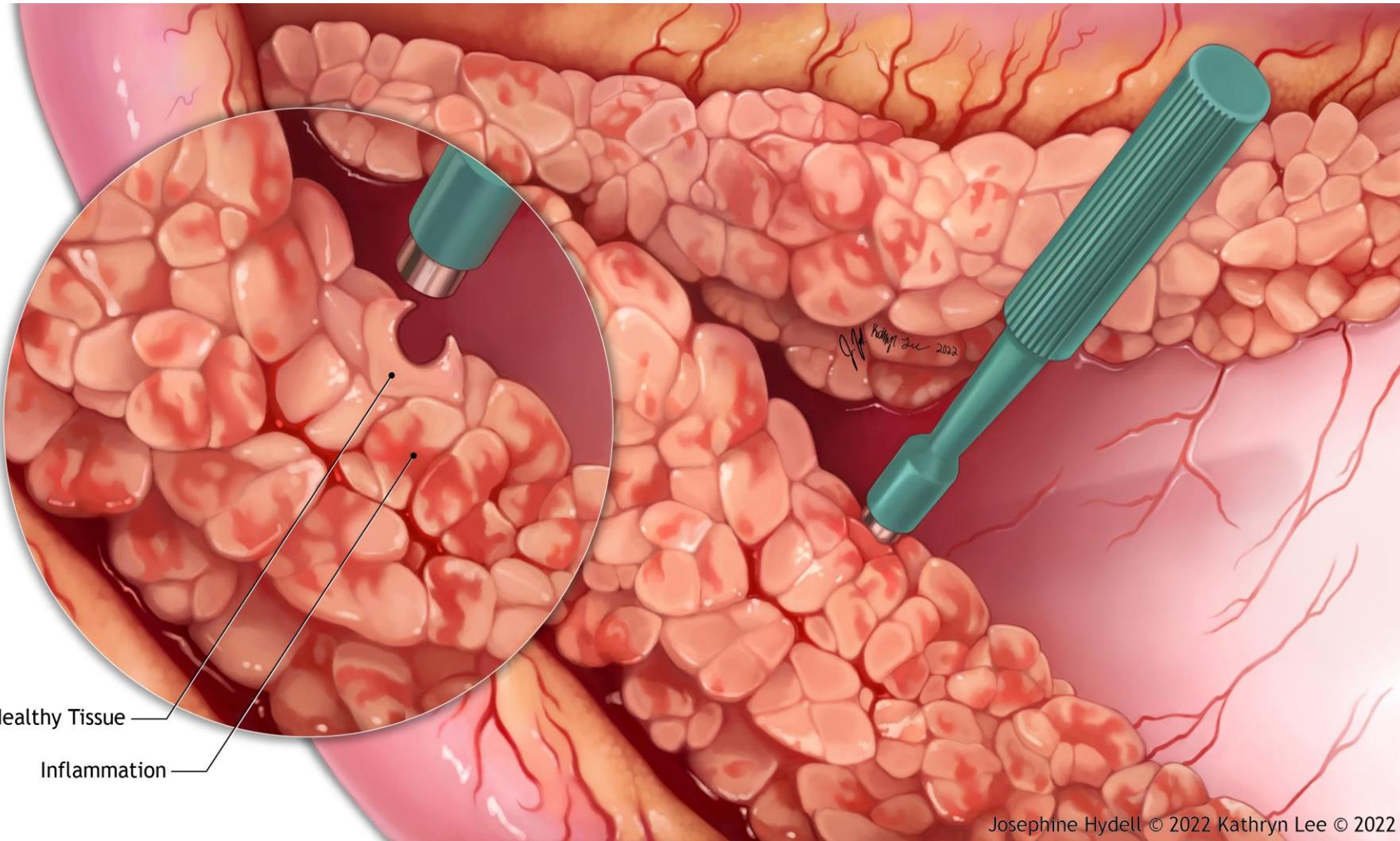
Chronic Pancreatitis

Lymphocytic or lymphoplasmacytic inflammation

Fibrosis & loss of acinar cells



Limitations of Histopathology



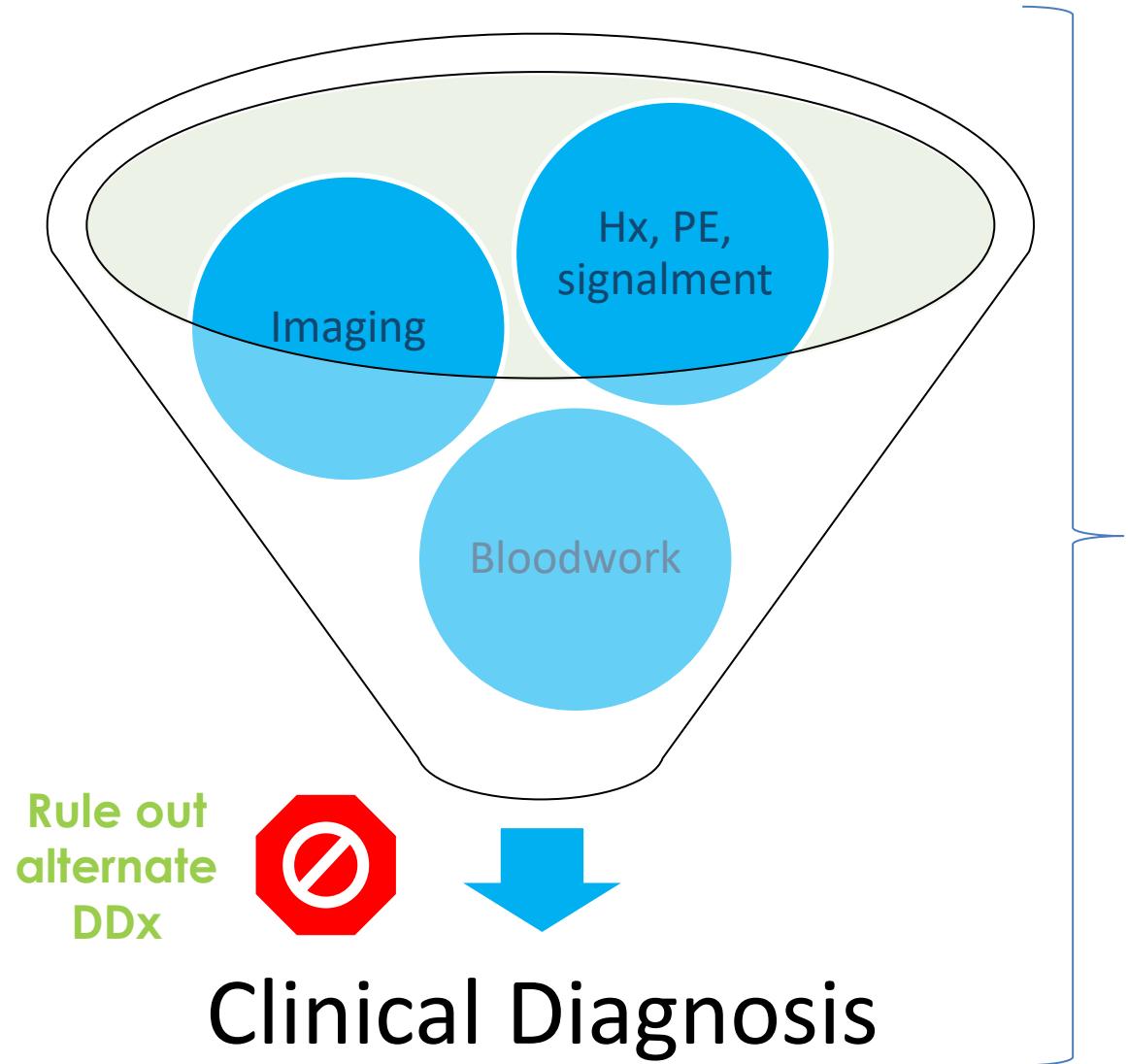
Localized lesions

Cost

Invasiveness

Interpretive Criteria (?)

Proposed Diagnostic Approach



Challenges

Mild C/S

AUS has low sensitivity

'Age-related' changes
mimic pathology on
AUS (?)

Pancreatic lipase is
less elevated vs. acute
disease

Risk of Under Diagnosis

Prevalence of Chronic Pancreatitis

Exact prevalence is unknown

- Uncommon diagnosis despite high prevalences of histopathologic lesions in necropsy studies.

Prevalence and breed distribution of chronic pancreatitis at post-mortem examination in first-opinion dogs

P. J. WATSON, A. J. A. ROULOS,
T. SCASE, P. E. J. JOHNSTON*,
H. THOMPSON* AND M. E. HERRTAGE

Journal of Small Animal Practice (2007)
48, 609–618
DOI: 10.1111/j.1748-5827.2007.00448.x

J Vet Intern Med 2004;18:488–493

Localization of Pancreatic Inflammation and Necrosis in Dogs

Shelley Newman, Jörg Steiner, Kristen Woosley, Linda Barton, Craig Ruaux, and David Williams

- 40% of dogs with acute fatal pancreatitis have features of chronicity

(Hess et al. *J Am Vet Med Assoc*. 1998; 213(5): 665-70).

Microscopic evidence of pancreatitis in 34% of non-autolyzed cases.

Microscopic evidence of pancreatitis in 64% of cases.

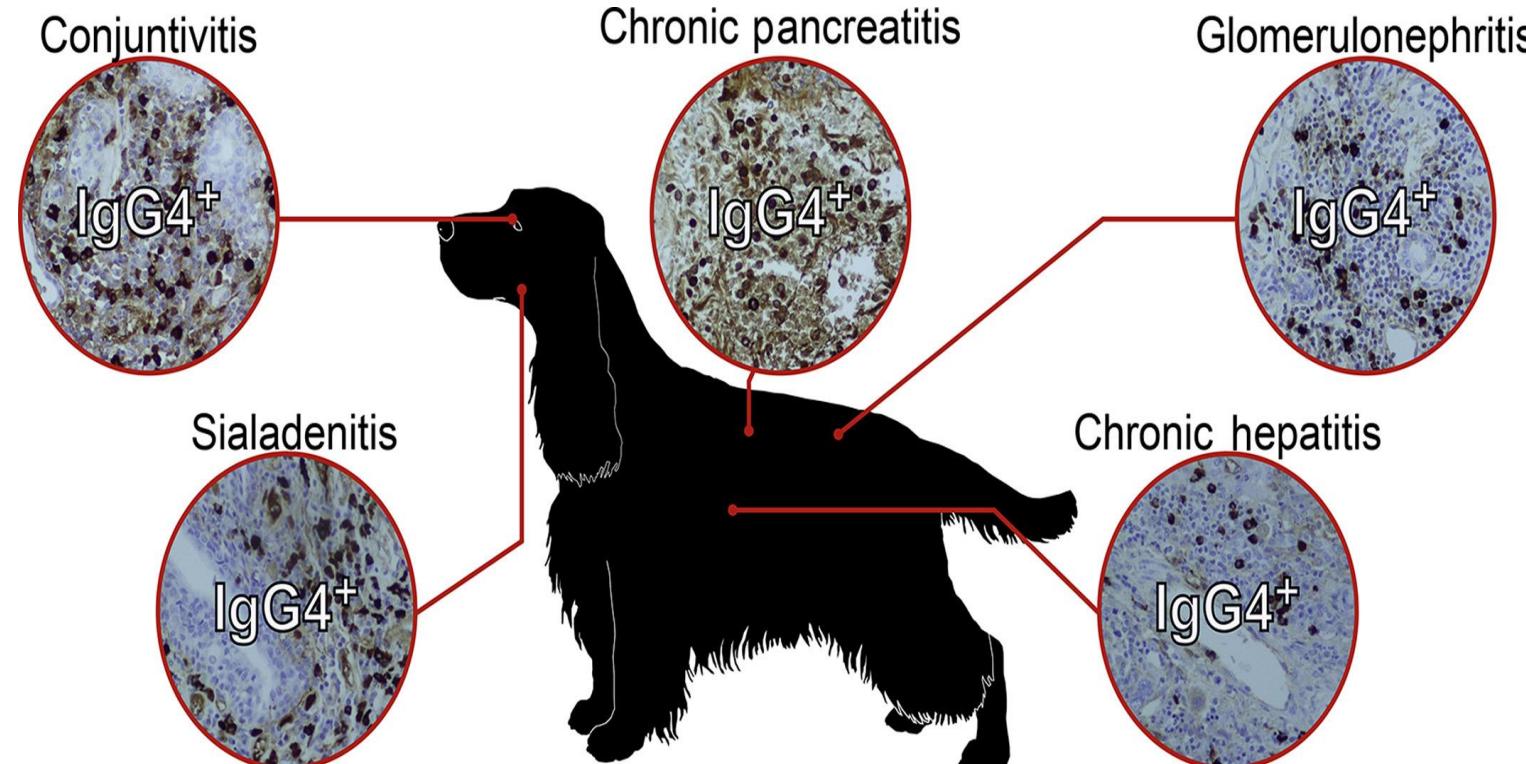


- Limited research
- Presumed idiopathic vs. immune-mediated in the majority of cases
- Diet and underlying endocrinopathies may play a role



Etiology & Risk Factors

- Specific auto-immune disease identified in English Cocker Spaniels



Coddou, et al. *J Comp Path.* 2020; 177: 18-33.



Signalment

Middle aged to older dogs are predisposed

Any breed can be affected

USA

- Non-sporting / toy breeds
- Labradors, boxers, miniature schnauzers, collies, & dachshunds over-represented (*Bostrom, et al. Vet J. 2013; 195: 73-9*)

UK

- Boxers, collies, and spaniel breeds are over-represented
- English cocker spaniel has a unique pattern of disease consistent with IgG4 related disorder in humans



Clinical Presentation

	Clinical pancreatitis no. (%)	Incidental pancreatitis no. (%)	Odds ratio (95% CI)	P*
Vomiting	35/40 (88)	NA	NA	NA
Decreased appetite	39/40 (98)	NA	NA	NA
Lethargy	37/40 (93)	8/16 (50)	12.3 (2.22, 77.7)	<0.001
Diarrhea	19/39 (49)	1/16 (6)	14.3 (1.66, 318)	0.003
Prior history of vomiting	16/36 (44)	2/16 (13)	5.60 (1.01, 56.3)	0.025
Fever	9/37 (24)	3/15 (20)	1.29 (0.26, 8.63)	1.0
Abdominal pain	13/37 (35)	1/15 (7)	7.58 (0.91, 345)	0.043
Diabetes without ketosis	4/40 (8)	0/18 (0)	Undefined ^a	0.299
Diabetic ketosis	7/40 (18)	1/18 (6)	3.61 (0.40, 172)	0.413
Biliary obstruction	6/40 (15)	0/18 (0)	Undefined ^a	0.163
Pancreatic mass	8/40 (20)	2/18 (11)	2.00 (0.34, 21.3)	0.708
Antiepileptic drugs	4/38 (11)	2/15 (13)	0.76 (0.10, 9.46)	1.0
Antibiotics	30/38 (79)	4/15 (27)	10.3 (2.18, 54.1)	<0.001
Plasma	6/38 (16)	0/15 (0)	Undefined ^a	0.167
Corticosteroids	12/38 (32)	6/15 (40)	0.69 (0.17, 2.96)	0.560





Clinicopathologic Data

Table 3

Results of hematologic analysis in 47 of 61 dogs with histopathological evidence of chronic pancreatitis included in this retrospective case series.

Variable	RI	No. of dogs	No. (%) of dogs > RI	No. (%) of dogs < RI	Median (range)
PCV (%)	31-56	47	0 (0)	9 (19)	39.3 (10.1-54.7)
WBCs (cells/ μ L)	6000-17,000	47	21 (45)	3 (6)	16.3 (0.7-76.4)
Segmented neutrophils (cells/ μ L)	3000-11,500	47	28 (60)	2 (4)	14,181 (238-63,412)
Bands (cells/ μ L)	0-300	47	9 (19)	NA	0 (0-4202)
Lymphocytes (cells/ μ L)	1000-4800	47	1 (2)	29 (61)	903 (0-5439)
Monocytes (cells/ μ L)	150-1250	47	17 (36)	1 (2)	828 (134-13,754)
Eosinophils (cells/ μ L)	100-1250	47	0 (0)	22 (47)	101 (0-1200)
Basophils (cells/ μ L)	0	47	4 (8)	NA	0 (0-2292)
Platelets (cells/ μ L)	200,000-500,000	37	4 (11)	7 (19)	332,000 (9000-713,000)

PCV, packed cell volume; WBC, white blood cells; RI, reference interval; NA, not applicable.



Clinicopathologic Data

Table 4

Results of select variables from serum biochemical analysis in dogs with clinical or incidental chronic pancreatitis.

Variable	RI	No. of dogs	No. (%) of dogs > RI	No. (%) of dogs < RI	Median (range)	P*
Cholesterol	120–247 mg/dL					
Clinical		35	19 (54)	3 (9)	262.5 (0–852)	0.032
Incidental		10	2 (20)	1 (10)	186 (81–281)	
ALP	24–147 U/L					
Clinical		35	30 (85)	2 (6)	1103 (21–7667)	0.016
Incidental		10	7 (70)	1 (10)	246.5 (20–2013)	
ALT	10–130 U/L					
Clinical		35	20 (57)	0 (0)	187 (21–2835)	0.006
Incidental		10	2 (20)	0 (0)	38 (11–224)	
GGT	0–25 U/L					
Clinical		35	18 (51)	NA	27 (0–320)	0.008
Incidental		10	2 (20)	NA	8 (5–30)	
Bilirubin	0–0.8 mg/dL					
Clinical		35	17 (49)	NA	0.9 (0–19.6)	0.018
Incidental		10	0 (0)	NA	0.3 (0.1–1.3)	

ALP, alkaline phosphatase; ALT, alanine aminotransferase; GGT, gamma-glutamyltransferase; RI, reference interval; NA, not applicable.

* Based on the comparison of clinical and incidental values using Mann-Whitney *U* tests.



PANCREATIC LIPASE

1

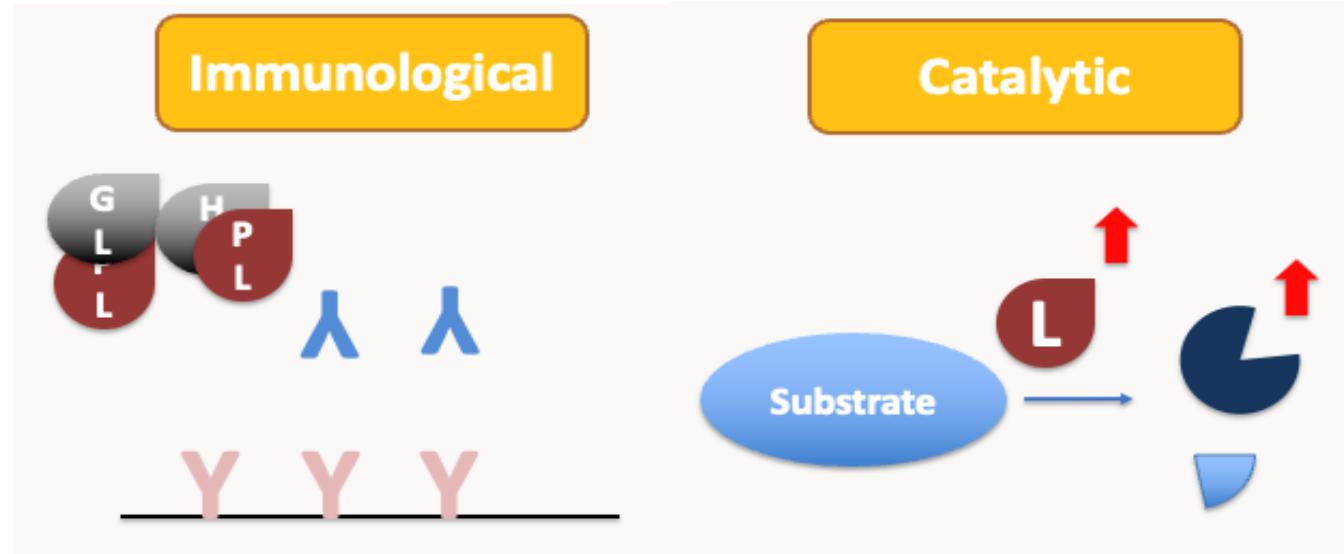
Immunologic assays

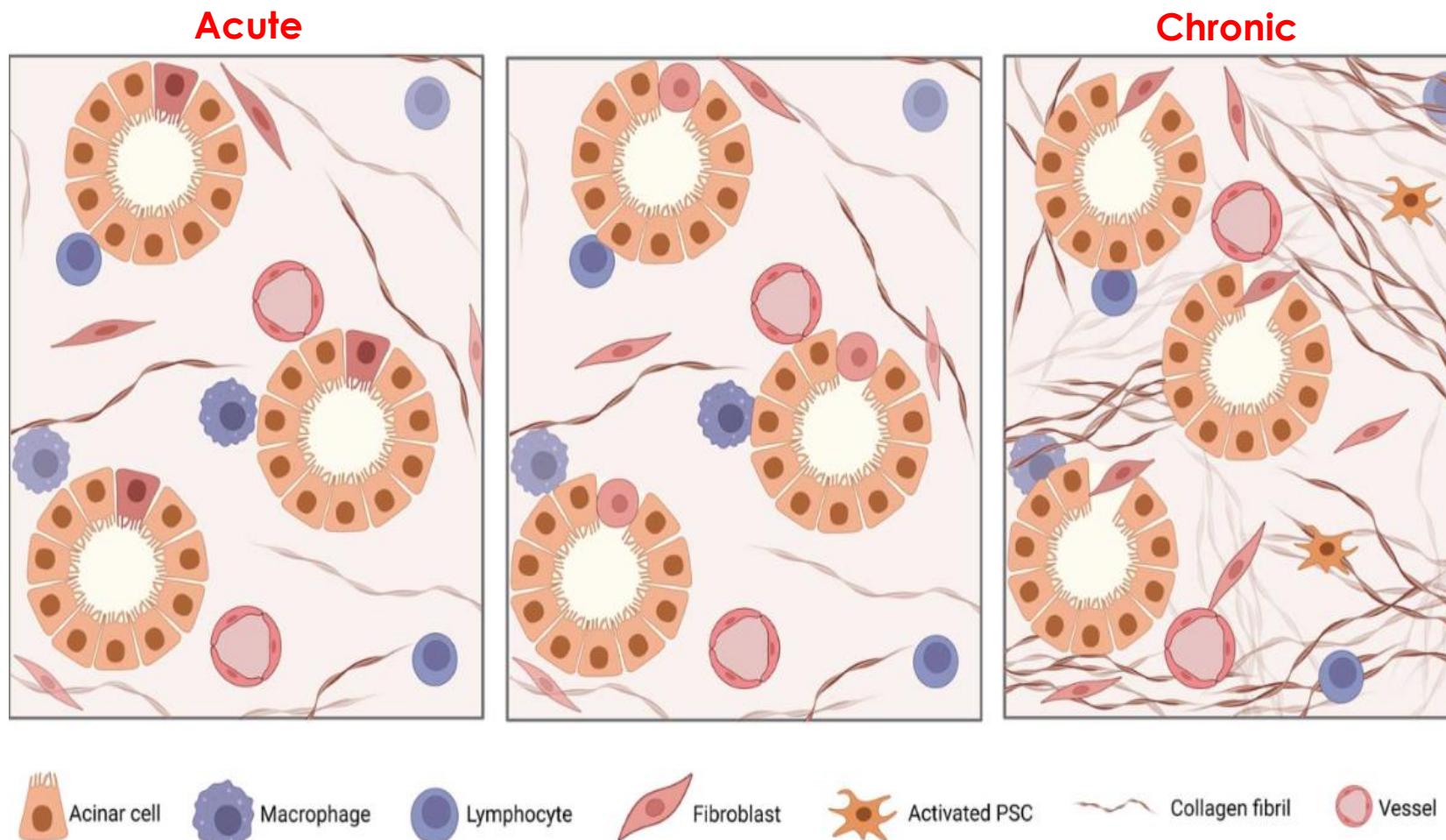
- Spec cPL

2

Catalytic assays

- 1,2-diglyceride (older)
- DGGR (Catalyst PL, Precision PSL)
- Triolein





Due to fibrosis ± loss of acinar cells there is ↓ leakage of pancreatic lipase from acinar cells in chronic pancreatitis vs acute disease

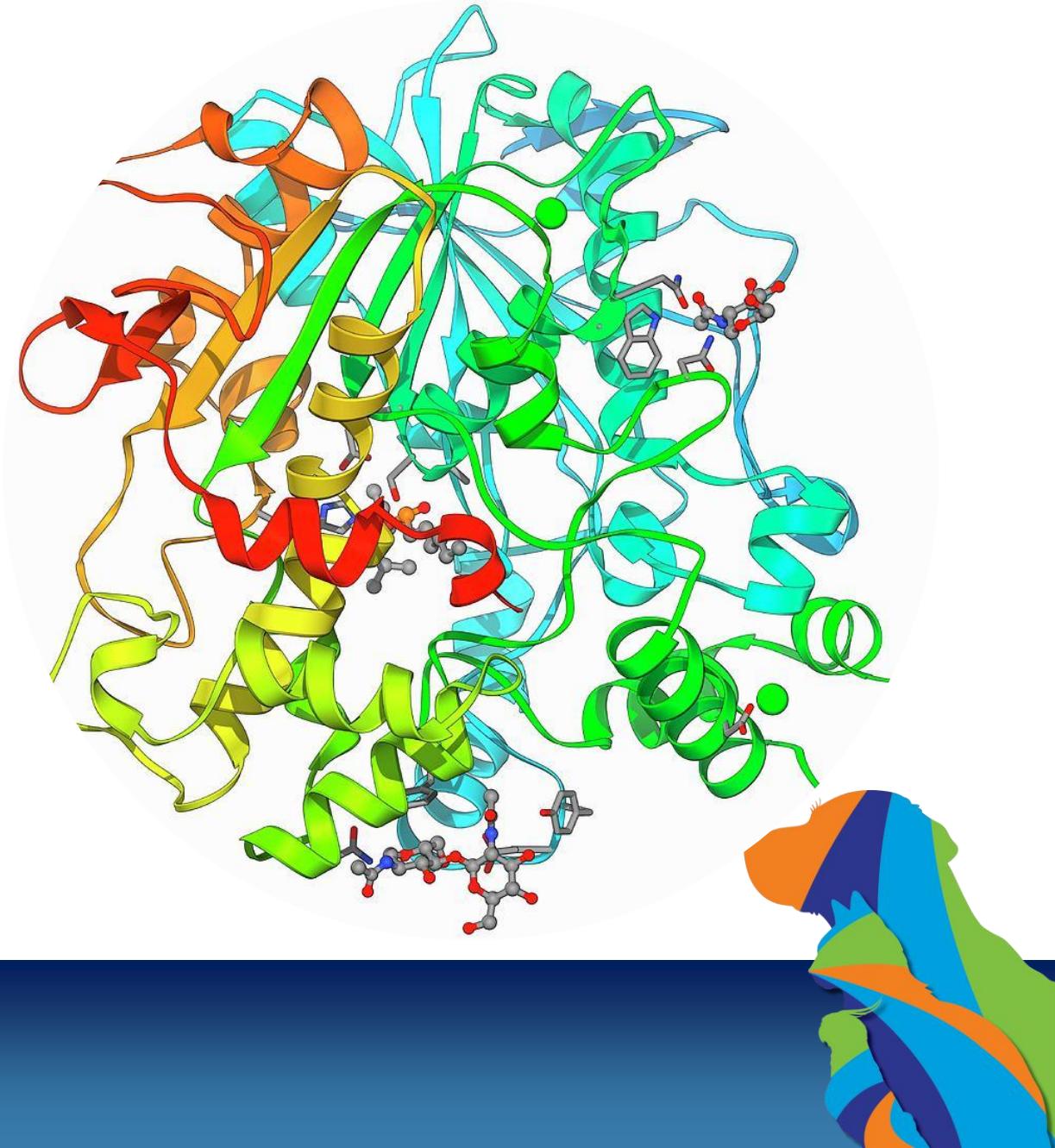


↓ diagnostic sensitivity

MAY NEED TO TEST ON A FEW DIFFERENT OCCASIONS

LIPASE IMMUNOASSAYS

- Use an antibody to bind to specific moieties of the pancreatic lipase molecule
- Helps avoid detection of non-pancreatic lipases



Catalyst PL (IDEXX) – a DGGR based assay

- Internal analytical validation 
- External analytical validation (Texas A&M University GI Lab) 
- Provides a numerical result scaled to align with that of the Spec cPL assay
- Point-of-care / rapid results

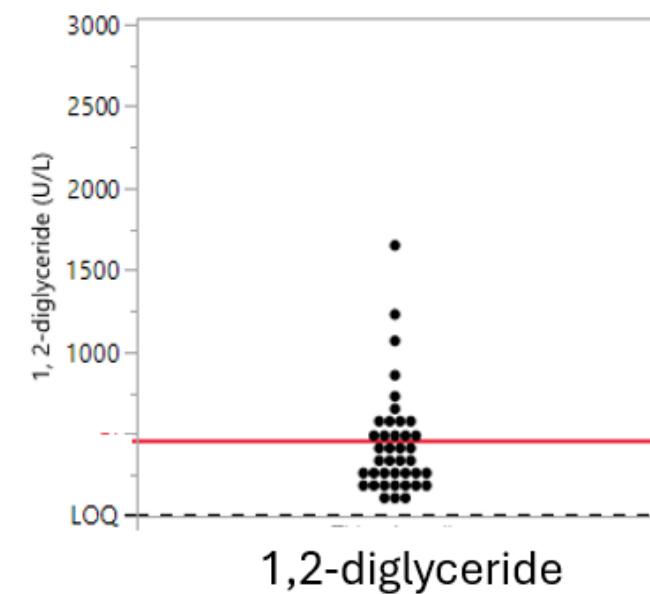
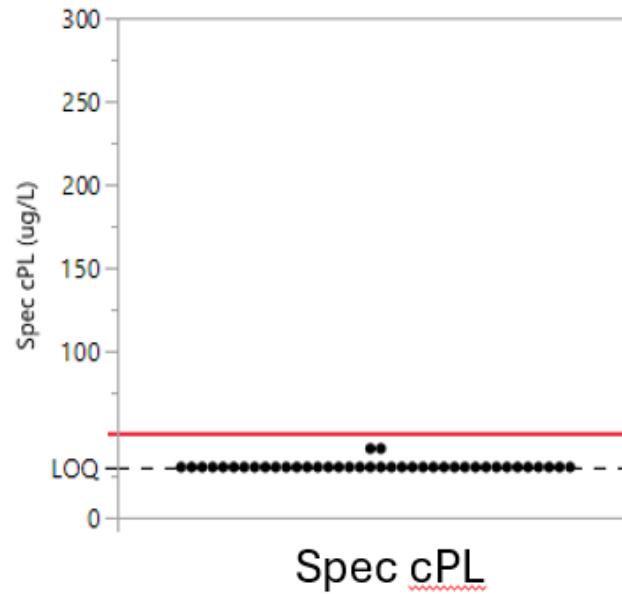
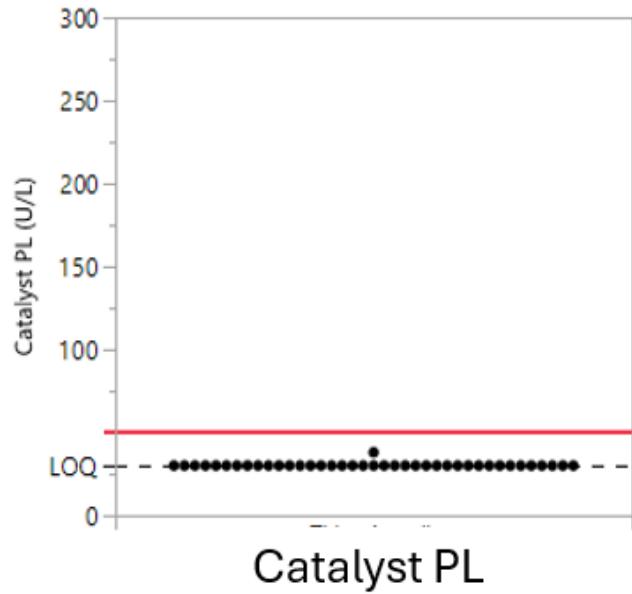


EXTERNAL VALIDATION 1

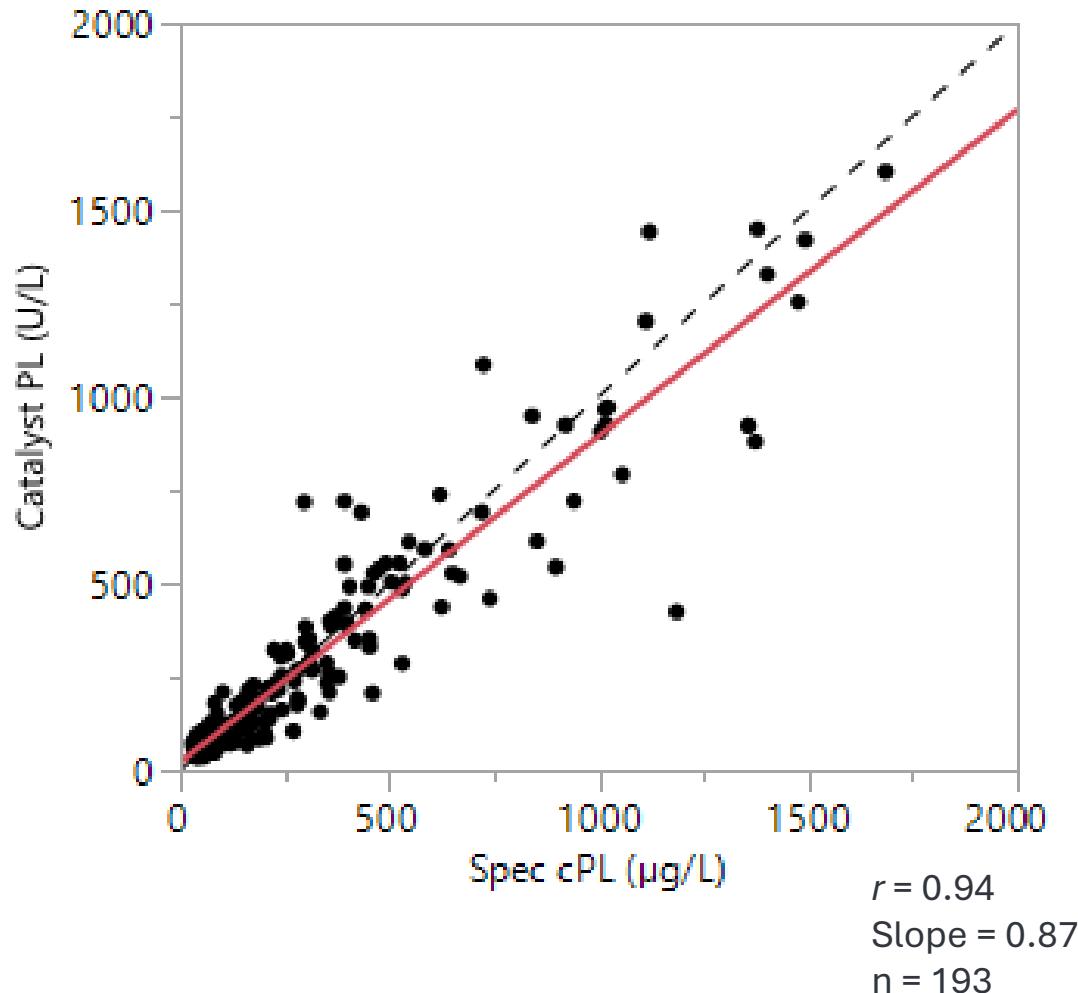
- Precision:
 - intra-assay variability dog: 5.2%
 - intra-assay variability cat: 5.2%
- Reproducibility:
 - inter-assay variability dog: 6.6%
 - inter-assay variability cat: 6.1%



EVALUATION OF SPECIFICITY IN A POPULATION OF PAA DOGS



METHOD COMPARISON & INTERPRETATION

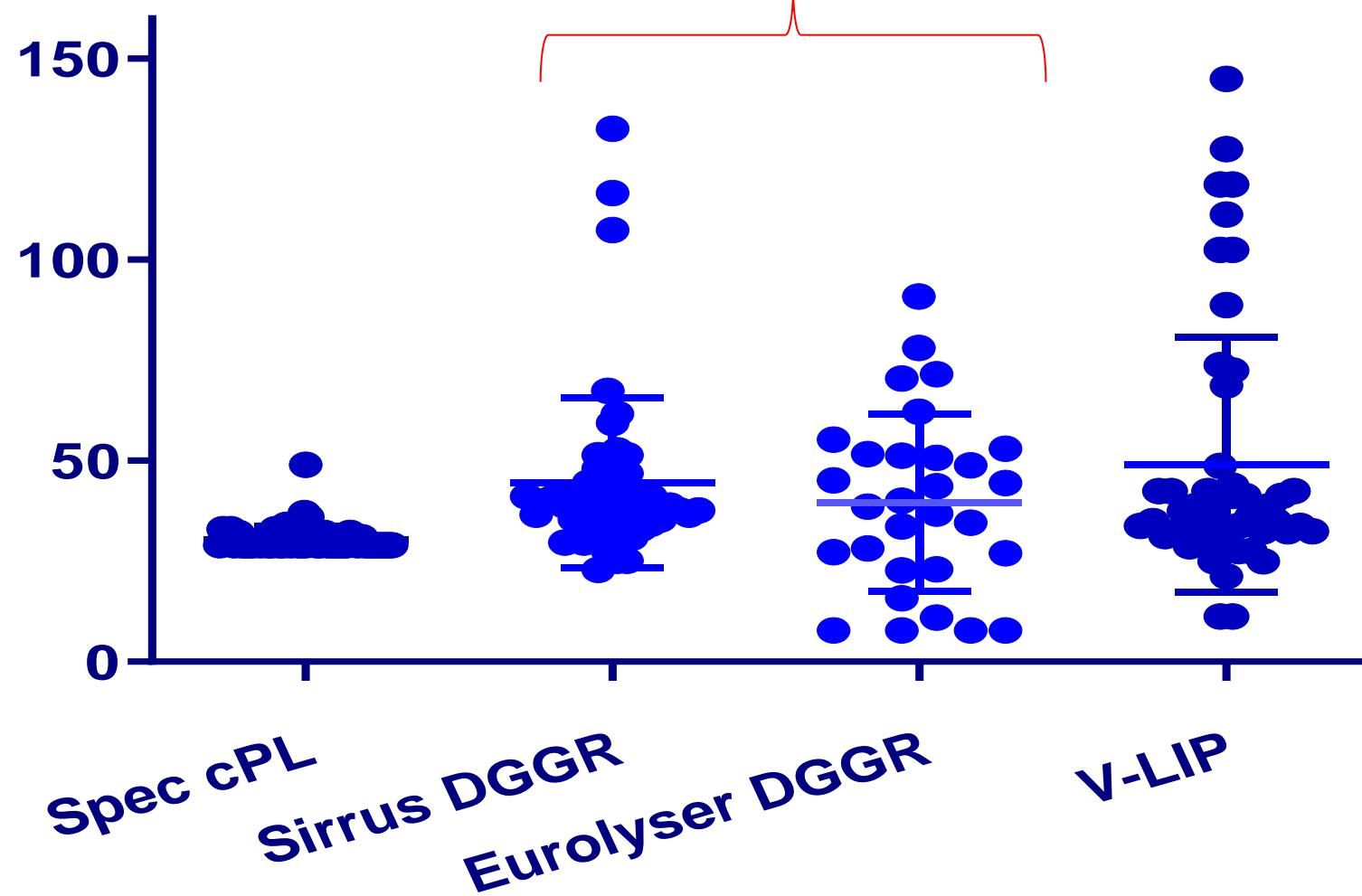


Dogs

- + **$\leq 200 \text{ U/L}$:** Normal reference interval
- + **$201\text{--}399 \text{ U/L}$:** Pancreatic lipase concentration is in the equivocal range, and pancreatitis is possible. Investigate for other diseases and consider additional diagnostics and/or treatment if clinical signs or other evidence of disease exist. Recheck with the Catalyst Pancreatic Lipase Test in 2–3 weeks.
- + **$\geq 400 \text{ U/L}$:** Pancreatic lipase concentration is consistent with pancreatitis. If clinical signs are present, treat appropriately and investigate for risk factors and concurrent diseases, including gastroenteritis or foreign body. Monitor with the Catalyst Pancreatic Lipase Test to assess response to treatment. If clinical signs are not present, consider additional diagnostics, instruct owner to monitor closely, and recheck with the Catalyst Pancreatic Lipase Test in 2–3 weeks.



Analytical specificity is ASSAY specific NOT substrate specific



WHICH LIPASE TEST SHOULD I USE?

Point-of-care	Reference lab	Use
Catalyst® Pancreatic Lipase Test	Spec cPL® Test	<ul style="list-style-type: none">+ Specifically designed to quantitatively measure pancreatic lipase+ Rule out, <u>diagnose, and monitor</u> pancreatitis
SNAP® cPL™ Test		<ul style="list-style-type: none">+ Specifically designed to semiquantitatively measure pancreatic lipase+ <u>Rule out</u> pancreatitis+ Follow up with a Spec cPL Test to diagnose

DIAGNOSTIC IMAGING – ROLE OF ULTRASOUND

- Non-invasive diagnostic tool
- Two roles in diagnostic approach to suspect CP:
 1. Help rule out DDx
 2. Assess pancreatic morphology for features consistent with CP



Image from West Hills Veterinary Centre



Imaging Findings in Chronic Pancreatitis

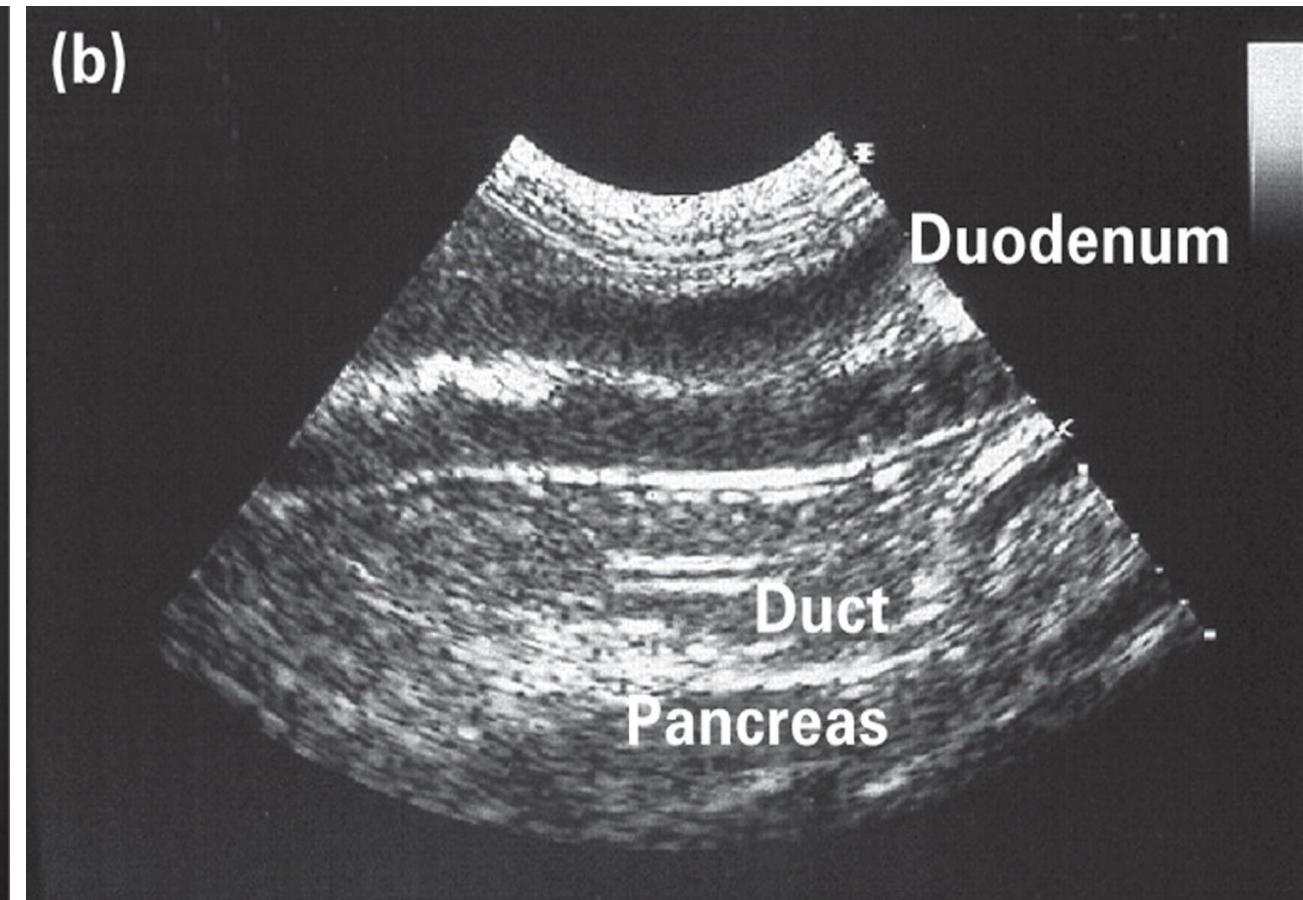
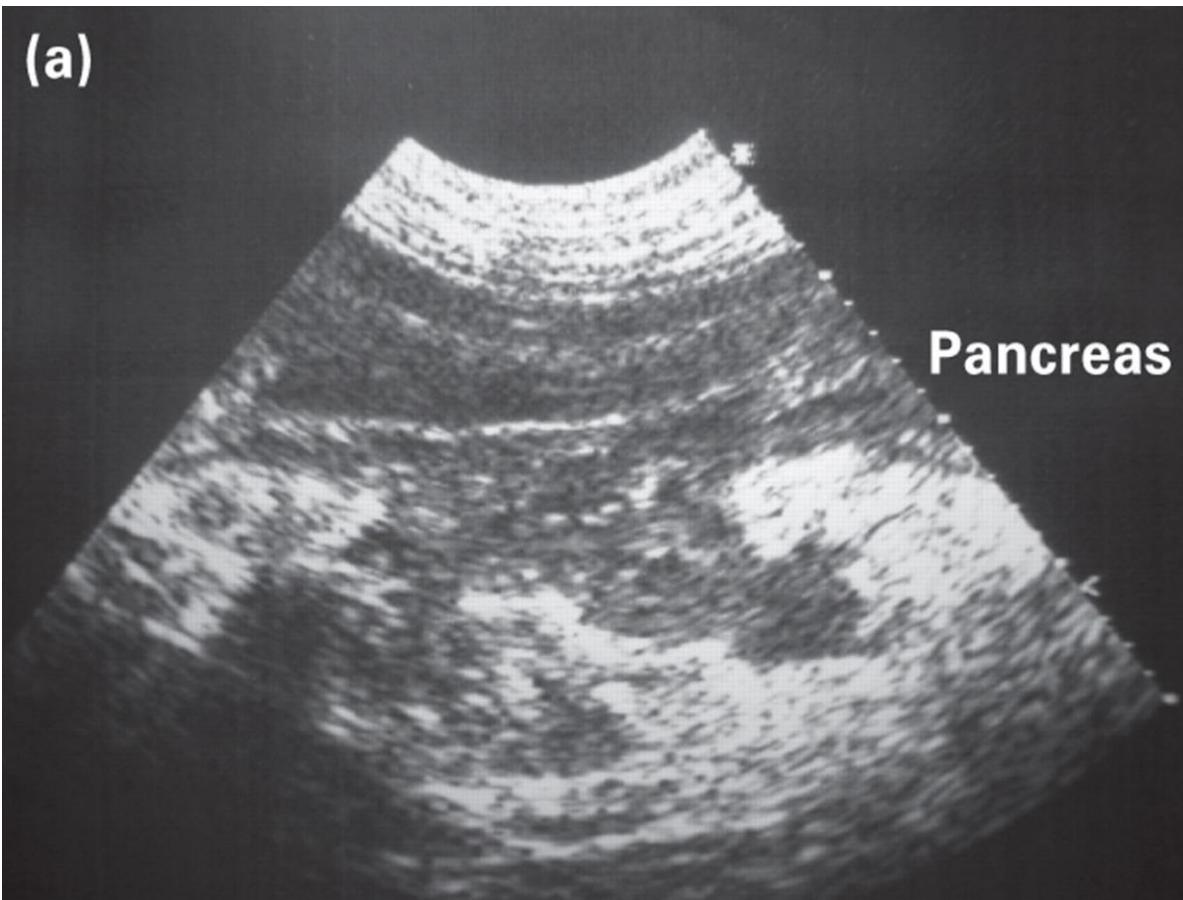
- Low sensitivity → pancreatic abnormalities seen in only 8/14 dogs with CP in an observational study (*Watson et al. Vet Rec. 2010; 167(25): 968-76*).
- Potential sonographic abnormalities (*)
 - Mild thickening of pancreas
 - Irregular pancreatic margins
 - Hyperechoic or heterogenous parenchyma
 - Peri-pancreatic inflammatory changes
- Inflammatory mass lesions (that can mimic neoplasia) can be found in English Cocker Spaniels (and likely other breeds)

* Limited evidenced based data exists on ultrasonographic abnormalities in chronic pancreatitis



Imaging Findings in Chronic Pancreatitis (*)

* Limited evidenced based data exists on ultrasonographic abnormalities in chronic pancreatitis



Abnormally mottled pancreas with a mixed hyperechoic and hypoechoic pattern

Normal right pancreas with duct visualized

Imaging Findings in Acute vs Chronic Pancreatitis (*)

* Limited evidenced based data exists on ultrasonographic abnormalities in chronic pancreatitis – anecdote underlies several reported findings

	Acute Pancreatitis	Chronic Pancreatitis
Pancreatic Size	↑↑↑	±↑ or ↓ (fibrosis)
Pancreatic Echogenicity	Hypoechoic	Hyperechoic or Heterogenous ± Nodular change
Margins	Ill-defined	Irregular but well-defined
Surrounding Mesentery	Hyperechoic	± Hyperechoic
Peritoneal effusion	Often present	Rarely present

Abdominal Ultrasound – Age related changes?

Ultrasonographic measurement of the pancreas and pancreatic duct in clinically normal dogs

Objective—To obtain ultrasonographic reference values for the thickness of the pancreas and the diameter of the pancreatic duct in clinically normal dogs.

Animals—242 adult dogs with no clinical signs of gastrointestinal tract disease.

Procedures—The maximum pancreatic thickness and the diameter of the pancreatic duct were recorded ultrasonographically at the level of the left lobe, body, and right lobe of the pancreas.

Results—Mean \pm SD pancreatic thickness measurements were as follows: left lobe, 6.5 ± 1.7 mm (n = 214); body, 6.3 ± 1.6 mm (155); and right lobe, 8.1 ± 1.8 mm (239). The mean pancreatic duct diameter was 0.6 ± 0.2 mm (n = 42) in the left lobe and 0.7 ± 0.2 mm (213) in the right lobe. The right pancreatic duct was visible in 213/242 (88.0%) dogs, and the left pancreatic duct was visible in 41/242 (16.9%) dogs. However, the body was visible in only 16/242 (6.6%) dogs. Pancreatic thickness and diameter of the pancreatic duct significantly increased with body weight in all lobes, but age was not correlated with the measurements.

Conclusions and Clinical Relevance—Ultrasonographic reference values for the pancreas and pancreatic duct of dogs were determined. Results of this study indicated that the pancreatic duct was visible, especially in the right lobe of the pancreas. These values may be useful for the assessment of pancreatic abnormalities, such as chronic pancreatitis and exocrine pancreatic insufficiency. (Am J Vet Res 2013;74:433–437)

Dogs → pancreatic duct size is not correlated with age (unlike cats)

Median age of dogs with heterogenous pancreas → 12 years

r/o age change vs subclinical CP

Median age of dogs with hyperechoic foci → 14 years

r/o age change vs subclinical CP

Overall Diagnostic Criteria

If a dog has repeated episodes of:

- Consistent clinical signs
- Elevated PLI (even mild/grey-zone)
- Abnormal pancreatic ultrasound (can be subtle)

= presumptive CP (*) (no need for biopsy)

(* rule out DDx for related C/S)

Development of EPI in a non-predisposed breed warrants consideration of potential underlying CP



Image created with Google Gemini



Treat Underlying Risk Factors / Prevent Recurrent Pancreatitis

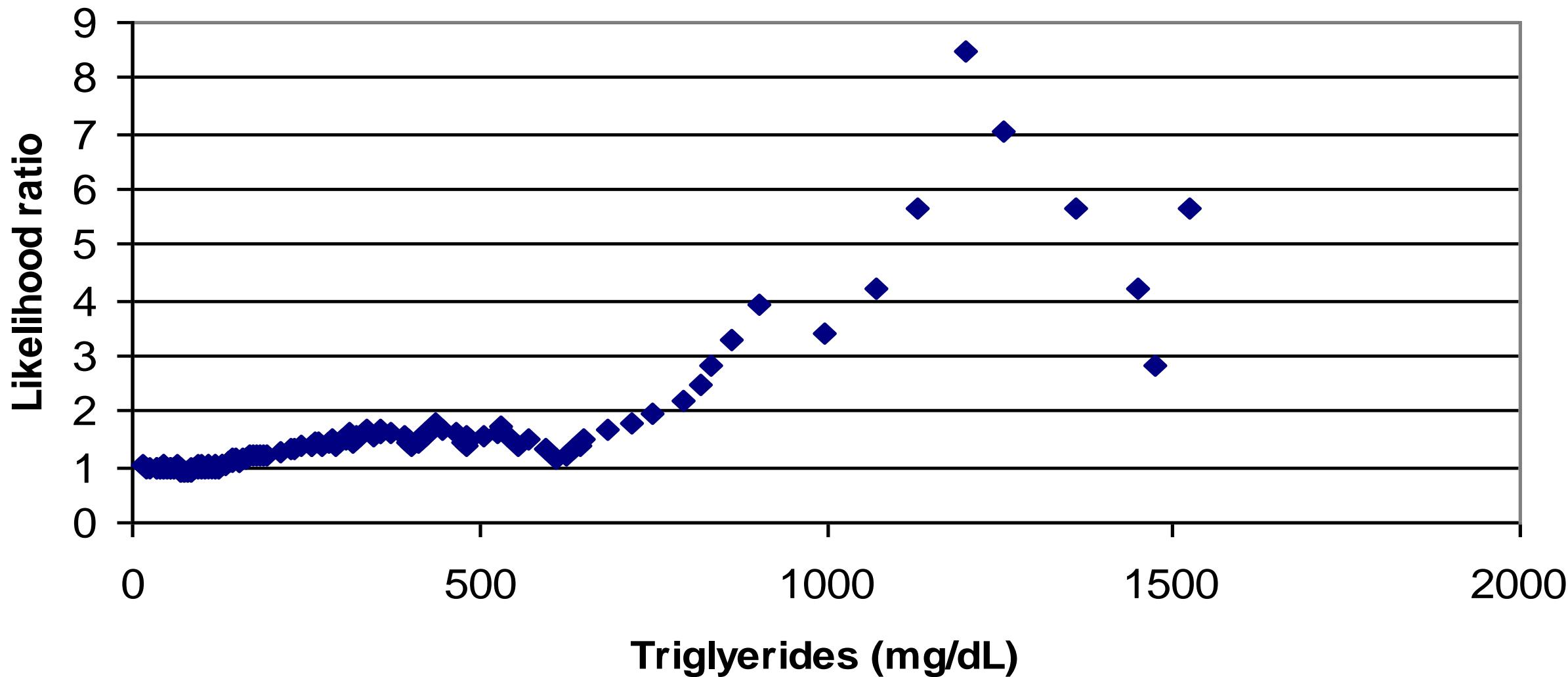
Category	Potential Risk Factor
Lipid disorders 	Hypertriglyceridemia (but not hypercholesterolemia)
Hereditary	Miniature Schnauzer, Terrier breeds, etc.
Dietary factors 	High fat diet
Endocrinopathies 	Hyperadrenocorticism, hypothyroidism, diabetes mellitus
Calcium Disorders	Hypercalcemia
Auto-immune IgG4 related disease	English Cocker Spaniel ± other breeds

Association Between Serum Triglyceride and Canine Pancreatic Lipase Immunoreactivity Concentrations in Miniature Schnauzers

The objective of this study was to investigate possible associations between serum triglyceride and canine pancreatic lipase immunoreactivity (cPLI) concentrations in miniature schnauzers. One hundred and ninety-five miniature schnauzers were enrolled and divided into two groups based on whether they had normal (group 1) or increased (group 2) serum triglyceride concentrations. Serum cPLI concentrations were measured and compared between groups. A significant positive correlation was seen between serum triglyceride and cPLI concentrations (Spearman $r=0.321$; $P<0.0001$). Miniature schnauzers with hypertriglyceridemia had a significantly higher median serum cPLI concentration (99.5 $\mu\text{g/L}$) than miniature schnauzers with normal serum triglyceride concentrations (median cPLI concentration 39.3 $\mu\text{g/L}$; $P=0.0001$). A cutoff value of 862 mg/dL was selected for serum triglyceride concentrations based on receiver operator characteristic analysis. Miniature schnauzers with severe hypertriglyceridemia (≥ 862 mg/dL) were 4.5 times more likely to have a serum cPLI concentration consistent with pancreatitis (≥ 200 $\mu\text{g/L}$) than miniature schnauzers with a normal serum triglyceride concentration. The present study supports an association between hypertriglyceridemia (especially when severe [≥ 862 mg/dL]) and high cPLI concentrations in miniature schnauzers. *J Am Anim Hosp Assoc* 2010;46:229-234.



Likelihood ratio of different triglyceride concentrations for cPLI $\geq 200 \mu\text{g/L}$





hypertriglyceridemia

SCENARIO 1:

If pancreatitis CAUSES hypertriglyceridemia →
serum triglyceride concentrations would be
NORMAL after pancreatitis resolves

pancreatitis

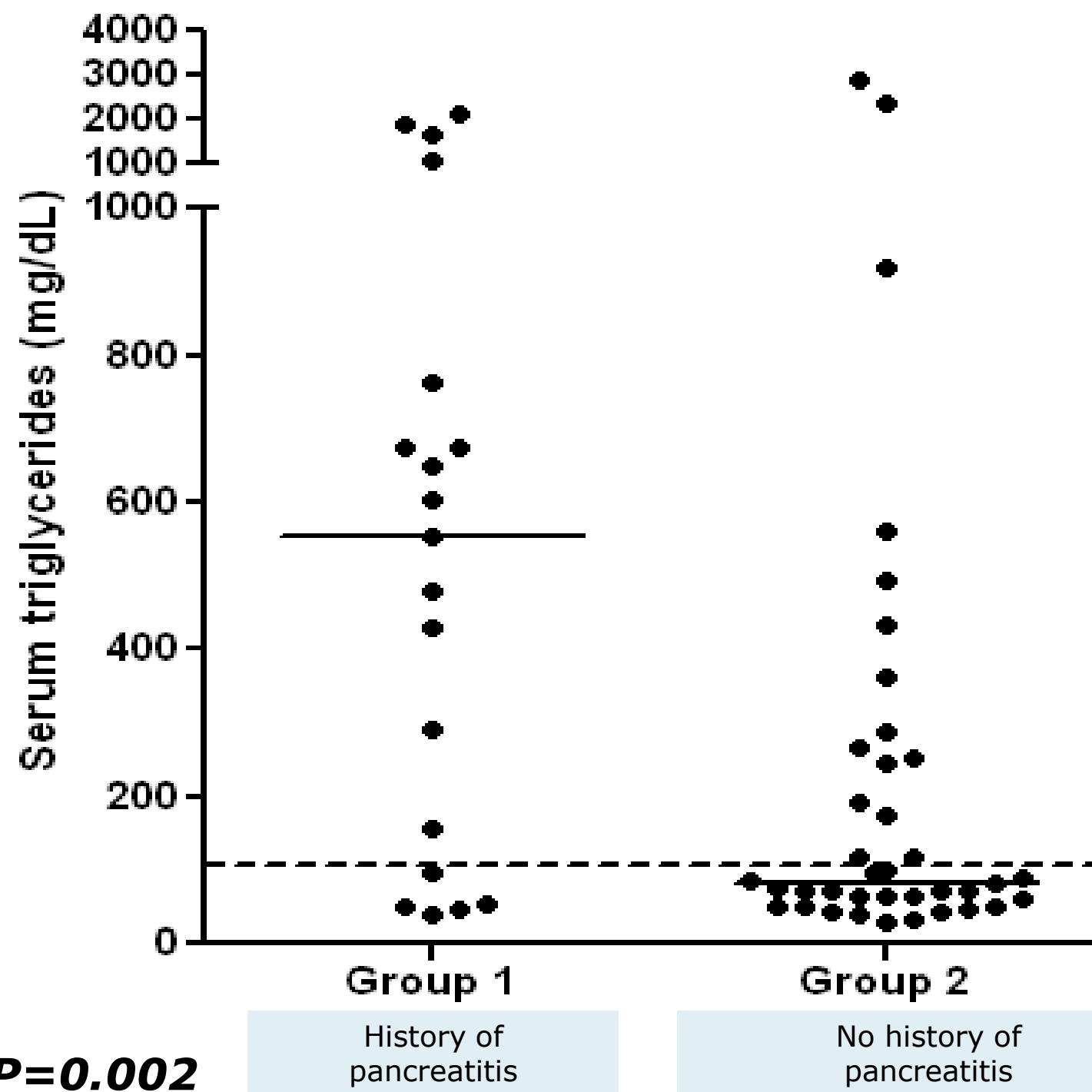
hypertriglyceridemia

SCENARIO 2:

If pancreatitis IS SECONDARY TO hypertriglyceridemia → serum triglyceride concentrations would be ABNORMAL even after pancreatitis resolves

pancreatitis





Efficacy of a micronized, nanocrystal fenofibrate formulation in treatment of hyperlipidemia in dogs

Abstract

Background: Safe, effective, and readily available drug therapies are required for the management of hyperlipidemia and its associated complications in dogs.

Objectives: To investigate the efficacy of a micronized, nanocrystal formulation of fenofibrate (Tricor) in the treatment of hyperlipidemia in dogs.

Animals: Ten client-owned dogs with primary ($n = 7$) and secondary ($n = 3$) hyperlipidemia. All dogs had hypertriglyceridemia at baseline; 3 dogs also had hypercholesterolemia.

Methods: Prospective dose-escalation study. Dogs were treated with fenofibrate orally once daily in up to 3 cycles of 21 days each. Fenofibrate dose was increased at the end of each cycle if hypertriglyceridemia persisted and adverse effects were not documented. Complete blood count, biochemistry, and urine protein:creatinine ratio were collected serially. Baseline (T0) parameters were compared to time of maximal reduction in serum triglyceride concentrations (T1) and reported as median (range).

Results: Triglycerides normalized in all dogs (T0 = 662 mg/dL [189-2391]; T1 = 113 mg/dL [81-132]; $P = .002$). Fenofibrate dose at T1 = 6.4 mg/kg PO q24h (range, 2.2-13.5). T1 was achieved at 3 ($n = 4$), 6 ($n = 4$), and 9 ($n = 2$) weeks. Serum cholesterol concentrations decreased in 9 of 10 dogs. Quiet demeanor and firm stools in 1 dog were the only reported adverse reactions. Fenofibrate administration resulted in a significant reduction in median alkaline phosphatase activity ($P = .049$).

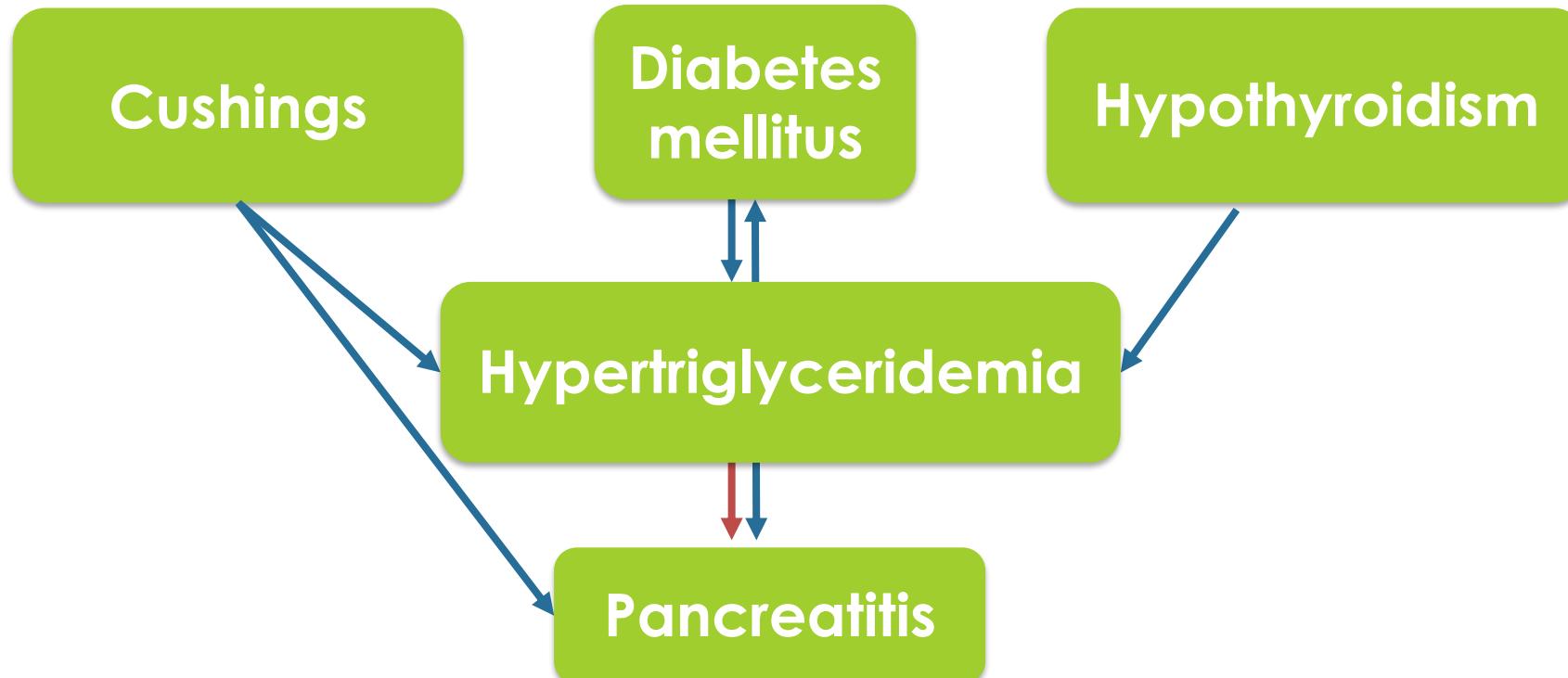
Conclusions and Clinical Importance: Over 21 to 63 days, Tricor was effective in the management of primary and secondary hyperlipidemia in dogs.



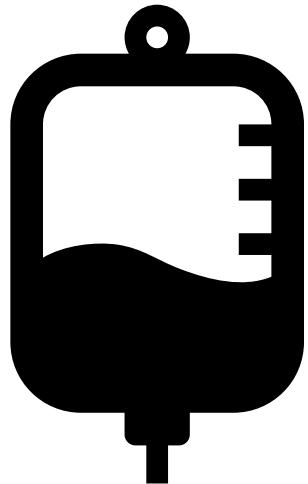
Henry Schein
Medical



UNDERLYING ENDOCRINOPATHIES

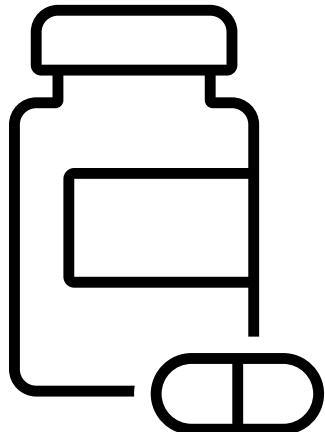


TREATMENT

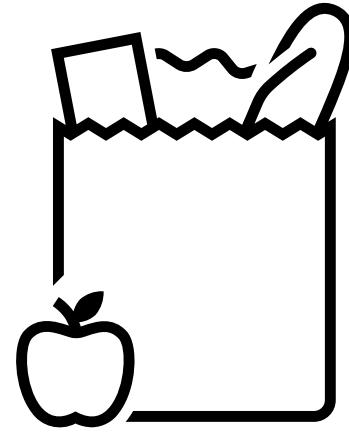


Fluid Therapy

-typically, at time of acute or chronic dz



Analgesia



Nutrition

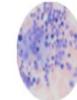
-low-fat highly digestible diet

many many tests

What's the problem?



Let's start steroids



vetinternalmedicine

± immunosuppression



ANALGESIA

Chronic Disease / Outpatient Options?

Gabapentin?
Tramadol?
Butorphanol?
Opioids?
Others?



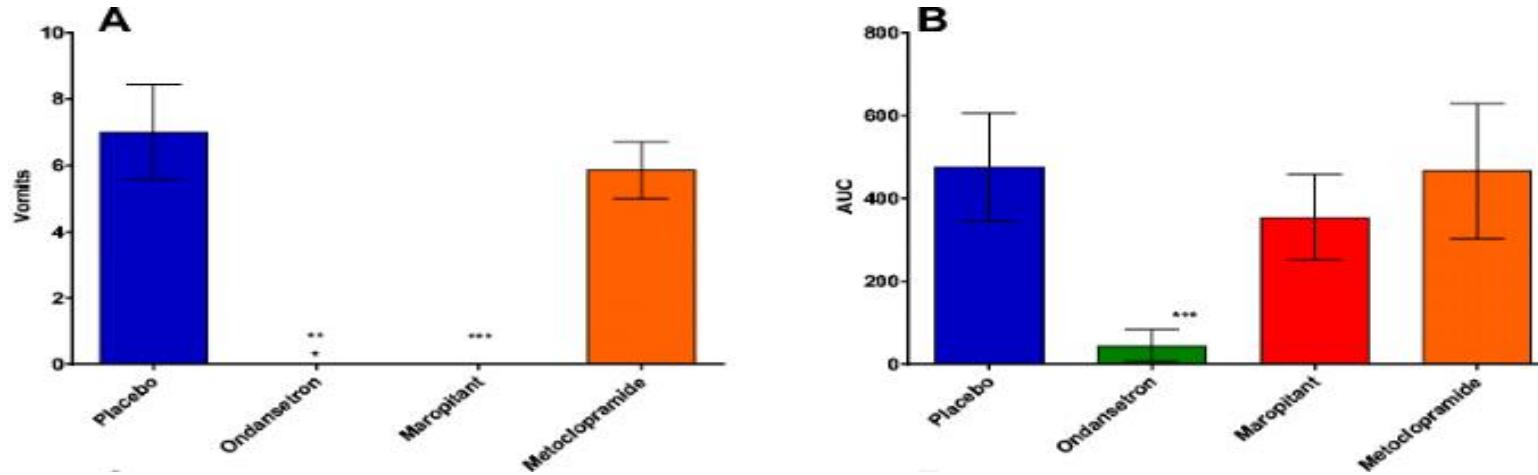
ANTI-EMETICS

Comparative Study

› BMC Vet Res. 2017 Aug 16;13(1):244. doi: 10.1186/s12917-017-1156-7.

Anti-nausea effects and pharmacokinetics of ondansetron, maropitant and metoclopramide in a low-dose cisplatin model of nausea and vomiting in the dog: a blinded crossover study

Hannah Kenward ¹, Jonathan Elliott ¹, Terry Lee ², Ludovic Pelligand ^{3 4}

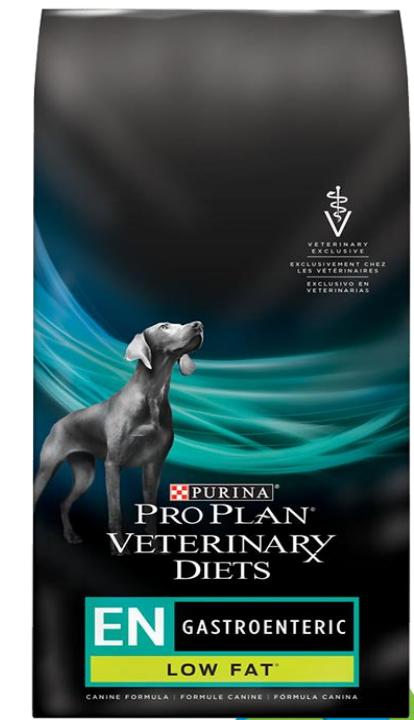


NUTRITIONAL MANAGEMENT

- Low-fat diet (< 2 g fat/100 kcal)



- Only low-fat treats:
 - vegetables
 - fruits
 - low-fat treats
 - home-made treats

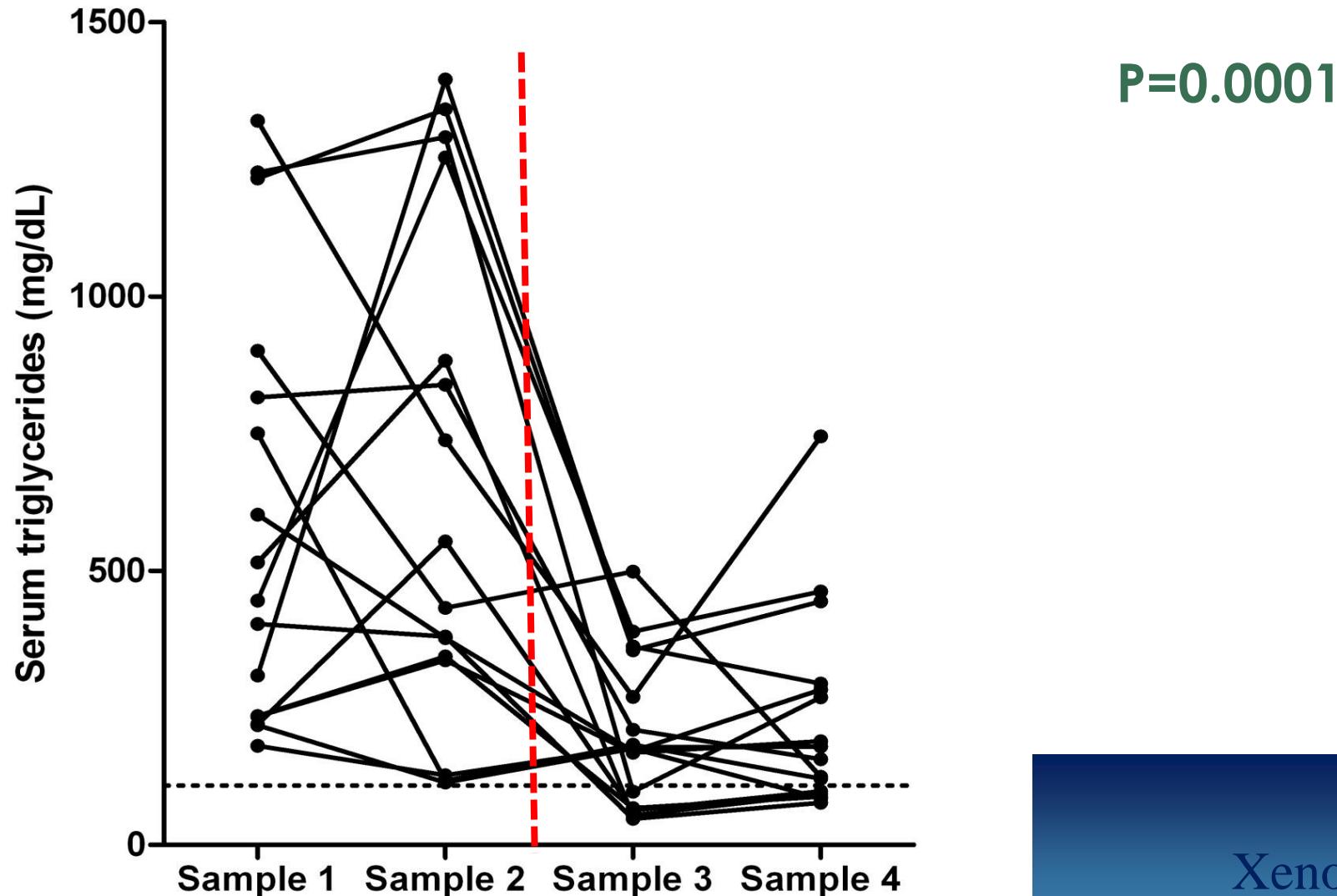


EFFECT OF DIET

- 15 healthy MS with hypertriglyceridemia
 - the diet of the dogs was changed to the study diet
 - fat content: 18.6 g/1,000 Kcal
- 4 blood samples were collected from each dog
 - 2 samples before the diet change
 - 1 sample ~2 months after the diet change
 - 1 sample ~3 months after the diet change

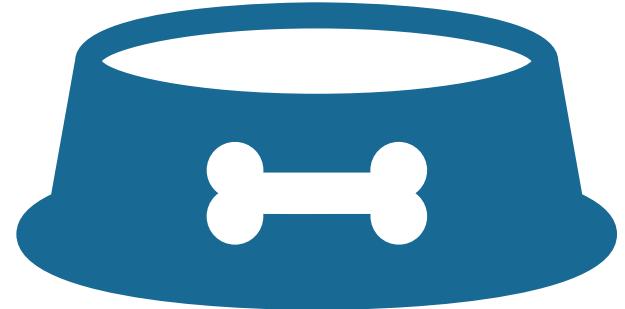


SERUM TG OVER TIME



HOW TO IMPROVE FOOD INTAKE

- Warm food
- Highly palatable & digestive foods
- Many textures & types of food (kibble vs can etc.)
- Appetite stimulants



MMI Nutrition Guide

Review summaries of nutritional management and compare products for specific disease conditions

Species

Select

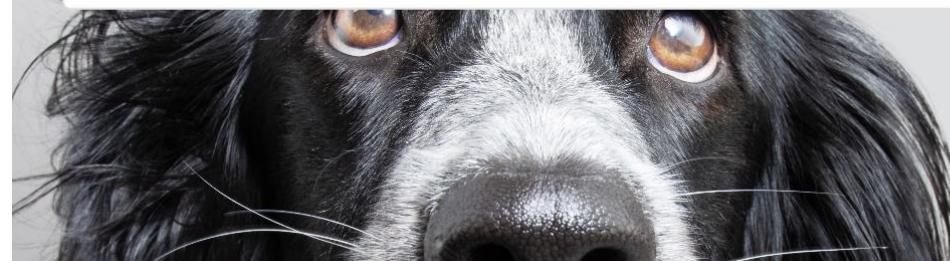
Age

Select

Disease Condition

Select

Search



Please note that product values shown are for the United States only and are not valid in other regions.

Diets are presented in random order. To sort by a specific nutrient, click on the nutrient name at the top of the column.

PRODUCT	CALORIC DENSITY (KCAL/KG) KCAL/KG AF	CALORIC DENSITY (VOLUME - UNIT) KCAL/VOL UNIT	PROTEIN G/100 KCAL	FAT G/100 KCAL	TOTAL DIETARY FIBER G/100 KCAL
Purina Pro Plan Veterinary Diets EL Elemental dog dry	3316	411	6.97	2.65	0.580
Royal Canin Veterinary Health Nutrition Gastrointestinal Low Fat Small Dog dry	3203	263	6.46	2.06	2.47
Purina Pro Plan Veterinary Diets EN Gastroenteric Low Fat dog dry	3216	290	8.08	1.95	1.78
Purina Pro Plan Veterinary Diets OM Select Blend Overweight Management dog dry	2941	219	10.0	2.18	8.83
Hill's Prescription Diet c/d Multicare Low Fat Vegetables & Turkey Stew Dog Food canned	785	278	6.50	2.30	2.90
Royal Canin Veterinary Health Nutrition Gastrointestinal Low Fat dog dry	3210	247	6.46	2.05	2.41
Hill's Prescription Diet z/d Low Fat Hydrolyzed Soy Recipe Dog Food dry	3198	317	7.70	2.10	2.60
Purina Pro Plan Veterinary Diets EN Gastroenteric Low Fat dog canned	956	363	12.0	2.45	2.98

IMMUNOSUPPRESSION?

Rationale for Immunosuppressive Therapy

- CP may have an immune-mediated component
- Persistent lymphocytic or lymphoplasmacytic infiltration suggests a role for immune dysregulation
- Aim: reduce ongoing pancreatic inflammation and fibrosis progression

Steroids or Modified Cyclosporine

- Monitor Catalyst PL or Spec cPL + clinical signs during treatment → evaluate efficacy



Management of Complications – Exocrine Pancreatic Insufficiency

- Dogs with CP should be monitored for EPI
- Pancreatic enzyme supplementation recommended
- Even if TLI is normal, consider supplementation for:
 - Weight loss
 - Steatorrhea
 - Poor coat quality
- ± Cobalamin supplementation



Mountainside Medical Equipment

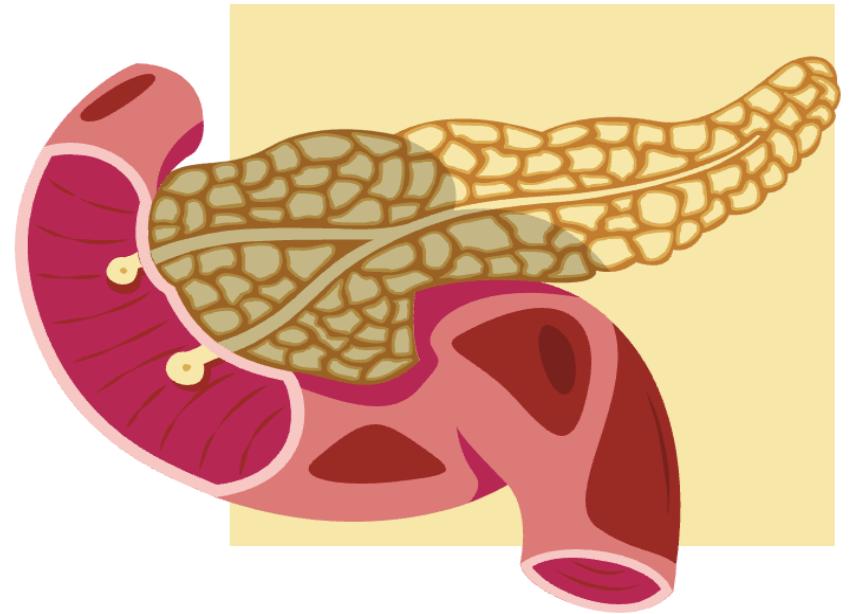
Management of Complications – Diabetes Mellitus

- Standard DM treatment applies
- Avoid high fiber diabetic diets
- If enzymes are added to treatment, it may increase digestion leading to increased insulin requirements
- Dogs with CP do not appear to have a worse prognosis than other DM cases

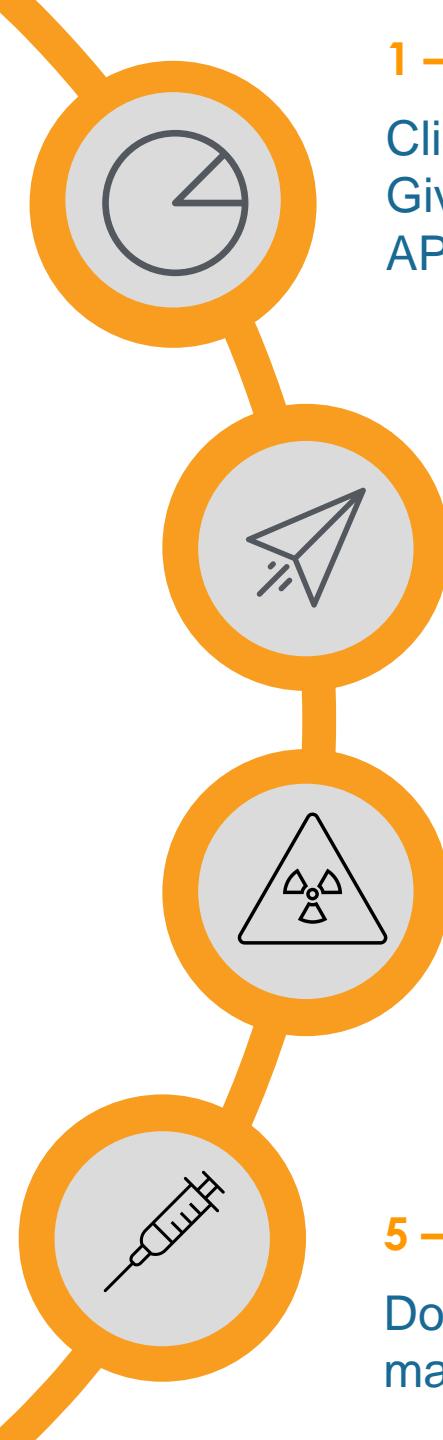


PROGNOSIS

- Long-term prognosis depends on the extent of pancreatic damage and presence of 2ndry complications
- Early detection and intervention are critical in improving outcomes



SUMMARY



1 – DEFINITIONS

Clinical and histopathologic definitions often do not overlap
Given infrequent use of pancreatic biopsies I utilize clinical definitions of AP and CP

2 – SUBTLE CLINICAL SIGNS

CP dogs typically have mild waxing and waning C/S
Post-prandial pain may be under-estimated and only noted after resolution with analgesic trial.

3 – DIAGNOSIS

Diagnosis is made based on integration of all available data.
Repeat lipase testing may be needed.

4 – TREATMENT OF UNDERLYING RISK FACTORS & CP

Identifying underlying risk factors may help to prevent recurrence
Treatment of CP involves nutrition and analgesia. Fluids may also be needed

5 – ACTIVE SCREENING & TX OF SECONDARY COMPLICATIONS IS NEEDED

Dogs with CP may develop EPI &/or DM which need separate management



THANK YOU

DON'T FORGET TO RATE YOUR SPEAKER
AND SESSION IN THE APP!

Presented By

NAVC
YOUR VETERINARY COMMUNITY

